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Research report

Prism adaptation alters spatial remapping in healthy individuals: Evidence from double-step saccades☆

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ABSTRACT

The visual system is able to represent and integrate large amounts of information as we move our gaze across a scene. This process, called spatial remapping, enables the construction of a stable representation of our visual environment despite constantly changing retinal images. Converging evidence implicates the parietal lobes in this process, with the right hemisphere having a dominant role. Indeed, lesions to the right parietal lobe (e.g., leading to hemispatial neglect) frequently result in deficits in spatial remapping. Research has demonstrated that recalibrating visual, proprioceptive and motor reference frames using prism adaptation ameliorates neglect symptoms and induces neglect-like performance in healthy people – one example of the capacity for rapid neural plasticity in response to new sensory demands. Because of the influence of prism adaptation on parietal functions, the present research investigates whether prism adaptation alters spatial remapping in healthy individuals. To this end twenty-eight undergraduates completed blocks of a double-step saccade (DSS) task after sham adaptation and adaptation to leftward- or rightward-shifting prisms. The results were consistent with an impairment in spatial remapping for left visual field targets following adaptation to leftward-shifting prisms. These results suggest that temporarily realigning spatial representations using sensory-motor adaptation alters right-hemisphere remapping processes in healthy individuals. The implications for the possible mechanisms of the amelioration of hemispatial neglect after prism adaptation are discussed.

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1. Introduction

As we move our gaze to explore the world, the image projected on the retina rapidly changes with every saccade. Our perception does not reflect this frenzy of movement; instead we experience a stable visual scene. With each new fixation the brain must integrate the old and new retinal images with information about the current eye position and the magnitude and direction in which gaze was displaced by the most recent saccade. This integration of different points of view over time and space is referred to as spatial remapping.

Neurons with spatial remapping properties were first identified in the lateral intra-parietal area of monkeys by Duhamel et al. (1992). Using single cell recordings, they observed neurons that responded when a saccade brought the location of a target into their receptive field, even when the target was extinguished before the eye movement. Furthermore, other cells responded to targets outside their current receptive field, but only if the target would be brought into the receptive field by an imminent saccade, suggesting predictive shifts in the cortical representations of visual stimuli (see also Colby et al., 1996; Gnadt and Andersen, 1988). The maintenance of target coordinates between saccades, and the anticipation of the retinal consequences of eye movements, suggest that the parietal cortex has a role in the generation and updating of visual representations. This probably occurs in concert with other visual areas in which neurons with spatial remapping properties have been subsequently found: the superior colliculus (Walker et al., 1995), frontal eye field (Umeno and Goldberg, 1997, 2001) and striate and extrastriate cortex (Nakamura and Colby, 2002).

Spatial remapping has frequently been studied using the double-step saccade (DSS) paradigm (Becker and Jürgens, 1979; Hallet and Lightstone, 1976; Mays and Sparks, 1980; Westheimer, 1954). In this paradigm, participants make two successive saccades to the location of targets that briefly appear one after another and disappear before the first saccade can be initiated (see Fig. 1). The first eye movement can be performed based on the retinal coordinates of the remembered location of the target, but in order to perform the second saccade correctly its retinal coordinates must be updated to account for the change in gaze direction after the first saccade. This is thought to occur through the integration of information regarding eye muscle potentiation (i.e., current gaze direction) and corollary discharge signals (i.e., internal copies of imminent ocular-motor commands; Wurtz and Sommer, 2004). Thus, as shown in the bottom right panel of Fig. 1, both the direction and amplitude of the second saccade are revised with respect to the first saccade. The observation that healthy individuals are able to perform this task smoothly and accurately indicates the capacity of the visual system to remap the relative retinal locations of objects over successive saccades in reference to the environmental locations (Becker and Jürgens, 1979; Heide and Kömpf, 1998).

In contrast to healthy individuals, patients with lesions to the parietal cortex such as those that lead to hemispatial neglect ('neglect') show either a complete failure of, or inaccuracies in, executing the second saccade when it requires updating of spatial locations from a contralesional location (Duhamel et al., 1992; Heide et al., 1995). Heide et al. (1995) found that this was true for patients with left or right posterior parietal cortex (PPC) lesions but not lesions to the left or right frontal eye fields, left supplementary motor area or left dorsolateral prefrontal cortex (see also Heide and Kömpf, 1998). These results also revealed an asymmetry between the spatial remapping properties of the two cerebral hemispheres: whereas patients with both left PPC and right PPC lesions had deficits in remapping from contralesional to ipsilateral locations, the right hemisphere patients also had deficits in remapping in an ipsilateral direction *even if* the first saccade was in the ipsilateral (right) visual field (Heide et al., 1995). Recent evidence from a patient with damage to the right PPC accompanied by a focal collosal lesion reinforces this result (Pisella et al., 2011).

These neuropsychological findings have been complemented by research demonstrating deficits in DSS performance in healthy participants following Transcranial Magnetic Stimulation (TMS) to the right PPC (van Donkelaar and Müri, 2002; Morris et al., 2007). Furthermore, when TMS over the PPCs of the left and right hemispheres were directly compared, only right-hemisphere stimulation altered spatial remapping of attentional inhibition (van Koningsbruggen et al., 2010). Therefore converging evidence supports a leading role of the right hemisphere in spatial remapping, consistent with a large body of work suggesting right-hemisphere dominance for spatial and attentional functions (Heilman and van der Abell, 1980; Husain and Rorden, 2003; Kinsbourne, 1993; Vogel et al., 2003).

Pisella and Mattingley (2004) proposed that a deficit in spatial remapping is a major contributor to the impairments shown by patients with neglect. The cardinal symptom of neglect is difficulty orienting towards objects and events on the contralesional side of space. The disorder can be observed after lesions to a broad range of left and right fronto-parietal areas that together constitute a bilateral attention network (Mesulam, 1981, 1999). Neglect, however, has greater incidence, chronicity and severity following damage to the right parietal lobe centred around the temporo-parietal junction and inferior parietal lobe (Farnè et al., 2004; Mort et al., 2003). The dominant role of the right hemisphere in spatial remapping may be one reason for the higher prevalence of neglect following right compared to left hemisphere lesions. This was first suggested by Pisella and Mattingley (2004), who stated that since retinotopic maps are updated or overwritten with each saccade, an impairment in remapping would mean that patients would be unable to keep track of the number and locations of objects as they move their gaze over a scene, (but see Vuillermier et al., 2008, for an alternative view on how a spatial remapping deficit may contribute to neglect). Along with the lateralised orientation bias, a remapping deficit would explain, for example, why neglect patients can be unaware of objects on the left side of space even when they have recently explored them with direct vision, and why they repeatedly revisit right-sided distractors in visual search tasks.

