



HAL
open science

Attentional modulation of peripheral pointing hypometria in healthy participants: An insight into optic ataxia?

Tristan Jurkiewicz, Audrey Vialatte, Yaffa Yeshurun, Laure Pisella

► To cite this version:

Tristan Jurkiewicz, Audrey Vialatte, Yaffa Yeshurun, Laure Pisella. Attentional modulation of peripheral pointing hypometria in healthy participants: An insight into optic ataxia?. *Neuropsychologia*, 2025, 208, pp.109084. 10.1016/j.neuropsychologia.2025.109084 . hal-04951058

HAL Id: hal-04951058

<https://hal.science/hal-04951058v1>

Submitted on 17 Feb 2025

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29

Attentional modulation of peripheral pointing hypometria in healthy participants: an insight into optic ataxia?

Tristan Jurkiewicz^{1,2}, Audrey Vialatte¹, Yaffa Yeshurun^{*3}, Laure Pisella^{*1}

1: Centre de Recherche en Neurosciences de Lyon (CRNL), INSERM U1028, CNRS UMR5292, Université Claude Bernard Lyon 1, 69500 Bron, France

2: Centre d'Exploration de la Rétine Kléber (CERK), 50 cours Franklin Roosevelt, 69006 Lyon, France

3: School of Psychological Science, University of Haifa, Haifa, Israel

*: same contribution

Corresponding author mail: tristan.jurkiewicz@inserm.fr

Phone: +33 6 84 89 39 43

Number of figures: 8 (+ 10 supplementary figures)

Number of tables: 1

Declaration of Competing Interest:

The authors have no conflicts of interest to declare.

Financial support:

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

30

31 **Abstract**

32 Damage to the superior parietal lobule and intraparietal sulcus (SPL-IPS) causes optic ataxia
33 (OA), characterized by pathological gaze-centered hypometric pointing to targets in the
34 affected peripheral visual field. The SPL-IPS is also involved in covert attention. Here, we
35 investigated the possible link between attention and action. This study investigated the
36 effect of attention on pointing performance in healthy participants and two OA patients.

37 In invalid trials, targets appeared unpredictably across different visual fields and
38 eccentricities. Valid trials involved cued targets at specific locations. The first experiment
39 used a central cue with 75% validity, the second used a peripheral cue with 50% validity. The
40 effect of attention on pointing variability (noise) or time was expected as a confirmation of
41 cueing efficiency. Critically, if OA reflects an attentional deficit, then healthy participants, in
42 the invalid condition (without attention), were expected to produce the gaze-centered
43 hypometric pointing bias characteristic of OA.

44 Results revealed main effects of validity on pointing biases in all participants with central
45 predictive cueing, but not with peripheral low predictive cueing. This suggests that the
46 typical underestimation of visual eccentricity in OA (visual field effect) at least partially
47 results from impaired endogenous attention orientation toward the affected visual field.

48

49

50 **Keywords:** Superior parietal lobule; Intraparietal sulcus; Covert attention; Reaching; Positional
51 encoding; Peripheral vision; Spatial attention

52

53 **1- Introduction:**

54 Following damage to the superior parietal lobule and intraparietal sulcus (SPL-IPS), optic
55 ataxia (OA) is characterised by difficulty reaching toward and grasping objects presented in
56 peripheral vision, whereas actions in free vision are generally accurate (Garcin et al., 1967;
57 Perenin & Vighetto, 1988). More precisely, unilateral OA reveals visual reaching errors
58 associated with a combination of hand and field effects (Perenin & Vighetto 1988; Blangero
59 et al., 2008), that are now considered distinct and dissociable components of the deficit
60 (Pisella et al., 2006, 2008, 2009, 2017, 2021). Reaching errors corresponding to the OA field
61 effect reveal pathological underestimation of the visual eccentricity of targets while reaching
62 toward the contralesional peripheral visual field (Blangero et al., 2010; Vindras et al., 2016).
63 This gaze-centered hypometric pointing is characteristic of the OA field effect and reflects
64 inaccurate positional encoding of the target position in oculocentric reference frame (Khan
65 et al., 2005a,b). Reaching errors associated with the OA hand effect most likely result from a
66 mislocalization of the contralesional (ataxic) hand, arising from impaired positional encoding
67 in oculocentric reference frame from proprioceptive information (Blangero et al., 2007;
68 Mikula et al 2021). Having both hand and target positions in a common oculocentric
69 reference frame within the SPL-IPS network allows direct visuo-manual transformation
70 (Buneo et al., 2005).

71

72 Jackson et al. (2009) proposed that it is the ability to simultaneously represent multiple
73 spatial locations that is impaired in OA patients. For example, visuospatial information about
74 the target and fixation object must be compared and might compete for limited processing
75 resources. This is reminiscent of simultanagnosia, a deficit of simultaneous visual processing
76 described in Balint case and present at the acute stage in bilateral OA patient I.G. (Pisella et

77 al., 2000). This visual extinction is considered as a deficit of attention affecting visual
78 perception (Khan et al., 2015; Medina et al., 2020; Vialatte et al., 2021), but can also explain
79 an inability to de-couple reach direction from gaze direction, a phenomenon called
80 “magnetic misreaching” (Carey et al., 1997; Jackson et al., 2005) that was observed in
81 unilateral OA patient C.F. at the acute stage and evolved toward gaze-centered hypometric
82 pointing errors at the chronic stage.

83

84 Indeed, there has been converging evidence in recent years for the co-occurrence of
85 peripheral reaching deficit characteristic of OA and impairments of covert spatial attention,
86 leading to the view that they could be understood as part of the same anatomo-functional
87 deficit (Pisella et al., 2007, 2008, 2009, 2021; Striemer et al., 2007; McIntosh et al., 2011;
88 Martin-Arevalo et al., 2021; Knights et al., 2024; but see Striemer et al., 2009). The view that
89 the core function of the SPL-IPS appears to be attention and only consequently affects
90 “vision for action” (Pisella et al., 2013, page 319) is in line with the view that the dorsal visual
91 stream processes location (Holmes, 1918; Ratcliff, 1991; Ratcliff & Davies-Jones, 1972;
92 Ungerleider & Mishkin, 1982) and that covert attention improves spatial resolution in
93 peripheral vision (Yeshurun & Carrasco, 1998, 1999). This is also reminiscent of the debate
94 between attentional (spatial coding or “where”, Colby & Goldberg, 1999) and intentional
95 (motor planning or “how”, Andersen & Buneo, 2002) interpretations of the function of the
96 posterior parietal cortex in the non-human primate literature.

97

98 Aguilar Ros et al. (2021) were the first to investigate this issue by testing healthy
99 participants. In a dual-task paradigm, their participants performed a central task while also
100 reaching to peripheral visual targets, and they tested whether the gaze-centered pointing

101 hypometria characteristic of the OA field effect could be elicited by the attentional
102 engagement at the fixation point. They positively showed more under-estimation of
103 peripheral visual target eccentricity in the pointing responses during the dual-task condition.
104 This important hypothesis that OA may be 'simulated' in healthy participants by limiting
105 their ability to shift covert attention can be tested directly by investigating the effect of valid
106 versus invalid attentional cueing on pointing performance. With this in mind, we recruited a
107 group of 14 healthy participants in this study, as well as bilateral OA patient I.G. and
108 unilateral OA patient C.F. Whether the gaze-centered pointing hypometria of OA can be
109 explained by a deficient ability to orient covert attention toward the visual peripheral target
110 in the ataxic field was evaluated by testing 1) whether removing attention by an invalid
111 cueing toward the opposite visual field, before the presentation of the pointing target,
112 simulates OA in healthy participants, and 2) whether a valid cueing of the visual peripheral
113 target position improves pointing accuracy in the OA patients. This would suggest that
114 pointing inaccuracy is linked to inaccurate or slowed covert attentional orienting toward the
115 ataxic visual field.

116 Because spatial covert attention can be allocated either voluntarily according to
117 participants' goals or in a stimulus-driven fashion (reviewed in Carrasco, 2011), we examined
118 whether a deficit in either type of attention allocation mediates the pointing errors observed
119 in OA. To this aim, we designed two peripheral pointing experiments with random visual
120 target presentation, i.e., both in terms of eccentricity and visual field, as in the classical
121 assessment of peripheral reaching performance in OA (Vindras et al., 2016; Striemer et al.,
122 2009), in which the performance of healthy controls and two patients with OA could be
123 hindered or improved by invalid or valid covert attentional cueing (Posner, 1980). More
124 precisely, invalid trials involved target presentation at random visual eccentricity (horizontal

125 distance from gaze fixation: 15, 20, 25, 30, or 35°) opposite to the cued visual field, whereas
126 valid trials involved target presentation in the cued visual field at the same blocked visual
127 eccentricity. In one experiment (Figure 1), the cue consisted of a central arrow which was
128 75% predictive of the target location. In the other experiment (Figure 2) the cue was a
129 peripheral line flashed at one of the possible target locations. This was a low predictive cue
130 as it flashed at the target location in only 50% of the trials. Still, it should automatically grasp
131 the participant's attention toward the target position before its actual presentation in the
132 valid trials.

