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Research Report

Trans-saccadic memory after right parietal brain damage

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ABSTRACT

Introduction: Spatial remapping, the process of updating information across eye movements, is an important mechanism for trans-saccadic perception. The right posterior parietal cortex (PPC) is a region that has been associated most strongly with spatial remapping. The aim of the project was to investigate the effect of damage to the right PPC on direction specific trans-saccadic memory. We compared trans-saccadic memory performance for central items that had to be remembered while making a left- versus rightward eye movement, or for items that were remapped within the left versus right visual field.

Methods: We included 9 stroke patients with unilateral right PPC lesions and 31 healthy control subjects. Participants memorized the location of a briefly presented item, had to make one saccade (either towards the left or right, or upward or downward), and subsequently had to decide in what direction the probe had shifted. We used a staircase to adjust task difficulty (i.e., the distance between the memory item and probe). Bayesian repeated measures ANOVAs were used to compare left versus right eye movements and items in the left versus right visual field.

Results: In both conditions, patients with right PPC damage showed worse trans-saccadic memory performance compared to healthy control subjects (for the condition with left- and rightward gaze shifts, $BF_{10} = 3.79$; and when items were presented left or right, $BF_{10} = 6.77$), regardless of the direction of the gaze or the initial location of the memory item. At the individual level, none of the patients showed a direction specific deficit after leftward versus rightward saccades, whereas two patients showed worse performance for items in the left versus right visual field.

Conclusion: Damage in the right PPC did not lead to gaze direction specific impairments in trans-saccadic memory, but instead caused more general spatial memory impairments.

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1. Introduction

We perceive the external world as coherent, yet being rich in detail. However, detailed information is processed only over a few degrees of visual angle at the fovea. We, therefore, continuously execute eye movements in order to create a complete representation of the world around us. Nevertheless, we do not experience this rapid change of visual input as the brain continuously integrates the visual information that is obtained from one fixation to another. The process of transferring visual information across eye movements is known as trans-saccadic perception (Prime, Vesia, & Crawford, 2011). There are two central processes in trans-saccadic perception: the visual information must be stored in memory across saccades (i.e., trans-saccadic memory) and the retinotopic coordinates of visual information must be updated. Thus trans-saccadic perception is the process of integrating the content of individual fixations over space and time into a stable, internal representation of the environment (Pisella et al., 2011; Pisella & Mattingley, 2004).

The posterior parietal cortex (PPC) is one of the brain regions that is strongly associated with trans-saccadic perception. Studies in monkeys (Duhamel, Colby, & Goldberg, 1992) showed that the receptive fields of neurons in the PPC are remapped *before* an eye movement is made, and neurons respond when an eye movement brings a previously flashed location into the receptive field (i.e., a memory trace; Colby & Goldberg, 1999; Duhamel et al., 1992). This property is called spatial remapping of receptive fields and might underlie the updating of retinotopic representations. Neurons demonstrating spatial remapping have, however, also been found in other brain areas, such as the macaque analogue of the human intraparietal sulcus (IPS), the frontal eye fields (FEF), and area V4 (e.g., Andersen, Bracewell, Barash, Gnadt, & Fogassi, 1990; Neupane, Guitton, & Pack, 2016; Umeno & Goldberg, 2001; Wang et al., 2016).

Human functional neuroimaging studies in healthy participants confirm the role of the PPC in trans-saccadic perception as they have shown that activity in the PPC is associated with encoding and updating information in a gaze-centred reference frame (Medendorp, Goltz, Vilis, & Crawford, 2003; Merriam, Genovese, & Colby, 2003). In particular the *right* PPC has been associated with updating of retinotopic coordinates: TMS over the right PPC disrupts this process (Chang & Ro, 2007; Morris, Chambers, & Mattingley, 2007; Prime, Vesia, & Crawford, 2008; Van Donkelaar & Müri, 2002; van Koningsbruggen, Gabay, Sapir, Henik, & Rafal, 2010), whereas TMS over the left PPC has no effect (Prime et al., 2008; van Koningsbruggen et al., 2010).

Furthermore, lesions in the right PPC have been associated with deficits in trans-saccadic perception (e.g., Pierce & Saj, 2018; Pisella & Mattingley, 2004; Ten Brink, Van der Stigchel, Visser-Meily, & Nijboer, 2016; Van der Stigchel et al., 2018; but Rath-Wilson & Guitton, 2015 argued otherwise). Direction specific deficits in trans-saccadic perception might play an important role in post-stroke cognitive disorders such as visuospatial neglect (Pierce & Saj, 2018; Pisella & Mattingley, 2004), a disorder in lateralized attention, and constructional apraxia (Russell et al., 2010; Van der Stigchel et al., 2018), a disorder in constructional skills such as drawing and copying. Visuospatial neglect and constructional apraxia are heterogeneous, multicomponent disorders that can result from different brain lesions, typically with a right-hemispheric dominance. A similarity between these disorders is that patients have difficulty maintaining or updating an accurate representation of the spatial array, which could be explained by impaired spatial remapping properties at the neural level. Vuilleumier et al. (2007) found that in seven neglect patients with a single focal right-hemispheric stroke, trans-saccadic memory was disproportionally impaired after *rightward* gaze-shifts, thus when the memory item required updating into a leftward position within internal gaze-central maps. For a memory item that is initially encoded at fixation, an eye movement towards the right should update the information about the item's location *leftwards* in retinotopic terms. This information is then transferred to the right hemisphere. If the neurons that represent the leftward locations within retinotopic maps are damaged, such remapping might be disturbed. The effect of initial target side did not interact or correlate with the trans-saccadic cost caused by the rightward gaze, indicating distinct and additive sources for these two effects (Vuilleumier et al., 2007). Comparable results were found in eight right brain-damaged patients with constructional apraxia (Russell et al., 2010). The patients were significantly impaired in trans-saccadic memory performance, particularly when the first saccade of the sequence was towards the ipsilesional side (Russell et al., 2010). An important drawback of both studies, however, is that two eye movements had to be made instead of one (i.e., ipsi-contra and contra-ipsi; Russell et al., 2010; Vuilleumier et al., 2007), which makes it difficult to disentangle which eye movement direction, and thus the direction of updating of in trans-saccadic perception (i.e., ipsilesional or contralesional) caused the observed trans-saccadic memory deficit. Furthermore, in both studies, eye movements were not measured.

In both studies (Russell et al., 2010; Vuilleumier et al., 2007), it is explicitly suggested that the observed trans-saccadic memory deficit in patients with neglect and apraxia can possibly be explained by right parietal involvement, and in particular the right PPC. We aimed to directly test this specific hypothesis by selecting patients based on having a lesion in the right PPC rather than the presence of neglect or apraxia. We investigated whether damage in the right PPC results in direction specific impairments in trans-saccadic memory by using a staircase controlled, single-eye movement design. We hypothesized that, as in prior studies (Russell et al., 2010; Vuilleumier et al., 2007), if damage in the right PPC affects trans-saccadic memory specifically when eye movements are made towards the ipsilesional field, we would see impaired trans-saccadic memory performance after *ipsilesional* (i.e., rightward) eye movements. A secondary aim was to investigate whether trans-saccadic memory would be reduced for items presented and updated *within* the left compared to the right hemifield. We expected worse trans-saccadic memory accuracy for items that were presented in the left compared to the right hemifield, due to attention deficits that could result from right hemispheric lesions (Corbetta & Shulman, 2011;

Heilman, Abell, & Van Den Abell, 1980; Kinsbourne, 1987; Mesulam, 1981). Third, we aimed to study which brain areas within the PPC are related to the expected direction specific trans-saccadic memory deficits by using lesion-symptom mapping.

