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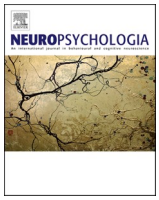

Gaze and attention: Mechanisms underlying the therapeutic effect of optokinetic stimulation in spatial neglect☆

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ABSTRACT

Left smooth pursuit eye movement training in response to large-field visual motion (optokinetic stimulation) has become a promising rehabilitation method in left spatial inattention or neglect. The mechanisms underlying the therapeutic effect, however, remain unknown. During optokinetic stimulation, there is an error in visual localisation ahead of the line of sight. This could indicate a change in the brain's estimate of one's own direction of gaze. We hypothesized that optokinetic stimulation changes the brain's estimate of gaze. Because this estimate is critical for coding the locus of attention in the visual space relative to the body and across sensory modalities, its change might underlie the change in spatial attention. Here, we report that in healthy participants optokinetic stimulation causes not only a directional bias in the proprioceptive signal from the extraocular muscles, but also a corresponding shift of the locus of attention. Both changes outlasted the period of stimulation. This result forms a step in investigating a causal link between the adaptation in the sensorimotor gaze signals and the recovery in spatial neglect.

1. Introduction

Spatial neglect is an attention disorder caused by unilateral brain lesions. Up to one-third of acute stroke patients lack awareness of objects in the contralesional side of space despite adequate sensory abilities (Hammerbeck et al., 2019). Although some individuals appear to recover spontaneously, residual, subtle deficits in spatial attention typically persist in the long-term (Bonato, 2015). The presence of spatial neglect significantly impacts the independence (Hammerbeck et al., 2019; Tarvonen-Schröder et al., 2020) and the quality-of-life post-stroke (Buxbaum et al., 2004). Despite intensive efforts to find an effective treatment, meta-analyses to date have shown little evidence of success (Bowen et al., 2013; Longley et al., 2021).

A promising intervention highlighted by recent systematic reviews is left smooth pursuit eye movement training in response to large-field visual movement (optokinetic stimulation) (Hill et al., 2015; Liu et al., 2019). During optokinetic stimulation the patients look at dots that move on a computer screen towards the neglected visual hemifield (Pizzamiglio et al., 1990a; Vallar et al., 1993, 1995; Pizzamiglio et al., 2004; Kerkhoff et al., 2006, 2012a, 2014a). In individuals with normal vision large-field visual motion of various stimulus velocities and stimulus field sizes elicits eye movements with an initial, slow phase that resembles smooth pursuit in the direction of motion followed by a second, fast phase that resembles a saccade in opposite direction (van Hof-van Duin and Mohn, 1986). When applied for 1 h daily over the course of five days, optokinetic stimulation alleviates symptoms of spatial neglect as measured by neuropsychological tests such as target cancellation, line bisection tasks, and reading (Kerkhoff et al., 2013). The therapeutic effect of this intervention crosses sensory modalities, improving both tactile (Kerkhoff et al., 2014) and auditory (Kerkhoff et al., 2012a) neglect. The benefit of optokinetic stimulation in patients with spatial neglect lasts up to 8 weeks after treatment (Hill et al., 2015). The mechanisms that underlie the therapeutic effect of optokinetic stimulation in spatial neglect remain unclear.

During voluntary smooth pursuit eye movement of one single target healthy individuals systematically mislocate the target in the direction of movement (Mitrani et al., 1979; Rotman et al., 2004) and orient attention ahead of the line of sight (Khan et al., 2010). Likewise,

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watching wide-field drifting gratings (optokinetic stimulation) elicits smooth pursuit eye movement, during which briefly flashed visual targets are mislocated in the direction of the drift (Kaminiarz et al., 2007; Tozzi et al., 2007). The current interpretation is that attention is allocated predictively to the eye movement target (Khan et al., 2010; Van Donkelaar and Drew, 2002)). However, because the predictive allocation of attention to the eye movement target can only be measured immediately before the onset of an eye movement (Zirnsak et al., 2014), this phenomenon cannot explain the rehabilitative effect of optokinetic stimulation in spatial neglect.

Kerkhoff and colleagues have advanced the hypothesis that optokinetic stimulation might alter a modality-independent, attentional priority map (Kerkhoff et al., 2013).

Organisms receive a wealth of sensory inputs that far exceed their neural processing resources. To deal with this bottleneck, the most important stimuli are given preferential access. This process is thought to be implemented by the attentional priority map. For each location, this neural representation codes the importance of an object that can appear there (Fecteau and Munoz, 2006; Gottlieb et al., 1998; Koch and Ullman, 1985; Mirpour and Bisley, 2012; Ptak and Fellrath, 2013). To attend to an object, one needs to know its location. All sensory modalities provide location cues. However, their coordinate systems differ, e.g. the location of visual objects is coded relative to the centre of the retina while that of sounds is relative to the head midline. Therefore, to obtain a single estimate of location, the brain uses knowledge about the relative positions of one's sensory organs, to transform each senses' coordinate system. The rotation of the eyes in the head and that of the head on the trunk provide an estimate of the direction of one's own gaze. This estimate is thus a critical input of the attentional priority map. At neural level, there is evidence for several mechanisms for integrating gaze information into the attentional priority map. Gain-field neurons in the posterior parietal cortex, for instance, are thought to be involved in coding the locus of attention because they respond to visual stimuli only when they are behaviourally relevant. These neurons receptive field is retinotopic and their response scales with the direction of gaze (Andersen and Mountcastle, 1983). Here we hypothesized that the estimate of own gaze direction is changed by optokinetic stimulation. Importantly, a shift in perceived gaze direction in the direction of the optokinetic stimulation would also explain the visual mislocalisation in the direction of the large field visual motion observed by (Kaminiarz et al., 2007; Tozzi et al., 2007) (Fig. 1).

Because gaze direction is a component of the attentional priority map, an error in gaze direction would alter the allocation of attention. We have observed that all interventions tested so far that alter extraocular muscle proprioception displace the locus of attention. First, inhibitory repetitive transcranial magnetic stimulation (rTMS) over an eye proprioceptive representation in the human postcentral gyrus (Balslev et al., 2011a)causes visual localisation errors that are indicative of an underestimation of the angle of gaze (Balslev and Miall, 2008; Odoj and Balslev, 2013). Importantly, the same intervention also shifts the locus of attention in the same direction as the shift in perceived gaze (Balslev et al., 2011b; Odoj and Balslev, 2013, 2016). Secondly, passive rotation of the non-viewing, dominant eye in healthy individuals using a scleral lens, which is an intervention that stretches the extraocular muscles, stimulating the eye proprioceptors in the absence of a corresponding oculomotor command (Ilg et al., 1989; Gauthier et al., 1990; Knox et al., 2000), changes not only the perceived angle of gaze towards the direction of the passive rotation, but also increases the detection of visual targets that appear in the same vs. opposite direction from fixation, at equal retinal eccentricity (Balslev et al., 2012b). Finally, a focal lesion of the eye proprioceptive area in the somatosensory cortex causes an error in visual localisation that can be observed after an eye-press (Balslev et al., 2012a), as well as a change the allocation of attention (Balslev et al., 2013).

