

More (corrective) consecutive saccades after a lesion to the posterior parietal cortex

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Abstract

To reach a target, primary saccades (S1s) are often followed by (corrective) consecutive saccades (S2, and potentially S3, S4, S5), which are based on retinal and extraretinal feedback. Processing these extraretinal signals was found to be significantly impaired by lesions to the posterior parietal cortex (PPC). Recent studies, however, added a more nuanced view to the role of the PPC, where patients with PPC lesions still used extraretinal signals for S2s and perceptual judgements (Fabius et al., 2020; Rath-Wilson & Guitton, 2015). Hence, it seems that a PPC lesion is not disrupting extraretinal processing per se. Yet, a lesion might still result in *less reliable processing* of extraretinal signals. Here, we investigated whether this lower reliability manifests as *decreased* or *delayed* S2 initiation. Patients with PPC lesions ($n = 7$) and controls ($n = 26$) performed a prosaccade task where the target either remained visible or was removed after S1 onset. When S1 is removed, accurate S2s (corrections of S1 error) rely solely on extraretinal signals. We analysed S2 *quantity* and *timing* using linear mixed-effects modelling and additive hazards analyses. Patients demonstrated slower S1 execution and lower S1 amplitudes than controls, but their S2s still compensated the S1 undershoot, also when they only relied on extraretinal information. Surprisingly, patients showed an increased amount of S2s. This deviation from control behaviour can be seen as suboptimal, but given the decreased accuracy of the primary saccade, it could be optimal for patients to employ more (corrective) consecutive saccades to overcome this inaccuracy.

KEYWORDS

additive hazards analysis, extraretinal signals, oculomotor control, secondary saccades

Abbreviations: AG, angular gyrus; CT, computer tomography; CVA, cerebrovascular accident; GLMM, generalized linear mixed-effect model; IPG, inferior parietal gyrus; LMM, linear mixed-effect model; MRI, magnetic resonance image; PPC, posterior parietal cortex; SPL, superior parietal lobule; SMG, supramarginal gyrus; SRT, saccadic reaction time (latency); S1(s), primary saccade(s); S2(s), (corrective) consecutive saccade(s); UMC, university medical centre.

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1 | INTRODUCTION

Imagine yourself in a dark forest. Suddenly an alarm light flickers between the trees. Your eyes move towards the estimated position of the light, but when your eyes land the light is nowhere to be found. It is windy and a waving tree is occluding it. Without retinal feedback, you need to rely on some internally evoked extraretinal signal to monitor whether your eyes are at the right spot and to make a consecutive saccade to reach the target.

Two error signals contribute to the execution of a (corrective) consecutive saccade (S2, and potentially S3, S4, S5). First, *retinal* information signals whether the target projection is too far off the fovea. Alternatively, *extraretinal* information (i.e., corollary discharge, efference copy or eye proprioception) facilitates an internally evoked error alert that signals the discrepancy between the motor record of the planned saccade and the executed saccade (Guthrie et al., 1983).

Classical lesion studies using the double-step saccade paradigm proposed that neurons in the posterior parietal cortex (PPC) carry extraretinal information about this motor command of the saccade-to-be-made (Duhamel et al., 1992; Heide et al., 1995). In this task, two targets are flashed in rapid succession and need to be foveated sequentially. As the targets have already disappeared by the time the first saccade is initiated, calculations of the redirection are reliant on extraretinal signals. After a lesion to the PPC, redirecting towards contralesionally presented targets was impaired. The PPC was therefore considered of great importance in extraretinal signal processing; damage would interfere with the regular generation of sequential saccades.

However, the impairment that a PPC lesion would bring to processing extraretinal signals was recently nuanced (Rath-Wilson & Guitton, 2015). The authors suggested that subtler changes in saccadic behaviour might have been missed because of too strict cut-offs, data filtering and the paradigm (e.g., double-step saccade task) employed. They found that patients were still able to reach a second target in a double-step task, but that multiple saccades (more than two) were needed. This suggests that a lesion to the PPC is not disrupting extraretinal processing per se, yet, it is still possible that this area is needed to *optimally* use extraretinal signals, with damage to the PPC introducing difficulties in both perception and action.

Results of our previous study indicated that patients with a lesion in the PPC were indeed still able to use extraretinal signals to make perceptual judgements (Fabius et al., 2020). Being only slightly less accurate than controls, patient performance showed that a PPC lesion resulted in a suboptimal rather than a completely

distorted performance in the perceptual domain. But what about motoric consequences?

The overarching aim here is to investigate whether patients with a lesion to the right PPC show suboptimal generation of consecutive saccades (here, specifically, the second one; S2) in terms of quantity (i.e., fewer) and timing (i.e., slower) when processing extraretinal signals as compared with controls. Potential impairments might further differ for targets presented to the contra- (left) or ipsilesional (right) hemifield because of hemispheric attentional asymmetries. To investigate this, we use a simple visually driven localization task that evokes naturally occurring consecutive saccades (i.e., S2, S3 and so forth), and discards the complex nature of double-step paradigms used in earlier studies assessing the effects of a PPC lesion. Inspired by Ohl and Kliegl (2016), we complement regular analysis of saccadic behaviour with adopting an additive hazards analysis (Aalen, 1980), which allows to assess subtle, time-dependent effects that are often overlooked. If a lesion to the PPC would impair extraretinal signal processing (to any degree), patients should exhibit a decrease in – but not necessarily an absence of – S2 generation to make up for a primary saccade error when there are only extraretinal signals to rely on, and/or an increase in the time it takes to process extraretinal signals to initiate an S2. Patients that participated were the same as in our previous study (Fabius et al., 2020) in which we showed that these patients can use extraretinal signals in perceptual judgements. Now, we take a closer look at characteristics of their oculomotor behaviour.

2 | MATERIALS AND METHODS

2.1 | Subjects

Twelve patients in the chronic phase post-stroke onset (>4 months) with chronic stroke damage (see same recruitment description also in Fabius et al., 2020) and 26 healthy control subjects participated. These sample sizes were determined as the maximum possible given available resources. Patients were invited for participation after inspection of their clinical imaging data (MRI or CT scan) from existing databases at the UMC Utrecht that are available for scientific purposes. This database contains patients who had been admitted because of (suspected) cerebrovascular problems. Patients included in this database provided informed consent to have their imaging data be inspected for scientific purposes. Patients were included when there appeared to be a lesion to the right PPC. In practice, the right PPC was defined as lesions found A) posterior to the

postcentral gyrus, B) dorsal to the posterior horn of the right lateral ventricle and C) not posterior to the parieto-occipital sulcus. Later, lesion locations were determined exactly by an expert neurologist. Patients were not included when they had exhibited clinical signs of visual field defects, a history of substance abuse or an inability to understand the task instructions.

2.2 | Experimental setup

Visual stimuli were presented on an Asus ROG Swift PG278Q computer monitor (27 in., 60.1×34.0 cm, 120 Hz, 2560×1440 px) in a darkened room. Participants were seated in a chair with their head supported by a chin and forehead rest in order to facilitate eye-tracking and to maintain a fixed viewing distance of 70 cm between the projection screen and the eyes. An EyeLink1000 eye-tracker (SR Research Ltd.) was used to measure the left eye gaze position with a sampling frequency of 1000 Hz. The EyeLink1000 was calibrated using a 9-point grid at the beginning of the experimental blocks and recalibrated when needed. As participants needed to fixate for a minimum of 250 ms for a target to appear, indirectly this served as a drift check. If any blinks occurred during this time frame, the clock was set to 0 again.