2. Methods

We report how we determined our sample size, all data exclusions, all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all measures in the study.

2.1. Participants

Patients were recruited via the University Medical Centre Utrecht and De Hoogstraat Rehabilitation, Utrecht (the Netherlands). The sample size calculation was based on the study by Vuilleumier et al. (2007). The crucial within-subject effect (remapping after a leftward saccade *vs* remapping after a rightward saccade) in their sample of 5 patients was reported as $t(4) = 3.38$. This *t*-value was entered into the program on the psychometrica website (https://www.psychometrica.de/effect_size.html) in order to estimate the effect size. Unfortunately, no correlation between the two conditions was reported by Vuilleumier et al. (2007). We set the correlation to a neutral value ($r = .5$) and calculated the effect size (Cohen's $d = 1.51$). We used this effect size to calculate sample size using G*Power 3.1.9.2 (Faul, Erdfelder, Lang, & Buchner, 2007). With alpha set to .05 (one-tailed) and desired power set to .8, a sample size of minimal 5 patients was required. Because this sample size estimation was based on an estimated correlation between conditions, we wanted to add 5 more subjects to our sample to make sure that we had sufficient power, in case the actual correlation would differ from our estimation. Hence, we aimed for a sample size of 10 patients. Inclusion criteria for the current study were established prior to data analysis, and were: (1) clinically diagnosed ischemic stroke affecting the right posterior parietal cortex (confirmed with a CT or MRI scan), first or recurrent; (2) in subacute/chronic phase (i.e., at least 6 weeks have passed since stroke); (3) 18–85 years of age; (4) being able to understand the instructions of the experimental task; (5) no lesions in the left hemisphere; (6) no history of substance abuse; and (7) no clinical signs of visual field defects (see '2.2 Apparatus, stimuli, procedure, and design'). A group of neurologically healthy individuals with a comparable age distribution served as experimental controls. They were recruited through staff and relatives of the researchers. All reported normal or corrected-to-normal visual acuity. No part of the study procedures or analyses were pre-registered prior to the research being conducted. The experiment was performed in accordance with the Declaration of Helsinki and in accordance with the Medical Research Involving Human Subjects Act (WMO). The study was approved by the Medical Ethical Committee of the University Medical Centre Utrecht (protocol number 15-314/M). All participants gave written informed consent.

2.2. Apparatus, stimuli, procedure, and design

Before start of the experiment the following data were obtained: age, sex, handedness, and date of stroke. Clinical signs of visual field defects were verified by showing the participant stimuli near the edge of a computer screen. Patients who could not see the stimuli were excluded from the study. We used the same experimental procedure as described by Ten Brink, Nijboer, Fabius, and Van der Stigchel (2019). The script of the experiment can be found at https://osf.io/xqvkb/. Participants were seated in a light and sound attenuated room at 70 cm from a computer monitor (60.7 × 35 cm). Their head was stabilized using a chin rest. Monocular eye movement data was collected at 1000 Hz using an SR Research EyeLink 1000 eye tracker, located at 600 mm from the eye. Participants were tested having both eyes open, and the left eye was monitored. We used nine-point calibrations at the beginning of the experiment and between trials when necessary. Experimental tasks were programmed in MATLAB (version R2015a), using the Psychophysics Toolbox extensions (Brainard, 1997).

Stimuli were presented against a black background. Participants were instructed to fixate a central cross (.5°, white, luminance 33.3 cd/m^2). From the moment of fixation, there was a random delay of 500–1000 msec where after a memory item (filled circle, Ø1°, red or blue, luminance 3.20 and 3.77 cd/m^2 respectively) appeared for 250 msec. The item was presented at a random distance in between 5 and 8° either above, below, left, or right from fixation, depending on the experimental condition. In the "gaze left/gaze right" conditions, the item was presented either above or below fixation after which participants had to make a leftward ("gaze left"; contralesional) or rightward ("gaze right", ipsilesional) eye movement (Fig. 1). In the "item left/item right" conditions, the item was presented either left ("item left"; contralesional field) or right ("item right"; ipsilesional field) from fixation, after which participants had to make an up- or downward eye movement. Participants had to name the colour of the memory item to verify whether the item had been identified. The experimenter entered the answer using the keyboard ('r' for red and 'b' for blue). Subsequently, the fixation cross shifted 6° towards the left or right in the gaze left/gaze right conditions, and up or down in the item left/item right conditions. The trial continued when participants re-fixated the cross. After a 1000 msec delay, a probe appeared (same colour and size as the memory item), and remained on screen. The probe was shifted relative to the memory item, either up or down in the gaze left/gaze right conditions or left or right in the item left/item right conditions. Participants had to verbally report the direction of the shift to avoid a motor bias by having to reach leftward or rightward when providing the response. The experimenter entered the answer using a keyboard (using arrow keys).

The size of the shift between the memory item and probe was defined based on a staircase algorithm. We used Accelerated Stochastic Approximation (ASA), a non-parametric adaptive procedure that, by quickly reducing step size, rapidly converges to any accuracy level (Kesten, 1958). In the first trial, the probe always shifted 5° relative to the memory item. The ASA staircase gradually decreases step size in two

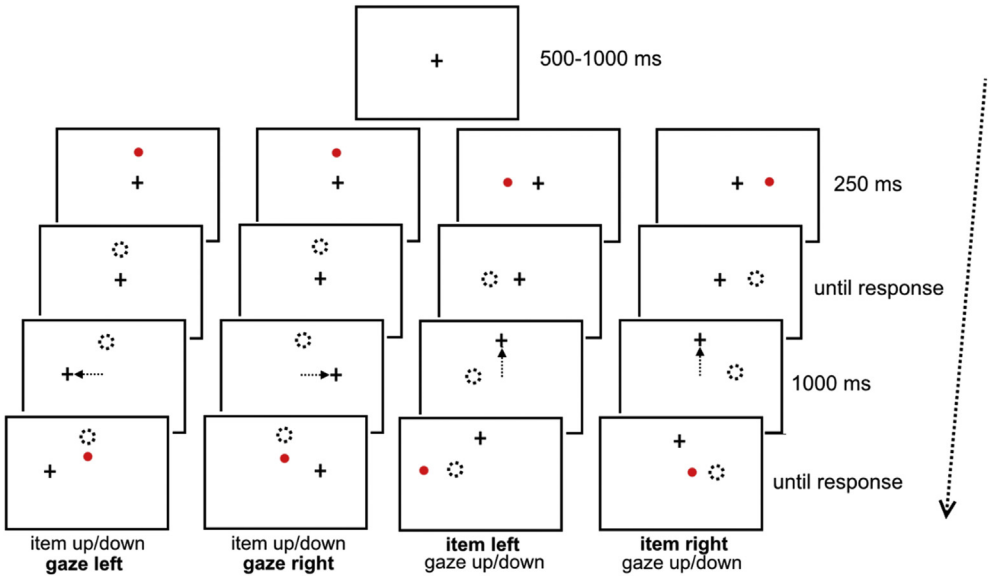

Fig. 1 – Illustrative sequences of events for the four different conditions. From fixation, a random delay of 500–1000 msec was introduced. Then, a memory item appeared for 250 msec. Participants had to name the colour of the item and memorize its location. After a response was given, the fixation cross shifted. When participants fixated the cross again, there was a delay of 1000 msec. A probe appeared and remained onscreen until participants indicated in which direction the probe had shifted relative to the memory item (represented by the dotted circle in this figure).