The aim of this study was to test whether optokinetic stimulation causes a shift in the estimate of gaze in the direction of the optokinetic stimulation that outlasts the duration of the intervention, and whether this change is associated with a corresponding displacement in the locus of attention.

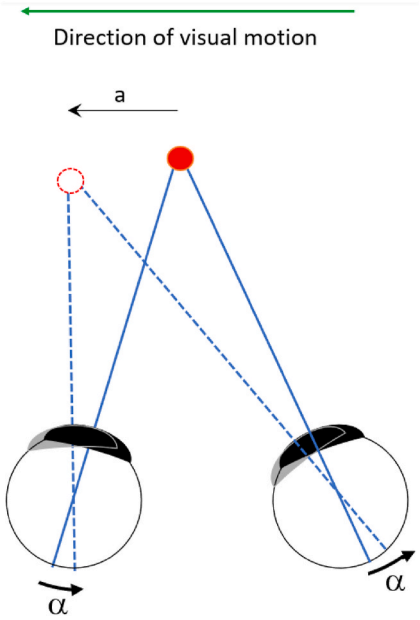

Fig. 1. The hypothesis. Visual mislocalisation in the direction of visual motion ('a') observed during smooth pursuit eye movement of a single target or during optokinetic stimulation could indicate a shift in the perceived gaze in the same direction ('α'). Filled circle – actual location of the visual target relative to the head and body, when the visual target is foveated. Empty circle – perceived location of the visual target relative to the head and body, for the same retinotopic location. Solid line– actual direction of gaze. Dashed line – perceived direction of gaze. Black arrows indicate the direction of the mislocalisations.

2. Material and methods

2.1. Participants

Healthy, right-handed adults participated after giving written informed consent. Ethical approval was obtained from the local ethics committee at the University of St. Andrews (PS11859). Thirty-eight participants (24 women, 13 men, 1 non-binary; median age 21; age range 18–34) were recruited for Experiment 1 eighteen participants (14 women, 4 men; median age 21; range 18–29) were recruited for Experiment 2. Handedness was assessed by self-report. The participants were asked which hand they preferred to use for skilled activities like writing. This was deemed appropriate because a single-item assessment of handedness shows a good classification concordance with more extended inventories (Coren, 1993). Vision was examined using a Snellen chart. All participants had normal or corrected-to-normal vision (20/20). For Experiment 1, motor (sighting) eye dominance was assessed using the hole-in-the-card test (Mapp et al., 2003). Twenty-one participants were right eye dominant, while the remaining were left eye dominant. Sample size was calculated a priori using G*power (Faul et al., 2007). Effect size was estimated from our previous studies which used the same tasks to examine the effect of an intervention that specifically targets the eye proprioceptive signal in healthy participants (repetitive transcranial magnetic stimulation over an eye proprioceptive area in the somatosensory cortex) (Balslev and Miall, 2008; Odoj and Balslev, 2016). In those studies the effect size was dz = 0.84 (large) for the visual localisation task (Experiment 1) and dz =0.64 (medium-large) for the cross-modal attention task (Experiment 2). To detect effects of this size with a one-tailed, paired-samples *t*-test with alpha = 0.05, power = 0.80, samples of eleven and seventeen participants,

respectively, are needed. Because the estimate of the effect size was based on a different intervention, we used these figures as indicators of the minimum sample size and included as many participants as practical.

2.2. Study design

This study assessed the aftereffect of a short session of optokinetic stimulation on the eye proprioceptive signal of gaze direction (Experiment 1) as well as on the allocation of attention during a cross modal attention task (Experiment 2). Each participant completed two optokinetic stimulation sessions on separate days, at least one day apart. The sessions differed by the direction of the visual motion (left or right). The order of presentation (left optokinetic stimulation first or right optokinetic stimulation first) was counter-balanced across participants.

The advantage of this crossover design is that each participant acts as their own control, and for this reason, it requires a smaller sample size than a parallel design. The disadvantage, however, is that the effects may carry over, and if so, the difference between the two interventions (left or right optokinetic stimulation) would be smaller. To control for carry over effects, the dependent variables were measured both before and after each optokinetic stimulation session. This allowed us to establish a session-specific, individual baseline.

2.3. Optokinetic stimulation

Optokinetic stimulation was conducted in dim light. The participants had both their eyes open. Stimuli were presented on computer monitor placed 45 cm in front of the participant and subtending 45° visual angle. The participant's head was fixed in a chin rest. The centre of the screen was aligned with the participant's midsagittal plane. Visual stimuli were adapted from (Kerkhoff et al., 2014). Small light-grey squares randomly scattered on the screen moved slowly in the same direction and at the same speed on a black background. Their quantity (50–70), size (0.05–2°) and velocity (3.1–12.6°/s) changed randomly every 30 s. An optokinetic stimulation session lasted 30 min with a short break after the first 15-min-block. Participants were instructed to follow with their eyes the direction of visual motion (Ilg, 1997). To encourage participants to look at the dots, a coloured square (red or blue) was presented at a random location every 30 s (pseudorandomised). After up to 10 presentations (pseudorandomised), the question "Was the coloured dot red or blue?" was presented on the screen. Participants responded by pressing a blue or red coloured key on a keyboard. The accuracy of responses was recorded. Optokinetic stimulation blocks where the average accuracy of the responses was below 70% were repeated. To prevent boredom, the participants could listen to music during the task with headphones placed in/over both ears.

2.4. Experiment 1: The aftereffect of optokinetic stimulation on the eye proprioceptive signal

2.4.1. Experiment 1: Rationale

Participants aligned an LED with the location of their own finger in complete darkness. This task requires information about the rotation of one's eyes in the orbits. Several sources provide this information: the feedback from proprioceptive receptors in the extraocular muscles (Balslev and Miall, 2008; Gauthier et al., 1990; Han and Lennerstrand, 1999; Knox and Whalley, 1997; Skavenski, 1972; Velay et al., 1994; Wang et al., 2007), the corollary discharge, which is a copy of the oculomotor command (Sommer and Wurtz, 2002) and visual cues, such as coherent visual motion (Poletti et al., 2010). To remove visual cues that could convey the direction of gaze, the experiment was conducted in complete darkness.