One promising treatment for neglect involves recalibrating spatial representations. During prism adaptation participants reach to targets that are viewed through prismatic

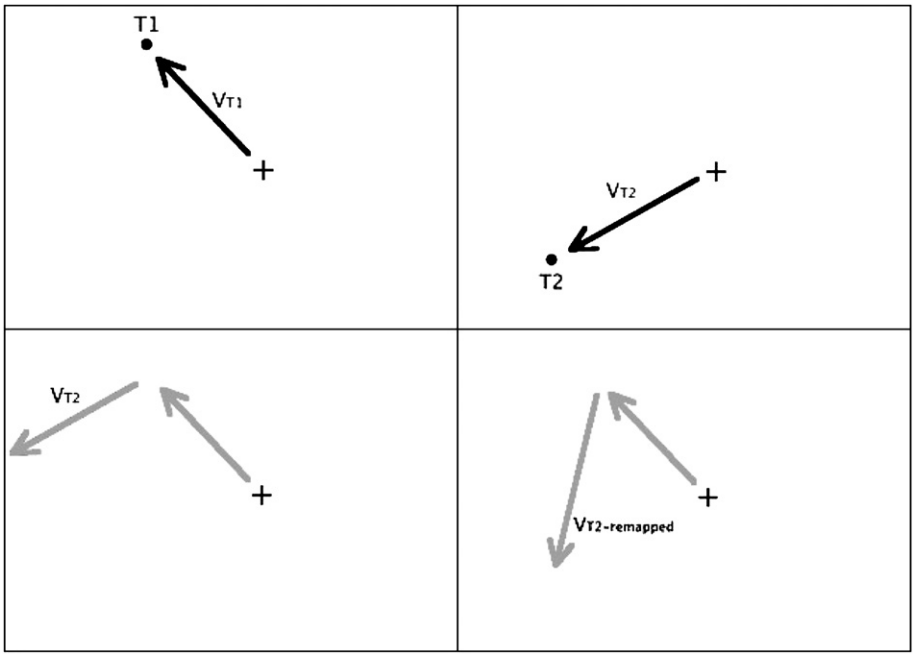

Fig. 1 – An example of a DSS trial. Targets T1 and T2 are briefly flashed on the screen (upper panels) at vectors V_{T1} and V_{T2} relative to fixation (black arrows). The lower left panel shows eye movements (grey arrows) that would be made in the absence of spatial remapping. Here, vector V_{T2} results in a saccade to an incorrect location, above and leftward of the true T2 location. However, with spatial remapping (lower right panel) a new vector, $V_{T2\text{-remapped}}$, is calculated for the second eye movement, resulting in a correct saccade.

lenses that bend the light before it reaches the eyes, shifting the visual image to one side. Since their movements are programmed based on shifted visual information, participants initially point to one side of the target. With successive trials, however, pointing accuracy is re-established as the visual system recalibrates visual, proprioceptive and motor reference frames (Redding et al., 2005). In practical terms, this constitutes a shift in pointing movements in the opposite direction of the visual shift. Once the prisms are removed pointing errors are made in the opposite direction to the prismatic shift: the adaptation after-effect. Adaptation to rightward visual shifts, resulting in a leftward recalibration of reaching movements, reduces the severity of neglect on a broad range of clinical and experimental measures (Berberovic et al., 2004; Farnè et al., 2002; Mcintosh et al., 2002; Nijboer et al., 2008; Rossetti et al., 1998; Schindler et al., 2009).

In healthy individuals adaptation to leftward-shifting prisms, giving a rightward after-effect, induces neglect-like patterns of performance on line bisection (Berberovic and Mattingley, 2003; Colent et al., 2000; Michel et al., 2003; Nijboer et al., 2008), haptic exploration (Girardi et al., 2004), spatial representations of numbers and letters (Loftus et al., 2008; Nicholls et al., 2008) and computerised reaction-time tests of spatial attention (Striemer et al., 2006). Interestingly, a consistent finding is that neglect symptoms are ameliorated by adaptation to rightward- but not leftward-shifting prisms, while neglect-like changes in healthy participants are seen only after adaptation to leftward-shifting prisms (although, see Berberovic and Mattingley, 2003; Striemer et al., 2006). The reasons for this are yet to be determined, but are probably related to the same mechanisms that cause the greater prevalence of neglect following right than left hemisphere damage. Overall, the findings from both neglect patients and healthy individuals suggest that prism adaptation is one example of the brain's capacity for rapid rewiring in response to new sensory demands (Boudreau et al., 2007), and that these plastic processes affect more than just low-level representations of visual coordinates and arm proprioception.

Metabolic (Clower et al., 2001; Luauté et al., 2006; Shiraishi et al., 2008) and functional imaging (Chapman et al., 2010; Danckert et al., 2008; Luauté et al., 2006) studies of healthy individuals and brain-lesioned patients undergoing prism adaptation have implicated parietal areas of both the left and right hemisphere, especially the anterior intra-parietal sulcus. It is therefore likely that perturbation of parietal lobe function is a major component of the mechanism by which prism adaptation reduces neglect, and that other right-parietal deficits may be similarly affected. In previous work, we explored the possibility that prism adaptation reduces deficits caused by right parietal dysfunction other than the lateralised attention bias (Bultitude and Woods, 2010; Bultitude et al., 2009). We demonstrated that prism adaptation ameliorated one such deficit: the local processing bias. Specifically, prism adaptation reversed the tendency for patients with lesions to the temporo-parietal junction to be hyperattentive to local details of a visual scene in preference to the global structure (Bultitude et al., 2009). Prism adaptation also induced a neglect-like increase in attention to local

detail in healthy participants (Bultitude and Woods, 2010). These results demonstrated that the influence of prism adaptation on spatial cognition extends beyond a simple lateral shift in proprioception and orienting, which might underlie the broad generalisation and longevity of the cognitive changes induced.