ways. In the second and third trial, the size of the shift between the memory item and probe on the next trial (d_{k+1}) was given by:

$$d_{k+1} = d_k - \frac{3}{k}(Z_k - 0.8) \quad (1)$$

where d_k is the distance used in the current trial, 3 is the staircase constant to determine the shift in degrees of visual angle between the memory item and probe, k is the trial number, Z_k is 1 when a correct response was provided in the current trial or 0 when an incorrect response was provided, and .8 is the desired accuracy level. For trial $n > 3$ step size changes as a function the number of changes in 'response category' (i.e., switch trials from consecutive correct to incorrect, or vice versa):

$$d_{k+1} = d_k - \frac{3}{2 + m_{switch}}(Z_k - 0.8), \; k > 3 \quad (2)$$

where m_{switch} is the number of switch trials. The minimum and maximum step sizes were set at .1° and 5° respectively. Ideally, the final threshold estimate is taken from the staircase estimates when the step size reaches a predefined lower limit. However, this means that the duration of the task is undefined. As there was no criterion to decide whether the staircase converged, we collected as many trials as possible depending on the time available, fatigue of the participant, and the amount of invalid trials, resulting in 64–112 trials per condition. Note that for each condition (i.e., gaze left, gaze right, item left, item right), a separate staircase procedure was used throughout the experiment. Per condition, the estimated distance in visual degrees at which participants were able to detect the direction of the probe shift in 80% of trials ("discrimination threshold") was computed based on the final 1/3 of trials. Later trials put more weight in the equation based on a linear relationship, the sum of the weights is 1. Thus, the last trial is the most influential single trial in our threshold computation. We used the weighted average for two reasons. First, we could not include all trials (see below for exclusion criteria of trials) and had a time constraint on the experiment, which does not guarantee that the staircase converged. Second, we put a constraint on the minimum step size to keep subjects engaged in the experiment. The calculated step size could theoretically be smaller than the minimal physical step size on our screen, effectively resulting in a step size of 0.

The four conditions were divided into two block types (i.e., gaze left/gaze right and item left/item right) that alternated throughout the experiment. The direction of the gaze shift, colour of the memory item, initial location of the item, and direction of the item shift were counterbalanced across the conditions and randomized within blocks. The order of the block types was randomized across participants. One block consisted of 32 experimental trials. At the start of each block, two additional (randomly picked) trials were presented. Responses provided in these trials were not included in the staircase algorithm to make sure that potential costs of switching between blocks did not affect the outcome. The shift size in these trials was based on the previous distance from the staircase procedure of the given condition. Prior to the experiment, practice trials were provided until the participant was able to perform the task.

Participants were instructed to fixate the cross throughout the task, and make no eye movements towards the memory item or the probe. Eye movements within 2° from a stimulus (i.e., either the fixation cross, memory item, or probe) were considered fixations at this stimulus and were detected online by the experimental program or the experimenter. A

trial was aborted on-line and not included in the staircase procedure when the memory item or probe was fixated or when the colour of the memory item was incorrectly named. In retrospect, per participant, we kept the same number of trials per condition to avoid possible outcome differences due to unequal amount of trials. Thus, the condition with the lowest number of trials was indicative of the number of trials to maintain for each condition.

2.3. Generation of lesion maps

The procedure for the generation of lesion maps has been previously described elsewhere (Biesbroek et al., 2015, 2014, 2016; Ten Brink, Biesbroek, et al., 2016). A trained researcher (NAW) who was blinded to the behavioural data manually segmented infarcts on transversal slices of either follow-up CT ($n = 6$), DWI ($n = 2$), or on T2 FLAIR sequences of MRI scans ($n = 5$). Infarct segmentations were transformed to the Montreal Neurological Institute (MNI)-152 template (Fonov, Evans, McKinstry, Almli, & Collins, 2009) using the standardized image processing pipeline RegLSM (publicly available at www.metavcimap.org), which applies the elastic toolbox (Klein, Staring, Murphy, Viergever, & Pluim, 2010). RegLSM provides custom-fit settings for CT (Kuijf, Biesbroek, Viergever, Biessels, & Vincken, 2013), FLAIR (Biesbroek et al., 2013) and DWI sequences (Zhao et al., 2018). The output format of the pipeline is the same for each setting, i.e., a lesion map in MNI-152 space. This allows lesion data from CT and MRI to be combined into one dataset. In short, the registration procedure consisted of linear registration followed by nonlinear registration. As an intermediate step, registration to an age-specific MRI template was performed (Rorden, Bonilha, Fridriksson, Bender, & Karnath, 2012) which has shown to result in a more accurate registration if brain atrophy is present. These registration steps were combined into a single step, in which the original lesion maps were registered directly to MNI-152 space, in order to prevent intermediate interpolations and thereby improve registration accuracy. Quality checks of the registration results were performed by comparing the native scan to the lesion map in MNI-152 space. For one patient, registration was not possible due to insufficient quality of the scan. For eight patients, the co-registered lesion maps were manually adjusted to correct for slight registration errors using ITK-SNAP v3.6.0 (www.itksnap.org; Yushkevich et al., 2006) by NAW. The voxel size after normalisation was $1 \times 1 \times 1$ mm.

The PPC was defined based on the AAL brain atlas (Tzourio-Mazoyer et al., 2002). Lesions in the superior parietal lobule (SPL), inferior parietal gyrus (IPG), supramarginal gyrus (SMG), and angular gyrus (AG) were considered to be part of the PPC. We reported total lesion volume in ml, percentage of lesion volume within the PPC and within each of the PPC sub regions.

2.4. Statistical analysis

We report Bayes factors (*BF*) using the Savage–Dickey density ratio method, which can be interpreted as the weight of evidence for one hypothesis over another (Wagenmakers, Lodewyckx, Kuriyal, & Grasman, 2010; Wagenmakers, Marsman, et al., 2018). Specifically, we report BF_{10}, the evidence in favour of the alternative hypothesis. Note that BF_{01}, the evidence in favour of the null hypothesis, is related to this value and can be computed by the following formula: $BF_{01} = 1/BF_{10}$. Kass and Raftery (1995) have provided guidelines to interpret the *BF* as weight of evidence: a *BF* of 1–3 is described as providing evidence that is 'not worth more than a bare mention', a *BF* of 3–20 provides 'positive' evidence, 20 to 100 'strong' evidence, and above 100 'very strong' evidence. Data was analysed using JASP version .9.0.1 (Team JASP, 2018; Wagenmakers, Love, et al., 2018). We used the default settings for ANOVA designs to set the prior distribution (Rouder, Morey, Speckman, & Province, 2012). The Bayes factor of the interaction effect was computed by selecting the option 'Effects/Across matched models' in JASP. All data and analysis scripts can be found at https://osf.io/xqvkb/.