To examine eye proprioception, we used the eye-press method (Bridgeman and Delgado, 1984; Ilg et al., 1989; Stark and Bridgeman, 1983). The participants placed their own index finger on the closed eyelid, at the outer canthus. When instructed they pressed briefly (<1 s) on the eyeball towards the nose. The push causes passive deviation of the eye in the absence of a corresponding oculomotor command (Balslev et al., 2022; Ilg et al., 1989). The eye moves in the direction of the push, then back, presumably due to the elasticity of the orbital tissue. The amplitude of this movement measured in a separate experiment was 11.7°±3.5° (mean ± standard deviation) during the eye press and 11.7° ±3.25° during rebound (Balslev et al., 2022). Participants reported no discomfort during the eye press in this or in previous studies (Balslev et al., 2012a, 2022; Balslev and Miall, 2008).

Because the eye is moved passively, corollary discharge does not convey the rotation of the eye in the orbit, so eye proprioception is the only source that can provide this information accurately. When eye proprioception is accurate, the eye press (compared with the condition with no eye press) does not increase the mean or the standard deviation of the estimate of eye position, measured using the visual localisation error in complete darkness (Balslev et al., 2012a). However, when eye proprioception is inaccurate, e.g. after a focal lesion of the primary somatosensory cortex, this intervention causes a visual localisation error (Balslev et al., 2012a). Based on this previous finding, an error in locating a visual target relative to the body in complete darkness in the presence vs. the absence of an eye press provides a measure of the error in eye proprioception. Our prediction was that optokinetic stimulation would cause an error in visual localisation opposite the visual motion (i.e. the visual target will appear further in the direction of the visual motion, Fig. 1) and that this error would be larger in the presence (vs. the absence) of an eye press, to reflect an error in eye proprioception.

2.4.2. Experiment 1: Visual localisation task

The task was similar to a visual localisation task used previously to assess the effect on the eye proprioceptive signal after transcranial magnetic stimulation (Balslev and Miall, 2008) or a focal lesion of the extraocular muscles' representation in the human primary somatosensory cortex (Balslev et al., 2012a).

The participants were seated with their head fixed in a chinrest. A lit LED was presented using a custom-made 100 cm long horizontal array placed at 168 cm in front of the participant, aligned with their body midline The array comprised of 96 red diodes spaced 1 cm (0.34° visual angle) apart. LEDs were switched on and off by the experimenter who pressed keys in response to the participant's verbal instructions. The LED array was controlled via Psychophysics Toolbox v. 3 (Brainard, 1997) using a computer interface (USBDIO96H, Measurement Computing).

The non-dominant eye was covered with a patch and the array was viewed through the dominant eye only. On some trials an eye press was applied to the viewing eye. The estimate of eye rotation typically relies on the eye proprioceptive input from both eyes during monocular vision (Balslev and Miall, 2008; Velay et al., 1994).An alteration of the eye proprioceptive input from the dominant (compared with the non-dominant) eye causes the largest visual localisation error (Velay et al., 1994).

The task was to align the red lit LED with a body landmark in complete darkness. This landmark was their index finger, placed by the researcher on one of three small, Velcro patches (1 cm × 1 cm) at 20 cm in front of the participant. One patch aligned to the participant's midsagittal plane and the other two were placed 15 cm to the left and right of it. The index finger on the side of the non-dominant eye was used. For instance, a participant who was right-eye dominant would place the left index finger on the Velcro patches and use the right index finger to press on the dominant eye.

Each trial started with the presentation of a lit LED. The lit LED could appear at one of six locations in a pseudorandomised order (−6.05, −4.94, −3.91, 2.49, 4.27, 5.34° from body midsagittal plane). The participants instructed the experimenter to move the LED using verbal commands ('left', 'right' and 'stop') to be directly in line with their hidden finger location. The participants were told 'Imagine that your

finger is long enough to touch the array. Let us know when the LED touches the tip of your finger'. Once the participant decided on which direction the LED needed to be moved, they said 'Left' or 'Right' and the experimenter pressed keys to move the LED in that direction until the participant said 'Stop'. They were allowed to correct the LED position until they felt it was directly in front of their finger. The position of the LED was recorded, then a new trial started.

The participants were asked to complete the visual localisation task under two different conditions, with and without an eye press. During the eye press (*push*) condition the participants placed the index finger on the eyelid, at the outer corner of their closed, dominant eye. At the beginning of each trial a pre-recorded audio ('push') prompted the participants to passively displace the eyeball towards the nose by pressing briefly (<1 s) on their eyelid. Then an LED on the array was lit, and the participant was instructed to open their unpatched (dominant) eye to carry out the visual localisation task. Before the experiment, participants practiced pressing their dominant eye gently while viewing an object through both eyes and increased the force gradually until they experienced double vision. An eye press of a strength that produces double vision during normal binocular viewing was assumed to be sufficient to passively displace the eyeball in complete darkness. The reason why the displacement is much smaller in normal light conditions than in darkness is thought to be the retinal slip, which triggers an extraocular muscle contraction that opposes the push (Ilg et al., 1989). The experimenters verified that the pushed eye moved, while the participants tried the task in normal light conditions, with both eyes open, prior to data collection.

The timing of the LED presentation relative to the eye press was the same as in our previous experiments, which identified errors in visual localisation after transcranial magnetic stimulation (Balslev and Miall, 2008) or a focal brain lesion (Balslev et al., 2012a) of an eye proprioceptive area in the human somatosensory cortex.

Each participant completed six trials of each condition, twice for each finger location. The condition without an eye press (*no-push*) was always presented first for all participants and sessions (before and after optokinetic stimulation). The order of the blocks for each condition (finger location center, left, right) was the same across participants.

2.4.3. Experiment 1: Visual localisation task: Data analysis

Visual localisation error for each trial was calculated as the signed difference between the location of the LED when the participant perceived it as being straight in front of their finger minus the location of the finger on a left-right axis (negative values indicated that the LED was mislocated to the left of the finger). After averaging across trials of the same condition, this error was compared across conditions using a repeated measures 2 × 2 × 2 ANOVA with factors i) optokinetic stimulation direction (left vs. right) ii) session (pre vs. post optokinetic stimulation) and iii) eye push (present vs. absent). If optokinetic stimulation affects the estimate of eye-rotation (proprioception, corollary discharge, or both), then an interaction between optokinetic stimulation direction and session would be expected. If optokinetic stimulation affects eye proprioception, then furthermore, a three-way interaction of optokinetic stimulation direction, session and eye-press would be expected, driven by a directional increase in localisation error after the eye-press.

