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Deficits in peripheral visual attention in patients with optic ataxia.

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Abstract

Previous research has suggested that optic ataxia – a deficit in reaching in peripheral vision, can be isolated from Balint’s syndrome as it is primarily a visuomotor disorder, independent of perceptual or attentional deficits. Yet, almost no research has examined the attentional abilities of these patients. We examined peripheral visual attention in two patients with unilateral optic ataxia. Results indicated that both patients were slower to respond to targets in their ataxic visual field, irrespective of cuing condition (i.e., validly, invalidly, and no cue conditions), consistent with an overall decrease in the salience of stimuli in the ataxic field. Attentional deficits in peripheral vision are therefore an important factor to consider when examining visuomotor control deficits in optic ataxia.

Keywords: optic ataxia, covert attention, intraparietal sulcus, parietal lobe, stimulus salience

Introduction

Lesions of superior parietal cortex often lead to optic ataxia – impaired reaching under visual guidance [1]. Optic ataxia patients typically misreach for peripheral targets, whereas actions in central vision are spared [2]. In addition, the visuomotor deficits are not thought to be associated with perceptual or attention deficits [1]. Although the visuomotor abilities of optic ataxia patients have been studied extensively in the periphery, their ability to direct attention towards the periphery has received less attention [2,3]. In the current study we examined reflexive and voluntary attention in the periphery in optic ataxia patients.

Reflexive and voluntary attention were explored using the covert orienting of visual attention task in which participants fixate centrally while attending to locations in the periphery. Attention is cued by brightening one location or presenting a central arrow to direct attention to the left or right. On ‘valid’ trials, targets appear at the cued location. On ‘invalid’ trials, targets appear at the uncued location with a response time advantage for valid over invalid targets [4]. Faster response times for valid trials reflect the fact that attention has been directed to the cue. On invalid trials attention must be disengaged from the cue and moved to the uncued location [5].

Previous studies of covert attention in parietal patients *without* optic ataxia demonstrated a deficit in ‘disengaging’ attention from ipsilesional cues in order to detect contralesional targets [5,6]. Neuroimaging and monkey neurophysiology data have identified regions in the intraparietal sulcus and superior parietal lobe that are important for attending to locations in space, and preparing and executing saccades and reaching movements [7,8]. It is possible that lesions in this area will disrupt both attention *and* visuomotor control.

Methods

Participants

Two patients with optic ataxia completed voluntary and reflexive covert orienting tasks. C.F. is a 27 year-old right-handed male who suffered bilateral lesions of the superior parietal lobule (right lesion larger than the left) at age 24 (Fig 1). C.F. presented with Balint's syndrome post stroke, but at the time of testing only displayed symptoms of optic ataxia in left visual space [9]. M.E. is an 88 year-old female tested 8 months post-stroke affecting the right superior parietal lobule with minor extension into the angular gyrus (Fig 1). Initial bedside testing demonstrated neglect which resolved by the time of testing. M.E. showed optic ataxia in left visual space.

--insert Figure 1 here--

Optic ataxia was assessed by asking the patient to grasp a pen placed in either peripheral or central vision while maintaining central fixation [1]. Both patients made reaching errors when grasping targets with either hand in the left space but not for grasping targets in central vision.

Seventy-one healthy individuals (26 male, 3 left-handed; mean age 19.8 years) completed the voluntary orienting task and 60 healthy individuals (22 male, 5 left-handed; mean age 20.8 years) completed the reflexive orienting task. These served as control groups for C.F. In addition, six healthy elderly individuals (4 male, 1 left-handed, mean age 76.8 years, range 71-85) completed both tasks and served as controls for M.E. All persons gave informed consent prior to participation in accordance with the Helsinki declaration. All protocols were approved by the University of Waterloo Ethics Committee, the Tri-Hospital Research Ethics Board of Kitchener, and the French Ministry of Research.