2.4.1. Demographical and stroke-related characteristics

We compared groups (i.e., stroke and healthy controls) regarding age using Bayesian independent samples *t*-test, and regarding sex and handedness using Bayesian contingency tables with the Poisson sampling scheme (Jamil et al., 2017).

2.4.2. On-line trial exclusion

We compared the amount of trial loss (due to incorrect naming of the colour and due to making eye movements towards the memory item or probe) between groups and conditions. We performed two Bayesian repeated measures ANOVAs with Group as between subject factor (i.e., stroke patients *vs* controls) and Gaze direction (i.e., gaze left *vs* gaze right) or Item side (i.e., item left *vs* item right) as within subject factor.

2.4.3. Off-line evaluation of eye movements

Because we used a staircase algorithm it was not possible to exclude trials after the experiment had finished. Therefore, we checked the quality of the data in retrospect. We analysed how well participants had fixated the fixation cross throughout the task, separately for the first half of the trial (i.e., from the appearance of the memory item until the fixation shift) and the second half of the trial (i.e., from the appearance of the memory probe until the answer was provided). We used the marks that were provided by the EyeLink on-line parser (SR Research Ltd., 2019, Mississauga, Ontario, Canada) to distinguish between the onset and end of fixations, saccades, and blinks. Based on all fixations that (partly) occurred within the predefined periods, we computed the average deviation from the fixation cross on both the *x*-axis and *y*-axis and the percentage of fixations that were more than 3° away from the fixation cross. As on-line trial exclusion is less accurate compared to off-line trial exclusion, we used a stricter threshold for on-line trial exclusion (2°) than the threshold we used when we evaluated fixations off-line (3°). Bayesian independent samples *t*-tests (two-tailed) were used to compare fixation accuracies between groups (i.e., stroke *vs* healthy controls). Note that these analyses were only meant to evaluate the data quality and no additional trials were excluded based on the results.

For each condition, we computed the average of participant's median latency and accuracy (i.e., the absolute distance between the endpoint and the shifted fixation cross) of the

gaze shift, defined as the first eye movement of >3° after the fixation cross had shifted. We performed two Bayesian repeated measures ANOVAs with 'group' as between subject factor (i.e., stroke patients *vs* controls) and 'condition' (either gaze left/gaze right or item left/item right) as within subject factor.

2.4.4. *Trans-saccadic memory accuracy*

2.4.4.1. GROUP COMPARISONS. We performed two Bayesian repeated measures ANOVAs with Group as between subject factor (i.e., stroke patients *vs* controls) and Gaze direction (i.e., gaze left *vs* gaze right) or Item side (i.e., item left *vs* item right) as within subject factor. We expected an interaction effect between Group * Gaze direction, with worse performance for the patients in the gaze right condition compared to the gaze left condition, and an interaction effect between Group * Item side, with worse performance for the patients in the item left compared to the item right condition.

2.4.4.2. INDIVIDUAL COMPARISONS. We computed the difference in discrimination threshold between the gaze left and gaze right conditions (difference score = discrimination threshold gaze left − discrimination threshold gaze right) and between the item left and item right conditions (difference score = discrimination threshold item right − discrimination threshold item left). A negative value indicates worse performance (i.e., a higher discrimination threshold) for the gaze right compared to gaze left condition, or the item left compared to item right condition.

To determine whether patients showed a deficit or not, individual mean scores and individual difference scores were compared with the average mean score and difference score of the control group by using Bayesian Crawford statistics (Crawford & Garthwaite, 2007). We reported the point estimate of the percentage of the control population that would exhibit a lower score than the patient. We also provided an interval estimate of this quantity (i.e., the credible limit), which indicates with 95% confidence that the percentage of people who have a lower score than the patient is between the lower and upper limits. A point estimate of 5% or 95% (i.e., 5% of the control population would obtain a score lower or higher than the patient) was used as an indication for a deficit.

3. Results

3.1. *Demographical and stroke-related characteristics*

Thirteen patients with right parietal damage due to an ischemic stroke were tested. Four patients were not included in the analyses because they were not able to maintain fixation at the fixation cross when the memory item appeared (see Supplementary Tables 1 and 2 for their characteristics and Supplementary Figure 1 for their lesion maps). We decided to exclude these patients prior to data analyses, as we aborted task administration. Thus, 9 stroke patients were included in the analyses. Fig. 2 shows a lesion overlay plot of all patients and lesion maps of each individual patient. In addition, 31 healthy controls were included. Demographic and stroke-related characteristics are depicted in Table 1. It was inconclusive whether patients and controls differed regarding age, $BF_{10} = .50$, sex, $BF_{10} = .89$, or handedness, $BF_{10} = .40$.

3.2. *On-line trial exclusion*

There was a trial loss of 8.5% in the gaze left and 7.8% in the gaze right conditions for the control participants, and of 13.6% in the gaze left and 12.5% in the gaze right conditions for the patients. There was no clear main effect of group, $BF_{10} = 1.41$, and no clear interaction effect of Group * Gaze direction, $BF_{10} = .40$ ($BF_{01} = 2.5$). There was a trial loss of 10.3% in the item left and 10.9% in the item right conditions for the control participants, and of 24.4% in the item left, and 13.8% in the item right conditions for the patients. There was a main effect of group, $BF_{10} = 5.04$, and an interaction effect of Group * Item side, $BF_{10} = 5.13$. Thus, more trials had to be removed in the patient group compared to the control group when items occurred left versus when items occurred right. Trial loss was mainly due to fixations at the memory item. After exclusion, a median of 70 trials was available per condition (range: 47 to 78 trials).

3.3. *Off-line evaluation of eye movements*

The characteristics of the fixations and eye movements are shown in Table 2. We found no clear differences between the groups regarding fixation characteristics, saccade latency, and saccade accuracy, as all Bayes factors were inconclusive (BF_{10} ranging from .28 to 1.91; see Supplementary Tables 3 and 4 for all Bayes factors). Note that we did not exclude trials based on this off-line evaluation of eye movements, as this was not possible due to the staircase procedure.

3.4. *Trans-saccadic memory accuracy*

3.4.1. *Group comparisons*

Mean discrimination thresholds are depicted in Fig. 3. The output of the Bayesian repeated measures ANOVA can be found in Tables 3–6.

Tables 3 and 5 show the 'Modal Comparison' output, listing all possible models (i.e., the null model, the models with one or both main effects, and the model with the interaction effect) and their relative adequacy (Wagenmakers, Love, et al., 2018). The column p(M) shows the prior model probabilities (we have set them equal across all models as we had no prior knowledge on possible results) and the column p(M|Y) shows the updated model probabilities based on the data (higher probabilities indicate that the model is more likely). The column BF_M indicates the degree to which the data have changed the prior model odds. The column BF_{10} lists the Bayes factor for each model against the null model.