In summary, a large body of research suggests that the parietal lobe, particularly in the right cerebral hemisphere, has a critical role in remapping of saccadic coordinates, and that this may be one reason for the deficits in spatial exploration demonstrated by patients with hemispatial neglect. Prism adaptation has been identified as a promising means of ameliorating neglect symptoms on a broad range of tasks, and also influences at least one other right-parietal function that is associated with neglect (i.e., global/local processing). Because of these observations, and the involvement of the parietal lobe in prism adaptation, the present study examined whether prism adaptation alters spatial remapping in healthy individuals using a DSS task.

In a between-groups design, participants underwent adaptation to leftward-shifting (experimental group) or rightward-shifting (control group) prisms before completing a DSS task. In each trial two targets (T1 and T2) were presented in quick succession within the same visual field. We predicted that adaptation to leftward-shifting prisms, which induces neglect-like performance in healthy individuals, would lead to impaired spatial remapping reminiscent of that produced by disruption of the right parietal lobe by a lesion or TMS. Based on the studies reviewed above, the induced deficit could be restricted to the left – 'contralateral' – visual field (e.g., Duhamel et al., 1992; Morris et al., 2007); or evident across both visual fields, albeit with the possibility of larger effects in the left visual field (LVF) (e.g., Heide et al., 1995; van Koningsbruggen et al., 2010; Pisella et al., 2011). Errors in updating the retinal coordinates of the T2 location after the first saccade could lead to T2 saccade endpoints that are shifted away from fixation with respect to the true T2 location, such as in the leftward remapping error shown for the LVF trial in Fig. 1. Therefore, disruption to spatial remapping by adaptation to leftward-shifting prisms could result in outward shifts in the endpoints for T2 saccades.

In keeping with previous studies on effects of prism adaptation in healthy participants (e.g., Colent et al., 2000; Loftus et al., 2008; Loftus et al., 2009), no changes in saccadic remapping were expected for the rightward-shifting prism groups.

2. Methods

2.1. Participants

Twenty-eight undergraduates from Utrecht and Bangor Universities participated in the experiment for €7 or £6 per hour (twelve males; mean age = 23.6, *SEM* = .73). All participants had normal or corrected-to-normal acuity and were right-handed (mean handedness quotient = −.84, *SEM* = .03; where a score of −1 indicates absolute right-handedness and a score of +1 indicates absolute left-handedness; Oldfield, 1971).

2.2. General procedure

The experiment took place in a dimly lit room. Participants were seated in a standard rotating office chair that enabled easy movement between two tables placed at 90° to each other. The equipment for prism adaptation was positioned upon one of these tables, and the monitor for the DSS task was positioned on the other (see below for the full procedures for these tasks). The experiment was explained and informed consent received in accordance with the 1964 Declaration of Helsinki.

The overall procedure is outlined in Fig. 2. A repeated-measures design was used in which participants underwent sham adaptation and prism adaptation within a single session. Participants completed four experimental blocks of the DSS task with eighty trials per block in an AABB design: two sham adaptation blocks to establish baseline performance followed by two prism adaptation blocks. As a result of pilot testing, two sets of shorter blocks were used, each preceded by sham or prism adaptation, to ensure that the adaptation after-effect was maintained for the entire DSS task (see Schindler et al., 2009, for a similar design). In addition to these, there was one practice block before the first sham adaptation session, during which the experimenter gave verbal feedback on the participant's performance.

2.2.1. Prism adaptation and open-loop pointing measures

For prism adaptation, participants faced a 90 cm wide × 35 cm high × 70 cm deep box similar to that described in Bultitude and Woods (2010). The box had a removable lid and was open at two opposite sides at which the participant and the experimenter were positioned. The targets for prism adaptation were markers on the base of the box at arm's reach from the participant at −10°, 0° and +10° from their mid-saggital plane (negative numbers indicate a leftward deviation). Participants rested their chin on the edge of the box and were fitted with welding goggles containing Risley biprisms set to induce no visual shift (0°; sham adaptation), or to induce a 15° visual shift to the left or right (prism adaptation). The direction of prismatic shift (leftward or rightward) was varied between participants, resulting in two groups of fourteen participants for each prism direction.

Starting with their right hand resting directly in front of their torso, participants were asked to reach out and touch with their right index finger each of the three targets in a specific pattern (left-middle-right-middle), repeated continuously for a total of 150 pointing movements. Pointing was performed in time with a metronome set to 1 Hz to minimise online correction of pointing trajectory. Participants returned their hand to their torso before pointing to the next target.

To confirm visuo-motor adaptation, open-loop pointing errors were measured immediately before and after each sham/prism adaptation session, and after the second DSS block for each type of adaptation (see Fig. 2). The lid was placed on the box to occlude the participant's pointing arm from their vision, and they performed twelve open-loop pointing movements to −10°, 0° and +10° visual targets. Pointing errors were recorded to the nearest .5° by the