2.5. Experiment 2. The aftereffect of optokinetic stimulation on the allocation of attention in the visual space

2.5.1. Experiment 2: Rationale

In line with the attentional priority map hypothesis, coding the locus of attention in the body-centered space requires an estimate of the rotation of the eyes in the orbits (Andersen and Mountcastle, 1983; Pouget and Driver, 2000). Experiment 2 therefore asked whether optokinetic stimulation displaces the locus of attention in the visual space relative to a somatosensory (hand proprioceptive) cue, the location of one's own index finger in complete darkness. Participants discriminated a visual target (a letter). They were told the letter would appear at the location of their left index finger hidden from view. In addition to this instruction to voluntarily orient attention at the location of the hand, hand location (visible or hidden) is known to involuntarily affect the allocation of attention in space. Stimuli near the hand are prioritised over those located further away. Visual and/or proprioceptive signals of hand location are responsible for this effect (Reed et al., 2006).

To probe the allocation of attention in space, unbeknown to the participants, targets were presented not only at that location, but also at 1°, 2° and 3° from it, to the left or to the right (seven locations in total) with equal probability. Additionally, some trials showed target letters at random locations outside this range so that the participants could not predict the location of the non-visual cue from the spatial distribution of the visual targets. To assess the benefit of the location cue, we calculated the difference in reaction time for visual discrimination in its presence vs. the absence. The locus of attention was defined as the finger location corresponding to the largest benefit of the cue. 'Cueing error' was calculated as the difference between the locus of attention and the actual location of the cue.

If gaze contributes to coding the locus of attention, optokinetic stimulation should increase cueing error by shifting the locus of attention in the same direction as the shift in perceived eye position measured by Experiment 1. This hypothesis was based on the findings of our previous experiments, in which we assessed the effect on visual attention of an error in the eye proprioception signal. Those previous experiments examined the allocation of attention after passive extraocular muscle stretch (Balslev et al., 2012b), a lesion of the proprioceptive projection of the extraocular muscles in the human primary somatosensory cortex in a patient with a focal stroke (Balslev et al., 2013) or transcranial magnetic stimulation an eye proprioceptive area in the somatosensory cortex (Odoj and Balslev, 2016).

We have previously observed that a change in eye proprioception has a larger effect on the attentional priority map than it has on other representations of visual location, for instance that used to reach to a visual target in the absence of visual feedback about the location of one's own hand (Odoj and Balslev, 2016). Therefore, to find out whether optokinetic stimulation predominantly affects the attentional priority map, Experiment 2 included a control task (Pointing task) in which the participants pointed to visual targets using their unseen hand. If optokinetic stimulation predominantly affects attention, then the cueing error after optokinetic stimulation should be larger than the pointing error. The tasks used in Experiment 2 were the same as those used by (Odoj and Balslev, 2016).

2.5.2. Experiment 2: Cross-modal spatial attention task

The experiment took place in complete darkness. Participants sat with their head fixed in a chin rest and cheek pads. They had both their eyes open. An OLED (no backlight) display was placed 45 cm in front of them with the center of the screen aligned to their body's midsagittal plane. The display was placed on a stand. The participant wore a black glove on their left hand. Their left index finger rested on the stand just under the base of the screen at 7° left and right of the screen centre. The participants were told to expect the visual target to appear at the location of their left index finger.

At the beginning of each trial, participants fixated on a central cross (white, 1° × 1°) presented on black background near the bottom of the screen (2° from the bottom). Fixation was verified with a head-mounted eye tracker (EyeLinkII). All trials where the fixation deviated by more than 1.5° in the 200 ms before visual target presentation were discarded from further analysis. After 500–650 ms (randomized) the fixation cross disappeared. 100 ms later, a target letter ('A' or 'H', 1° visual angle) appeared for 100 ms. The target appeared at one out of seven possible locations, at −3°, −2°, −1°, 0°, 1°, 2°, 3° from the location of the finger. Actual target locations for trials with the index finger at −7° were −10°,

−9°, −8°, −7°, −6°, −5°, and −4° and for the trials with the finger at 7° they were 4°, 5°, 6°, 7°, 8°, 9°, 10°. Targets were presented at the same offset with equal probability (11 times). In addition, four random targets were presented outside this range. Participants were instructed to respond as quickly and as accurately as they could using their right hand. They pressed one of two keys to indicate which letter was presented. Reaction time and accuracy of the response were recorded.

Trials with the same finger location were grouped in blocks (left-cue, right-cue, and no-cue). Their order was pseudo-randomised. To assess the baseline distribution of attention as well as visual accuracy, participants performed the same visual discrimination task with their left index finger placed near their body (at 45 cm away from the screen). During this no-cue block the targets appeared randomly, six times at each target location (14 locations in total). Trials within each block differed by the location of the target. Their order was pseudo-randomised. At the end of each block, participants were instructed to close their eyes. The experimenter then moved the participants' index finger at the next cue location and started a new block.

2.5.3. Experiment 2: Cross-modal spatial attention task: Data analysis

Healthy participants typically perform this task at ceiling (>95% accuracy) (Odoj and Balslev, 2016). The study therefore focused on the change in reaction time for visual discrimination at the same retinal eccentricity to examine any changes in the allocation of attention. Only trials where the participant's response was accurate were included in the analysis. The locus of attention was the center of mass (COM) of all visual locations that showed a benefit of that cue (the largest decrease in RT for discriminating the visual target in the presence vs. the absence of the finger cue for the expected location of the visual target).

The method for calculating the center of mass is also described in (Balslev and Odoj, 2019; Odoj and Balslev, 2016). Two separate centers of mass were computed, corresponding to the left or the right location cue. We first calculated for each visual target location (L) the average reaction time (RT) with a cue ($meanRTcue_L$) and the average RT without a cue ($meanRTnocue_L$) across all trials that presented the target at that location. A benefit of the cue was defined as a negative difference ($meanRTcue_L$-$meanRTnocue_L$). Then we calculated the centre of mass (equation (1)) by taking the mean of all (n) target locations where the participant showed a benefit of the cue ($L < 0$) weighted by the size of that benefit.

$$COM = \left(\sum \left((L < 0) * (meanRTcue_L - \ meanRTno\ cue_L) \right) \right) \Big/ \left(n(L < 0) \right) \quad (1)$$

The advantage of this method (compared with a more principled approach such as fitting a gaussian curve) is that it can be used with a relatively small number of data points. For each target location the participants completed 6 trials for the condition without a location cue and 11 trails for the condition with a location cue. Experiment 2 lasted ~2h. Estimating a Gaussian with 95% accuracy requires ~20 trials/location (Psutka and Psutka, 2015). Because the center of mass was calculated in the same way in all conditions (before or after, left or right optokinetic stimulation), a difference in this variable across conditions cannot be attributed to the method of calculating the center of mass.