Tables 4 and 6 show the 'Analysis of Effects' output, including three effects (i.e., each main factor and the interaction effect). The first columns are based on model averaging: the evidence for the presence of a particular effect is combined across models that include that effect. As we selected 'Across matched models', the interaction-effect is not included in the averaging of the main effects. For instance, the effect 'Group', without the interaction effect, features in two out of the five models. Therefore, the prior inclusion

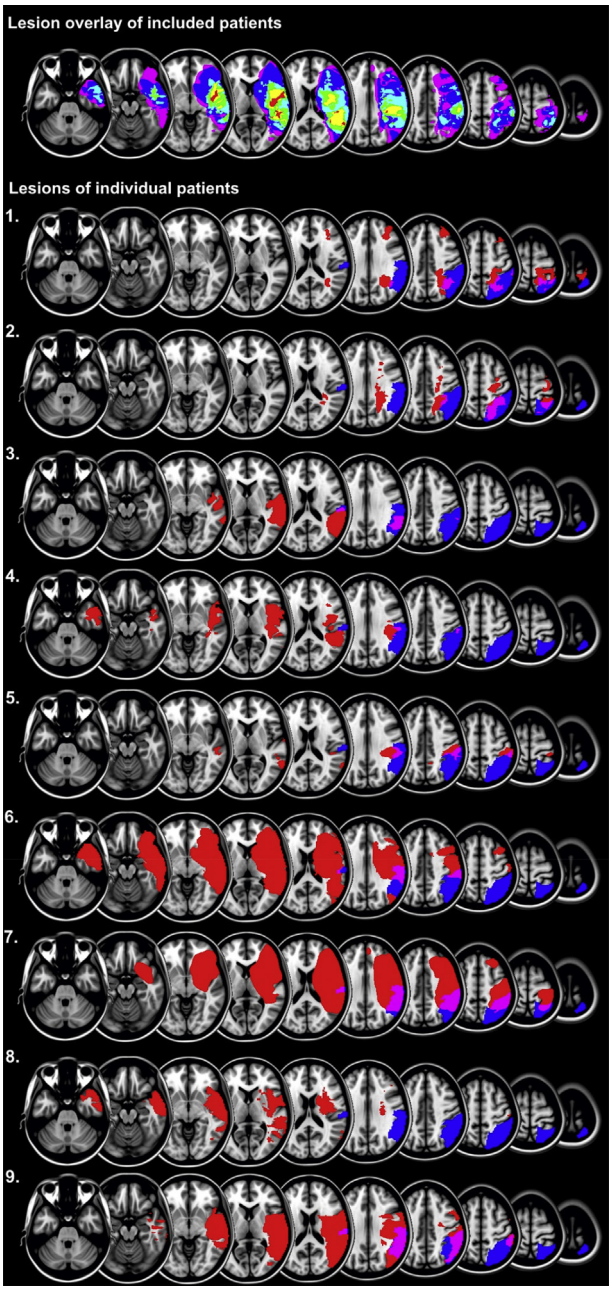

Fig. 2 – Upper panel: Lesion overlay of the included stroke patients. Colours indicate the number of patients with damage at a given location, ranging from pink (1 patient) to red (6 patients, which was the maximum number of patients with brain damage at the same location). Lower panels: Lesion maps of individual patients in red, the right PPC in blue (*lesions* in the right PPC are depicted in pink). Note that patient 8 has very small lesions in the right PPC in slices that are not depicted here. The right hemisphere is depicted on the right.

probability P(incl) equals .2 + .2 = .4. Similarly, the posterior inclusion probability P(incl|data) is the sum of the posterior probabilities of all models that include the effect. The change from prior to posterior inclusion odds is given in the column $BF_{inclusion}$.

For the gaze left/gaze right conditions, there was a suggestive evidence for a main effect of Group, $BF_{10} = 3.79$, indicating that stroke patients performed worse than control subjects. The result for the interaction effect of Group * Gaze direction was inconclusive, $BF_{10} = .45$ ($BF_{01} = 2.22$). For the item left/item right conditions, there was suggestive evidence for a main effect of Group, $BF_{10} = 6.77$, indicating that stroke patients performed worse than healthy subjects. The result for the interaction effect of Group * Item side was inconclusive, $BF_{10} = 1.88$. Thus, on a group level, damage in the right PPC did not lead to gaze direction specific impairments in trans-saccadic memory, but instead caused more general spatial memory impairments when compared to healthy control subjects.

3.4.2. *Individual comparisons*

The scores across conditions for each stroke patient and for the control group are depicted in Figs. 4 and 5. The mean scores can be found in Table 7. Three patients showed a general trans-saccadic memory impairment compared to the control group (patient 7 and 9 for all conditions and patient 2 for the item left/item right conditions). The difference scores can be found in Table 8. For the gaze left/gaze right conditions, none of the patients obtained an abnormal difference score compared to the control group. Thus, individual differences do not explain the inconclusive results found for the interaction Group * Gaze direction. Two patients (6 and 9) showed worse trans-saccadic memory performance when items were presented left versus items that were presented right, compared to asymmetries seen in healthy control subjects. The individual differences between patients could explain why we found inconclusive results for the interaction Group * Item side.

Thus, at an individual level, four of the nine stroke patients with right PPC damage showed trans-saccadic memory deficits compared to the healthy control group, either in general (patient 2, 7 and 9) or specifically when items were presented in the left versus the right visual field (patient 6 and 9). None of the patients showed direction-specific trans-saccadic memory impairment after leftward versus rightward eye movements. The four patients who showed trans-saccadic memory deficits had generally larger lesions (patient 2: 37 ml, patient 6: 293 ml, patient 7: 291 ml, and patient 9: 167 ml) than the other patients (ranging from 26 to 84 ml). Looking at lesion size within PPC, patient 2 (23.7%), 6 (22.1%), 7 (67.7%) and 9 (52.0%) had larger PPC lesions compared to the other stroke patients (ranging from .2% to 14.1%, except patient 1: 25.5%) which could explain why these patients showed impaired performance and other patients did not. There seems no clear pattern in lesion volume of the different PPC sub regions when comparing patients with and without a trans-saccadic memory deficit.

4. Discussion

The aim of the project was to investigate the effect of damage to the right PPC on direction specific trans-saccadic memory. We administered a trans-saccadic memory task in 9 chronic stroke patients with right PPC damage and 31 healthy control

Table 1 – Demographic and stroke-related characteristics for individual stroke patients and for the different groups (mean ± SD).