Stimuli were generated using Matlab R2015a (Mathworks; MATLAB, 2015) and the Psych Toolbox (version 3.0.11; Brainard, 1997; Kleiner et al., 2007). Target stimuli consisted of red dots (radius = 0.1°) presented at 15° (with a jitter of $\pm 1^\circ$ on x- or y-axis) to the left or the right side of a fixation stimulus (red dot, radius = 0.1°). The fixation stimulus could be placed either left ($-15 \pm 1^\circ$) from the middle, right ($15 \pm 1^\circ$) from the middle, or in the middle ($0 \pm 1^\circ$) of the screen. Each trial's initial fixation point was where the previous trial ended. This implies that for trials starting with fixation on the left side of the screen, the target stimulus would always appear to the right and vice versa. Target stimuli were jittered with 1° to reduce predictability of the vector length. The small target stimulus size was chosen to ascertain that foveal projection of the object was needed to obtain an accurate representation.

2.3 | Task

Participants were explicitly instructed to fixate at the fixation stimulus and redirect their gaze as accurately as possible to the presented target stimulus (see Figure 1). Participants were asked to keep fixating on the target stimulus if it was still available after saccade execution (retinal feedback; ON), and to fixate at the perceived

location of the earlier presented target stimulus whenever retinal feedback was absent (OFF). Another trial type was present in the experiment (displacement of the target stimulus during saccade execution), but the current study only focuses on the former two (ON and OFF) given that these are most relevant for the research question.

One block (48 trials) of practice trials was completed to familiarize the subjects with the task. The experiment consisted of 32 blocks (third condition included) with 48 trials each. A block lasted around 2 min. Within blocks, trials were counterbalanced and randomized for retinal feedback condition (ON, OFF) and target side (Left, Right). Participants were given a break between every block. During breaks, a white screen with increasing luminance was presented and the room was illuminated. This procedure was implemented to prevent the visual system from adapting to the darkened environment, which takes approximately 5 to 10 min (Cao, 2017). Adaptation to the dark should be prevented, as this leads to a shift in the projection location of the retina that gives the most accurate perception, meaning that after a while, foveal projection would not be advantageous and S2s might not be employed.

2.4 | Data analysis

2.4.1 | Data preparation

A saccade detection algorithm was applied (Nyström & Holmqvist, 2010). This allowed for separation of the primary and consecutive saccades. For the algorithm, we used a minimum saccade duration of 10 ms and a minimum of interval of 40 ms between saccades. Only the primary (S1) and secondary saccade (S2) were investigated in the current study.

2.4.2 | Exclusion criteria

Over 33 participants, 50,688 trials were planned to be collected. Six hundred twenty-four trials were missing because of the early termination of the experiment by two patients (23 and 28 blocks completed, respectively) as a result of fatigue. Valid trials were intentionally quite liberally defined as trials in which the S1 (1) had an amplitude of $\geq 1^\circ$ and $< 50^\circ$ (in line with the possibility that patients need multiple small saccades in double-step paradigms to reach the target), (2) was in the target direction, (3) had a latency ≥ 80 ms and < 1000 ms and (4) did not reach the target before target stimulus offset when extraretinal feedback should not be present (see Supplementary Figure S1). Thereafter, trials for the

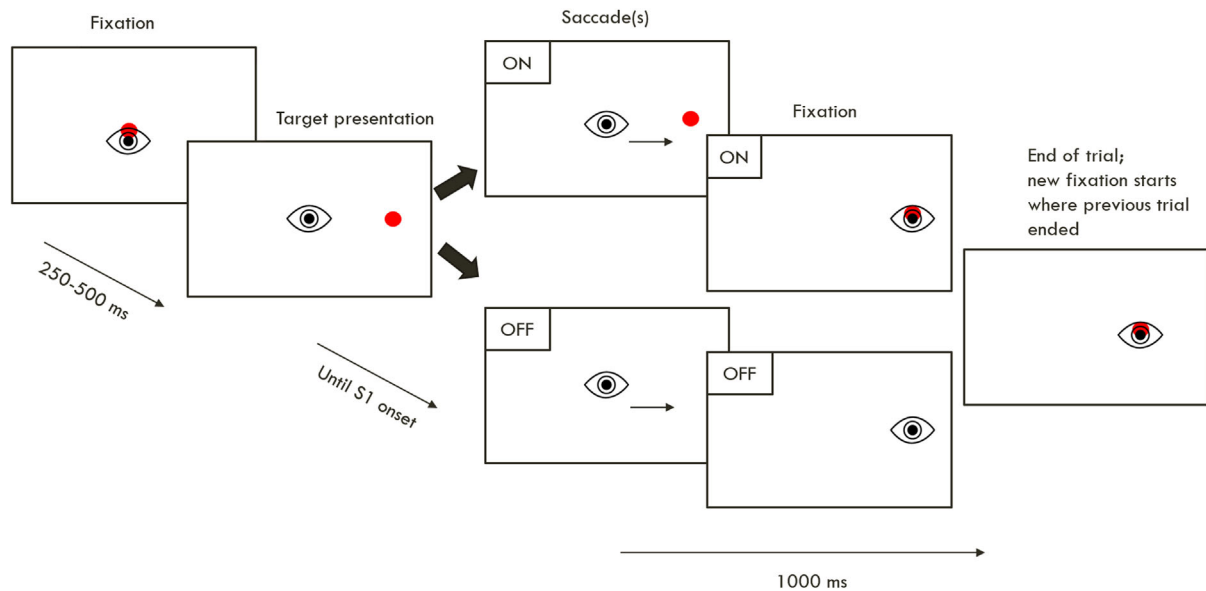


FIGURE 1 The fixation stimulus was presented gaze-contingently and participants were required to maintain stable fixation for a random duration between 250 and 500 ms. Fixation target offset was equal to target onset. Target stimulus presentation duration depended on S1 initiation and condition: ON) targets remained visible until the end of the trial (1000 ms), OFF) targets disappeared directly after S1 initiation. The trial ended 1000 ms after S1 was detected. Trial conclusion lasted for 500 ms.

displacement condition (as described above) were filtered out. After exclusion and filtering, 24,517 trials were available for analysis.

2.4.3 | Analyses rationale

Instead of using traditional methods for data-analysis (e.g., analysis of variance (ANOVA)), we used sophisticated statistical models: generalized linear mixed models (GLMM), linear mixed-effect models (LMM) and additive hazard models. We used these models to deal with unequal group sizes (26 controls and only seven patients) and unequal amounts of datapoints within these groups, as not all trials were valid or yielded good eye-tracking data quality. LMM (used for S1 and S2 latency and accuracy, and S1 peak velocity analysis) and GLMM (used for S2 occurrence analysis), take these obstacles into account by weighing factors according to the number of observations. Additionally, they take into account individual differences and fluctuations across conditions. Although advanced modelling is sometimes regarded complicated and unnecessary, they are a powerful tool in analysis. For the analysis of proportions of categorical data specifically (hence, S2 occurrence), GLMMs respect the nature of the outcome variable without the need to transform and maintain higher power (Jaeger, 2008). Because of the complexity of these models, we decided to only run them for the main variables of interest, which

are described in the text body. Other basic outcomes are given in a descriptive table.

Furthermore, we implemented a time-course analysis. This type of analysis allows to study the time-dependent probability of the occurrence of a specific event based on certain factors. Translated to oculomotor behaviour, we are interested in the probability of S2 to occur depending on an interplay of factors (e.g., patient S2s when S1 error was large but no visual feedback was available etc.). Interestingly, the additive hazards analysis (Aalen, 1980) is able to illustrate *when* a certain factor exerts its influence. Thereby, as Ohl and Kliegl (2016) rightly put forward, this analysis adds nuance to the analysis of S2 generation, as it takes into account trials where no S2s are generated – which are kicked out of the equation when analysis concerns mere means or medians, biasing estimate results – and it allows to assess time-dependent effects that are normally lost. Additive hazard analyses could reveal that certain factors (e.g., extraretinal signals) are still at play after a lesion but have a *delayed* – suboptimal – impact on S2 generation in patients as compared with healthy controls. Instead of only offering S2 generation proportions, it adds the elegant display of S2 generation time-course and underlying factors — both with and without a lesion to the PPC.