Apparatus and Procedure

For voluntary orienting an 80% predictive central arrow cue was used. For reflexive orienting a non-informative abrupt-onset peripheral cue was used. In both tasks, target locations were indicated by green circles subtending 2° of visual angle, presented 12° left and right of fixation. Targets were red circles presented within the green circles. Response times were measured by button press of the right hand. Participants were seated 50cm from the monitor with their head in a chin rest. Stimuli were presented on Pentium IV computer with a 19 inch CRT monitor. Participants maintained fixation and while this was not monitored in controls, patients were monitored by an eye-tracker or by the experimenter. For voluntary orienting, after a stimulus onset asynchrony (i.e., time between cue and target onset; SOA) of 300ms or 500ms, targets appeared at the cued (valid) or uncued (invalid) location. For reflexive orienting we used SOAs of 50ms and 150ms [10]. We included no-cue trials (targets appeared without cues) in both tasks to examine response times for simple target detection.

Data Analysis

Mean response times were calculated for each trial type for each participant. For controls, response times less than 150ms or more than 2 standard deviations above the participant's overall mean were discarded. Response time data were analyzed separately for controls and patients using a three-way ANOVA with cue (valid, invalid), target (left, right), and SOA as within subject factors. Data from the patients was analyzed using the same procedure with response times removed if they were below 150ms or above 2000ms. Post hoc tests were carried out using Fisher's LSD tests with Bonferroni correction. In addition, to examine the cost for reorienting attention left vs. right we calculated cuing effects. For left shifts we subtracted

response times to valid right targets from response times to invalid left targets. For right shifts we subtracted response times to valid left targets from response times to invalid right targets [11]. To examine the difference between response times to targets in left and right space for each cue type we computed a symmetry ratio using the following formula [12]:

$$\text{Symmetry Ratio} = (\text{right field response time} - \text{left field response time} / \text{right field response time} + \text{left field response time})$$

Symmetry ratios determine the speed with which participants detect left versus right visual targets in each condition. Negative ratios indicate longer response times for left space, whereas positive ratios indicate longer response times for right space. The cuing effect and symmetry ratio data are presented graphically, but were not analyzed statistically as they concur with response time data. In all graphs C.F.'s data is plotted in reference to young controls whereas M.E.'s data is plotted in reference to elderly controls.

Results

Healthy Individuals: Voluntary Orienting

Mean response time, directional cuing effects, and symmetry ratios are shown in Figure 2.

--- insert Figure 2 about here ---

Young controls showed main effects of cue, target, and SOA (F 's >5 , all p 's $<.025$). Valid targets (300ms) were detected faster than invalid targets (331ms), left targets (313ms) were detected faster than right targets (318ms), and response times were faster at the 500ms SOA (312ms vs. 319 at the 300ms SOA). Non-cued trials were responded to slightly faster for left (382ms) vs. right (389ms) targets ($t(70)=1.9$, $p=.07$). Elderly controls showed main effects of cue, and SOA (F 's >9 , p 's $<.03$). Valid targets (377ms) were responded to faster than invalid targets (433ms) and targets at the 300ms SOA (395ms) were responded to faster than targets at the 500ms SOA (415ms). There was a significant Target x SOA interaction ($F(1,5)=9.5$, $p=.03$) such that left targets (430ms) were responded to more slowly than right targets (398ms) at the 300ms SOA ($t(6)=6.7$, $p<.001$). Elderly controls were also slower to respond to left (494ms) vs. right (464ms) non-cued targets ($t(5)=2.9$, $p=.03$).

Patients: Voluntary Orienting

C.F.'s analysis showed a significant Cue x Target interaction ($F(1,305)=4.3$, $p=.04$) such that he was slower to detect invalid targets on the left (384ms) vs. the right (308ms; $t(305)=16.9$, $p<.001$). C.F. was also slower to detect valid targets on the left (341ms) versus the right (297ms; $t(305)=9.6$, $p<.001$). Finally, C.F. was slower to detect non-cued targets on the left (428ms) vs. the right (373ms; $t(37)=3.0$, $p=.005$). M.E.'s analysis revealed a significant Cue x Target interaction ($F(1,343)=11.4$, $p<.001$) where she was slower for invalid targets on the left (777ms) vs. the right (565ms; $t(343)=16.9$, $p<.001$). M.E. was also slower to detect valid targets on the left (555ms) vs. right (495ms; $t(343)=4.8$, $p<.001$). Finally, M.E. was slower to detect non-cued left (733ms) versus right (575ms) targets ($t(33)=2.4$, $p=.023$).

Healthy Individuals: Reflexive Orienting

Mean response time, directional cuing effects, and symmetry ratios are shown in Figure 3.