ID	Sex	Age (years)	Handedness	Time post stroke (years)	Total lesion volume (ml)	Percentage of sub region that is damaged				
						PPC	SPL	IPG	SMG	AG
1	Male	78	Right	8.0	48	25.5	54.8	20.5	0	28.3
2[a]	Male	48	Right	7.5	37	23.7	56.2	36.6	2.2	4.4
3	Female	42	Right	7.3	48	13.0	0	.8	20.8	27.0
4	Female	64	Right	8.3	64	5.1	0	.0	14.4	3.3
5	Male	58	Right	3.7	26	14.1	0	8.3	42.4	0
6[a]	Male	64	Right	4.0	293	22.1	0	.1	66.9	9.6
7[a]	Female	48	Left	4.8	291	67.7	34.7	96.2	99.5	42.8
8	Male	36	Right	3.1	84	.2	0	.5	.3	0
9[a]	Female	56	Right	3.4	167	52.0	.1	41.1	94.8	65.0
Patients ($N = 9$)	56% Male	54.9 ± 12.9	11% Left	5.6 ± 2.2	118 ± 107					
Controls ($N = 31$)	39% Male	50.4 ± 12.4	10% Left	–	–					

Abbreviations. PPC, posterior parietal cortex; SPL, superior parietal lobule; IPG, inferior parietal gyrus; SMG, supramarginal gyrus, AG, angular gyrus.
[a] The patient showed a deficit in trans-saccadic memory.

Table 2 – Characteristics of the fixations and eye movements of stroke patients and healthy controls, depicted as means (SD).

	Stroke patients (N = 9)	Healthy controls (N = 31)
Fixation in first half of trial[a]		
Deviation from fixation on x-axis (visual degrees)	−.06° (.47°)	.28° (.40°)
Deviation from fixation on y-axis (visual degrees)	−.23° (.42°)	−.17° (.50°)
Percentage fixations >3° from fixation cross	2.52% (1.64%)	2.97% (2.94%)
Fixation in second half of trial[b]		
Deviation from fixation on x-axis (visual degrees)	−.02° (.51°)	.30° (.40°)
Deviation from fixation on y-axis (visual degrees)	<.01° (.52°)	−.12° (.56°)
Percentage fixations >3° from fixation cross	8.50% (7.27%)	5.33% (4.38%)
Saccade latency (msec)		
Gaze left	239 (51)	256 (62)
Gaze right	262 (63)	264 (67)
Item left	277 (68)	266 (64)
Item right	274 (64)	264 (58)
Saccade accuracy[c] **(visual degrees)**		
Gaze left	1.23° (.28°)	1.43° (.65°)
Gaze right	1.27° (.32°)	1.49° (.52°)
Item left	1.51° (.32°)	1.36° (.31°)
Item right	1.49° (.32°)	1.42° (.37°)

[a] The first half of the trial lasts from the appearance of the memory item until the shift of the fixation cross.
[b] The second half of the trial lasts from the appearance of the memory probe until the answer is entered.
[c] Saccade accuracy is defined as the absolute distance in visual degrees between the saccade endpoint and fixation cross.

subjects. Patients with right PPC damage showed worse trans-saccadic memory performance compared to healthy control subjects, regardless of the direction of the gaze shift. When we compared performance of each individual stroke patient with the healthy control group, four out of nine stroke patients showed impaired trans-saccadic memory performance. These patients had larger lesions in the PPC compared to the patients who did not show a deficit.

None of the individual patients showed asymmetric trans-saccadic memory performance when making leftward versus rightward eye movements. As such, damage to the right PPC does not seem to lead to *gaze direction specific* impairments in trans-saccadic memory, but instead causes general memory impairments for spatial locations. One patient showed specific deficits when memory items were presented either left or right (after which an up/downward gaze shift had to be made), while not showing deficits when items were presented up or down (after which a left/rightward gaze shift had to be made). Two patients showed worse performance in all conditions when compared to the healthy control group. These findings could either relate to a general memory encoding deficit of spatial locations, or to excessive costs of eye movements on spatial memory. As we did not include a no-gaze condition, we cannot exclude one explanation over the other. One of the aforementioned patients showed even worse trans-saccadic memory for items that were presented left than for items that were presented right. A fourth patient showed this asymmetry as well, while showing normal performance in all other conditions. This asymmetry could be explained by reduced attention for the contralesional, left side, due to the lesion in the right hemisphere (as seen in hemispatial neglect), which could impair memory performance for the item in the left versus right visual field.

Our results are in contrast with those of Vuilleumier et al. (2007) and Russel et al. (2010). Their designs and main findings are summarized in Supplementary Table 5. In those studies, direction specific trans-saccadic memory deficits were reported, in addition to general trans-saccadic memory impairments. This could be explained by the differences in experimental design. In the experiment of Vuilleumier et al. (2007), the first target appeared equally often in the ipsilesional or contralesional field, so possible attention effects (i.e.,

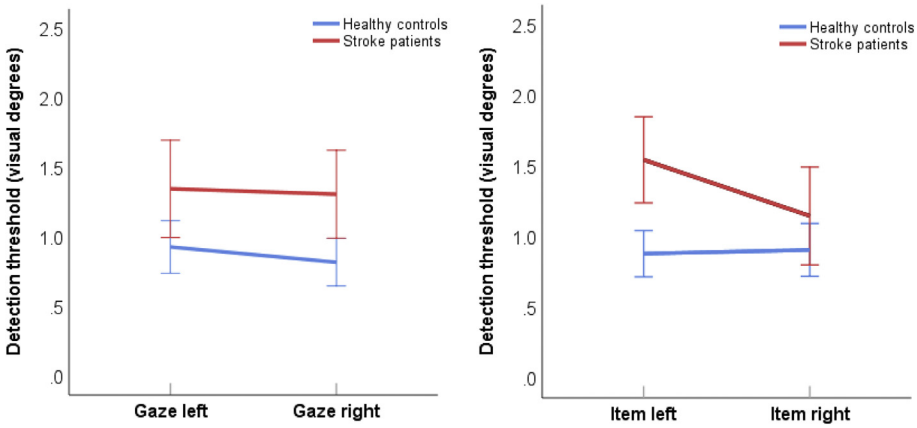

Fig. 3 – Mean discrimination threshold scores for the gaze left/gaze right conditions (left panel) and the item left/item right conditions (right panel), split for group. Error bars indicate 95% confidence intervals.

Table 3 – Model Comparison for the gaze left/gaze right conditions.

Models	P (M)	P (M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	.200	.104	.462	1.000	
group	.200	.392	2.577	3.785	2.882
Gaze	.200	.077	.332	.741	.859
Group + Gaze	.200	.295	1.671	2.847	3.036
Group + Gaze + Group*Gaze	.200	.133	.615	1.288	9.603

Note. All models include subject.

Table 4 – Analysis of Effects for the gaze left/gaze right conditions.

Effects	P (incl)	P (incl|data)	$BF_{Inclusion}$
Group	.400	.686	3.809
Gaze	.400	.371	.750
Group*Gaze	.200	.133	.453

Note. Compares models that contain the effect to equivalent models stripped of the effect. Higher-order interactions are excluded.

Table 5 – Model Comparison for the item left/item right conditions.

Models	P(M)	P (M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	.200	.072	.308	1.000	
group	.200	.484	3.753	6.767	.697
Item	.200	.022	.090	.308	.967
Group + Item	.200	.147	.688	2.052	1.198
Group + Item + Group*Item	.200	.276	1.521	3.852	2.875

Note. All models include subject.

Table 6 – Analysis of Effects for the item left/item right conditions.