We are convinced that these analyses methods – albeit complex and less straightforward – fit our data well and yield outcomes that are robust and not detectable when more traditional analyses are used.

2.4.4 | Primary saccades (S1)

As a first exploration, we analysed S1 characteristics (latency, accuracy, velocity) and possible differences between groups, as we were primarily interested in detecting potential deviant eye movement behaviour in patients. Further, as all patients had damage to the right parietal cortex, potential group differences in target localization between hemifields might arise as right parietal damage is often associated with neglect for the contralesional hemifield. Although patients with neglect were excluded from participation, damage to the right PPC might still have led to attentional asymmetries that could impact S1 in this specific direction in terms of both speed and accuracy. We therefore included the target presentation side (Left, Right) in the equation.

Regarding the target visibility condition (ON/OFF), another reasoning applies. S1s in our experiment were *always* evoked by a visual target, and target visibility was only manipulated *after* onset of the S1. Therefore, no differences were expected between trials in which the target stayed on screen (Condition ON) or was removed after initiation of S1 (Condition OFF), but we have still included those to make sure potential effects would not drive S2 characteristics that were analysed later on.

Primary saccade latency

S1 latency was calculated as the difference in time between target onset and saccade onset in milliseconds. A log transformation was applied to S1 latency to overcome skewness. Model comparisons were performed for several linear mixed-effect models (LMM; Singmann & Kellen, 2019) including effects of factors Group (control, patient), Target Side (left, right) and Condition (ON, OFF) and by-subject random intercepts and slopes (see Supplementary Table S1). All LMM were fit and analysed using the lmer function (lme4 package; Bates et al., 2015) in R (R Core Team, 2017). A likelihood ratio test (ANOVA function of the ltm package; Rizopoulos, 2018) was used for model comparison to investigate which model outperformed the others in explaining the data (see Supplementary Table S2 for model comparison outcomes). χ^2 with $\alpha < .05$ and lowest AIC/BIC values were leading in deciding on the most informative model. In the absence of an lmer *p*-value outcome, a value of $t > 2.0$ was used to judge significance of factors within the chosen model. To make the outcomes more intuitive, S1 latency log values can be de-transformed by using its inverse following the formula:

$$SRT = \exp(L) \quad (1)$$

with *SRT* denoting saccadic reaction time (latency) in milliseconds and *L* denoting the log estimate.

Primary saccade accuracy

Among many ways to express saccadic accuracy (e.g., amplitude, numerical or absolute error in degrees visual angle) is saccadic gain. Here, S1 accuracy was expressed as the value of log transformed S1 gain, calculated from the vector that was needed to reach the target (distance between saccade starting point and target) compared with the vector actually executed (distance between saccade starting point and saccade landing point). The absolute value of log transformed S1 gain indicates the magnitude of the S1 landing error from the target position (e.g., two times the desired distance, half the desired distance). As saccadic gain does not only take the saccade endpoint but also the starting position into account, it yields a more fine-grained measure of saccadic behaviour than, say, subtracting the saccade endpoint from the target location.

We also coded a variable Shoot to indicate whether the saccade over- or undershot the target. See 'Code and software' for the available code on OSF for the (computational) transformations. The same model comparison procedure (LMM) and factors (Group, Target Side, Condition) were used for accuracy as for latency (see Supplementary Table S3 and S4).

Primary saccade peak velocity

S1 peak velocity was calculated as the maximum velocity (change in distance/change in time). The same model comparison procedure (LMM) and factors (Group, Target Side, Condition) were used for velocity as for accuracy and latency (see Supplementary Table S5 and S6).

2.4.5 | Secondary and subsequent saccades (S2, S3, S4, S5)

Pre-processing

Secondary saccades (S2s) were defined as all saccades that were initiated within 1 s after the offset of S1. Importantly, we did not only take into account corrective secondary saccades but also all secondary saccades, hence, the term 'secondary' over 'corrective'. S3s, S4 and S5s were defined as all saccades that were initiated within 1 s after the offset of S2, S3 and S4, respectively.

Secondary and subsequent saccade proportions

S2, S3, S4 and S5 proportions were calculated as the amount of trials in which a S2, S3, S4 or S5 occurred divided by the total amount of trials.

Secondary saccade latency

S2 latency was calculated as the difference in time between primary saccade offset and secondary saccade onset in milliseconds. The analysis procedure for S2 latency was the same as for S1 latency, with factors Group, Target Side and Condition taken into account (see Supplementary Table S7 and S8).

Secondary saccade accuracy

Like S1 accuracy, S2 accuracy was expressed as the value of log transformed S2 gain, calculated from the vector that was needed to reach the target (distance between secondary saccade starting point and target) compared with the vector actually executed (distance between secondary saccade starting point and saccade landing point). Contrary to other expressions of saccadic accuracy (i.e., numerical or absolute error in degrees visual angle) saccadic gain does not only take into account the saccade endpoint but also the starting position. Therefore, it deals with unequal starting points of S2s given the variability in S1 landing points and yields a more fine-grained measure of saccadic behaviour than accuracy expressed as mere distance from the target location.

For analysis, the same model comparison procedure (LMM) and factors (Group, Target Side, Condition) were used as for S2 latency (see Supplementary Table S9 and S10).

Secondary saccade incidence

Factors contributing to secondary saccade initiation. To investigate which factors underlie the incidence of secondary saccades, we fitted logistic linear mixed-effect models to our data of the binomial variable S2 present (0 = absent, 1 = present) with various configurations of the fixed factors Group (Control, Patient), Condition (ON, OFF), Target Side (Left, Right), S1 Error (continuous) and S1 Shoot (under-, overshoot; Models 1–6, see Supplementary Table S11).

As the error tolerance is found to be lower when retinal feedback is present (Tian et al., 2013), the likelihood of an S2 to occur was expected to be higher with retinal feedback as compared with when this feedback is absent. It is particularly interesting to compare S2 occurrence for controls and patients over the different feedback conditions. As a reflection of the hypothesized suboptimal functioning of extraretinal signals, patients were expected to display the same likelihood of an S2 to occur in the *presence* of retinal feedback, but a smaller S2 occurrence in the *absence* of retinal feedback as compared with controls. Additionally, we expected more S2s to occur with increasing S1 error and when S1 undershot the target as compared with when it overshoot the target (Ohl & Kliegl, 2016). Based on the

above, we fitted a model with hypothesized interaction effects of Group*Condition, Condition*S1 error and three-way interactions of Group*Condition*S1 error and Group*Condition*Target Side, while also controlling for the random effect of Subject ID (Model 7, see Supplementary Table S11).

All GLMM were fitted and analysed using the glmer function of the lme4 package (Bates et al., 2015) in R (R Core Team, 2017). A likelihood ratio test (ANOVA function of the ltm package; Rizopoulos, 2018) was used to compare hierarchical models and to investigate which model performed best in explaining our data (see Supplementary Table S12 for model comparison outcomes). χ^2 with $\alpha < .05$ and lowest AIC/BIC values were leading in deciding on the most informative model. After fitting the model, the significance of factors was judged by a value of $t > 2.0$ as glmer does not provide p -values.

Speed–accuracy trade-off. We analysed the relationship between S1 latency and the probability of S2 execution with a linear regression for both patients and controls to observe a potential speed–accuracy trade-off. Speed–accuracy trade-offs are widely described in psychophysical research, where slower responses lead to higher accuracy while quicker responses yield less accurate responses. Here, slower S1s might yield more accurate responses, reducing the need to execute an S2. Vice versa, quicker S1s might yield more inaccurate responses, and therefore a higher need – and thus probability – to employ a corrective saccade.

Survival analysis of secondary saccade time course

S2 latency was calculated as the difference in time between S1 offset and S2 onset in milliseconds. With information about S2 occurrence and S2 latency, it is possible to analyse the time course of S2 generation by using survival analyses. Survival analyses study the occurrence of a specific event in a time range t_{start} to t_{end} . Here, we were interested in the ‘survival’ of S1 in the time range of a trial: if an S2 was made, S1 was not sufficient to ‘survive’ the trial. The question is what factors contribute to the generation of an S2 both without and after a lesion to the PPC.