133 Crucially, we focused our analysis on the pointing horizontal error, as systematic
134 pointing biases toward the fixation point characterizes peripheral pointing performance in
135 OA (Blangero et al., 2010; Vindras et al., 2016). We also analysed pointing variability and
136 response time, as well as ocular positions throughout the experiments in each participant, as
137 a validation that we actually manipulate covert attention.

138

139 **2- Methods**

140 **2-1. Participants:**

141 The required sample size was estimated using the G*Power V.3.1.7 program (Franz Faul,
142 Christian-Albrechts-Universität, Kiel, Germany). The options selected were: a paired t-test
143 with a one-tailed hypothesis that pointing parameters would be better in the valid than in
144 the invalid trials for each eccentricity condition. An effect size of 0.9 was estimated for this
145 study based on pilot data, with a probability of an alpha error of 0.05 and a power of 0.95 (1
146 - probability of beta error). The proposed sample size was 14 participants for an actual
147 power of 0.952. Accordingly, the same group of 14 healthy participants was recruited for

148 the two experiments. They were between the ages of 21 and 42 years (mean age = $26.79 \pm$
149 6.12 years) and had no neurological history or psychiatric disorder.

150

151 Two stroke patients exhibiting chronic OA and willing to participate in research protocols
152 also took part in these two experiments. Patient I.G. was 54 years old when she was included
153 in this study. This woman suffered in 1998 from an ischemic stroke related to acute
154 vasospastic angiopathy in the posterior cerebral arteries. The lesion involved mainly
155 Brodmann areas 18, 19, 7, a limited part of area 39, as well as the intraparietal sulcus of both
156 hemispheres. Chronically, she demonstrates bilateral OA (e.g. Pisella et al., 2000; Gréa et al.,
157 2002; Khan, Pisella, Rossetti, et al., 2005; McIntosh et al. 2011; Mikula et al. 2021). Patient
158 C.F. was 44 years old when he was included in this study. This man suffered in 2003 from a
159 posterior watershed infarct resulting in distributed and asymmetrical bilateral lesions of
160 Brodmann areas BA 18, 19, 7, 5, and 2 with a minute extension to the centrum semiovale.
161 Chronically, he demonstrates isolated unilateral OA in his left visual field, thought to be the
162 consequence of larger damage in the right hemisphere of both BA 7 and the intra-
163 hemispheric parieto-frontal fibers (Blangero et al., 2008; Striemer et al., 2009; Khan et al.
164 2005, 2007, 2013; Mikula et al. 2021; Cheviet et al. 2021).

165

166 All participants were right-handed, had normal vision or vision corrected-to-normal using
167 contact lenses, and were able to read Parinaud 2 with their optical correction. Informed
168 written consent was collected from all participants in accordance with CPP Northwestern 1
169 046/2017 registration number 2017-A02562-51.

170

171 **2-2. Experimental design:**

172 Participants were seated on a height-adjustable chair with a chin-rest at 35 centimeters from
173 the touch screen (Iiyama ProLite TF3215MC-B1, dimension 31.5" (73.9 x 43.4 cm), refresh
174 rate: 75Hz, response time: 8 ms, resolution: 1920x1080). They were informed that visual
175 targets would be presented at various visual eccentricities along the horizontal axis at the
176 same height of the ocular fixation in their left or right visual field. The peripheral pointing
177 task was carried out in a dimly illuminated room. A tactile marker was stuck on the table to
178 ensure that all the participants started their right-hand movement from the same position at
179 the bottom center of the screen. An eye-tracker (EyeLink Portable Duo; SR Research,
180 Ottawa, Ontario, Canada) was used. Ocular fixation had to be maintained on a central
181 fixation cross for 250ms to start each trial, and was checked throughout.

182

183 The instruction was to fixate the cross at the center of the screen throughout the pointing
184 blocks. The target was presented only if the participant's gaze remained within a $\pm 2^\circ$ square
185 zone around the fixation cross for at least 250 ms. Target duration was 50 ms, preventing
186 any significant change in ocular position during its presentation and thereby ensuring its
187 visual eccentricity. Furthermore, fixation was monitored throughout the experiment by the
188 examiner allowing for a recall of the instruction if necessary. The fixation area (mean and
189 standard deviation of each participant) of each block was also recorded and checked offline
190 using data viewer software V4.4.1 (SR Research, Canada). The task consisted of immediate
191 peripheral pointing with the right hand, as precisely as possible, at the place on the screen
192 where the target was perceived. The peripheral cueing experiment was performed first by
193 healthy participants and patients, followed by the central cueing experiment. Before each
194 experiment, a training block of 15 trials was performed. Five blocks per experiment were
195 performed with targets presented either in the right or left visual field, at blocked

196 eccentricities (15, 20, 25, 30, or 35° of visual eccentricity) in the valid trials, and at unblocked
197 eccentricities (15, 20, 25, 30, and 35° of visual eccentricity) in the invalid trials.

198

199 2-2-1. Central cueing experiment (Figure 1):

200 The experiment included five blocks of 80 trials. For each block, there were 60 valid trials (30
201 per visual field at one blocked eccentricity) and 20 invalid trials (10 per visual field
202 distributed across the 5 unblocked eccentricities), corresponding to 75% valid trials and 25%
203 invalid trials. This led to 400 trials overall per participant. In terms of timing, the central cue
204 was flashed for 50 ms, then the fixation cross reappeared for 250 ms, and the target was
205 finally presented for 50 ms. The trial ended when the participant's index finger touched the
206 screen.

207

208 2-2-2. Peripheral cueing experiment (Figure 2)

209 The experiment included five blocks of 60 trials. For each block, there were 30 valid trials (15
210 per visual field at one blocked eccentricity) and 30 invalid trials (15 per visual field
211 distributed across the 5 unblocked eccentricities), corresponding to 50% of valid trials. This
212 led to 300 trials overall per participant. In terms of timing, the peripheral cue was flashed for
213 50ms, then the fixation cross reappeared for 50ms and the target was finally presented for
214 50ms. The trial ended when the participant's index finger touched the screen.

215

216 **2-3. Data analysis:**

217 The pointing was a forced-choice response. Sometimes a participant spontaneously reported
218 that s/he did not perceive the visual target at all, because s/he was blinking or fully
219 inattentive for example. In this case, the trial was excluded offline from further analysis

220 (0.28% of trials for healthy participants, 0.6% for the bilateral patient, and 0.33% for the
221 unilateral patient on average throughout the two experiments). Outlier trials were also
222 removed as defined by endpoints time or x-coordinate greater than 2.5 standard deviation
223 (SD) compared to the individual mean (5.30% of trials on average in the central cueing
224 experiment [ranging between 2.5% to 7.25% among participants] and 4.55% on average in
225 the peripheral cueing experiment [ranging between 1.33% to 7.0% among participants],
226 7.75% and 7.33% of trials for patient IG, 3% and 4% of trials for patient CF, for the two
227 experiments, respectively).

228

229 In order to study the gaze-centered pointing biases, the x-coordinate (in pixels) of the
230 participant's endpoint was subtracted from the actual x-coordinate of the visual target in
231 each trial. This difference provided a signed horizontal error translated in cm (1 pixel=
232 0.03637cm). A negative error corresponds to gaze-centered hypermetria, i.e., a pointing
233 movement that ends beyond the target visual eccentricity. A positive error corresponds to
234 gaze-centered hypometria, i.e., a pointing movement that ends in-between the gaze and the
235 visual target, as classically observed in the contralesional field of unilateral OA patients
236 (Blangero et al. 2010). All significant interactions are listed in the results section, but only the
237 figures illustrating the effects linked to our hypotheses are shown. The other significant
238 interaction figures are included as supplementary information.

239

240 Two other parameters were computed for each condition of target validity and eccentricity
241 in order to study pointing performance: the endpoint intra-individual variability along the
242 horizontal axis (SDx), as a measure of pointing noise, and the time between visual target
243 presentation and the touch of the screen (response time).

244

245 **2-4. Statistical analysis of peripheral pointing performance:**

246 Descriptive statistics (mean and standard deviation) and ANOVAs were performed using
247 Statistica V14.0.0.15 software for Windows (StatSoft, Inc., Tulsa, Oklahoma). For the group
248 of healthy participants, we ran repeated measures ANOVAs on the horizontal pointing bias,
249 our main dependent variable, but also on intra-individual pointing variability (SDx) and
250 response time (RT), with target Eccentricity (15/20/25/30/35°) and cueing Validity
251 (valid/invalid) as factors. LSD pairwise comparisons were used to test the specific least
252 squares difference means to further explore significant interactions.