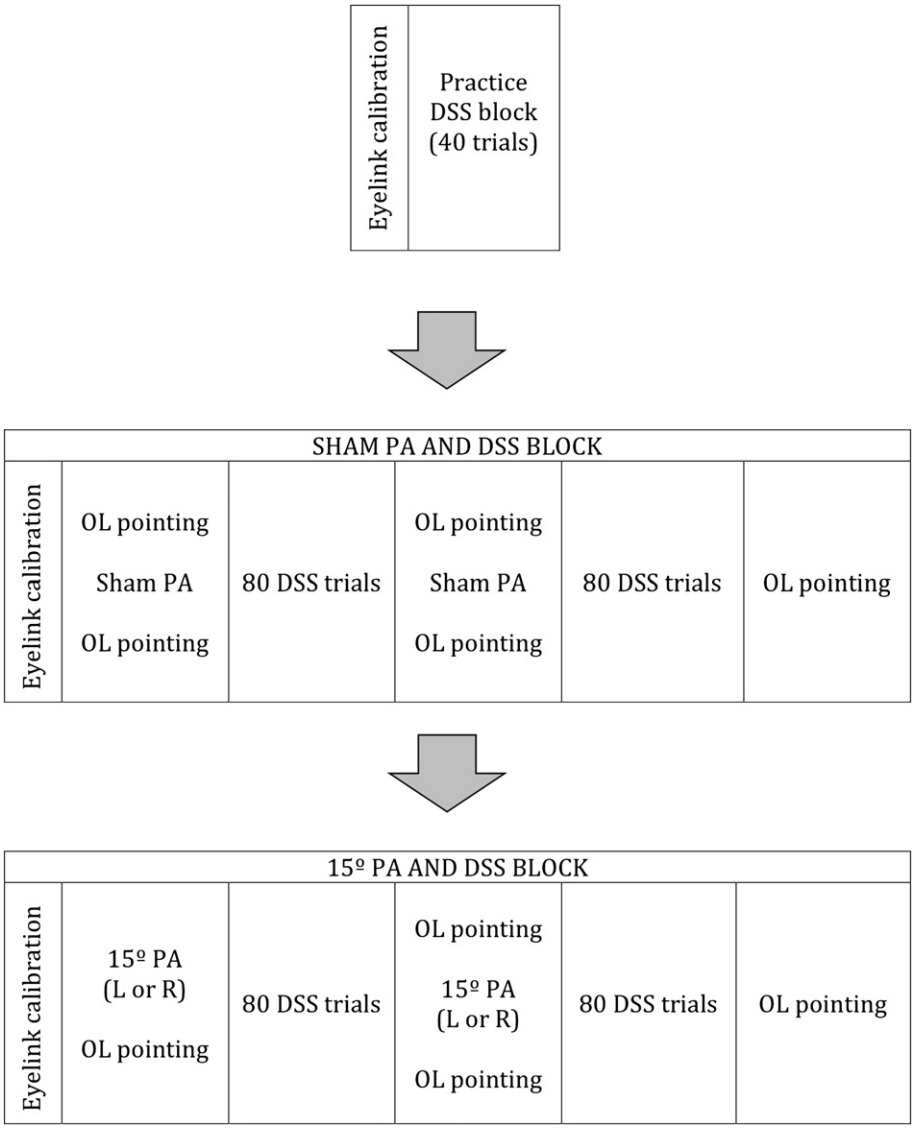

Fig. 2 – Flowchart for the overall experimental design.

experimenter with the aid of markings drawn on the underside of the lid of the box.

To discourage de-adaptation, participants kept their eyes closed between the prism adaptation and open-loop pointing tasks, and when moving between the prism adaptation box and the computer for the DSS task.

2.2.2. *DSS task*

For the DSS task, participants were seated in front of a computer with their head held stationary by a chin rest positioned 57 cm from the computer screen. All visual stimuli were presented as white images on a black background. Eye movements were recorded by an Eyelink 1000 infrared eye tracker (SR Research Ltd, Canada) at a temporal resolution of 1000 Hz and a spatial resolution of .01°. A nine-point calibration was performed immediately prior to each of the practice DSS blocks, the first sham adaptation block, and the first prism adaptation block. Recalibration was kept to a minimum as it was assumed that this may result in de-adaptation (Paap and Ebenholtz, 1976; Wallace and Fisher, 1984). Calibration, however, was generally stable and recalibration was required mid-block in <5% of cases.

The order of stimulus presentation for the DSS task is shown in Fig. 3. Each trial commenced with the appearance of a central fixation dot. On a second computer screen the experimenter monitored eye movements and initiated the trial with a button press when the participant was fixating the dot. The trial would not be initiated if the participant's gaze deviated from fixation by more than 1.0°. Once the trial was initiated, the central dot was replaced by a .72° × .72° fixation cross, indicating the start of the trial. After a time that varied randomly between 1500 msec and 2500 msec, the fixation cross disappeared and two .72° circular targets appeared sequentially for 140 msec (T1) and 80 msec (T2), based on the design of Duhamel et al. (1992). These were replaced by a blank screen.

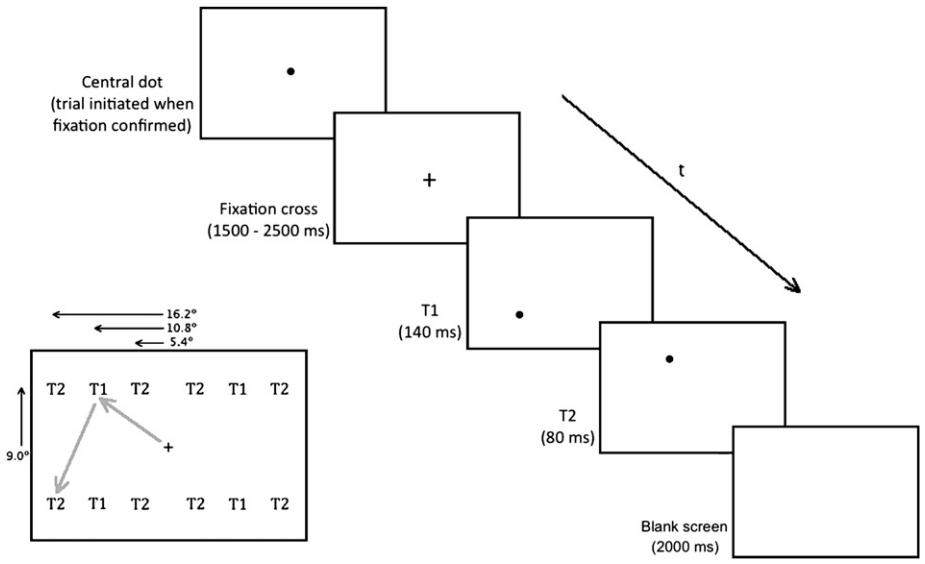

Fig. 3 – The trial time course and stimulus locations for the DSS task. Note that for clarity the illustration is given as black figures on a white background, however stimuli for the task were white figures on a black background. The eye movement sequence for one example trial is shown in grey arrows.

The possible T1 and T2 target locations are illustrated in Fig. 3. There were four possible T1 target locations: 9.0° above or below, and 10.8° left or right of fixation. There were eight possible T2 locations: 9.0° above or below and 16.2° and 5.4° to the left or right of fixation. For each T1 target there were two possible T2 locations, in the same horizontal visual field and the vertically opposite quadrant. For example, for the T1 location in the upper left quadrant, the T2 location could be one of the two locations in the lower left quadrant. Participants were instructed to move their eyes to the T1 location, and then immediately to the T2 location. After an interval of 2000 msec the central dot appeared for the beginning of the next trial.