Inspired by Ohl and Kliegl (2016), Aalen’s additive hazards model (Aalen, 1980) was used. This model can uncover time-dependent effects of certain factors on S2 generation. In other words: this model illustrates *at which point(s) in time a specific factor is promoting the execution of a secondary saccade most*. The model allows for the inclusion of trials in which *no* S2 was generated, and, thus, in which S1 ‘survived’.

Statistical analyses were performed using the aalen (Surv) function of the timereg package (Scheike et al., 2010) in the R environment. The survival analysis was performed for both groups separately. We included the

factors Target Side (Left, Right), Condition (ON, OFF), S1 Error (continuous) and Shoot (under-, overshoot). Interactions of S1 Error*Shoot, S1 Error*Condition and Shoot*Condition were also included in the model (see Supplementary Table S13). Condition was included to explore the speculated differential effect of Condition between patients and healthy controls. S1 Error and S1 Shoot were included to replicate the findings of Ohl and Kliegl (2016). Target Side was included to explore the differences in timing for both sides across groups, as we expect S2 generation after a contralesional S1 to be suboptimal for patients, but not for controls. Subjects were included as a cluster variable. Goodness of Fit was assessed by computing the residuals in the Aalen analysis and subsequent resampling ($n = 1000$) of cumulative residuals (Ohl & Kliegl, 2016; Martinussen & Scheike, 2006, p. 154). After, the model was stripped down to the essential factors. Resampling ($n = 10,000$) provided p -values and tests for time-varying effects. A seed was set to make the analysis reproducible.

2.4.6 | Code and software

Raw and processed data (.edf, .mat, .asc and .csv files) and analysis scripts (R Core Team, 2017) are available on Open Science Framework: <https://osf.io/hua6t/>.

3 | RESULTS

3.1 | Demographics

Twelve patients were recruited to partake in the intra-saccadic displacement task (Fabius et al., 2020) and

were again invited for participation in the current study. Lesion characteristics of these 12 can be found in Supplementary Figure S2. Of these 12 patients, two were excluded because of a different lesion location (not PPC; patient B and E in Fabius et al., 2020) or because of an unavailable scan (and as such no certainty of lesion location; patient F in Fabius et al., 2020). Patient J and L did not partake in the current experiment.

Seven patients with chronic right parietal damage following stroke (>4 months, four male, $M = 58.3$ years, $SD = 11.61$ years, range 41–76) were included for further analysis in the current study. Twenty-six healthy controls (nine male, $M = 51.2$, $SD = 10.6$, range 22–66) participated. See Table 1 for the resulting demographic data of all patients and a summary of the healthy controls. All participants were without known visual field deficits and had normal or corrected to normal vision. Participants gave written informed consent prior to the start of the experiment in accordance with the Declaration of Helsinki and Utrecht University and Medical Research Ethics Committee requirements.

3.2 | Primary saccade performance

3.2.1 | Primary saccade latency

Table 2 displays S1 latency values split on group and condition aggregated by median. A linear mixed-effects model was fit to primary saccade latency (as described in Methods, see again Supplementary Tables S1 and S2). The model included main effects of fixed factors Group ($t = 2.06$) and Target Side ($t = 2.27$), concluding that for

TABLE 1 Participant demographics and lesion characteristics.

ID	Age	Sex	Modified Rankin scale (after 3 months)	Years since CVA	Scan	Lesion volume (ml)	Percentage damaged				
							PPC (54.1 ml)	SPL (14.3 ml)	IPG (10.4 ml)	SMG (16.0 ml)	AG (13.3 ml)
A (27)	65	F	3	4.43	MRI	187.6	47.08	0.08	43.53	93.18	42.14
C (29)	76	M	1	5.43	CT	48.2	25.47	54.79	20.51	0.04	28.29
D (30)	57	M	2	2.53	MRI	26.4	14.12	0	8.28	42.42	0
H (32)	63	F	2	3.48	CT	64.2	5.05	0	0	14.39	3.26
I (33)	41	F	1	5.92	MRI	47.5	12.97	0	0.80	20.80	27.05
K (34)	47	M	2	6.10	MRI	37.2	23.69	56.16	36.59	2.18	4.44
M (35)	59	M	2	0.34	MRI	6.6	4.77	0	5.71	0	14.88
Average	58.29										
Controls	51.24 [22, 66]										

Abbreviations: AG, angular gyrus; CT, computer tomography; CVA, cerebrovascular accident; IPG, inferior parietal gyrus; MRI, magnetic resonance image; PPC = posterior parietal cortex, with areas SMG, supramarginal gyrus; SPL, superior parietal lobule.

both hemifields, patients are slower (left: 234 ms, right: 243 ms) to initiate S1s than controls (left: 207 ms, right: 215 ms). No effect was found for factor Condition. See Figure 2 for a display of group and individual (median) values.

3.2.2 | Primary saccade accuracy

Table 2 displays S1 accuracy values split on group and condition. A linear mixed-effects model was fit to S1 accuracy expressed in gain (as described in Methods, see again Supplementary Tables S3 and S4). The model showed significant effects for Group ($t = -2.49$), where patients were less accurate than controls. No main effect for Target Side was found ($t = 0.47$), but there was a significant interaction between Group and Target Side ($t = 2.15$) with patients being less accurate than controls for targets presented to the left (i.e., contralesional). This difference between groups was absent for rightward saccades.

Additionally, a surprising effect of Condition was found ($t = 4.34$): in the condition where the target remained visible on the screen during and after the saccade, participants' undershoot (and therefore, their error) was more pronounced than when the target was removed during the saccade. Although the effect is significant, it only has a small effect (factor estimate = 0.08). See Figure 2 for a display of group and individual (median) values across conditions.

One patient systematically made S1s with smaller amplitudes towards targets presented to the left as compared with the other patients (see Figure 2b). This patient systematically made multiple stepwise saccades with small amplitudes to left-sided targets instead of following the 'normal' pattern of an S1 with a large amplitude followed by smaller ones. We fit the model again after exclusion of this patient and still found a main effect of Group ($t = -2.10$), a main effect of Condition ($t = 3.69$), but no main effect of Target Side ($t = 1.77$) nor an interaction effect of Group*Target Side ($t = 0.74$). We, therefore, infer that the previously found interaction was mainly driven by this one patient. Nevertheless, as we try to characterize patients' eye-movement behaviour, we like to get the full scope and chose to perform our subsequent analyses both with and without this outlier. A similar multiple-step pattern that may be typical for patients with parietal damage found by Rath-Wilson and Guitton (2015) substantiates this decision.

Primary saccade under- or overshoot

Out of 24,517 observations (outlier included), 19,483 times (79.5%) S1 undershot the target. This information will be used in consecutive analyses to see how under- or overshooting may influence S2 generation.

3.2.3 | Primary saccade peak velocity

Table 2 displays S1 peak velocity values split on group and condition aggregated by median. A linear mixed-effects model was fit to S1 peak velocity (as described in Methods, see again Supplementary Tables S5 and S6). The model showed no main effect for Group ($t = -1.27$), but it did for Target Side ($t = -2.18$) and Condition ($t = 2.52$). Here, continuous visual feedback yielded slightly lower peak velocity values than when visual feedback was removed during the primary saccade. This finding contradicts earlier findings (Tian et al., 2013) but does add up with our own (surprising) finding where the removal of visual feedback yielded more accurate S1s. More accurate S1s were closer to the target, thus yielded larger amplitudes, and therefore, if any, should evoke *higher* peak velocities (Lebedev et al., 1996). S1 peak velocities did not differ for patients as compared with controls and no interactions were found to be significant predictors.