253

254 For the patients, we ran factorial ANOVAs on horizontal pointing bias and RT. For the patient
255 with bilateral OA, we assessed the same two factors as for healthy participants: Eccentricity
256 and cueing Validity. For the patient with unilateral OA, the Visual field (left/right) factor was
257 added. Note that the intra-individual pointing variability of each patient corresponds to a
258 single value and therefore it can only be compared visually between the valid and invalid
259 conditions of the patient, and to that of the healthy participants.

260

261 Bonferroni corrections for multiple comparisons were applied by dividing the alpha
262 threshold initially set at 5% by the number of comparisons performed. In case of comparison
263 of the cueing effect for each of the 5 visual target eccentricities, the significant threshold for
264 the p-value was $0.05/5$ i.e., 0.01. In the case of visual field by cueing validity condition
265 comparisons for the unilateral patient, the significant threshold for the p-value was $0.05/4$
266 i.e., 0.0125.

267

268 **3- Results:**

269 **3-1. Eye movement analysis**

270 In order to confirm that we were manipulating covert (and not overt) attention, horizontal
271 ocular positions (i.e., the distance from the center of each fixation) were analysed offline
272 during all test blocks. For each participant and experiment, the mean position and standard
273 deviation were calculated. Table 1 shows that all participants nicely fulfilled the requirement
274 to maintain gaze fixation during both peripheral pointing experiments.

275

276 Table 1. Mean and standard deviation of horizontal ocular positions (°) throughout the
277 blocks of central cueing and peripheral cueing experiments, for each healthy individual and
278 the two patients. The fixation cross was at 0°, leftward positions were scored negative, and
279 rightward positions positive. P = participants, UP = Unilateral Patient, BP = Bilateral Patient

280

		P1	P2	P3	P4	P5	P6	P7	P8	P9	P10	P11	P12	P13	P14	UP	BP
Central cueing	mean °	0.74	0.36	0.70	-0.05	0.11	1.64	-0.64	1.55	0.33	0.75	0.65	0.38	-0.02	0.25	-0.11	0.31
	STD °	0.75	0.41	0.65	0.97	0.92	0.88	0.64	0.70	1.57	0.90	0.85	0.65	1.24	0.74	0.83	0.95
Peripheral cueing	mean °	0.44	0.18	0.90	-0.21	-0.26	0.36	0.03	0.61	0.34	0.57	-0.05	0.38	0.83	0.47	-0.05	0.70
	STD °	0.85	0.65	0.72	1.06	0.75	0.65	0.65	0.82	1.15	0.95	0.79	0.69	1.03	0.88	0.82	0.70

281

282

283 **3-2. Central cueing experiment**

284 3-2-1. Healthy participants:

285 As can be seen in Figure 3 (left column) healthy participants in valid trials produced gaze-
286 centered hypometric pointing with a bias of 1.71 cm on average and a mean pointing
287 variability of 1.15 cm. In the invalid trials, a condition that potentially simulates OA in

288 healthy participants, hypometria increased to a mean of 1.94 cm with a pointing variability
289 of 1.27 cm.

290

291 The repeated measures ANOVA assessing horizontal pointing bias revealed a main effect of
292 Validity condition ($F(1,13)=6.86$, $p=0.021$, $\eta^2= 0.345$) corresponding to a significant increase
293 of gaze-centered hypometria in invalid cueing trials illustrated in Figure 3. There was also a
294 main effect of visual Eccentricity ($F(4,52)=20.61$, $p<0.001$, $\eta^2= 0.613$; illustrated in Fig. Supp
295 1-Left panel), the higher the target's eccentricity, the more hypometria. The two-way
296 interaction was not significant ($F(4,52)=2.06$, $p=0.10$), $\eta^2= 0.137$).

297

298 For the pointing variability (SDx), repeated measures ANOVA also revealed a main effect of
299 Validity condition ($F(1,13)=10.17$, $p=0.007$, $\eta^2= 0.439$) illustrated in Figure 3 (bottom left), a
300 main effect of Eccentricity ($F(4,52)=48.32$, $p<0.001$, $\eta^2= 0.788$; illustrated on Fig. Supp 1-
301 Right panel), and no significant interaction ($F(4,52)=1.00$, $p=0.42$, $\eta^2= 0.0714$).

302

303 The repeated measures ANOVA assessing RT also revealed a main effect of Validity
304 ($F(1,13)=16.62$, $p=0.0013$, $\eta^2= 0.561$), the mean RT was 1184 ± 171 ms in the valid condition
305 and increased to 1242 ± 167 ms in the invalid condition. There was also a main effect of
306 Eccentricity ($F(4,52)=13.21$, $p<0.001$, $\eta^2= 0.504$) and a significant two-way interaction
307 ($F(4,52)=5.73$, $p<0.001$, $\eta^2= 0.305$; illustrated on Fig. Supp 2); post-hoc analyses showed a
308 significantly longer RT in the invalid than valid condition only at 25° , 30° and 35° of
309 eccentricity (respectively $F(13)=15.36$; $p=0.0018$, $F(13)=28.83$; $p<0.001$, $F(13)=13.54$;
310 $p=0.0028$).

311

312 To sum up, removing attention from the visual field where the visual target is presented
313 (invalid trials) by a central cue that is highly informative (high validity) led healthy
314 participants to produce systematically larger gaze-centered hypometric pointing biases, as
315 well as larger pointing noise and a slowing of the movements aimed to targets presented at
316 eccentricities further than 20°.

317

318 3-2-2. Bilateral OA patient:

319 The question with the OA patient was whether a highly predictive central cue can improve
320 peripheral pointing performance. As can be seen in Figure 3 (right column) patient I.G.
321 displayed large horizontal hypometria of 3.11 cm on average and large pointing variability of
322 2.59 cm in the invalid condition, which corresponds to the typical finding in OA. In the valid
323 condition, gaze-centered hypometria decreased to 2.31 cm, while the pointing noise was
324 2.86 cm on average.

325

326 The factorial ANOVA assessing horizontal pointing bias revealed a main effect of Validity
327 ($F(1,357)=4.98$, $p=0.026$, $\eta^2= 0.0138$) illustrated in Figure 3; the valid central cue significantly
328 decreased gaze-centered hypometria in comparison to the invalid condition. There was
329 neither a significant main effect of target Eccentricity ($F(4,357)=2.55$, $p=0.39$, $\eta^2= 0.0278$) nor
330 a two-way interaction ($F(4,357)=2.83$, $p=0.05$, $\eta^2= 0.0307$).

331

332 The factorial ANOVA assessing RT also revealed a main effect of Validity ($F(1,357)=20.76$,
333 $p<0.001$, $\eta^2= 0.0550$) with RT of 1596 ± 217 ms on average in the invalid condition and a
334 decrease to 1497 ± 181 ms in the valid condition (Fig. Supp 3-Left panel). There was also a
335 main effect Eccentricity ($F(4,357)=19.03$, $p<0.001$, $\eta^2= 0.176$), RT increased with target

336 eccentricity (Fig. Supp 3-Right panel), and no significant interaction ($F(4,357)=1.98$, $p=0.096$,
337 $\eta^2= 0.0217$).

338

339 To sum up, a valid central cue shifted the pointing hypometria of patient I.G. toward the
340 performance of the healthy participants; it also reduced her pointing time but not her
341 pointing noise.

342

343 3-2-3. Unilateral OA patient:

344 As can be seen in Figure 4, the gaze-centered hypometria of 1.99 cm and pointing variability
345 of 2.04 cm observed in the invalid trials in the left (ataxic) visual field of patient C.F.
346 decreased to 0.65 cm and 1.43 cm, respectively, with valid central cueing.

347

348 The factorial ANOVA conducted for the horizontal pointing bias showed a significant main
349 effect of Validity ($F(1, 372)=32.26$, $p<0.001$, $\eta^2= 0.0798$), consisting of a general decrease of
350 gaze-centered hypometria in the valid condition compared to the invalid condition. There
351 was no main effect of visual field ($F(1, 372)=0.89$, $p=0.77$, $\eta^2= 0.00239$) but there was a
352 significant Validity x Visual field interaction ($F(1, 372)=7.16$, $p=0.0078$, $\eta^2= 0.0189$). Clarifying
353 this interaction, Figure 4 shows that the asymmetry of pointing error in the unilateral OA
354 patient between the left and right visual fields in the invalid condition ($F(372)=5.54$, $p=0.019$)
355 was absent in the valid condition ($F(372)=2.97$, $p=0.086$). Post-hoc analyses revealed that
356 this was achieved by a significant decrease of hypometric pointing bias specifically in the left
357 (ataxic) visual field ($F(372)=34.76$, $p<0.001$) between invalid and valid conditions.

358 There was also a significant main effect of Eccentricity ($F(4,372)=3.7542$, $p=0.0052$, $\eta^2=$
359 0.0388), and another significant two-way interaction (Validity x Eccentricity: $F(4, 372)=3.75$,

360 $p=0.0053$, $\eta^2= 0.0388$) illustrated in Fig. Supp 4. Post-hoc analyses indicated that the
361 decrease in pointing bias in the valid condition reached significance bilaterally at 25° and 35°
362 ($p<0.001$). The three-way interaction was not significant ($F(4, 372)=1.68$, $p=0.15$, $\eta^2=$
363 0.0177).