Cueing error was calculated as the signed distance between the center of mass and the location of the finger. A positive cueing error denotes a center of mass (i.e. a locus of attention) to the right of the location cue. After averaging across the two cue locations, this error was compared across conditions. Using a repeated measures 2 × 2 ANOVA with factors i) optokinetic stimulation direction (left vs. right) and ii) session (pre vs. post optokinetic stimulation). If optokinetic stimulation affects the allocation of attention in space as one would predict based on the change in the gaze direction signal (Experiment 1) and the attentional priority map hypothesis, then an interaction between optokinetic stimulation direction and session would be expected.

If optokinetic stimulation causes an attention shift in the same direction as the direction of visual motion, then one should see a positive cueing error after right optokinetic stimulation and a negative cueing error after left optokinetic stimulation.

2.5.4. Experiment 2: Pointing task: pointing to a visual target in the absence of visual hand feedback

This task too was conducted in complete darkness and the participant wore a black glove on their left hand. A Polhemus Fastrak sensor was taped at the tip of their left index finger. The same experimental set-up was used, as for the cross-modal attention task. The participant's left index finger was placed on the stand just under the base of the screen. At the beginning of each trial, a cross appeared at the centre of the screen (2° from the bottom), and after 500–650ms (randomised) the cross disappeared and a target (letter 'X', 1 × 1°) was presented for 100ms. The target could appear at two possible locations, 7° left or right of the centre. Participants were asked to move their left index finger along the stand, until they felt the finger was just under the visual target and to say "There" to confirm location. The researcher pressed a key to record finger position via the motion sensor attached to the participant's fingertip. A beep then prompted the participant to move their finger back to a location they felt was roughly in front of their body midsagittal plane. This location was not pre-defined, to encourage the participants to match the position of their finger with the location of the target (rather than move the finger a pre-defined distance). At the end of data collection, a short calibration was run. The participant closed their eyes and the researcher turned on the light and placed the participant's finger at the exact target location. A recording was made twice at each target location. This was used to calculate the participant's pointing error (finger location at the end of the pointing movement minus actual target location averaged over calibration trials). Trials for each target location were grouped into blocks. There were 10 trials per block. The order of pointing and cross-modal attention tasks was counterbalanced across participants.

2.5.5. Experiment 2: Pointing task: Data analysis

Pointing error was averaged across the two target locations. We conducted a 2 × 2 × 2 ANOVA with factors i) task (cross-modal attention vs. pointing), ii) optokinetic stimulation direction (left vs. right) and iii) session (before vs. after optokinetic stimulation). If optokinetic stimulation affects the attentional priority map specifically, then a statistically significant 3-way interaction would be expected.

2.6. Data and code availability

The anonymised data and code underpinning this research can be accessed at https://doi.org/10.17630/7ec04723-283f-4725-bccd-3b5e79f98807 (Balslev, 2023)

3. Results

3.1. Experiment 1: Accuracy

Visual localisation error was calculated on a horizontal, left-right, axis as the signed difference between the location of the LED when the LED was felt to be straight in front of the unseen finger minus the location of the finger (visual minus somatosensory location). Optokinetic stimulation caused a visual localisation error in the opposite direction of the visual motion (e.g., after left optokinetic stimulation training, an LED that was ~1° further to the right of the finger was perceived as being straight in front of the finger, Fig. 2). Repeated measures 2 × 2 × 2 ANOVA showed a statistically significant 2-way interaction between the factors i) optokinetic stimulation direction (left vs. right) and ii) session (pre vs. post optokinetic stimulation) with $F(1, 37) = 12.5$, $p = 0.001$. Furthermore, this difference was larger in the push condition than in the no-push condition. The three-way interaction with the third factor iii) eye push (push and no-push) was significant F

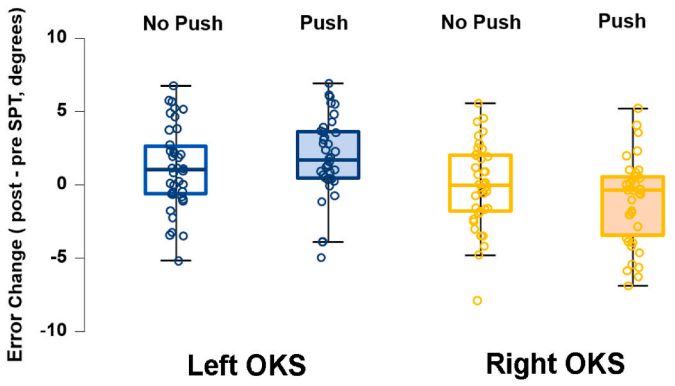

Fig. 2. Optokinetic stimulation (OKS) caused a visual localisation error in opposite direction of the visual motion. This change was larger after an eye press ('push') compared to the condition without an eye press ('no push'). Visual localisation error was calculated on a horizontal, left-right axis as the signed difference between the location of the LED when the LED was perceived to be straight in front of the unseen index finger minus the location of the finger. Negative values indicate that the location of the LED was to the left of the location of the finger. Box and whiskers plot showing median, interquartile range (box), range (whiskers), individual data (circles) and outliers.

(1, 37) = 5.53, p = 0.024. Because passive eye movement changes the proprioceptive input from the extraocular eye muscles independent of an oculomotor command and increases the weight of proprioception in the estimate of eye position (Balslev et al., 2012a), this three way interaction is a signature of a change in the eye proprioceptive input.

Planned post-hoc comparisons (paired-samples t-tests) showed a statistically significant change in the visual localisation error in the opposite direction of the visual motion from pre-to post-optokinetic stimulation in three out of the four conditions: left optokinetic stimulation/no eye press (p = 0.021), left optokinetic stimulation/eye press (p < 0.001) and right optokinetic stimulation/eye press (p < 0.025).

Before optokinetic stimulation, visual localisation error showed no statistically significant main effects or interaction between conditions (2 × 2 repeated measures ANOVA, all p > 0.15). The error before optokinetic stimulation across conditions was −1.3° ± 3.77° (mean ± standard deviation).