3.2.4 | Conclusion on primary saccade performance

Primary saccade latency and accuracy outcomes seem best explained by differences between groups and the influence of target location. As expected, patients had longer latencies and lower accuracy as compared with healthy controls. Across groups, leftward (contralesional) saccades unexpectedly seem to start faster, but for patients, they can be somewhat less accurate than rightward (ipsilesional) saccades. Primary saccade peak velocities do not differ between groups. We conclude that a lesion in the PPC can impair patients in saccadic accuracy and speed in their initial attempt to detect targets in the visual environment.

3.3 | Secondary (and consecutive) saccade generation

3.3.1 | Secondary and consecutive saccade proportions

Proportions of S2, S3, S4 and S5 occurrences (as a portion of the total amount of trials) are listed in

TABLE 2 Descriptive results for primary saccade (S1) and secondary saccade (\geq S2) characteristics. Values were aggregated by median for each participant in each condition for both groups, after which group values (median Mdn, interquartile range (IQR), mean, standard deviation (SD), range) were extracted.

Basic characteristics	Patients (n = 7)			Neurotypical controls (n = 26)		
	Mdn (IQR)	Mean (SD)	Range	Mdn (IQR)	Mean (SD)	Range
S1 latency (ms)						
-ON	227 (22)	229 (16.1)	210–254	205 (33.2)	211 (36.0)	162–328
-OFF	227 (27)	230 (19.1)	208–258	203 (30.5)	211 (36.3)	159–325
S1 amplitude (dva)						
-ON	13.5 (0.875)	13.3 (0.909)	11.8–14.4	14.2 (0.434)	14.1 (0.673)	11.9–15.2
-OFF	13.4 (0.474)	13.5 (0.566)	12.5–14.3	14.2 (0.42)	14.1 (0.654)	11.8–15.2
S1 error						
<i>Absolute dva</i>						
-ON	1.58 (0.632)	1.65 (0.789)	0.653–3.08	0.937 (0.346)	1.05 (0.489)	0.546–3.01
-OFF	1.53 (0.35)	1.44 (0.49)	0.6–2.15	0.907 (0.325)	1.03 (0.513)	0.485–2.94
<i>Gain (1 = perfect landing)</i>						
-ON	0.902 (0.054)	0.899 (0.049)	0.827–0.964	0.946 (0.025)	0.942 (0.041)	0.798–1.01
-OFF	0.9 (0.024)	0.906 (0.036)	0.85–0.967	0.949 (0.031)	0.941 (0.041)	0.799–1.01
S1 peak velocity ($^{\circ}$/s)						
-ON	411 (24.1)	422 (30.1)	392–483	433 (88.7)	440 (69.5)	271–562
-OFF	430 (35)	429 (30.7)	388–481	436 (94.8)	443 (70.5)	274–574
S2 latency (ms)						
-ON	151 (25.5)	150 (21.6)	118–180	159 (41.4)	172 (36.0)	113–261
-OFF	308 (97.8)	305 (80.7)	165–398	406 (152)	405 (115)	127–678
S2 amplitude (dva)						
-ON	1.33 (0.654)	1.41 (0.616)	0.55–2.37	0.898 (0.22)	0.996 (0.417)	0.546–2.68
-OFF	1.45 (0.341)	1.39 (0.451)	0.599–2.07	1.08 (0.44)	1.22 (0.507)	0.69–2.93
S2 error						
<i>Absolute dva</i>						
-ON	0.428 (0.075)	0.537 (0.326)	0.326–1.27	0.471 (0.195)	0.516 (0.223)	0.286–1.34
-OFF	0.737 (0.105)	0.822 (0.37)	0.58–1.65	0.731 (0.226)	0.827 (0.335)	0.496–1.96
<i>Gain (1 = perfect landing)</i>						
-ON	0.832 (0.103)	0.813 (0.106)	0.619–0.948	0.784 (0.145)	0.798 (0.114)	0.633–1.11
-OFF	0.873 (0.177)	0.839 (0.163)	0.623–1.12	0.933 (0.322)	0.997 (0.299)	0.601–1.6
S2 peak velocity ($^{\circ}$/s)						
-ON	159 (58.7)	164 (46.8)	98.8–236	120 (32.2)	125 (29.9)	90.7–199
-OFF	162 (45)	161 (44.4)	95.3–224	123 (53)	137 (39.1)	91.3–225
Proportion of trials with S2/S3/S4/S5 occurrence						
S2 proportion						
-ON	0.968 (0.042)	0.97 (0.026)	0.937–0.996	0.949 (0.059)	0.917 (0.1)	0.569–0.996
-OFF	0.867 (0.215)	0.819 (0.188)	0.466–0.998	0.71 (0.286)	0.685 (0.189)	0.327–0.955
S3 proportion						
-ON	0.755 (0.169)	0.694 (0.201)	0.344–0.976	0.547 (0.227)	0.543 (0.184)	0.069–0.856
-OFF	0.312 (0.322)	0.44 (0.287)	0.159–0.952	0.229 (0.268)	0.267 (0.203)	0.017–0.719

TABLE 2 (Continued)

Basic characteristics	Patients (n = 7)			Neurotypical controls (n = 26)		
	Mdn (IQR)	Mean (SD)	Range	Mdn (IQR)	Mean (SD)	Range
S4 proportion						
-ON	0.283 (0.204)	0.382 (0.195)	0.095–0.641	0.194 (0.182)	0.22 (0.129)	0.017–0.489
-OFF	0.083 (0.167)	0.186 (0.212)	0.048–0.589)	0.028 (0.087)	0.066 (0.087)	0–0.345
S5 proportion						
-ON	0.086 (0.101)	0.097 (0.086)	0.009–0.25	0.034 (0.059)	0.056 (0.061)	0–0.245
-OFF	0.018 (0.058)	0.048 (0.063)	0–0.168	0.007 (0.011)	0.011 (0.016)	0–0.061
Main sequence	Mdn (IQR)	Mean (SD)	Range	Mdn (IQR)	Mean (SD)	Range
S1						
-ON						
• <i>Intcpt</i>	231.1 (83.12)	229.5 (52.081)	165.5–300	242.3 (141.65)	252 (90.822)	82.2–410.5
• <i>Slope</i>	13.67 (8.04)	14.75 (5.122)	9.29–22.33	12.78 (8.26)	13.488 (5.188)	5.47–25.5
-OFF						
• <i>Intcpt</i>	239.6 (110.3)	247.0 (57.816)	187.5–311.8	281.27 (137.15)	266.43 (90.87)	97.25–403.85
• <i>Slope</i>	13.146 (8.97)	13.745 (5.867)	6.49–21.64	12.72 (6.52)	12.547 (4.924)	2.86–21.91
S2						
-ON						
• <i>Intcpt</i>	102.32 (41.8)	98.83 (46.322)	31.53–177.25	72.54 (29.62)	72.67 (25.173)	21.79–132.58
• <i>Slope</i>	37.53 (21.46)	48.7 (31.163)	15.14–112.11	45.6 (27.85)	52.38 (21.639)	24.21–107.6
-OFF						
• <i>Intcpt</i>	115.93 (75.38)	105.12 (48.582)	45.81–173.53	82.39 (40.93)	87.22 (33.578)	32.15–162.57
• <i>Slope</i>	30.86 (30.5)	39.95 (23.34)	15.9–76.31	38.97 (17.87)	40.87 (16.304)	18.43–77.79

Note: Graphs displaying individual main sequences for S1 and S2 with (ON) and without (OFF) visual feedback can be found in Supplementary Figure S3.