364

365 The factorial ANOVA on RT showed no main effect of Validity ($F(1,372)=0.00$, $p=0.84$, $\eta^2=$
366 0.0) and no significant interactions involving Validity (all $F_s<2.1$, $p_s>0.05$). There were main
367 effects of Visual Field ($F(1,372)=158.2$, $p<0.001$, $\eta^2= 0.298$), with faster responses in the right
368 (healthy) visual field than in the left (ataxic) visual field, and of Eccentricity ($F(4,372)=3.46$,
369 $p=0.0087$, $\eta^2= 0.0359$), with faster response to targets at closer visual eccentricities. There
370 was also a significant interaction between Visual Field and Eccentricity ($F(4,372)=3.26$,
371 $p=0.012$, $\eta^2= 0.0339$) illustrated in Fig. Supp 5, demonstrating a larger increase in RT when
372 target eccentricity increased in the left (ataxic) visual field. Post-hoc analyses revealed that
373 RT differed significantly between the visual fields only at the farthest visual eccentricities
374 ($p<0.001$ for 25°, 30°, and 35°). This indicates that patient C.F. compensated his OA by
375 slowing his pointing movement to targets presented at the farthest visual eccentricities in
376 his ataxic visual field.

377

378 To sum up, for the unilateral OA patient, the valid central cue reduced the pointing bias and
379 variability specifically in the left (ataxic) visual field, while no validity effect was revealed on
380 the RT asymmetry between visual fields.

381

382 **3-3. Peripheral cueing experiment**

383 3-3-1. Healthy participants:

384 As can be seen in Figure 5 (left column) healthy participants in the valid trials produced gaze-
385 centered hypometria of 1.19 cm on average with a mean pointing variability of 1.13 cm. In
386 the invalid trials, a condition that potentially simulates OA in healthy participants,
387 hypometria was 1.31 cm on average, and pointing variability was 1.40 cm.

388

389 The repeated measures ANOVA assessing the effects of Validity and target Eccentricity on
390 horizontal pointing bias showed no main effect of Validity ($F(1,13)=1.22$, $p=0.29$, $\eta^2= 0.0858$),
391 there was a significant main effect of Eccentricity ($F(4,52)=28.62$, $p<0.001$, $\eta^2= 0.688$) and a
392 significant two-way interaction ($F(4,52)=5.31$, $p=0.0012$, $\eta^2= 0.290$). Clarifying this
393 interaction, post-hoc analyses revealed that the peripheral cue had a significant effect only
394 at 15° of eccentricity ($F(13)=11.70$, $p=0.0046$): for this closest target position, there was a
395 small gaze-centered hypometria in the invalid condition but not in the valid condition (mean
396 of -0.06 cm in valid vs. +0.58 cm in invalid condition), as illustrated in Figure 6.

397

398 For pointing variability (SDx), the repeated measures ANOVA demonstrated significant main
399 effects of Validity ($F(1,13)=24.61$, $p<0.001$, $\eta^2= 0.654$) and of Eccentricity ($F(4,52)=49.22$,
400 $p<0.001$, $\eta^2= 0.791$), with no significant interaction ($F(4,52)=0.43$, $p=0.78$, $\eta^2= 0.00320$).
401 These main effects consisted of larger pointing noise in the invalid than valid conditions
402 (Figure 5 bottom left) and increasing pointing noise with target eccentricity (Fig. Supp 6).

403

404 The repeated measures ANOVA assessing RT also revealed a significant main effect of
405 Validity ($F(1,13)=11.75$, $p=0.045$, $\eta^2= 0.475$); RT increased from 1188 ms in the valid
406 condition to 1215 ms in the invalid condition. There were also a main effect of Eccentricity
407 ($F(4,52)=4.92$, $p=0.002$, $\eta^2= 0.275$) and a significant two-way interaction ($F(4,52)=8.98$,

408 $p < 0.001$, $\eta^2 = 0.409$) illustrated in Fig. Supp 7. Clarifying this interaction, post-hoc analyses
409 showed significantly longer RT for the invalid than valid condition at the farthest
410 eccentricities of 30° and 35° (valid: 1157 ± 224 and 1200 ± 218 ms, invalid: 1243 ± 249 and
411 1298 ± 255 ms, $F(13)=28.83$, $p < 0.001$; $F(13)=28.89$, $p < 0.001$, at 30° and 35° respectively).

412

413 To sum up, in this peripheral cueing experiment, the cue was low predictive of the target
414 location (validity of 50% only) and it produced limited modulation of pointing bias (only at
415 the closest visual eccentricity of 15°). Attracting automatic attention to the wrong visual field
416 with a peripheral cue rather produced non-specific detrimental effects like a general
417 increase of pointing noise and a slowing of the pointing response at the farthest
418 eccentricities (30 and 35°).

419

420 3-3-2. Bilateral OA patient:

421 Whether peripheral cueing can improve peripheral pointing performance in OA was tested in
422 this experiment. As can be seen in Figure 5 (right column) patient I.G. displayed horizontal
423 hypometria of 1.87 cm on average and pointing noise (SDx) of 3.06 cm in the invalid
424 condition. In the valid condition, gaze-centered pointing bias was shifted to a mean of -0.30
425 cm with a pointing noise of 2.41 cm.

426 The factorial ANOVA assessing the effect of Validity and Eccentricity on pointing bias
427 revealed a main effect of Validity condition ($F(1,267)=40.26$, $p < 0.001$, $\eta^2 = 0.131$) and a
428 significant main effect of Eccentricity ($F(4,267)=8.53$, $p < 0.001$, $\eta^2 = 0.113$), that were qualified
429 by a significant Eccentricity by Validity interaction ($F(4,267)=11.36$, $p < 0.001$, $\eta^2 = 0.145$)
430 illustrated in Figure 7: a drastic change was observed in horizontal pointing error direction
431 for all targets except at 15° of eccentricity. Post-hoc analyses showed significant changes for

432 targets presented at 20°, 30° and 35° of eccentricity in the valid condition (respectively
433 $F(267)=18.75$; $p<0.001$, $F(267)=12.52$; $p<0.001$, $F(267)=49.86$; $p<0.001$) with pointing biases
434 turning into negative (hypermetric) values.

435

436 The factorial ANOVA assessing RT revealed main effects of Eccentricity ($F(4,267)=12.25$,
437 $p<0.001$, $\eta^2= 0.155$; illustrated in Fig. Supp 8-Left panel) and Validity ($F(1,267)=21.50$,
438 $p<0.001$, $\eta^2= 0.0745$; illustrated in Fig. Supp 8-Right panel), with no significant two-way
439 interaction ($F(4,267)=1.73$, $p=0.14$, $\eta^2= 0.0253$), indicating that the patient produced faster
440 pointing movements with valid peripheral cueing at all visual target eccentricities.

441

442 To sum up, in the valid condition of the peripheral cueing experiment, pointing movements
443 of the bilateral OA patient were faster, less variable, and hypermetric.

444

445 3-3-3. Unilateral OA patient:

446 In the ataxic visual field, patient C.F. produced hypometric pointing biases of 2.08 cm on
447 average in the invalid condition (as compared to 1.28 cm in his healthy visual field).
448 Unexpectedly, the hypometric bias was even larger in the valid condition (mean 2.25 cm).
449 Similarly, pointing variability was 1.46 cm on average in the invalid condition and 1.57 cm in
450 the valid condition (Figure 8).

451

452 The factorial ANOVA assessing the effects of Visual Field, Eccentricity and Validity on gaze-
453 centered hypometria revealed no main effect of Validity ($F(1, 275)=0.00447$, $p=0.947$,
454 $\eta^2<0.001$) but main effects of Eccentricity ($F(4, 275)=12.60$, $p<0.001$, $\eta^2= 0.155$) and Visual

455 field ($F(1, 275)=36.80$, $p<0.001$, $\eta^2= 0.118$). There was also a significant interaction between
456 Validity and Eccentricity ($F(4, 275)=3.44$, $p=0.0091$, $\eta^2= 0.0447$) reflecting a paradoxically
457 higher gaze-centered pointing error in the valid than invalid condition at 30° of eccentricity
458 ($F(275)=7.90$, $p=0.0043$; Fig. Supp 9). There was no other significant two-way interaction
459 (Visual Field x Eccentricity: $F(4, 275)=0.60$, $p=0.67$, $\eta^2=0.00865$; Validity x Visual Field: $F(1,$
460 $275)=0.78$, $p=0.377$, $\eta^2=0.0112$) and the three-way interaction was not significant ($F(4,$
461 $275)=0.21$, $p=0.93$, $\eta^2= 0.00305$).