--- insert Figure 3 about here ---

Younger controls showed a 3-way Cue x Target x SOA interaction ($F(1,59)=6.3, p=.015$). At the 150ms SOA there was no response time advantage for valid over invalid targets in left space. At the 50ms SOA the response time advantage for valid over invalid targets was equivalent for left and right targets (Fig 3). Younger controls were faster at detecting non-cued targets on the left (377ms) vs. the right (383ms; $t(59)=1.9, p=.07$). Elderly controls showed a main effect of Cue ($F(1,5)=50.1, p=.001$) indicating that valid targets (426ms) were responded to faster than invalid targets (485ms). Finally, elderly controls responded more slowly to left (485ms) vs. right (465ms) non-cued targets ($t(5)=3.02, p=.03$).

Patients: Reflexive Orienting

C.F. demonstrated a Cue x Target interaction ($F(1,310)=17.5, p<.001$) such that invalid targets were detected more slowly on the left (408ms) vs. the right (326ms; $t(318)=21.7, p<.001$). C.F. was also slower to detect valid targets on the left (351ms) vs. the right (314ms; $t(318)=9.8, p<.001$). Finally, C.F. was slower to detect non-cued targets on the left (415ms) vs. right (327ms; $t(38)=5.6, p<.001$). M.E. showed a Cue x Target interaction ($F(1,150)=13.9, p<.001$) where invalid targets were detected more slowly on the left (798ms) vs. the right (541ms; $t(158)=12.98, p<.001$). M.E. was also slower to detect valid targets on the left (538ms) vs. right (490ms;

$t(158)=2.5, p=.015$) and was slower to detect non-cued targets on the left (573ms) vs. the right (479ms; $t(33)=2.7, p=.01$).

Cuing effects for both patients were larger for left than right shifts of attention compared to the range of the controls (Figs 2b & 3b). In addition, symmetry ratios for both patients were negative in all conditions indicating slower responses for left targets (Figs 2c & 3c).

Discussion

Optic ataxia has traditionally been considered a deficit of visuomotor control independent of perceptual or attention deficits [1]. However, the present results clearly demonstrate that optic ataxia patients also have marked deficits in orienting attention. Specifically, both patients were much slower to detect any target in their left (ataxic) field, regardless of cue type. This resulted in negative symmetry ratios in all cue conditions that were outside the range for controls, indicative of a left spatial deficit. This deficit was most evident for invalid trials when attention had to be disengaged from ipsilesional (i.e. right) cues (Figs 2b & 3b). One explanation for the dramatic slowing of responses to all cue types in the ataxic field would be a decrease in salience for contralesional stimuli. That is, when a cue or target appears in the ataxic field, attention may not be captured to the same extent as it is for ipsilesional stimuli. A decrease in salience is consistent with what is known about the intraparietal sulcus. Neurons in the lateral intraparietal region of the monkey intraparietal sulcus show increased firing rates only to task-relevant stimuli, reflecting a role for this region in representing salience of contralateral stimuli [13].

Interestingly, similar to patients with spatial neglect [14], both patients in our study had difficulty disengaging attention from ipsilesional cues. Previous studies suggest that the disengage deficit results from lesions to temporal-parietal cortex and not the superior parietal

lobe [15]. However, the current results question this conclusion given that neither patient's lesion extends into the temporal-parietal junction. It is important to emphasize that although C.F. and M.E. have a deficit in reorienting attention similar to neglect patients, they also have marked deficits in *orienting* attention. That is, they are much slower to detect valid and non-cued targets in their ataxic (left) visual field. This effect is often not present in patients with neglect [5,6].

One question raised by the current study is how the attention impairments in optic ataxia seen here may relate to deficits in visuomotor control. Recent research suggests that several regions within superior parietal cortex and the intraparietal sulcus are important for attending, motor planning, pointing, reaching, and grasping within a particular location in space [7,8,16]. Given that regions in superior parietal cortex are important for both spatial attention and control of actions, lesions to this region could lead to peripheral attention deficits that may interact with deficits in visuomotor control that are characteristic of optic ataxia.

Conclusion

Optic ataxia patients have attention deficits in their ataxic field. Although the link between peripheral attention and action in optic ataxia is unclear, we suggest that future studies consider these attention deficits when examining visuomotor control in these patients [17].