Each set of two blocks consisted of twenty repetitions of each visual field [LVF, right visual field (RVF)] by T1 location (upper, lower) by T2 location (left or right relative to T1) condition, giving a total of 160 trials for each of the sham and prism adaptation conditions.

2.3. *Analyses*

2.3.1. *Open-loop pointing errors*

The open-loop pointing errors for each subject were averaged by session and grouped for further analysis. To test for adaptation after-effects, these errors were subjected to Bonferroni-corrected paired-samples *t*-tests comparing different post-adaptation errors to baseline errors. There were two sets of comparisons: one for the sham adaptation open-loop pointing sessions, and one for the prism adaptation open-loop pointing sessions.

2.3.2. *Saccadic endpoints*

An eye movement was considered a saccade when either eye velocity exceeded 35/sec or eye acceleration exceeded 9500/sec^2. Saccades smaller than 2° were discounted from analysis. Trials for the DSS task were excluded if the starting point for the T1 saccade was more than 3° from fixation, saccades were made to only one of the target locations, or saccades were made to the T2 location before the T1 location (16.5% of trials). The remaining data were averaged across conditions for each subject and pooled for further analysis.

Since shifts in endpoint errors could result from changes in saccadic latencies, the RTs for T1 saccades and the T1-to-T2 inter-saccade intervals were subjected to separate omnibus repeated-measures Analyses of Variance (ANOVAs) of prism group (leftward-, rightward-shifting) × session (sham, prism) × visual field ('LVF', 'RVF') × T1 location (upper visual field, lower visual field) × remap direction (left of T1, right of T1).

Subsequently, the endpoints for the T1 and T2 saccades were considered separately. The dependent measures were horizontal shifts in saccade endpoint due to prism adaptation (in degrees of visual angle), which were calculated by subtracting pre-adaptation from post-adaptation horizontal endpoints for saccades made to the same VF × T1 × T2 locations (see Fig. 2). The T1 endpoint shifts were subjected to a three-way repeated-measures ANOVA with prism group (leftward-, rightward-shifting) as a between-subjects factor, and visual field (LVF, RVF) and T1 location (upper, lower) as within-subjects factors. A similar ANOVA was performed on the T2 endpoint shifts, with the addition of a fourth factor: T2 location (left of T1, right of T1).

Finally, follow-up Analyses of Covariance (ANCOVAs) were planned to determine if any changes in T2 were independent of those that would be expected by changes in low-level components of ocular-motor performance such as saccade magnitude, as reported in previous studies (Angeli et al., 2004a, 2004b; Datie et al., 2006; Dijkerman et al., 2003; Ferber and Murray, 2005; Ferber et al., 2003; Serino et al., 2006; Shiraishi et al., 2008). Any effects of prism adaptation on spatial remapping would influence only the remapped T2 saccades, however low-level ocular-motor shifts would affect

saccades regardless of whether or not they were remapped (i.e., both T1 and T2). Variability in T1 shifts can thus be used as a measure of the influence of low-level changes on T2 saccade endpoints. To this end, the ANCOVAs used T1 endpoint shift as a covariate to determine if any significant effects revealed by the ANOVA of T2 shifts were independent of general changes in ocular-motor behaviour (see van Donkelaar and Müri, 2002, for a previous example of analysing T2 saccade changes with respect to T1 variability).

3. Results

3.1. Open-loop pointing errors

Mean pointing errors across the nine open-loop pointing sessions for the leftward- and rightward-shifting prism group are shown in Fig. 4 (see also Tables 1 and 2 in Supplementary materials online). There was no significant change in the pointing errors of either group across the sham adaptation sessions ($ps > .0125$; Bonferroni-corrected). In contrast, the pointing errors for the leftward-shifting prism group were significantly rightward of baseline ($ps < .005$) in both of the sessions that immediately followed prism adaptation (post-prism 1 and post-prism 2), and in the sessions that followed DSS blocks (pre-prism 2 and late). Similarly, the pointing errors for the rightward-shifting prism group were significantly leftward of baseline in all four comparisons (post-prism 1, post-prism 2, pre-prism 2 and late; $ps < .005$). These analyses revealed, therefore, that although there was no change in pointing errors following sham adaptation, both groups showed significant shifts in pointing errors after prism adaptation that were in the opposite direction to that of the prismatic shift, and which were maintained throughout the entire DSS blocks. The magnitude of these shifts were similar to those reported in previous studies that used comparable methods of after-effect measurement (approximately one-third of the prismatic shift; Bultitude and Woods, 2010; Girardi et al., 2004; Loftus et al., 2008, 2009).

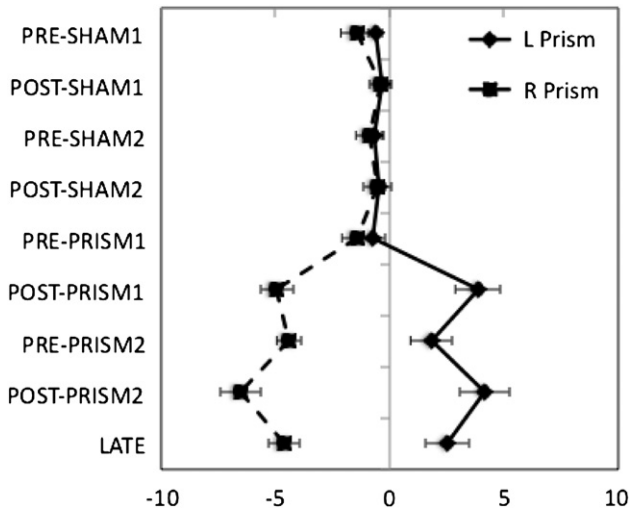

Fig. 4 – Pointing errors for the leftward- and rightward-shifting prism groups across the nine open-loop sessions. Error bars represent ±1SEM (across subjects).

3.2. DSS task

3.2.1. Latencies

There were no significant main effects nor interactions involving session or prism group in the analysis of T1 saccadic latencies, nor in the analysis of T1-to-T2 inter-saccade intervals ($ps > .05$). Therefore any differences in saccadic endpoints cannot be attributed to changes in saccadic reaction time (RT).