462

463 The factorial ANOVA revealed a significant main effect of Validity on RT ($F(1, 275)=7.22$,
464 $p=0.0076$, $\eta^2=0.0254$; Fig. Supp 10-Left panel), but no significant interaction that involves
465 validity (Validity x Visual Field: $F(1, 275)=3.45$, $p=0.064$, $\eta^2=0.0124$); Validity x Eccentricity:
466 $F(4, 275)=1.78$, $p=0.132$, $\eta^2=0.0252$; three-way: $F(4,275)=0.08$, $p=0.99$, $\eta^2<0.001$). There was
467 a significant interaction between Visual Field and target Eccentricity ($F(4,275)=4.39$,
468 $p=0.0019$, $\eta^2= 0.0600$; Fig. Supp 10-Right panel) consisting of a significant increase of RT in
469 the ataxic visual field at the farthest visual eccentricities (at 30° and 35°, $F(275)=11.39$;
470 $p<0.001$ and $F(275)=28.92$; $p<0.001$, respectively), demonstrating again that C.F was
471 compensating for his pointing bias.

472

473 To sum up, with the unilateral OA patient there was an overall effect of validity on RT but
474 neither the pointing performance nor the visual field asymmetry were improved in the valid
475 condition of the peripheral cueing experiment.

476

477 **4-Discussion:**

478 In order to test the hypothesis that OA visuomotor impairment can result, at least
479 partially, from an attentional deficit, we investigated 1) whether pointing performance
480 characteristic of OA can be mimicked by removing attention in healthy participants and 2)
481 whether providing attentional cues, which allow patients to shift their covert attention in
482 advance to peripheral target location, can reduce their pointing bias toward gaze fixation.
483 We planned to focus our analysis on the pointing horizontal error, as systematic pointing
484 biases toward the fixation point characterizes peripheral pointing performance in OA
485 (Blangero et al., 2010; Vindras et al., 2016), pointing variability and response time being
486 considered as non-specific effects of attention. Indeed, if attention just helps OA patients be
487 faster or even just reduces their pointing noise, but does not reduce their horizontal pointing
488 errors, one can say that this means attention is not specifically involved in their deficit, but
489 rather helps them as it helps healthy participants in most perceptual tasks. Following the
490 same logic, a specific increase of horizontal (gaze-centered) hypometric pointing bias was
491 expected in healthy participants, as a qualitative simulation of the specific pointing pattern
492 characteristic of OA in the invalid condition when the pointing movement results from
493 positional encoding of the goal without spatial attention.

494 Manipulations of attention in multiple variants of the Posner (1980) paradigm have
495 explored various methodological parameters (e.g., combinations of cue type, cue
496 predictiveness, cue-target delay, unique or various target locations, presence or absence of
497 flankers) and raised ongoing debates about the nature of attention depending on these
498 parameters (e.g., Wojciulik & Kanwisher, 1999; Eimer 1997; Tipples 2002; Chambers et al
499 2004; Macaluso & Doricchi, 2013). When considering OA, the study of Martin-Arevalo et
500 al. (2021) indicated that the type of cue matters in OA: in two experiments of covert
501 attention (adapted from the paradigm of Deubel & Schneider, 1996) involving target

502 identification among flankers with central versus peripheral cues, patient PE showed
503 decreased performance in her contralesional visual field only with the central cue. For the
504 present study, we therefore reasoned that the experimental conditions that would be the
505 most relevant to OA should involve the two most acknowledged and classical combinations
506 (Carrasco, 2011) of cue type, its predictiveness, and its timing, that tap into endogenous
507 attention and exogenous attention. Additionally, the field effect of OA consists of typical
508 pointing errors when the participants attempt to point at a specific peripheral location (i.e.,
509 the target location) not merely when they execute a standardized movement toward a side,
510 and importantly they occur whether the field is blocked, such that OA patients can put all
511 their “general” attention toward the affected side (Perenin & Vighetto, 1988) or unblocked,
512 such that OA patients cannot form any expectation on target location and side (Blangero et
513 al., 2010; Vindras et al., 2016). Thus, in both experiments of the present study, the
514 attentional cue indicated a specific location rather than a side.

515 The results revealed that the cueing procedure combining a directional central arrow
516 with 75% predictability of the target position was effective in both simulating OA horizontal
517 pointing errors in healthy participants (when the cue was invalid) and alleviating the bilateral
518 or unilateral pointing biases of OA patients (when the cue was valid). As for the low validity
519 peripheral cue, the fact that it speeded response time suggests that it indeed attracted
520 attention exogenously toward its location, that is to the opposite side when it was invalid or
521 to the target location when it was valid. However, when considering its effects on pointing
522 accuracy, the results were not as satisfactory. In healthy participants, gaze-centered
523 hypometria was produced only for the closest target at 15° in the invalid condition of the
524 peripheral cueing experiment. In OA patients, valid peripheral cueing did not produce
525 consistent effects on the pointing bias either. The patient with bilateral OA produced

526 unexpected hypermetric errors for targets further than 15°, which could reflect some
527 strategy of overcompensation following perceptual masking of the visual target by the
528 peripheral cue. Indeed, patient I.G. is aware of her pointing deficit in peripheral vision.
529 Moreover, she reported that she was “guessing more often” the visual target position in this
530 peripheral cueing experiment. We think that the flashing of the peripheral line above the
531 target position just before its presentation could have masked the target in the valid trials.
532 Consequently, the bilateral OA patient may have produced a force-choice pointing response
533 far away from gaze fixation only in the valid condition. As for the unilateral OA patient, the
534 visual field asymmetry of his pointing accuracy was not reduced by the peripheral cueing,
535 neither in terms of pointing bias nor pointing variability, even though the cue was efficient in
536 allowing the patient to produce overall faster movements in the valid trials.

537 The effects of attentional cueing on pointing variability had a different pattern from
538 that of pointing bias. Pointing variability was only modified by central cueing with the
539 unilateral OA patient. The reverse was observed with the bilateral OA patient: pointing
540 variability was only modified with peripheral cueing. Despite this difference, a change in
541 pointing bias was observed with both patients with central cueing. With healthy participants,
542 the pointing variability was modified with both central and peripheral cueing, whereas the
543 pointing bias was modified only with central cueing. These different patterns of results
544 suggest that pointing variability and pointing bias are dissociated measures. Indeed, a
545 systematic pointing error centered on current gaze direction has been postulated to reflect a
546 specific encoding bias of the visual target position in an oculocentric coordinate system
547 (Henriques et al., 1998; Medendorp & Crawford, 2002; Pouget et al., 2002; Khan et al.,
548 2005a,b). Pointing variability would reflect a non-specific pointing noise distributed equally
549 around this encoded target position (Revol et al., 2003). Therefore, a change in pointing

550 noise cannot explain a change in pointing bias. A modulation of reaching endpoints by visuo-
551 attentional cueing can affect, independently from each other, either visuomotor variability
552 or positional encoding bias of the visual target.

553 The interaction between reaching endpoints and response time is also a potential
554 issue, that has already been addressed in the literature on OA. Response time includes
555 movement reaction time and duration, which have both been shown by Striemer et al.
556 (2009) to be increased for pointing toward the ataxic visual field in unilateral OA.
557 Nevertheless, Striemer et al. (2009) also observed that accuracy of endpoints was strongly
558 modulated by target eccentricity contrary to reaction time, whether the latter involved the
559 latency of a button press response or of a reaching response. They therefore considered that
560 pointing accuracy and reaction time were dissociated measures, the latter possibly reflecting
561 perception (target detection) rather than action. The observed modulation of response time
562 by visuo-attentional cueing in the present study could indeed have been mediated by
563 enhanced visual acuity and perceptual target detection (Yeshurun & Carrasco, 1999) or
564 motor preparation, parameters that are all non-specific of OA (Pisella et al., 2000). However,
565 a paradoxical improvement of pointing accuracy has been shown in OA when a delay is
566 introduced between peripheral target presentation and go signal (Milner et al., 1999; Revol
567 et al., 2003; Khan et al., 2005b). A longer reaction time has been interpreted to allow a
568 change of reference frame for target localization from gaze-centered (specifically impaired in
569 OA) to a more stable one. Pointing accuracy and reaction time would thus not be so
570 independent. Our experimental design in which pointing responses were required with no
571 temporal constraint could have minimized 1) the pointing errors in OA patients and 2) the
572 effects of attention on the pointing errors in all participants. With the unilateral OA patient,
573 there was a significant visual field difference in response time only at the farthest visual

574 eccentricities, the further away the targets were presented in his left (ataxic) visual field, the
575 more cautious his response was in both experiments. However, this did not interact with
576 validity: he seems to have a compensatory strategy for his unilateral deficit involving slower
577 responses when pointing toward the left visual field, independent of his ability to allocate
578 covert spatial attention to a given location based on visual cues. Consequently, the beneficial
579 effect of the central attentional cue on the positional encoding bias characteristic of his
580 unilateral OA can hardly be explained by the general slowing of his leftward pointing
581 movement. With the bilateral OA patient and healthy participants, the effect of central
582 cueing on horizontal pointing bias was also significant, regardless of its response time
583 modulation. With both central and peripheral cueing, an interaction of validity and
584 eccentricity was observed on response time with the healthy participants. In the invalid
585 condition, their pointing response was even slower at the farthest eccentricities (30 and
586 35°). This possibly reflects an increased perceptual uncertainty in the invalid conditions.
587 Importantly, because these cueing effects on response time were found with both cue types,
588 they suggest that we could successfully modulate attention in both experiments. Thus, the
589 fact that attentional modulation of the gaze-centered pointing bias was observed with the
590 high predictive central cue but not with the peripheral cue cannot be attributed to a failure
591 to manipulate attention with the latter.