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Figure Captions

Figure 1. Upper panel: T1 MRI of C.F.'s bilateral lesions to superior parietal lobule and intraparietal sulcus. Lower panel: CT scan of M.E.'s lesion of right intraparietal sulcus with minor extension into inferior parietal cortex. CS=central sulcus; IPS=intraparietal sulcus.

Figure 2. **A.** Mean response time for the young controls and C.F. (left) and elderly controls and M.E. (right). **B.** Cuing effects for left and right attention shifts for patients and their respective controls (mean \pm SD). **C.** Symmetry ratios for patients in relation to mean (\pm SD) of controls. Positive ratios = longer response times for right targets; negative ratios = longer response times for left targets.

Figure 3. **A.** Mean response time for the young controls and C.F. (left) and elderly controls and M.E. (right). **B.** Cuing effects for left and right attention shifts for patients and their respective controls (mean \pm SD). **C.** Symmetry ratios for patients in relation to mean (\pm SD) of controls. Positive ratios = longer response times for right targets; negative ratios = longer response times for left targets.

Figure1

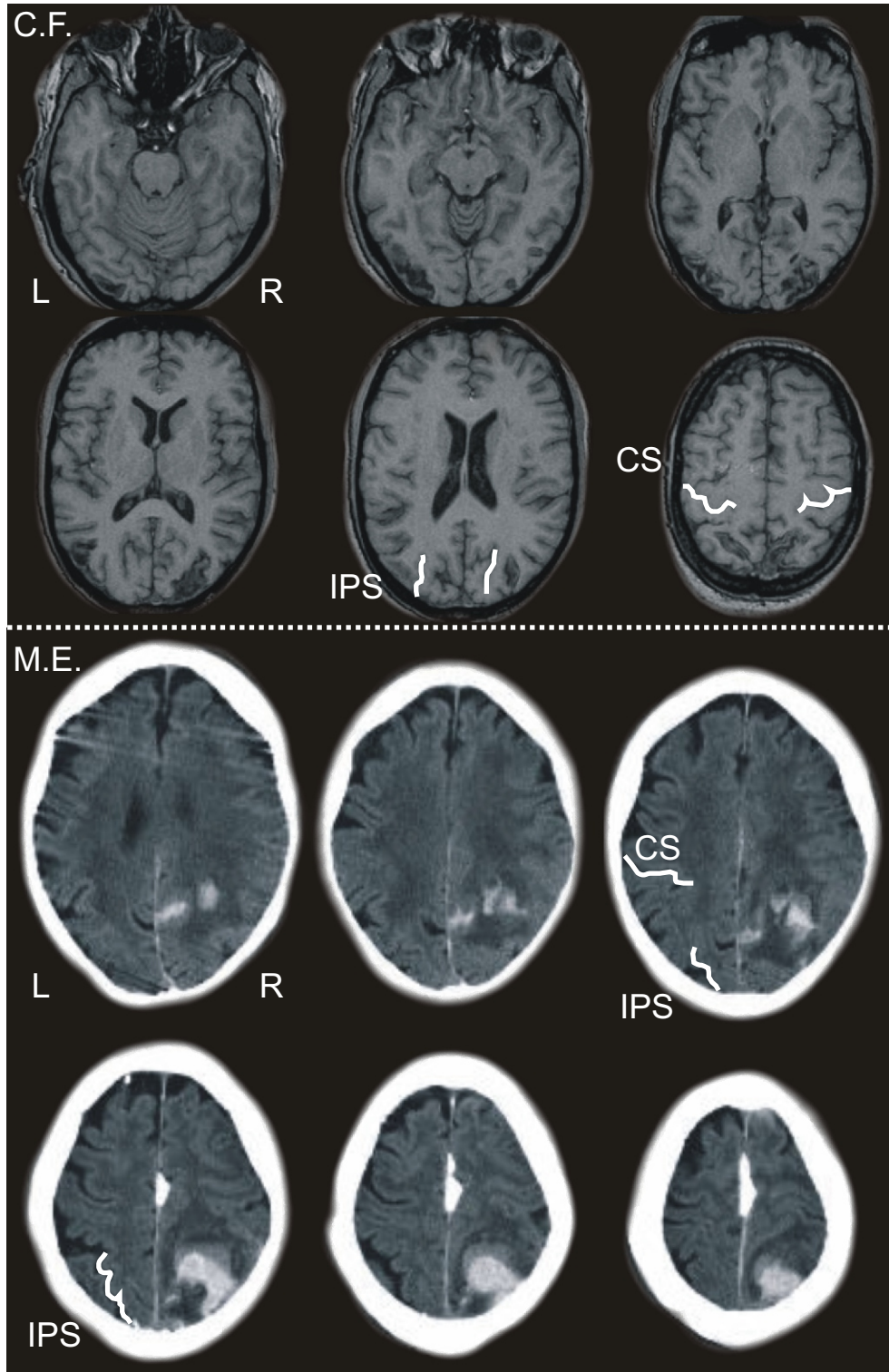


Figure 2

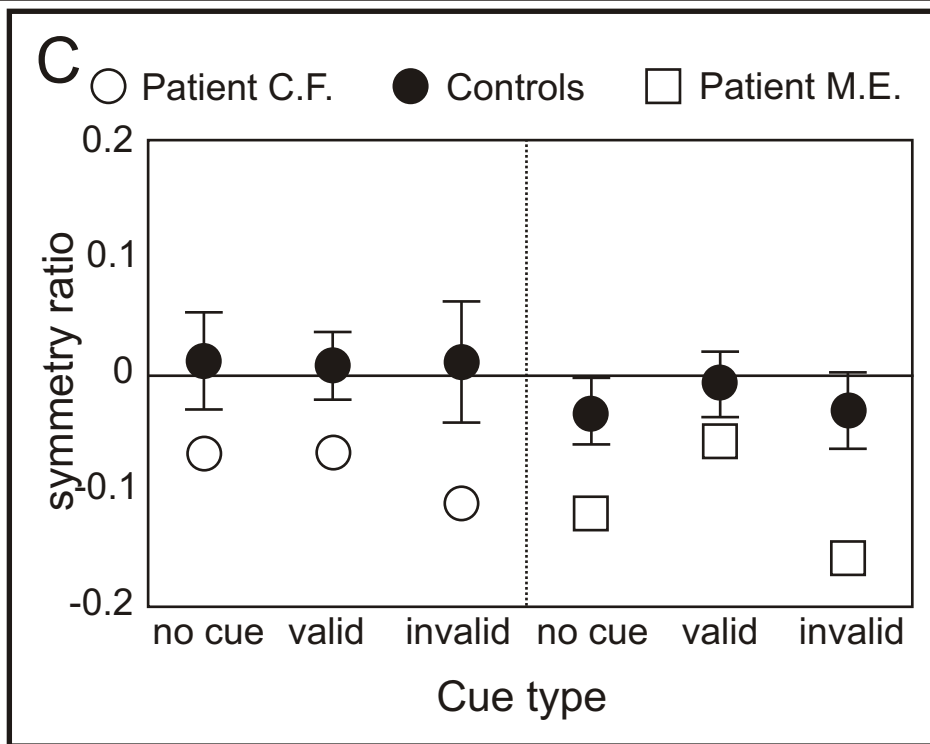
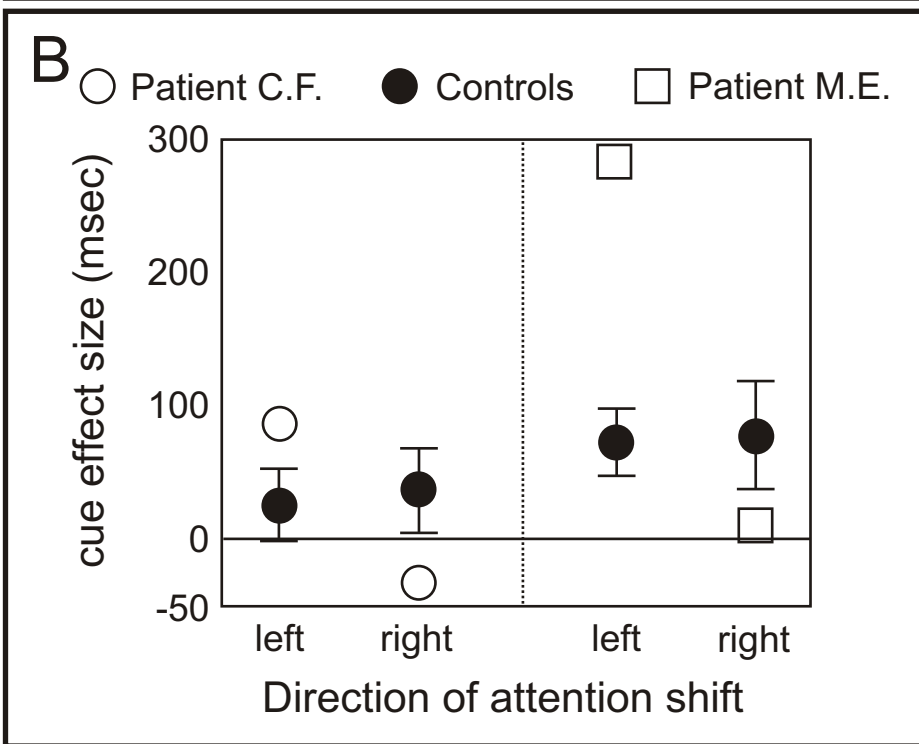
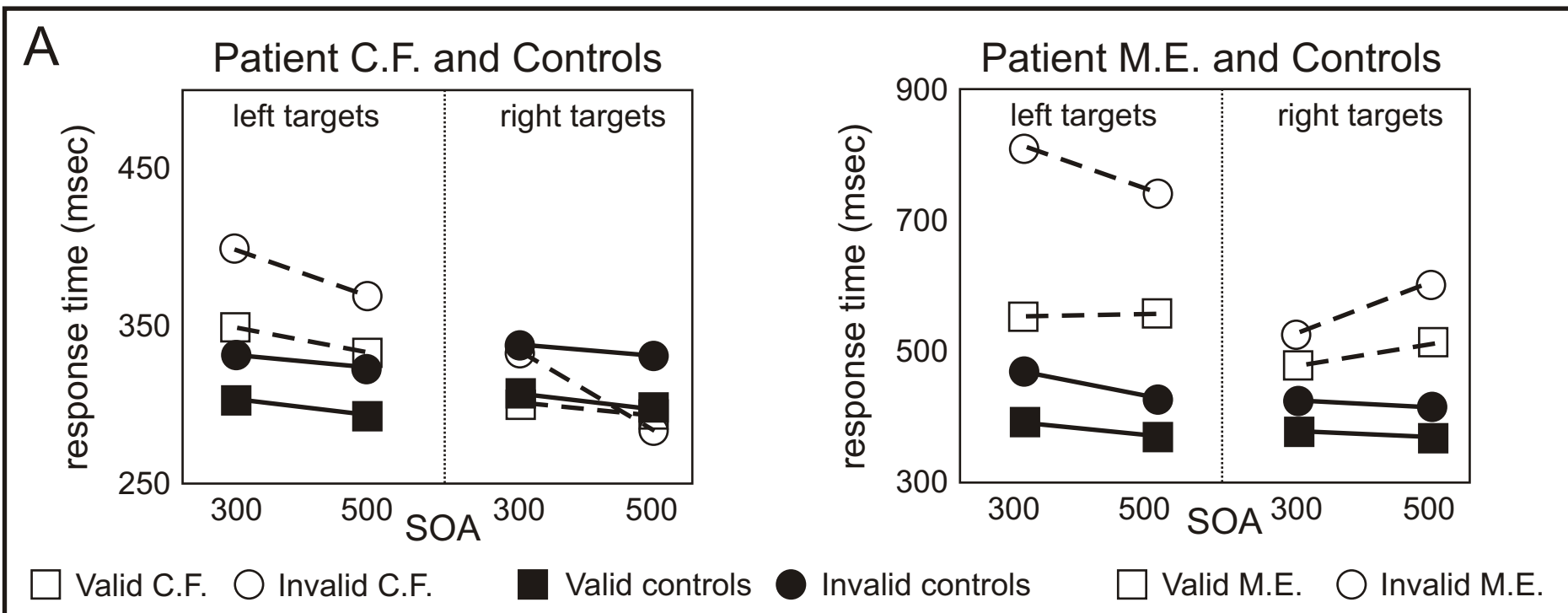


Figure 3