3.2. Experiment 2. The aftereffect of optokinetic stimulation on the allocation of attention in the visual space

3.2.1. Cross modal attention: Cueing error

Optokinetic stimulation shifted the locus of attention in the visual space relative to the location of the cue (the unseen index finger) in the direction of the visual motion. For instance, after left optokinetic stimulation training, the locus of attention was to the left of the location of the finger (Fig. 3). Cueing error was calculated in the same way as the visual localisation error for Experiment 1, by subtracting visual (center of mass) minus somatosensory (finger) location. Noteworthy, the misalignment of the visual and the somatosensory spaces for cross modal attention was in the opposite direction with that observed in Experiment 1. This suggests that the effect on the attentional priority map cannot be explained by a more general mislocalisation of the visual target relative to the hand.

For cueing error, repeated measures 2 × 2 ANOVA showed a statistically significant interaction between the factors i) optokinetic stimulation direction (left vs. right) and ii) session (pre vs. post optokinetic stimulation) with F(1, 17) = 5.05, p = 0.038. None of the main effects were significant. Planned post-hoc comparisons (paired-samples t-tests) showed a statistically significant shift in the locus of attention leftwards after left optokinetic stimulation (p = 0.03). The shift after right optokinetic stimulation did not reach statistical significance (p = 0.21).

Before optokinetic stimulation cueing error showed no significant

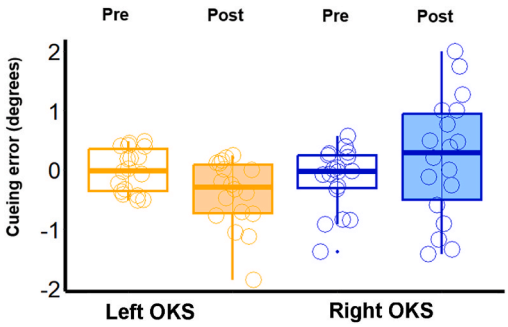

Fig. 3. Optokinetic stimulation (OKS) caused a shift of attention in the direction of visual motion. Cueing error was calculated as the signed difference between the locus of attention in the visual space and the location of the finger. Negative values indicate locations to the left of the finger. Conventions like in Fig. 2.

difference between conditions (paired-samples *t*-test, p > 0.4).

3.2.2. Cross modal attention: Accuracy

Response accuracy for the letter discrimination task was at ceiling (>95%). All participants were able to maintain their gaze on the fixation cross before the presentation of the visual target in over 73% of the trials/condition.

3.2.3. Pointing task: Accuracy

Pointing error (Fig. 4) was calculated as the signed difference between the location of the finger hidden from view when the finger was perceived to be in alignment with the visual target minus the actual location of the visual target (somatosensory minus visual location). We found no statistical evidence for a change in pointing error after optokinetic stimulation. Repeated measures 2 × 2 ANOVA showed no statistically significant interaction between the factors i) optokinetic stimulation direction (left vs. right) and ii) session (pre vs. post optokinetic stimulation) with F(1, 17) = 1.4, p = 0.253. The main effects were not statistically significant either. Finally, we conducted a three-way 2 × 2 × 2 repeated measures ANOVA to assess whether the misalignment between the visual and somatosensory spaces was task dependent. Note the sign of the pointing error (somatosensory minus visual location) was the opposite to that of the cueing error (visual minus somatosensory location). To compare results across tasks in this final analysis we then calculated visual minus somatosensory error for both tasks. The three-way interaction between i) task (cross-modal attention vs pointing), ii) optokinetic stimulation direction (left vs. right) and iii) session (pre vs. post optokinetic stimulation) was statistically significant with F(1, 17) = 6.33, p = 0.022. Like the difference between the results of Experiment 1 and the cross modal attention task, this interaction

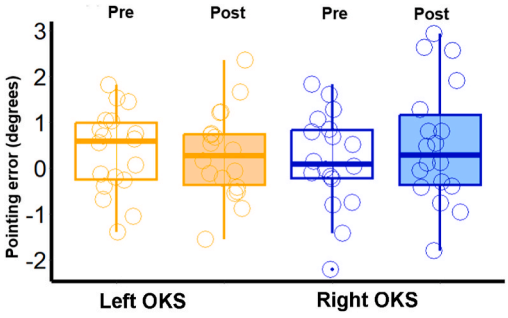

Fig. 4. Pointing error before and after optokinetic stimulation (OKS). Pointing error was calculated as the signed difference between the location of the finger hidden from view when participants felt the finger was in alignment with the visual target minus the location of the visual target. Negative values indicate that the finger was to the left of the visual target.

suggests that the effect on the attentional priority map cannot be explained by a more general mislocalisation of the visual target relative to the hand.

4. Discussion

This study found that a short period of optokinetic stimulation in healthy participants caused an adaptation in the eye proprioception and a lateral bias in spatial attention.

4.1. Adaptation in the eye proprioceptive system

Experiment 1 showed that after optokinetic stimulation, an LED in complete darkness was perceived as being further in the direction of the visual motion than its actual location (Figs. 2 and 5A). For instance, after left optokinetic stimulation, an LED that was further to the right of the finger was perceived as being straight in front of the finger. The effect of optokinetic stimulation on visual localisation error was significantly larger after an eye press, which is an intervention that increases the weight of eye proprioception in the estimate of eye position (Balslev et al., 2012a). The increase in visual localisation error after optokinetic stimulation in the presence vs. the absence of an eye-press indicates a change in eye proprioceptive input.