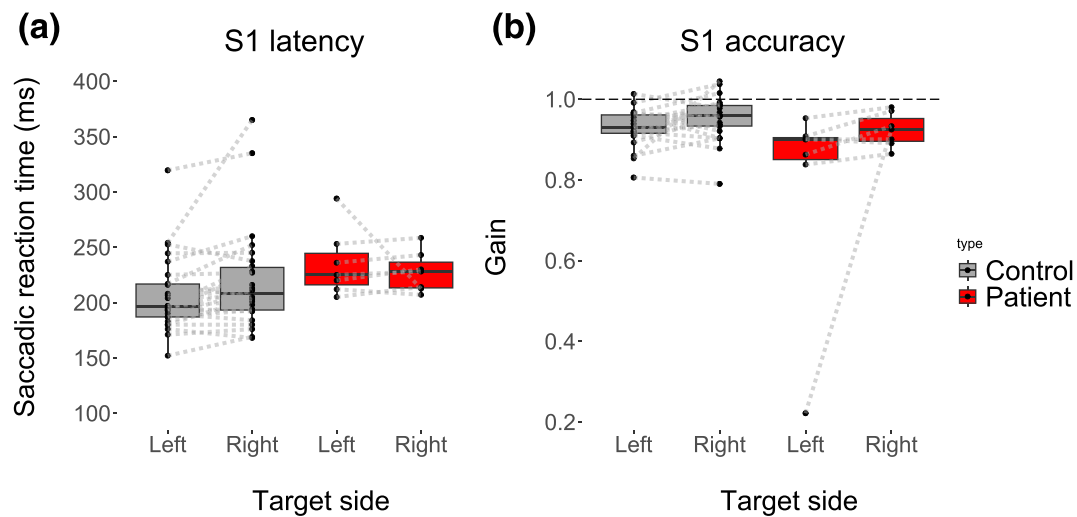


FIGURE 2 Visualization of (a) median primary saccade (S1) latency in milliseconds and (b) median S1 accuracy in gain for controls (grey) and patients (red) split on the side of target presentation (left or right), where a value of 1 depicts perfect landing. Dots and lines represent individual values across target side presentation.

Table 2. S2–S5s were counted per participant, and proportions per participant were calculated. These individual proportions were aggregated by median for each group in both conditions (visual feedback on or off).

3.3.2 | Secondary saccade latency

Table 2 displays S2 latency values split on group and condition. A linear mixed-effects model was fit to secondary saccade latency (as described in [Methods](#), see again Supplementary Tables S7 and S8). The model with the best fit included factors Group and Condition, where only Condition yielded a significant effect ($t = 15.95$): S2s in response to targets that were removed (visual feedback OFF) were slower than when the target remained visible (visual feedback ON). See Figure 3 for a display of group and individual (median) values on trials where visual feedback either remained available or disappeared.

3.3.3 | Secondary saccade accuracy

Table 2 displays S2 accuracy values split on group and condition. Figure 3b shows median S2 accuracy expressed in gain on group and individual level. A

linear mixed-effects model was fit to S2 gain (as described in [Methods](#), see again Supplementary Tables S9 and S10). The model yielded no effect of Group ($t = -0.29$) nor Target Side ($t = 1.80$), but showed a main effect of Condition ($t = 14.98$). Again, surprisingly, participants S2s were more accurate (gain = 1 indicates perfect landing) when visual feedback was removed. Additionally, an interaction between Group and Condition was found ($t = -6.99$). Related to this interaction, Figure 3b shows increased variability in S2 gain, specifically in the controls group where overshooting became more common in the condition without visual feedback (OFF). Yet, on a group level, controls were more accurate than patients when visual feedback was removed. There was no interaction with Target Side.

3.3.4 | Corrective secondary saccades

S2s occur often. Out of 24,517 observations, an S2 was performed in 20,320 trials (82.9%; the reader is referred to Supplementary Table S14 for proportion distribution over groups, conditions and target presentations side, and to Supplementary Figure S4 for patterns across individuals). Still, it could be the case that S2s occur, but that they are not directed to the target and therefore do not have a corrective nature. This would have

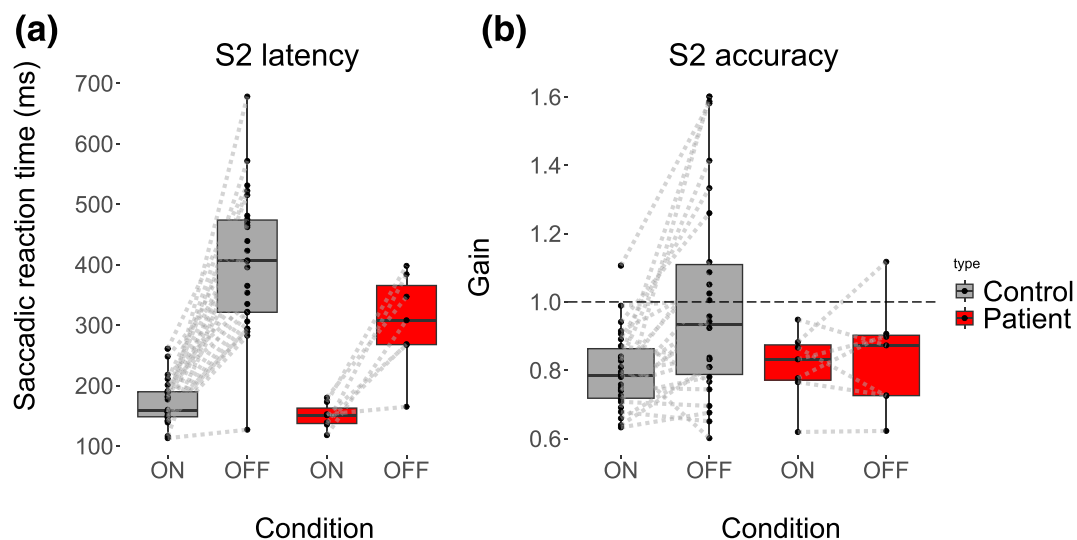


FIGURE 3 Visualization of (a) median secondary saccade (S2) latency in milliseconds, and (b) median S2 accuracy in gain for controls (grey) and patients (red) split on visual feedback condition (ON/retinal+extraretinal or OFF/extraretinal), where a value of 1 depicts perfect landing. Dots and lines represent individual values across conditions.

implications for our interpretation of the results and the answer to the question how patients deal differently with an S1 error than controls when they only have extraretinal signals to rely on. Therefore, we analysed the corrective nature of S2s. We calculated the distance to the target after S1 and S2. S2 was considered corrective if the absolute error after S2 was smaller than the error after S1. 70.7% of executed S2s was corrective, with 67.2% (range 43.8–96.25%) corrective S2s for controls as compared to 80.6% (range 67.3–96.4%) corrective S2s for patients. We found that the two groups are equal ($t = 1.23$) in the corrective nature of their S2s in both retinal and extraretinal signal conditions ($t = -0.29$; also see Supplementary Table S15 for models and model comparison to substantiate this result). Further interpretation based on the assumption that *S2s are equally corrective (or non-corrective, for that matter) for both groups* is therefore well-grounded.

3.3.5 | Secondary saccade incidence

As a next step, we investigated what factors (e.g. extraretinal signals) urge the generation of a (corrective) S2 and whether or not this holds after a lesion to the PPC. Apart from assessing group differences in general, we explored whether S2 generation occurs differently when there are both retinal and extraretinal signals to

rely on (condition ON) as compared to when there are only extraretinal signals to rely on (condition OFF). If any, we hypothesized patients to show hampered S2 generation – and thus, incidence – in response to sole extraretinal signals, as the PPC was thought to specifically be involved in maintaining and acting upon these extraretinal signals. Furthermore, we included target presentation side as lesions were unilateral, and we included S1 error to investigate potential differences in action threshold.

Model comparison (as described in [Methods](#), see again Supplementary Tables S11 and S12) yielded a best fit logistic linear mixed-effect model to the data (including outlier) of the binomial variable S2 presence (0 = absent, 1 = present). Table 3 shows GLMM output for the model's fixed effects. (Note: we performed the same procedure to fit the data without the outlier patient: still, the same model could best explain the data. Therefore, in the remainder of this section, the results including the outlier patient are presented.)