3.2.2. Saccadic endpoint shifts

There were no significant main effects nor interactions in the analysis of T1 endpoint shifts ($ps > .05$). In contrast, the analysis of T2 saccadic errors revealed a main effect of prism group [$F(1,26) = 4.7$, $p = .04$]. This reflected a non-significant .11° rightward shift [$SEM = .16$, $CI^{.95}$=(−.22, .44)] in the remapped T2 saccadic endpoints for the leftward-shifting prism group, and a significant .37° leftward shift [SEM=.16, $CI^{.95}$=(−.70, −.05)] in the remapped T2 saccadic endpoints for the rightward-shifting prism group.

This main effect was driven by a significant prism group × visual field interaction [$F(1,26) = 6.829$, $p < .05$]. The interaction is plotted in Fig. 5, along with the T1 shifts for the same (non-significant) interaction. Paired t-tests revealed that for the leftward-shifting prism group there was a significant difference between T2 shifts for the LVF [$M = -.19$, $SEM = .20$, $CI^{.95}$=(−.61, .22)] and RVF [$M = .42$, $SEM = .20$, $CI^{.95} = (.01, .83)$], $t(13) = 2.4$, $p < .05$. In comparison, the rightward-shifting prism group had no significant difference between T2 shifts for the LVF [$M = -.23$, $SEM = .20$, $CI^{.95} = (-.64, .19)$] and RVF [$M = -.52$, $SEM = .20$, $CI^{.95} = (-.94, -.11)$], $t(13) = 1.25$, $p = .233$. Two-sample t-tests also confirmed that the shift in T2 saccadic endpoints for the leftward- and rightward-shifting prism groups were significantly different for RVF trials [$t(26) = 3.31$, $p < .005$], but not for LVF trials [$t(26) = .11$, $p = .91$]. In summary, the interaction stemmed from an overall leftward shift in saccadic endpoints for the rightward-shifting prism group, and endpoints that shifted in opposite directions in the two visual fields (away from fixation) for the leftward-shifting prism group. There were no further significant main effects or interactions in the analysis of T2 endpoint shifts.

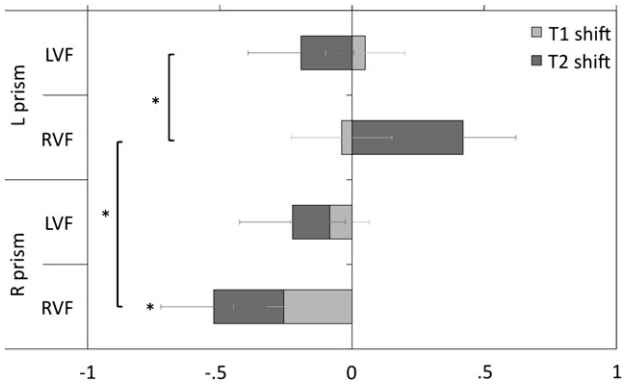

Fig. 5 – T1 and T2 endpoint shifts due to prism adaptation in the left and right visual fields for the two prism groups. Error bars represent ±1SEM (across subjects). Significance indicators (* = $p < .05$) refer to T2 endpoint shifts only.

To determine to what extent the above changes were due to altered spatial remapping over and above changes in low-level ocular-motor performance, T2 saccade endpoints for the leftward- and rightward-shifting prism groups were subjected to separate ANCOVAs, with T2 endpoint shift as the dependent variable, T1 endpoint shift as a covariate, and visual field (LVF, RVF) and T1 location (upper, lower) as fixed factors. For the leftward-shifting prism group there was a significant main effect of visual field [$F(1,12.8) = 7.0$, $p < .05$], indicating that there were changes in T2 saccadic endpoints over and above any low-level ocular-motor changes for the leftward-shifting prism group. In contrast, there were no significant main effects or interactions for the rightward-shifting prism group ($ps > .05$), suggesting that the changes in T2 saccadic endpoints can be attributed to low-level ocular-motor changes (The nature of the changes in saccadic endpoints following prism adaptation are better realised in Supplementary Fig. 1 online, which shows the T1 and T2 endpoint errors for individual trials pooled separately across prism group).

We further scrutinized the relationship between T1 and T2 endpoint shifts by constructing separate scatterplots for individual subject data in each visual field and for each prism group (Fig. 6). Results of linear regression analyses indicate significant positive correlations between T1 and T2 shifts in both the LVF ($r^2 = .55$, $p < .005$) and RVF ($r^2 = .71$, $p < .001$) for the rightward-shifting prism group, and in the RVF only ($r^2 = .70$, $p < .001$) for the leftward-shifting prism group. However, T1 and T2 shifts in the LVF were unrelated for the leftward-shifting prism group ($p = .22$). Together with ANCOVA analyses above, these results suggest that the T2 shifts in both visual fields for the rightward-shifting prism group, and in the RVF for the leftward-shifting prism group, are related to the T1 shifts. However, this is not the case for the LVF T2 shifts for the leftward-shifting prism group. Further illustration of this conclusion is provided in Fig. 7, which gives the mean T1 and T2 endpoints in given trial types for one individual from each of the leftward- and rightward-shifting prism groups. The Figure shows that the changes in T1 and T2 are similar in the four trial types following adaptation to rightward-shifting prisms, but that this is not the case for the participant who adapted to leftward-shifting prisms.

Finally, in neither group did T1 nor T2 endpoint shifts correlate with changes in open-loop pointing errors after the first set of prism adaptation ($ps > .05$), consistent with previous studies (Berberovic and Mattingley, 2003; Girardi et al., 2004; Frassinetti et al., 2002).

4. Discussion

This study examined whether adaptation to leftward-shifting prisms alters spatial remapping in healthy individuals. For the leftward-shifting prism group, the changes in endpoints for the remapped saccades following prism adaptation were different in the two visual fields, shifting away from central space. These were independent of T1 endpoint shifts. For the rightward-shifting group, there was an overall leftward shift in the endpoints of T2 saccades with no significant difference between the two visual fields, and this was not independent of T1 endpoint shift. Therefore, only adaptation to leftward-shifting prisms had a significant effect on spatial remapping above and beyond changes to general saccadic performance.