Effects	P (incl)	P (incl|data)	$BF_{Inclusion}$
Group	.400	.631	6.742
Item	.400	.169	.304
Group*Item	.200	.276	1.877

Note. Compares models that contain the effect to equivalent models stripped of the effect. Higher-order interactions are excluded.

reduced attention for contralesional stimuli, and thereby decreased trans-saccadic memory) were balanced between conditions. However, in trials in which an eye movement had to be made towards the right, ipsilesional field, the to-be-remembered location *always* shifted to a location contralesional from the second fixation location. If patients had reduced attention for the contralesional field, this could potentially explain the reduced performance for trans-saccadic memory. Vuilleumier et al. (2007) tried to resolve this issue by comparing performance for ipsilesional versus contralesional items in a no-remapping condition. This condition, however, is different compared to a condition in which eye movements had to be made, as the execution of a saccade results in a dual-task given the obligatory involvement of working memory in the programming of saccades (Schut, Van der Stoep, Postma, & Van der Stigchel, 2017; Van der Stigchel & Hollingworth, 2018). More importantly, in both studies (Russell et al., 2010; Vuilleumier et al., 2007), participants had to make two eye movements in each trial: one toward the ipsilesional or contralesional side, and one back to central fixation. Therefore, it remains unclear whether their finding regarding direction specific impaired trans-saccadic memory is due to the initial rightward gaze shift or the secondary, leftward gaze shift.

In addition, we included patients based on lesion location (i.e., right PPC) instead of the presence of neglect (Vuilleumier et al., 2007) or apraxia (Russel et al., 2010), both disorders that by definition involve difficulty maintaining or updating an accurate representation of the spatial array. Even though trans-saccadic deficits in neglect have been linked to lesions in the right parietal lobe (Saj, Verdon, Hauert, & Vuilleumier, 2018), damage to white matter tracts in the parietal lobe or other cortical regions could potentially contribute to trans-saccadic impairments (Saj et al., 2018). Possibly, the direction specific deficits in the aforementioned studies were

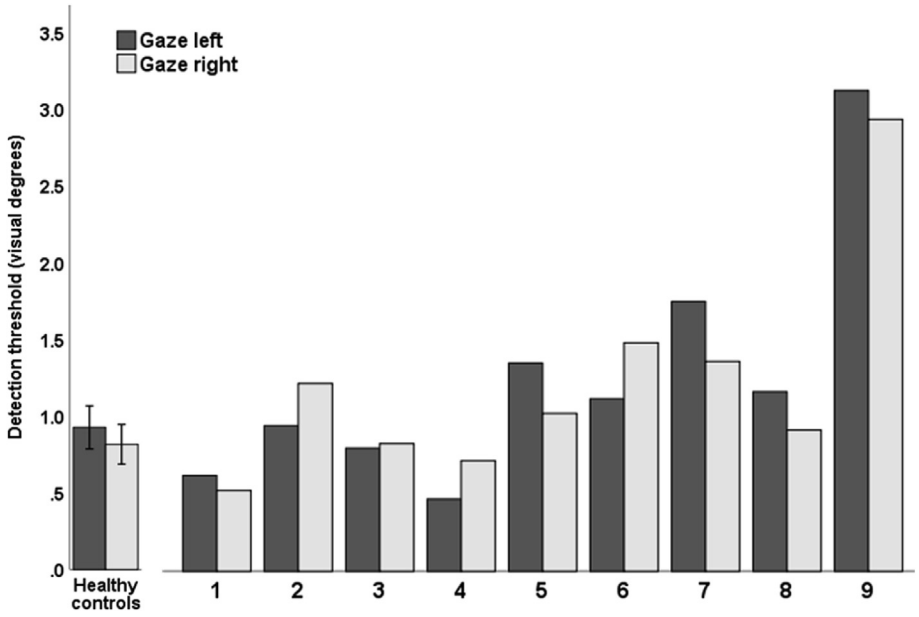

Fig. 4 – Mean discrimination threshold scores for the gaze left/gaze right conditions of the healthy control group (left panel), and the individual stroke patients (right panel). Error bars indicate 95% confidence intervals.

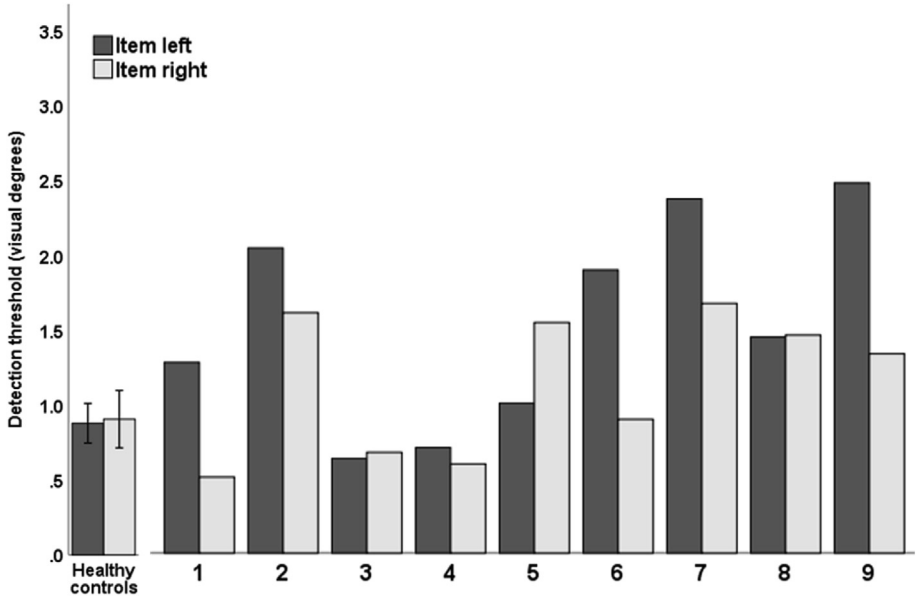

Fig. 5 – Mean discrimination threshold scores for the item left/item right conditions of the healthy control group (left panel), and the individual stroke patients (right panel). Error bars indicate 95% confidence intervals.

driven by patients with damage in other (combinations of) brain areas that are important for direction specific spatial remapping, and the PPC did not play a crucial role.

Finally, when we convert the *t*-values of the crucial comparisons of the previous studies (Russell et al., 2010; Vuilleumier et al., 2007) into Bayes factors (Supplementary Table 5), the evidence in the study of Vuilleumier et al. (2007) was moderate ($BF_{10} = 3.38$ and $BF_{10} = 4.96$), and in the study of Russell et al. (2010) would not be considered positive evidence ($BF_{10} = 2.19$). We should, therefore, be cautious with overinterpretation of these results.

It should be noted that to test spatial updating of retinotopic information in humans, the "double-step saccade paradigm" is typically used, which is different from the trans-saccadic memory task that was used here. In the double-step saccade paradigm two visual targets are quickly flashed successively in the periphery, after which the participant has to make a sequence of two saccades in the dark to the remembered target locations in the order they appeared (e.g., Heide, Blankenburg, Zimmermann, & Kömpf, 1995; Pisella et al., 2011). Both targets are extinguished before the first saccade is completed. Thus, the second saccade relies on the memorized location of the

Table 7 – Individual mean scores of the patients (upper rows) and the mean score of the healthy controls (lower row, mean ± SD). Bayesian point estimates (i.e., the percentage of the control population that would obtain a score lower than the patient) and credible limits are provided.