592 Most importantly, the present study revealed that at least the removal of top-down
593 endogenous attention in the invalid trials using the high predictive central cue allowed us to
594 simulate the hypometric gaze-centred pointing bias typical of OA in healthy controls. This
595 supports the hypothesis (see Pisella et al., 2013 and Martin-Arevalo et al., 2021) that OA may
596 emerge, at least partially, from impaired (possibly slowed or inaccurate) endogenous
597 orienting of covert attention. Further investigations are needed to determine whether this

598 effect can be extended beyond the specific expectancy, target-cue timing or the type of cues
599 we examined. Besides, several cueing manipulations of exogenous attention could be
600 interesting to test in the future and some of them may, indeed, produce a similar effect to
601 that found here with endogenous attention.

602 Another interesting issue arising concerning our results is the potential dissociation
603 between allocating endogenous attention to a location and forming expectations about the
604 most likely target location. In our central cue experiment, the central arrow was valid on
605 75% of the trials, and therefore, by itself, this manipulation does not allow disentangling
606 attention and expectations. One may wonder whether a paradigm with a 50% valid central
607 cue would allow disambiguating endogenous attention and expectations, because previous
608 studies (Eimer, 1997 and Tipples, 2002) have shown attentional effects with 50% valid
609 central arrows. However, Hein et al. (2006) showed that such central arrows drive
610 exogenous rather than endogenous attention. Importantly, when our findings are
611 considered alongside those from previous studies on OA showing a robust field effect in very
612 different conditions of expectancy (e.g., Perenin & Vighetto 1988; Blangero et al., 2010;
613 Vindras et al., 2016; Martin-Arevalo et al., 2021), attributing our results to the top-down
614 attention allocation internally-driven based on the central arrow appears to be the more
615 likely explanation. Our result and interpretation are in line with other findings, reviewed by
616 Macaluso & Doricchi (2013), leading them to conclude that *“attention selection entails top-
617 down signals that originate in the dorsal system and modulate activity in sensory areas that
618 represent the currently relevant location [...]. This, in turns, would yield to greater responses
619 when visual stimuli appear at the attended location, particularly so when the display also
620 contains other distracting visual stimuli [...].”* (page 2). Indeed, Bogler et al. (2011) and Nardo
621 et al. (2011) proposed that dorsal PPC represents the target selected from saliency maps

622 following a winner-takes-all mechanism for further perceptual or visuomotor processing
623 (covert or overt orienting of attention). A deficient winner-take-all mechanism following SPL
624 lesion could explain the increased spatial competition between visual fixation cross and
625 visual target in peripheral pointing condition, and the co-occurrence of bilateral optic ataxia
626 and simultanagnosia. This is in accordance with views of dorsal visual attention selection as a
627 competitive integration mechanism that biases competition between visual inputs toward
628 relevant information, both by enhancing the neural representation(s) of the relevant object
629 and by inhibiting the representation(s) of distractors (e.g., Wojciulik & Kanwisher 1999;
630 Jackson et al., 2005, 2009; Ouerfelli-Ethier et al., 2021, 2023).

631 As for the improvement in the OA patients in condition of valid endogenous
632 attentional cueing, despite damage to the SPL-IPS region corresponding to the posterior part
633 of the dorsal attentional network (Corbetta & Schulman 2002; Gillebert et al., 2010; Martin-
634 Arevalo et al., 2021), our interpretation is two-fold. First, since the exogenous cueing
635 employed here was not able to modulate the horizontal pointing error in controls, an
636 improvement of the typical pointing bias of OA patients by exogenous attention seems
637 unlikely, even if resources of exogenous attention remained available after their lesion.
638 Second, at the neurophysiological level, attentional orienting is based on the combined
639 action of different—though functionally related— frontal and parietal areas. This allows
640 hypothesizing residual resources of endogenous orienting in OA patients, that could be
641 activated by the valid central cue and could alleviate the horizontal pointing errors in
642 patients. The most likely would be via the frontal area that form the anterior part of the
643 dorsal attention network. As for the exact process and neurophysiological route underlying
644 this alleviation, alternative explanations exist, that further investigations should disentangle.
645 For example, unlike Martin-Arevalo et al. (2021) who used a paradigm involving accurate

646 spatial attentional cueing in order to identify a flanked visual target, Striemer et al. (2007)
647 used a classical Posner (1980) paradigm measuring reaction times to a single visual target
648 presentation and showed impairment in both endogenous and exogenous attentional cueing
649 paradigm. Moreover, this impairment was observed in valid, invalid and neutral trials, which
650 led the authors to hypothesize a global visual saliency decrease for targets presented within
651 the contralesional visual field. Applying this hypothesis to the improvement of pointing
652 performance observed in the present study for the two OA patients, possibly mediated via
653 the anterior attentional network, it could be conceived that the highly predictive central cue
654 has produced a top-down global increase of the cued position representation in frontal
655 priority maps and/or that it has increased the default saliency of contralesional targets by
656 boosting the Frontal Eye Field feedback modulation of visual areas (see Martin-Arevalo et al.,
657 2021 for neurophysiological argument in favour of the latter proposal).

658 Interestingly, exploring the interaction between perception and action programming
659 in patients with visual extinction with and without optic ataxia, Kitadono and Humphreys
660 (2007, 2011) also argued for strong coupling between endogenous visual and motor-based
661 attention. More precisely, the patients had to make pointing responses to left and right
662 locations, whilst identifying briefly presented shapes. Interaction between movement
663 direction and perceptual report was not found in the patient with OA, probably due to a
664 common deficiency of visuo-spatial orienting in the contralesional field affecting both
665 perceptual report and pointing. In the patient without OA, identification of contralesional
666 targets was ameliorated when the action was programmed to the contralesional side, and
667 programming an action to the ipsilesional side increased extinction (on 2-item trials) and
668 tended to induce neglect (on 1-item trials). Identification of contralesional targets was also
669 improved by an increase in the probability of occurrence of the visual target in the

670 contralesional visual field, but this advantage vanished when a concurrent movement was
671 planned toward the ipsilesional side. This was interpreted as a common system of visuo-
672 spatial orienting for endogenous attention and action.

673 To summarize, when the endogenous attention of healthy participants was directed
674 to the wrong location, their pattern of pointing to peripheral targets showed a gaze-
675 centered bias that is typical of OA. Accordingly, when the endogenous attention of OA
676 patients could be directed in advance to the target location, the typical pointing bias was
677 reduced. Taken together these findings support the hypothesis that OA reflects, at least
678 partially, a deficit in the allocation of endogenous attention.

679

680 Acknowledgements:

681 The authors wish to thank S Alouche and F Volland for their valuable technical support. This
682 research was supported by funds from Inserm, CNRS, Université de Lyon and HCL
683 “Mouvement et Handicap” Platform and partly supported by the Neurodis foundation
684 visiting grant to Y.Y.

685

686 **References:**

687 Andersen, R. A., Buneo, C. A., 2002. Intentional maps in posterior parietal cortex. Annual
688 review of neuroscience, 25, 189–220.

689 <https://doi.org/10.1146/annurev.neuro.25.112701.142922>

690

691 Aguilar Ros, A., Mitchell, A. G., Ng, Y. W., McIntosh, R. D., 2021. Attention attracts action in
692 healthy participants: An insight into optic ataxia?. Cortex; a journal devoted to the study of
693 the nervous system and behavior, 137, 149–159.

694 <https://doi.org/10.1016/j.cortex.2021.01.003>

695

696 Blangero, A., Ota, H., Delporte, L., Revol, P., Vindras, P., Rode, G., Boisson, D., Vighetto, A.,
697 Rossetti, Y., Pisella, L., 2007. Optic ataxia is not only 'optic': impaired spatial integration of
698 proprioceptive information. NeuroImage, 36 Suppl 2, T61–T68.