A possible interpretation of our results is represented in Fig. 8. Previous studies have demonstrated that prism adaptation shifts exploratory eye movements in the same direction as the adaptation after-effect, according to measures that include

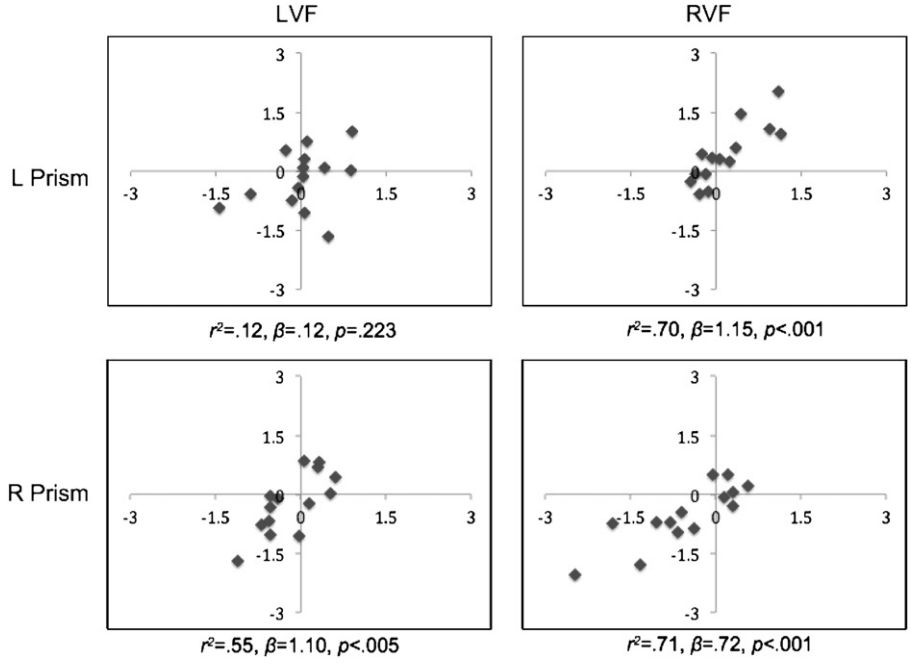

Fig. 6 – Scatterplots of individual participants' endpoint shifts for T1 saccades (x axis) versus T2 saccades (y axis) in each visual field.

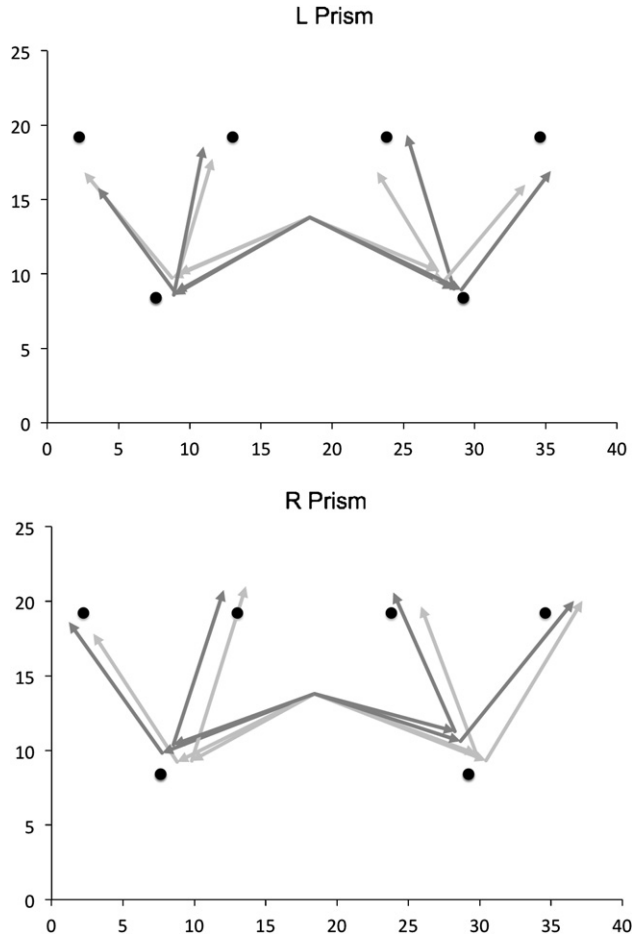

Fig. 7 – Mean pre- (light grey) and post- (dark grey) adaptation T1 and T2 endpoints for four example trial types for one individual from each of the leftward- and rightward-shifting prism groups.

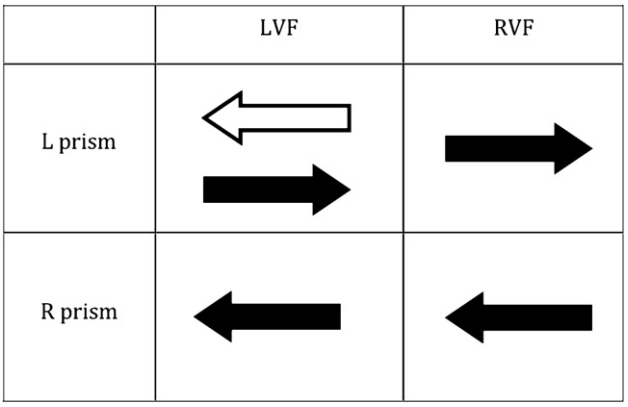

Fig. 8 – Representations of the possible influences of low-level ocular-motor shifts (black arrows) and changes in spatial remapping (white arrows) on the T2 saccadic endpoints of the leftward- and rightward-shifting prism groups.

the magnitude and number of leftward versus rightward saccades (Angeli et al., 2004a, 2004b; Dijkerman et al., 2003; Ferber and Murray, 2005; Ferber et al., 2003; Serino et al., 2006; Shiraishi et al., 2008). In Fig. 8, these low-level ocular-motor shifts are indicated by black arrows in both visual fields for both groups. The leftward shifts in saccadic endpoints that were observed for the rightward-shifting prism group can be attributed to these low-level changes. If adaptation to leftward-shifting prisms influences right hemisphere functions, as suggested by its effect on visuo-spatial processing, then any changes in spatial remapping may be either restricted to or larger in the LVF. A failure in spatial remapping would result in T2 saccades that have the same vector as the original retinal coordinates of T2 relative to fixation (see Fig. 1) – that is, leftward of the true retinal coordinates. Therefore, partial disruption of spatial remapping by adaptation to leftward-shifting prisms would result in leftward shifts in saccade endpoints (white arrow) that would negate the rightward low-level ocular-motor shift. Consistent with this interpretation, the 95% confidence intervals around the mean showed that the saccadic endpoint shifts in the LVF for the leftward-shifting prism group were not significantly different from zero, whereas there was a significant rightward shift in the RVF. Therefore, our results are consistent with both existing evidence of shifted low-level ocular-motor behaviour following prism adaptation, and additional changes in spatial remapping that are observed following right parietal brain lesions and TMS.