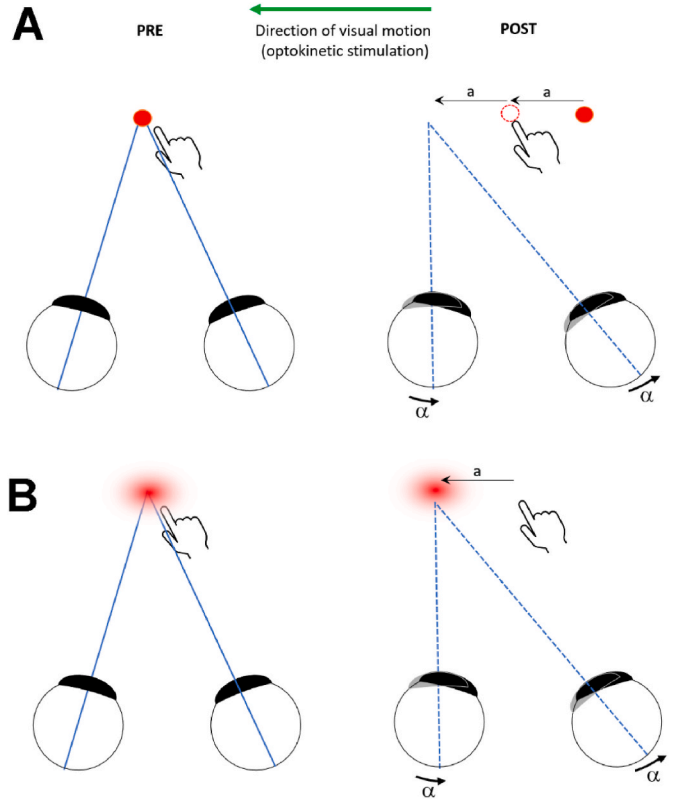

Fig. 5. Summary of the findings. A. After optokinetic stimulation healthy participants misalign an LED with their unseen index finger in complete darkness. The visual localisation error ('a') opposite the direction of visual motion suggests an error in the perception of own gaze ('α') in the direction of visual motion. After left optokinetic stimulation, a foveated visual object (red empty circle) at the location of the unseen hand appears displaced leftwards, and a visual object to the right of it, at distance ('a') from the fovea (solid red circle) appears to be in front of the hand. **B.** Optokinetic stimulation causes a shift of attention in the same direction as the gaze error. Attention is not allocated at the exact location of the cue (the participant's index finger, hidden from view), but further away, in the direction of the visual motion. Blue continuous line: actual direction of gaze. Blue dashed line: perceived direction of gaze. Red solid circle: LED location perceived by participants to be straight in front of their index finger, in complete darkness. Red blob: the locus of attention in the visual space, defined as the visual location with the largest reaction time difference for visual discrimination in the presence vs. the absence of the cue. PRE: before optokinetic stimulation. POST: after optokinetic stimulation. Black arrows indicate the direction of the mislocalisations. For clarity the hand is shown in front of the body and head midline. Distances are not drawn to scale.

There are several alternative explanations for these findings, which we think are unlikely.

First, it is known that the error in visual localisation varies with the retinal eccentricity of the visual target. Peripheral targets are typically misperceived to be located further in the periphery whereas foveated visual targets are located relatively accurately relative to the body (Bock, 1986). The participants moved their eyes freely in Experiment 1. Given previous findings, a difference in spontaneous gaze direction across conditions could have changed the eccentricity of the target on the retina and thus caused a difference in visual localisation error independent on a change in eye proprioception. For instance, left optokinetic stimulation could have caused a rightward spontaneous eye position during Experiment 1 and for this reason, a leftward mislocalisation of visual targets. If the difference in visual localisation error across conditions in Experiment 1 merely reflects a difference in the retinal eccentricity of the target, then the passive eye movement (the eye-press) should not affect mislocalisation error. This is because the eye press changes only transiently eye position. The eye moves in the direction of the eye press, then by an equal amount, in average, in opposite direction (Balslev et al., 2022). Because the eye press increased the difference in visual mislocalisation error between conditions, it is unlikely that a change in resting eye position (and retinal eccentricity), independent of a change in eye proprioception, explains the findings.

Likewise, the dependence of the mislocalisation error on the eye press rules out a change in the corollary discharge or proprioception from the landmark finger as alternative explanations. This is because neither corollary discharge nor proprioception from the landmark finger are altered by an eye press in complete darkness.

Finally, because the experiment took place in darkness and the finger-press was applied on a closed eye, there was no visual input during the eye press. For this reason, changes in the visual input cannot explain the effect of this intervention on visual localisation.

To detect whether a visual target aligns with the hidden hand (Experiment 1), we suggest, the brain transforms the retinotopic location of the target into body-centered coordinates. The eccentricity of the projection of the LED on the retina provides information about the distance of the LED from the centre of gaze. Limb proprioception conveys distance from the index finger to body midline. An estimate of gaze angle (eye-in-head rotation) is based on eye proprioception and corollary discharge. These signals (alongside head-on-trunk rotation and monocular depth-from-focus) together provide an estimate of location of the LED relative to the index finger. After optokinetic stimulation, a change in the eye proprioceptive signal would cause a shift in perceived gaze in the direction of visual motion. A foveated target would appear displaced in that direction causing an error in the opposite direction of optokinetic stimulation (Fig. 5A).

This study builds up on previous work that identified a misperception of visual target location in the direction of visual target movement during smooth pursuit of a single visual target (Mitrani et al., 1979; Rotman et al., 2004) or during the smooth pursuit phase of the optokinetic stimulation (Tozzi et al., 2007). We observed a mislocalisation error in the same direction after optokinetic stimulation. Because the effect was measured after the end of the intervention, it likely reflects plasticity in the oculomotor systems. Experiment 1 suggests that this plasticity involves a change in eye proprioception. Whereas our findings demonstrate that eye proprioception is affected, they do not rule out changes in the other component of the gaze estimate, corollary discharge, which the experiment did not explicitly test.

The mechanism of the aftereffect of optokinetic stimulation on eye proprioception is unclear. The repetitive pattern of extraocular muscle

contraction could be responsible. In support of the role of eye movements in this adaptation, it has been observed that 0.5–2 min of sustained extraocular muscle activity (fixating the gaze on a lateral target) caused an aftereffect on visual mislocalisation: perceived gaze shifted opposite the direction of fixation (Paap and Ebenholtz, 1976). Although the current experiment asked the participants to perform a different type of extraocular muscle activity, the findings of (Paap and Ebenholtz, 1976) may still be relevant. This is because they show that repetitive, directional extraocular muscle activity can cause lasting directional changes in the sense of eye position. To investigate which features of the eye movement in response to optokinetic stimulation is responsible for the aftereffect on eye proprioception future studies could record eye movements during the optokinetic stimulation to identify movement features that predict the behavioural effect on eye proprioception and spatial attention.

4.2. Adaptation in the allocation of attention

The cross-modal attention task of Experiment 2 showed a shift of attention after optokinetic stimulation in the direction of visual motion (Fig. 3). After left optokinetic stimulation, for instance, healthy participants did not allocate attention to visual targets at the exact location of the cue (their index finger hidden from view), but instead, further, to the left.

Previous work identified a shift in attention in the direction of visual motion during smooth pursuit of a single target (Khan et al., 2010). We identified a shift of attention in the same direction after the end of the optokinetic stimulation. Here the effect on spatial attention was observed during fixation, several minutes after the cessation of the eye movements. The transient, predictive orientation of attention immediately before a planned eye movement (Zirnsak and Moore, 2014) which could satisfactorily explain previous results (Khan et al., 2010; Van Donkelaar and Drew, 2002)), cannot account for the current findings.