699 <https://doi.org/10.1016/j.neuroimage.2007.03.039>

700

701 Blangero, A., Gaveau, V., Luauté, J., Rode, G., Salemme, R., Guinard, M., Boisson, D., Rossetti,
702 Y., Pisella, L., 2008. A hand and a field effect in on-line motor control in unilateral optic
703 ataxia. Cortex; a journal devoted to the study of the nervous system and behavior, 44(5),

704 560–568. <https://doi.org/10.1016/j.cortex.2007.09.004>

705

706 Blangero, A., Ota, H., Rossetti, Y., Fujii, T., Ohtake, H., Tabuchi, M., Vighetto, A., Yamadori, A.,
707 Vindras, P., Pisella, L., 2010. Systematic retinotopic reaching error vectors in unilateral optic
708 ataxia. Cortex; a journal devoted to the study of the nervous system and behavior, 46(1), 77–

709 93. <https://doi.org/10.1016/j.cortex.2009.02.015>

710

711 Brefczynski-Lewis, J. A., Datta, R., Lewis, J. W., DeYoe, E. A., 2009. The topography of
712 visuospatial attention as revealed by a novel visual field mapping technique. *Journal of*
713 *cognitive neuroscience*, 21(7), 1447–1460. <https://doi.org/10.1162/jocn.2009.21005>

714

715 Bogler, C., Bode, S., & Haynes, J. D., 2011. Decoding successive computational stages of
716 saliency processing. *Current biology : CB*, 21(19), 1667–1671.
717 <https://doi.org/10.1016/j.cub.2011.08.039>

718

719 Buneo, C. A., Andersen, R. A., 2006. The posterior parietal cortex: sensorimotor interface for
720 the planning and online control of visually guided movements. *Neuropsychologia*, 44(13),
721 2594–2606. <https://doi.org/10.1016/j.neuropsychologia.2005.10.011>

722

723 Carrasco, M. (2011). Visual attention: The past 25 years. *Vision Research*, 51(13), 1484–1525.

724

725 Carey, D. P., Coleman, R. J., Della Sala, S., 1997. Magnetic misreaching. *Cortex; a journal*
726 *devoted to the study of the nervous system and behavior*, 33(4), 639–652.
727 [https://doi.org/10.1016/s0010-9452\(08\)70722-6](https://doi.org/10.1016/s0010-9452(08)70722-6)

728

729 Cheviet, A., Pisella, L., Pélisson, D., 2021. The posterior parietal cortex processes visuo-
730 spatial and extra-retinal information for saccadic remapping: A case study. *Cortex; a journal*
731 *devoted to the study of the nervous system and behavior*, 139, 134–151.
732 <https://doi.org/10.1016/j.cortex.2021.02.026>

733

734 Colby, C. L., Goldberg, M. E., 1999. Space and attention in parietal cortex. Annual review of
735 neuroscience, 22, 319–349. <https://doi.org/10.1146/annurev.neuro.22.1.319>
736

737 Corbetta, M., Shulman, G. L., 2002. Control of goal-directed and stimulus-driven attention in
738 the brain. Nature reviews. Neuroscience, 3(3), 201–215. <https://doi.org/10.1038/nrn755>
739

740 Deubel, H., & Schneider, W. X., 1996. Saccade target selection and object recognition:
741 evidence for a common attentional mechanism. Vision research, 36(12), 1827–1837.
742 [https://doi.org/10.1016/0042-6989\(95\)00294-4](https://doi.org/10.1016/0042-6989(95)00294-4)
743

744 Eimer, M., 1997. Uninformative symbolic cues may bias visual-spatial attention: Behavioral
745 and electrophysiological evidence. Biological Psychology, 46(1), 67-71.
746

747 Faillenot, I., Toni, I., Decety, J., Grégoire, M. C., Jeannerod, M., 1997. Visual pathways for
748 object-oriented action and object recognition: functional anatomy with PET. Cerebral cortex
749 (New York, N.Y. : 1991), 7(1), 77–85. <https://doi.org/10.1093/cercor/7.1.77>
750

751 Faillenot, I., Decety, J., Jeannerod, M., 1999. Human brain activity related to the perception
752 of spatial features of objects. NeuroImage, 10(2), 114–124.
753 <https://doi.org/10.1006/nimg.1999.0449>
754

755 Garcin R, Rondot P, Recondo J. Ataxie optique localisée aux deux hémichamps visuels
756 homonymes gauches. Rev Neurol. 1967;116:707–714.
757

758 Gillebert, C. R., Mantini, D., Thijs, V., Sunaert, S., Dupont, P., Vandenberghe, R., 2011. Lesion
759 evidence for the critical role of the intraparietal sulcus in spatial attention. *Brain : a journal*
760 *of neurology*, 134(Pt 6), 1694–1709. <https://doi.org/10.1093/brain/awr085>
761

762 Gréa, H., Pisella, L., Rossetti, Y., Desmurget, M., Tilikete, C., Grafton, S., Prablanc, C.,
763 Vighetto, A., 2002. A lesion of the posterior parietal cortex disrupts on-line adjustments
764 during aiming movements. *Neuropsychologia*, 40(13), 2471–2480.
765 [https://doi.org/10.1016/s0028-3932\(02\)00009-x](https://doi.org/10.1016/s0028-3932(02)00009-x)
766

767 Hein, E., Rolke, B., & Ulrich, R., 2006. Visual attention and temporal discrimination:
768 Differential effects of automatic and voluntary cueing. *Visual Cognition*, 13(1), 29–50.
769 <https://doi.org/10.1080/13506280500143524>
770

771 Henriques, D. Y., Klier, E. M., Smith, M. A., Lowy, D., Crawford, J. D., 1998. Gaze-centered
772 remapping of remembered visual space in an open-loop pointing task. *The Journal of*
773 *neuroscience : the official journal of the Society for Neuroscience*, 18(4), 1583–1594.
774 <https://doi.org/10.1523/JNEUROSCI.18-04-01583.1998>
775

776 Holmes G. Disturbances of visual orientation. *Br J Ophthalmol*. 1918 Sep;2(9):449–516.
777

778 Jackson, S. R., Newport, R., Mort, D., Husain, M., 2005. Where the eye looks, the hand
779 follows; limb-dependent magnetic misreaching in optic ataxia. *Current biology : CB*, 15(1),
780 42–46. <https://doi.org/10.1016/j.cub.2004.12.063>
781

782 Jackson, S. R., Newport, R., Husain, M., Fowlie, J. E., O'Donoghue, M., Bajaj, N., 2009. There
783 may be more to reaching than meets the eye: re-thinking optic ataxia. *Neuropsychologia*,
784 47(6), 1397–1408. <https://doi.org/10.1016/j.neuropsychologia.2009.01.035>
785

786 Khan, A. Z., Pisella, L., Vighetto, A., Cotton, F., Luauté, J., Boisson, D., Salemme, R., Crawford,
787 J. D., Rossetti, Y., 2005. Optic ataxia errors depend on remapped, not viewed, target
788 location. *Nature neuroscience*, 8(4), 418–420. <https://doi.org/10.1038/nn1425>
789

790 Khan, A. Z., Pisella, L., Rossetti, Y., Vighetto, A., Crawford, J. D., 2005. Impairment of gaze-
791 centered updating of reach targets in bilateral parietal-occipital damaged patients. *Cerebral*
792 *cortex* (New York, N.Y. : 1991), 15(10), 1547–1560. <https://doi.org/10.1093/cercor/bhi033>
793

794 Kitadono, K., Humphreys, G. W., 2007. Interactions between perception and action
795 programming: evidence from visual extinction and optic ataxia. *Cognitive neuropsychology*,
796 24(7), 731–754. <https://doi.org/10.1080/02643290701734721>
797

798 Kitadono, K., Humphreys, G. W., 2011. Neuropsychological evidence for an interaction
799 between endogenous visual and motor-based attention. *Neurocase*, 17(4), 323–331.
800 <https://doi.org/10.1080/13554794.2010.509322>
801

802 Knights, E., McIntosh, R. D., Ford, C., Buckingham, G., Rossit, S., 2024. Peripheral and
803 bimanual reaching in a stroke survivor with left visual neglect and extinction.
804 *Neuropsychologia*, 201, 108901. <https://doi.org/10.1016/j.neuropsychologia.2024.108901>
805

806 Macaluso, E., & Doricchi, F., 2013. Attention and predictions: control of spatial attention
807 beyond the endogenous-exogenous dichotomy. *Frontiers in human neuroscience*, 7, 685.
808 <https://doi.org/10.3389/fnhum.2013.00685>
809

810 Martin-Arevalo, E., Guedj, C., Cotton, F., Rode, G., Reilly, K.T., Hadj-Bouziane, F., Pisella, L.,
811 2021. Neuropsychological assessment of a single-case with posterior parietal lesion using
812 behavioural testing and resting state fMRI. *OBM Neurobiology*, 5(3):20.
813 <https://doi.org/10.21926/obm.neurobiol.2103105>
814

815 Medendorp, W. P., Crawford, J. D., 2002. Visuospatial updating of reaching targets in near
816 and far space. *Neuroreport*, 13(5), 633–636. [https://doi.org/10.1097/00001756-200204160-](https://doi.org/10.1097/00001756-200204160-00019)
817 [00019](https://doi.org/10.1097/00001756-200204160-00019)
818

819 Medina, J., Jax, S. A., Coslett, H. B., 2020. Impairments in action and perception after right
820 intraparietal damage. *Cortex; a journal devoted to the study of the nervous system and*
821 *behavior*, 122, 288–299. <https://doi.org/10.1016/j.cortex.2019.02.004>
822