ID	Gaze left/gaze right			Item left/item right		
	Mean score	Point estimate	Credible limit	Mean score	Point estimate	Credible limit
1	.61	21.50%	11.22%–34.48%	.90	51.00%	37.22%–64.68%
2	1.15	80.21%	67.50%–90.02%	**1.83**[a]	**98.95%**	**95.97% to 99.93%**
3	.87	49.99%	36.21%–63.81%	.66	27.78%	16.30%–41.45%
4	.63	23.29%	12.62%–36.49%	.65	26.93%	15.55%–40.48%
5	1.27	88.60%	77.98%–95.68%	1.28	83.98%	72.02%–92.73%
6	1.39	93.97%	85.93%–98.44%	1.40	90.15%	80.13%–96.56%
7	**1.66**[a]	**98.93%**	**96.02% to 99.93%**	**2.03**[a]	**99.70%**	**98.48% to 99.99%**
8	1.11	76.70%	63.56%–87.35%	1.46	92.49%	83.59%–97.77%
9	**3.23**[a]	**100%**	**100%**	**1.91**[a]	**99.35%**	**97.22% to 99.97%**
Controls	.87 ± .32			.89 ± .38		

The mean scores, point estimates and credible limits that differ significantly are depicted in bold.
[a] The mean score significantly differs from the mean score in the healthy control group, using Bayesian Crawford statistics.

Table 8 – Individual difference scores of the patients (upper rows) and the mean difference score of the healthy controls (lower row, mean ± SD). Bayesian point estimates (i.e., the percentage of the control population that would obtain a score lower than the patient) and credible limits are provided.

ID	Gaze left/gaze right			Item left/item right		
	Difference score	Point estimate	Credible limit	Difference score	Point estimate	Credible limit
1	.10	49.36%	35.63%–63.21%	−.77	5.72%	1.42%–13.53%
2	−.29	14.63%	6.35%–26.21%	−.43	17.72%	8.45%–30.09%
3	−.03	41.76%	28.60%–55.73%	.04	51.09%	37.34%–64.85%
4	−.27	16.23%	7.38%–28.34%	−.11	39.14%	26.12%–53.13%
5	.35	73.41%	59.88%–84.77%	.54	84.91%	73.17%–93.39%
6	−.39	9.73%	3.35%–19.70%	**−1.00**[a]	**2.22%**	.28%–6.88%
7	.41	78.89%	66.06%–89.03%	−.70	7.38%	2.13%–16.20%
8	.26	65.89%	51.90%–78.37%	.01	49.01%	35.38%–62.83%
9	.20	59.30%	45.37%–72.40%	**−1.15**[a]	**1.14%**	.08%–4.24%
Controls	.11 ± .37			.03 ± .48		

The mean scores, point estimates and credible limits that differ significantly are depicted in bold.
[a] The difference score significantly differs from the mean difference score in the healthy control group, using Bayesian Crawford statistics.

second target, updated with respect to the new eye position after the first saccade to the first target. Direction specific deficits have been shown in PPC patients with or without neglect, showing specific impairments when the first target is flashed into the contralesional field and the second target in the ipsilesional field. The direction specific predictions for PPC patients in the current study (i.e., worse trans-saccadic memory after rightward compared to leftward gaze shifts) are opposite to the double-step results (i.e., failing to correctly saccade to a second rightward target after a first leftward gaze shift). In the double-step paradigm, the impairment might result from a loss of the anticipatory motor efference-copy (Duhamel, Goldberg, Fitzgibbon, Sirigu, & Grafman, 1992; Heide & Kömpf, 1998), which is not needed in the explicit perceptual task that was used here. Vuilleumier et al. (2007) argued that both tasks tap into different mechanisms with different timings, potentially recruiting different neural circuits. Furthermore, Rath-Wilson and Guitton (2015) questioned whether patients with damage in the parietal lobe show problems with spatial updating as measured with the double-step saccade paradigm, as findings could possibly be explained by contralesional visual processing deficits due to parietal lesions. When controlled for deficits other than corollary discharge, left and right parietal patients showed evidence of using corollary discharges for saccades in the ipsilesional and contralesional directions. This suggests that corollary discharges for left and right saccades are available to each cortical hemisphere (Rath-Wilson & Guitton, 2015).

For the stroke patients in the current study, more trials had to be excluded when the memory item was presented in the left versus right hemifield, indicating that they made more erroneous eye movements towards this memory item and were less able to inhibit their saccades (i.e., oculomotor inhibition). In addition, four stroke patients were not able to suppress their eye movements at all and were excluded from analyses (see Supplementary Table 2). This is possibly due to brain damage in areas that are associated with (lateralized) oculomotor inhibition, such as the dorsolateral prefrontal cortex and the frontal eye fields (Ettinger et al., 2008; Gaymard, Ploner, Rivaud, Vermersch, & Pierrot-Deseilligny, 1998; Pierrot-deseilligny, Milea, & Mu, 2004; Van der Stigchel, van Koningsbruggen, Nijboer, List, & Rafal, 2012). This impaired oculomotor inhibition could have caused a bias in the sample of stroke patients that was eventually included in our analyses. If brain areas that are important for oculomotor inhibition would also be associated with direction specific trans-saccadic memory (e.g., the FEF), we would not be able to

observe these direction specific deficits as patients with damage in those areas were not able to perform the task in the first place.

To conclude, patients with right PPC damage showed worse trans-saccadic memory performance for spatial locations compared to healthy control subjects, regardless of the direction of the gaze or the initial location of the memory item. This could either relate to a general spatial memory encoding deficit, or to excessive costs of eye movements on spatial memory. At individual level, four of nine stroke patients showed impaired trans-saccadic memory, which was related to the size of the PPC lesion. None of the patients showed a direction specific deficit after leftward versus rightward saccades, whereas two patients showed worse performance for items in the left versus right visual field. Thus, damage in the right PPC did not lead to gaze direction specific impairments in trans-saccadic memory, but instead caused more general spatial memory impairments.

Declaration of interest

The authors declare that they have no conflict of interest.

Open practices

The study in this article earned Open Materials and Open Data badges for transparent practices. Materials and data for the study are available at https://osf.io/xqvkb/.

CRediT authorship contribution statement

Antonia F. Ten Brink: Conceptualization, Methodology, Software, Formal analysis, Investigation, Data curation, Writing - original draft, Visualization. **Jasper H. Fabius:** Conceptualization, Methodology, Software, Formal analysis, Writing - review & editing. **Nick A. Weaver:** Methodology, Software, Formal analysis, Writing - review & editing. **Tanja C.W. Nijboer:** Resources, Writing - review & editing. **Stefan Van der Stigchel:** Conceptualization, Methodology, Resources, Writing - review & editing, Supervision, Project administration, Funding acquisition.

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Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cortex.2019.06.006.

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