While some descriptions of remapping deficits in patients with right PPC lesions have included deficits in both the LVF and RVF in the same individuals (Heide et al., 1995; Pisella et al., 2011), our interpretation of the present results is that there were changes to remapping within the LVF only. This is consistent with the disruption of saccadic remapping for LVF but not RVF trials following right parietal TMS (Morris et al., 2007)[1], and also with the dominant role of the right hemisphere in spatial remapping (Heilman and van der Abell, 1980; Husain and Rorden, 2003; Kinsbourne, 1993; Vogel et al., 2003).

Although any low-level ocular-motor changes should influence both unremapped and remapped saccades, the analysis of T1 shifts pooled across groups yielded no significant results. If the changes in T2 endpoints for the rightward-shifting prism group are due to low-level ocular-motor changes, for example, then why are there no equivalent changes to T1 saccades? It is possible that any low-level changes in ocular-motor performance could cumulate across successive saccades. Therefore, changes for the T1 saccades could be too small to reach significance in our group analysis, but would nonetheless covary with the larger ocular-motor changes for T2 saccades. The significant T1-to-T2 correlations that are represented in Fig. 6 are consistent with such a cumulative pattern. It is worth noting that previous studies reporting changes in ocular-motor performance after prism adaptation used tasks involving free exploration of pictures, text or words rather than directed saccades to a specific, pinpoint location as is the case in the present study. Prism effects may be smaller when the target endpoint is precise and

[1] In contrast to the results of Morris et al. (2007), who showed an increase in variance following parietal lobe TMS, we found no notable changes in the mean variance of the remapped saccadic endpoints following prism adaptation.

externally provided rather than broad and internally generated. Somewhat consistent with this is our previous research in which we found no effects of prism adaptation on latencies for directed saccades (Nijboer et al., 2010), although we have no information on saccade endpoints for this study.

Taken together with previous research, our results indicate that adaptation to leftward-shifting prisms induces both neglect-like shifts in spatial awareness and errors in saccadic remapping. Based on these results, it is reasonable to hypothesise that adaptation to rightward-shifting prisms, which ameliorates neglect in brain-lesioned patients, would also reduce the magnitude and/or variability of their remapping errors. An improvement in spatial remapping may have contributed to previous gains reported for neglect patients on tests of neglect dyslexia (Angeli et al., 2004b; Farnè et al., 2002), visual search (Saevarsson et al., 2009), and spatial dysgraphia (Rode et al., 2006) following adaptation to rightward-shifting prisms. Without improvements in the ability to maintain spatial representations across saccades *in addition to* a reduction in lateralised orienting bias, patients would be unable to integrate words across a paragraph, and would continue to revisit already-explored locations on visual search tasks. To the best of our knowledge, however, the results of the present study provide the first direct evidence for alteration of spatial updating by prism adaptation. This adds to previous findings that prism adaptation has a pervasive influence on parietal lobe functions that contribute to neglect impairments other than the lateralised orienting bias, including global/local processing (Bultitude and Woods, 2010; Bultitude et al., 2009), the disengage deficit (Striemer and Danckert, 2007; Striemer et al., 2006), spatial dysgraphia (Rode et al., 2006) and perseveration (Nys et al., 2008).

Corbetta et al. (2005) found that the degree to which neglect recovered over time in patients with right frontal lobe damage correlated with a rebalancing of activity in the (undamaged) parietal lobes. This suggests that the maintenance or reduction of neglect symptoms is determined by the extent to which normal performance of undamaged, functionally related areas is restored. The cause of the longevity and broad generalisation of prism adaptation may therefore lie in its recruitment of a broad cortical network. Metabolic and functional imaging studies of prism adaptation have implicated cerebellar and parietal areas of both hemispheres (Chapman et al., 2010; Clower et al., 2001; Danckert et al., 2008; Luauté et al., 2006; Luauté et al., 2009; Shiraishi et al., 2008). Also, prism adaptation is impaired in monkeys following muscimol deactivation of the ventral premotor cortex (Kurata and Hoshi, 1999) as well as in patients with lesions to the cerebellum (Pisella et al., 2005), white matter under the middle frontal gurus (Sarri et al., 2008), or dorsal premotor cortex (Lee and van Donkelaar, 2006). Imaging and neuropsychological studies are therefore consistent with the idea that the variety of behavioural changes following prism adaptation stems from its influence on a pervasive cortical network.

A suggested model for the prism adaptation (Striemer et al., 2008) is that sensory-motor discrepancy leads to the generation of bottom-up error signals from the cerebellum that trigger spatial realignment by parietal areas. In patients with neglect, this triggers leftwards reorienting mediated by the left superior parietal lobe. The subsequent sensory feedback would re-activate other left and residual right hemisphere parietal areas, leading to further cognitive effects such as improvement in spatial remapping. The absence of any correlation between the magnitudes of the pointing aftereffects and higher cognitive changes in our present results and previous studies supports such an indirect mechanism (Berberovic and Mattingley, 2003; Girardi et al., 2004; Frassinetti et al., 2002). Furthermore, a progressive restoration of function would explain reports of patients who initially show a paradoxical neglect of the *right* side of space immediately after prism adaptation, but had broader, more centralised allocations of attention with amelioration of neglect at later tests (Rode et al., 2001). If the process by which prism adaptation influences the broader attention network occurs particularly slowly in these patients, this could constitute an early leftward shift of their attention without a reduction in the local processing bias or spatial remapping deficit. As the broader bilateral attention network is activated these non-lateralised spatial deficits also reduce.

In conclusion, the results of the present study demonstrate that recalibrating sensory-motor representations of space using prism adaptation can also alter the ability of healthy individuals to maintain and integrate spatial representations across saccades. This adds to a growing body of research demonstrating that prism adaptation has a broad influence on higher-level attention and other visuo-spatial functions.

Supplementary material

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.cortex.2012.01.008.

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