823 Milner, A. D., Paulignan, Y., Dijkerman, H. C., Michel, F., Jeannerod, M., 1999. A paradoxical
824 improvement of misreaching in optic ataxia: new evidence for two separate neural systems
825 for visual localization. *Proceedings. Biological sciences*, 266(1434), 2225–2229.
826 <https://doi.org/10.1098/rspb.1999.0912>
827

828 Mikula, L., Blohm, G., Koun, É., Khan, A. Z., Pisella, L., 2021. Movement drift in optic ataxia
829 reveals deficits in hand state estimation in oculocentric coordinates. *Journal of experimental*

830 psychology. *Human perception and performance*, 47(5), 635–647.
831 <https://doi.org/10.1037/xhp0000901>
832
833 Nardo, D., Santangelo, V., & Macaluso, E., 2011. Stimulus-driven orienting of visuo-spatial
834 attention in complex dynamic environments. *Neuron*, 69(5), 1015–1028.
835 <https://doi.org/10.1016/j.neuron.2011.02.020>
836
837 Ouerfelli-Ethier, J., Salemme, R., Fournet, R., Urquizar, C., Pisella, L., & Khan, A. Z., 2021.
838 Impaired Spatial Inhibition Processes for Interhemispheric Anti-saccades following Dorsal
839 Posterior Parietal Lesions. *Cerebral cortex communications*, 2(3), tgab054.
840 <https://doi.org/10.1093/texcom/tgab054>
841
842 Ouerfelli-Ethier, J., Fournet, R., Khan, A. Z., & Pisella, L., 2023. Spatial bias in anti-saccade
843 endpoints following bilateral dorsal posterior parietal lesions. *The European journal of*
844 *neuroscience*, 58(6), 3488–3502. <https://doi.org/10.1111/ejn.16102>
845
846 Perenin, M. T., Vighetto, A., 1988. Optic ataxia: a specific disruption in visuomotor
847 mechanisms. I. Different aspects of the deficit in reaching for objects. *Brain : a journal of*
848 *neurology*, 111 (Pt 3), 643–674. <https://doi.org/10.1093/brain/111.3.643>
849
850 Pisella, L., Gréa, H., Tilikete, C., Vighetto, A., Desmurget, M., Rode, G., Boisson, D., Rossetti,
851 Y., 2000. An 'automatic pilot' for the hand in human posterior parietal cortex: toward
852 reinterpreting optic ataxia. *Nature neuroscience*, 3(7), 729–736.
853 <https://doi.org/10.1038/76694>

854

855 Pisella, L., Binkofski, F., Lasek, K., Toni, I., Rossetti, Y., 2006. No double-dissociation between
856 optic ataxia and visual agnosia: multiple sub-streams for multiple visuo-manual integrations.
857 *Neuropsychologia*, 44(13), 2734–2748.

858 <https://doi.org/10.1016/j.neuropsychologia.2006.03.027>

859

860 Pisella, L., Ota, H., Vighetto, A., Rossetti, Y., 2008. Optic ataxia and Bálint's syndrome:
861 neuropsychological and neurophysiological prospects. *Handbook of clinical neurology*, 88,
862 393–415. [https://doi.org/10.1016/S0072-9752\(07\)88020-1](https://doi.org/10.1016/S0072-9752(07)88020-1)

863

864 Pisella, L., Sergio, L., Blangero, A., Torchin, H., Vighetto, A., Rossetti, Y., 2009. Optic ataxia
865 and the function of the dorsal stream: contributions to perception and action.
866 *Neuropsychologia*, 47(14), 3033–3044.

867 <https://doi.org/10.1016/j.neuropsychologia.2009.06.020>

868

869 Pisella L., Blangero A., Tilikete C., Biotti D., Rode G., Vighetto A., Mattingley J.B., Rossetti Y.
870 (2013). Attentional disorders. Chapter 16. In: Kevin Ochsner and Stephen Kosslyn (Eds) : *The*
871 *Oxford Handbook of Cognitive Neuroscience: Volume 1 Core Topics*. Oxford University Press,
872 Oxford, UK, 319-350.

873

874 Pisella, L., Rossetti, Y., Rode, G., 2017. Optic ataxia in Bálint-Holmes syndrome. *Annals of*
875 *physical and rehabilitation medicine*, 60(3), 148–154.

876 <https://doi.org/10.1016/j.rehab.2016.01.003>

877

878 Pisella, L., Vialatte, A., Khan, A. Z., Rossetti, Y., 2021. Bálint syndrome. Handbook of clinical
879 neurology, 178, 233–255. <https://doi.org/10.1016/B978-0-12-821377-3.00011-8>
880

881 Posner M. I., 1980. Orienting of attention. The Quarterly journal of experimental psychology,
882 32(1), 3–25. <https://doi.org/10.1080/00335558008248231>
883

884 Pouget, A., Ducom, J. C., Torri, J., Bavelier, D., 2002. Multisensory spatial representations in
885 eye-centered coordinates for reaching. Cognition, 83(1), B1–B11.
886 [https://doi.org/10.1016/S0010-0277\(01\)00163-9](https://doi.org/10.1016/S0010-0277(01)00163-9)
887

888 Ratcliff, G., 1991. Brain and space: Some deductions from the clinical evidence. In J.
889 Paillard (Ed.), Brain and space (pp. 237–250). Oxford University Press
890

891 Ratcliff, G., Davies-Jones, G. A. B., 1972. Defective visual localization in focal brain wounds.
892 Brain, 95(1), 49- 60. <https://doi.org/10.1093/brain/95.1.49>
893

894 Revol, P., Rossetti, Y., Vighetto, A., Rode, G., Boisson, D., Pisella, L., 2003. Pointing errors in
895 immediate and delayed conditions in unilateral optic ataxia. Spatial vision, 16(3-4), 347–364.
896 <https://doi.org/10.1163/156856803322467572>
897

898 Striemer, C., Blangero, A., Rossetti, Y., Boisson, D., Rode, G., Vighetto, A., Pisella, L.,
899 Danckert, J., 2007. Deficits in peripheral visual attention in patients with optic ataxia.
900 Neuroreport, 18(11), 1171–1175. <https://doi.org/10.1097/WNR.0b013e32820049bd>
901

902 Striemer, C., Locklin, J., Blangero, A., Rossetti, Y., Pisella, L., Danckert, J., 2009. Attention for
903 action? Examining the link between attention and visuomotor control deficits in a patient
904 with optic ataxia. *Neuropsychologia*, 47(6), 1491–1499.

905 <https://doi.org/10.1016/j.neuropsychologia.2008.12.021>

906

907 Tipples, J., 2002. Eye gaze is not unique: Automatic orienting in response to uninformative
908 arrows. *Psychonomic bulletin & review*, 9(2), 314-318.

909

910 Ungerleider, L., Mishkin, M., 1982. Two Cortical Visual Systems. In D. Ingle, M. Goodale, & R.
911 Mansfield (Eds.), *Analysis of Visual Behavior* (pp. 549-586). Cambridge: MIT Press.

912

913 Vialatte, A., Salemme, R., Khan, A. Z., Pisella, L., 2021. Attentional limits in visual search with
914 and without dorsal parietal dysfunction: space-based window or object-based span?.

915 *Neuropsychologia*, 161, 108013. <https://doi.org/10.1016/j.neuropsychologia.2021.108013>

916

917 Vighetto, A., 1980. *Etude neuropsychologique et psychophysique de l'ataxie optique*. Univ.
918 Claude Bernard Lyon I [Thèse].

919

920 Vindras, P., Blangero, A., Ota, H., Reilly, K. T., Rossetti, Y., Pisella, L., 2016. The Pointing Errors
921 in Optic Ataxia Reveal the Role of "Peripheral Magnification" of the PPC. *Frontiers in*

922 *integrative neuroscience*, 10, 27. <https://doi.org/10.3389/fnint.2016.00027>

923

924 Vossel, S., Thiel, C. M., Fink, G. R., 2006. Cue validity modulates the neural correlates of
925 covert endogenous orienting of attention in parietal and frontal cortex. *NeuroImage*, 32(3),
926 1257–1264. <https://doi.org/10.1016/j.neuroimage.2006.05.019>

927

928 Wojciulik, E., & Kanwisher, N., 1999. The generality of parietal involvement in visual
929 attention. *Neuron*, 23(4), 747–764. [https://doi.org/10.1016/s0896-6273\(01\)80033-7](https://doi.org/10.1016/s0896-6273(01)80033-7)

930

931 Yeshurun, Y., Carrasco, M., 1998. Attention improves or impairs visual performance by
932 enhancing spatial resolution. *Nature*, 396(6706), 72–75. <https://doi.org/10.1038/23936>

933

934 Yeshurun, Y., Carrasco, M., 1999. Spatial attention improves performance in spatial
935 resolution tasks. *Vision research*, 39(2), 293–306. <https://doi.org/10.1016/s0042->

936 6989(98)00114-x

937