Highly significant effects are observed. Column 'Estimate' indicates predicted log odds values for S2 occurrence with regards to the specific factors included in the model. From these estimates, a proportion value can be calculated to make interpretation more intuitive (see Supplementary Table S16). Estimates given for *discrete* variables were combined to derive the estimated intercept for a specific condition (e.g. [1] + [2] + [3] + [6] + [7] + [8] for Patients, OFF, Right). Estimates

TABLE 3 Generalized linear mixed model estimates (in log odds) for secondary saccade occurrence as a function of factors group, condition, target side, S1 error (abslogS1gain) and S1 shoot. '(intercept)' (i.e. baseline) refers to trials for the control group with left sided targets where retinal feedback is available, with an error of 0 (perfect landing). 'Estimate' indicates predicted log odds values for the specific factor. Estimates for discrete variables are used in computation of intercepts of various situations (see table 9). Estimates linked to the continuous variable (S1 error, marked in grey) are used in computation of the predicted slopes (see Figure 4).

Fixed effects:	Estimate	Std. Error	z-value	Pr(> z)
[1](Intercept)	2.38617	0.21269	11.219	< 2e-16 ***
[2]groupPatient	1.75142	0.47989	3.650	0.000263 ***
[3]conditionOFF	-1.95737	0.07373	-26.549	< 2e-16 ***
[4]abslogS1gain (error)	4.70209	0.75084	6.262	3.79e-10 ***
[5]overshoot	-0.10862	0.02406	-4.515	6.33e-06 ***
[6]tarsideRight	0.17253	0.04229	4.079	4.52e-05 ***
[7]groupPatient:conditionOFF	-0.98670	0.18903	-5.220	1.79e-07 ***
[8]groupPatient:tarsideRight	-0.48468	0.10969	-4.419	9.92e-06 ***
[9]conditionOFF:abslogS1gain	-1.04904	0.80701	-1.300	0.193634
[10]groupPatient:conditionON:abslogS1gain	-3.15435	1.50517	-2.096	0.036111 *
[11]groupPatient:conditionOFF:abslogS1gain	5.53776	1.08927	5.084	3.70e-07 ***

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

given for the *continuous* variable (here `abslogS1gain`, marked in grey) were used in computation of the predicted slopes.

Figure 4 visualizes observed proportions of S2 occurrence (dots) and model predictions (lines) of S2 occurrence probability across the different conditions (ON = visual feedback, hence, both retinal and extraretinal signals; OFF = no visual feedback, hence, only extraretinal signals) separated on target presentation side (Left, Right). Note that that prediction lines are an expression of the statistical findings in Table 3 and Supplementary Table S16. In case the differences are hard to distinguish in the graph, Table 3 can be used to substantiate these. The main take-aways of the model predictions from the GLMM will be explained below, but a more detailed and complete overview of calculated estimates can be found in Supplementary Table S16 and the accompanying explanation. Additionally, Supplementary Table S17 displays the number of observations per binned S1 error magnitude per group,

condition and target side that was used as input for the model.

The core messages of the GLMM visualization (Figure 4) lie in the starting points of the prediction lines and the steepness of their slopes. In general (regardless of error signal type), patients are predicted to make more S2s than controls (Figure 4 all panels, compare starting point of red vs. black lines). Controls perform somewhat more S2s towards rightward instead of leftward targets (Figure 4a vs. b; Figure 4c vs. d, compare black line starting points), but patients execute more S2s when the target is presented contralesionally (to the left) than ipsilesionally (Figure 4a vs. b; Figure 4c vs. d, compare red line starting points). Regardless of S1 error size, undershooting a target results in more S2s than overshooting (for the sake of comprehensibility we chose not to display this in the graphs; but see Table 3 for substantiation). Again regardless of S1 error size, both patients and controls are more inclined to perform an S2 when retinal feedback is available as compared to when it is

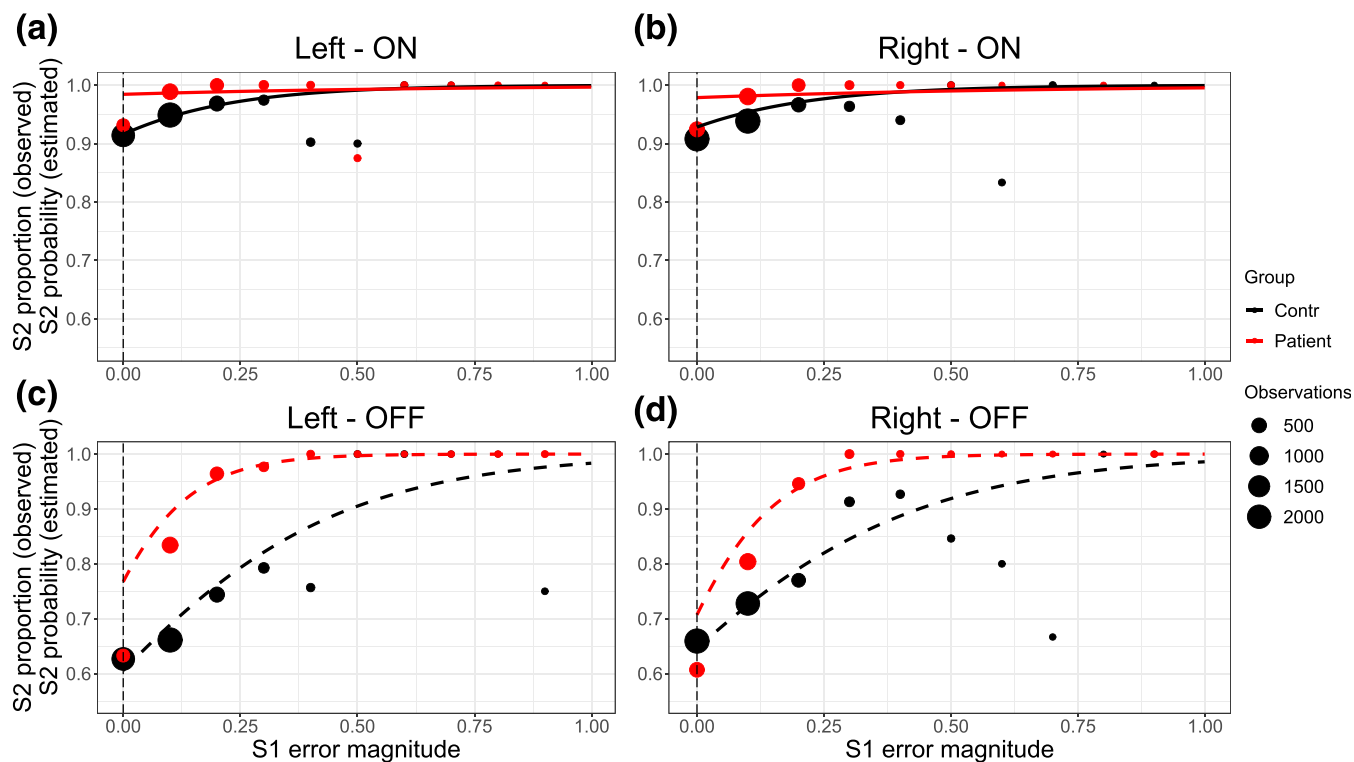


FIGURE 4 Visualization of varying effect for primary saccade (S1) error size on secondary saccade (S2) occurrence across specific conditions (a + b = retinal + extraretinal signals, condition ON; c + d = extraretinal signals only, condition OFF) for the two groups split on the side of target presentation (a + c = left, b + d = right). Dots represent the observed binned Centre means (binwidth = 0.1). Larger dotsizes indicate a larger amount of observations. Some small dots in the figure refer to a very small number of observations. For absolute number of observations per bin, see supplementary table S17. Lines represent the predicted proportions based on best fit GLMM across specific conditions (solid = condition ON, dashed = condition OFF) for the two groups (black = control, red = patients). The vertical dashed line corresponds to an ideal S1 (no error); the higher the value on the x-axis, the bigger the distance between S1 endpoint and the target.

unavailable (Figure 4a vs. c; Figure 4b vs. d, compare solid vs. dashed lines). When taking S1 error size into account (start to look at the steepness of the slopes instead of only their starting points), we see that the effect of S1 error is similar in the retinal and extraretinal condition for controls (Figure 4a vs. c; Figure 4c vs. d, compare steepness of black solid and black dashed lines; Table 3 shows the non-significant effect of `conditionOFF:abslogS1gain`). On the contrary, for patients the effect of S1 error does differ across those conditions (Figure 4a vs. c; Figure 4c vs. d, compare steepness of red solid and red dashed lines). When retinal feedback is available (Figure 4a + b, solid lines), the increase in S2s with increasing S1 error is smaller for patients than for controls (the three-way interaction in Table 3 `groupPatient:conditionON:abslogS1gain` adds up to a diminished effect of the primary saccade error when it comes to patients). When only extraretinal signals are available (Figure 4c + d), however, the opposite happens: here S1 error has a stronger effect on the generation of S2s for patients than for controls (the high positive estimate of Table 3 `groupPatient:conditionOFF:abslogS1gain` indicates a significantly steeper slope for the effect S1 error in the patient group than for controls when no retinal feedback is available).