The effect of left optokinetic stimulation on left spatial neglect (i.e., a re-balancing of attention towards the left visual hemispace) has been attributed to an illusory perception of head movement rightwards with the eyes pointing ahead which causes a shift in the perception of body midsagittal plane leftwards (Pizzamiglio et al., 1990a; Vallar et al., 1993a; Karnath, 1996). We did not measure the participants' perception of head-on-trunk rotation or midsagittal plane, so it remains unclear whether a change in these variables occurred alongside the change in eye proprioception. We do not think that the effect of optokinetic stimulation on spatial attention in Experiment 2, however, was mediated by a change in the perceived rotation of the head on the trunk for two reasons. Firstly, the error in the perception of gaze direction leftwards after left optokinetic stimulation (Experiment 1) does not suggest an illusory head movement rightward with the eyes pointing ahead. Secondly, it has been observed that neck-muscle vibration and vestibular caloric stimulation (intervention that alter the perception of head-on-trunk rotation) do not change the allocation of attention in the visual space (Rorden et al., 2001).

We found that optokinetic stimulation causes both a change in eye proprioception and a shift of attention in the visual space (Fig. 5). This dovetails with previous findings, where a change in eye proprioception led to a shift in spatial attention. This was observed after inhibitory transcranial magnetic stimulation over the somatosensory cortex (Balslev et al., 2011b; Odoj and Balslev, 2013, 2016), a focal lesion of the eye area in the primary somatosensory cortex (Balslev et al., 2013) or the passive stretch of the extraocular muscles using a scleral lens (Balslev et al., 2012b). Future intervention studies that combine optokinetic stimulation with a change in eye proprioception (for instance using transcranial magnetic stimulation over the somatosensory cortex) could answer whether the change in eye proprioception is causal for the effect of optokinetic stimulation on spatial attention.

Consistent with previous studies that altered eye proprioception, we found that the shift in spatial attention here (Experiment 2) was in the same direction as the shift in the perceived gaze (Experiment 1). The effect of optokinetic stimulation on attention cannot be explained by a mislocalisation of the visual targets relative to the body. This is because after left optokinetic stimulation a visual target was mislocated to the right of the hand, whereas the attention was allocated to the left of the hand, when the hand acted as a spatial attention cue (Fig. 5). It is unclear how the coordinate transformation for cross-modal attention differs from that used to align the hand with the visual target in Experiment 1. We propose that to orient attention, a retinotopic memory trace of salient visual locations is combined with an estimate of gaze direction when needed. In support of this conjecture, it has been observed that the brain maintains a retinotopic memory trace of salient locations, which updates over eye movements (Golomb et al., 2010; Henriques et al., 1998). One's own hand location (even when hidden from view) is a salient visual location, given that visual objects that appear near the location of unseen hand are typically prioritised for perception (Perry and Fallah, 2017; Reed et al., 2006). Sensorimotor signals of eye and hand movement i.e., the corollary discharge (Rolfs et al., 2011), the efference copy of the hand motor command and moment-to-moment changes in hand and eye proprioception could continuously update this retinotopic memory trace. When needed, the retinotopic memory trace could be transformed into cross-modal coordinates by combining the retinotopic location with an estimate of gaze direction (Fig. 6). After optokinetic stimulation, a shift in perceived gaze would cause an error in allocating attention (Fig. 6).

4.3. Clinical implications

A core symptom in spatial neglect is inattention towards people and objects located contralateral to the brain lesion. An error in the gaze direction input to the attentional priority map in spatial neglect is suggested by the following observation. When the patients with left spatial neglect are asked to focus their attention on a salient object, such as the location of their own hand hidden from view, visual perception does not improve at that exact location, but rather 1°-2° degrees to its right (Balslev and Odoj, 2019). The displacement is specific for attention. It does not reflect a general error in locating visual objects relative to the

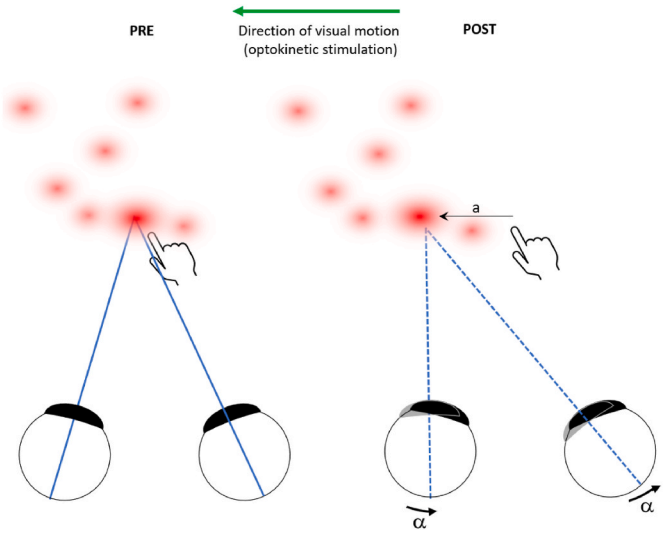

Fig. 6. Hypothesis about the coordinate transformation for allocating attention in the cross-modal space. A memory trace of salient visual locations (which includes hand location) is maintained in retinotopic coordinates and updated using signals of eye and hand movement. When needed, this retinotopic memory trace is combined with an estimate of eye position. If the retinotopic memory trace is accurate and eye position is misestimated, then attention would be deployed at incorrect locations. Red blobs - salient locations in retinotopic coordinates.

body, because the patients are accurate when pointing with their hidden hand to the visual targets.

It is yet unclear whether the eye proprioceptive error is causal for the attention bias in spatial neglect. If that is the case, then the effect of optokinetic stimulation on eye proprioception might be a substrate of the therapeutic effect of this intervention in spatial neglect (Hill et al., 2015; Liu et al., 2019). If so, then the therapeutic effect of left optokinetic stimulation could be improved by combining it with other interventions that affect eye proprioception, such as repetitive transcranial magnetic stimulation over the somatosensory cortex (Balslev and Miall, 2008; Odoj and Balslev, 2013).

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CRediT authorship contribution statement

H.H. Chan: Formal analysis, Investigation, Methodology, Software, Writing – original draft, Writing – review & editing, Data curation. **A.G. Mitchell:** Data curation, Formal analysis, Investigation, Methodology, Software, Writing – original draft, Writing – review & editing. **E. Sandilands:** Data curation, Formal analysis, Investigation, Methodology, Software, Writing – original draft, Writing – review & editing. **D. Balslev:** Conceptualization, Data curation, Formal analysis, Funding acquisition, Methodology, Project administration, Software, Supervision, Writing – original draft, Writing – review & editing.

Declaration of competing interest

None.

Data availability

https://doi.org/10.17630/7ec04723-283f-4725-bccd-3b5e79f98807 (Balslev, 2023).

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