One explanation for this difference might be that patients practically always execute S2s when retinal feedback is available (solid red line), even when the S1 error is minimal. Consequently, the slope cannot rise as much as in the extraretinal feedback condition (dashed red line), thereby making the additional effect of S1 error in the retinal feedback condition negligible.

A pattern where larger primary saccade errors lead to higher S2 probability has often been observed in previous studies (for example, Cohen & Ross, 1978; Ohl & Kliegl, 2016; Prablanc et al., 1978; Tian et al., 2013). Similarly, we clearly observe this pattern for our patient group, and we also observe this pattern for controls at smaller values of S1 error. This strengthens the belief that there is, in principle, a linear relationship between S1 error and the likelihood that an S2 is executed. However, a point to address is that the observations for controls do not fit the full length of the prediction lines (moving to larger S1 errors) as neatly as those of the patients. We explain this by possible ‘lapses’ in sensory decision-making. Lapses are described as occurrences where acting is not based on sensory evidence, but rather is an expression of a momentary lapse in attention or memory (Ashwood et al., 2022), such as disengagement from the task. The data points deviating from the model fit concern trials where very large primary saccade errors are made, that are not followed by an S2. An error magnitude of 0.5 means that one did not redirected gaze further than

half the distance towards the target. Error magnitudes of 1.0 mean that the primary saccade practically ends where the fixation were (albeit so far that it passed the filter criteria). One can speculate about whether primary saccades with such large errors were actually initiated with the intention to reach the target, or that they represent ‘lapses’ as described above.

All in all, we find that the S1 error urges a S2 in both groups in both the retinal and extraretinal feedback conditions, but that this effect varies depending on the specific group and availability of (extra)retinal signals. Interestingly, patients perform more S2s than controls when retinal feedback is unavailable (i.e. patients need to rely on their extraretinal signal). This finding goes against the hypothesis that patients cannot use extraretinal signals after a PPC lesion.

Speed–accuracy trade-off. We have analysed a possible speed–accuracy trade-off and found a significant relationship ($p < .001$) between S1 latency and S2 probability within our patient group. The longer S1 latency, the less probable the execution of an S2 (unstd. Estimate = -0.0036 , see Supplementary Figure S5). For controls, we found the same significant relationship ($p < .001$) between S1 latency and S2 probability (unstd. Estimate = -0.025).

Interim conclusion

Consistent with previous literature we find that S2s were initiated quicker when visual feedback remained available (Tian et al., 2013). With regards to S2s accuracy, on the other hand, we found that S2s were more accurate when based on extraretinal signals (i.e., when the target was removed). S2s were, however, more *likely* to be executed when retinal feedback was present as compared to when it was absent. Also, undershooting a target resulted in more S2s than overshooting, and S1 error was found to have a profound effect on whether to execute an S2 or not; the higher the error, the higher the likelihood that an S2 occurred. Importantly, patients generated more S2s after an S1 error than controls, both when retinal feedback was present and when not. This finding goes against the hypothesis that patients cannot use the extraretinal signals anymore due to a PPC lesion, which would result in a lack of initiation or a decreased initiation of S2s when retinal feedback is absent. Even more, the patients’ likelihood of generating an S2 is higher after executing a *contralesional* S1, whereas it was specifically hypothesized that a lesion in the PPC would distort *contralesional* extraretinal signal processing and would therefore interfere with initiating S2s after a *contralesional* S1. This effect could not be attributed to a bigger S1 error alone. In sum, patients’ initiation of S2s did not seem to be hampered by their PPC lesion in terms of quantity, and

their S2s were evenly quickly generated and were even accurate as those of controls. Nevertheless, in terms of timing, the current analyses only informed us about trials in which an S2 was actually executed. In other words, they discard trials in which only S1s were carried out, and do only partly shed light on the question of whether S2 generation is impaired in terms of timing. To also take into account trials in which no S2 was made, and to assess what factors contribute at *what moment in time* to the generation of S2s (with and without a PPC lesion), we carried out the following analysis.

3.3.6 | Secondary saccade time course

We have seen that secondary saccades based on extraretinal signal processing are still executed after a lesion to the PPC. This points towards evidence for a spared function of extraretinal signal processing after damage in this area. What the previous analysis does not tell, however, is whether or not more subtle changes occur in the generation of secondary saccades after a lesion to the PPC, e.g. in terms of timing. Therefore, our next analysis was aimed at assessing the *time course* of secondary saccade generation. This way, we investigated whether a lesion to the PPC resulted in suboptimal extraretinal signal processing expressed as a delayed S2 initiation.

Model and assumptions

A survival model was generated for each group separately. For the patient group, we ran an initial model once *including* outlier and once *excluding* outlier (so three models were generated in total). Based on our hypothesis, we included factors Target Side (left, right), Condition (ON, OFF), S1 Error (continuous) and Shoot

(under-, overshoot). Interactions of S1 Error*Shoot, S1 Error*Condition and S1 Shoot*Condition were also included in the models. With generating these initial models, we checked whether our continuous variable (S1 error) met the assumption of additivity. As suggested by Martinussen & Scheike, (2006, p.154), we performed a Goodness of Fit for each of the three models by computing the residuals in the Aalen analysis and subsequent resampling ($n = 1000$) of cumulative residuals. To evaluate the fit we transformed S1 error into a factor with 4 levels defined by the quartiles. Only one negligible violation of the additivity assumption was indicated (one $p = .049$, rest $p > .05$; see Supplementary Table S19). After securing this assumption, the initial model was stripped down to the essential factors (p -values $< .05$ for that factor within *all three* full models, see Supplementary Table S18). Only the side of target presentation appeared not to have a significant effect on S2 generation (all three $p > 0.7$) so this factor was left out of the new model.

Results

The model with the best fit showed that (absolute) S1 Error, Condition, Shoot, S1 Error*Shoot, S1 Error*Condition, and S1 Shoot*Condition significantly influenced secondary saccade generation in healthy controls and patients (Table 4).

All factors had a time-varying effect, meaning that their influence varied over different timepoints after primary saccade landing (Table 5). Again, the outlier patient did not influence the results for the group as a whole. In the remainder of this section the results including the outlier patient are presented.

Figure 5 visualizes the time course of S2 generation as a function of the different factors at play for both healthy

TABLE 4 Additive hazard model output for tests of non-significance of various factors included in the models (S1 error, condition, S1 shoot and interactions) for the three groups (controls, patients, patients excluding outlier). Values of $p < .05$ indicate a significant effect of the factor on the generation of a secondary saccade.

	Test for non-significant effects					
	Controls		Patients		Patients, excl. Outlier	
	Supremum-test of significance	p-value H ₀ : B(t) = 0	Supremum-test of significance	p-value H ₀ : B(t) = 0	Supremum-test of significance	p-value H ₀ : B(t) = 0
(intercept)	16.60	0.0000	7.63	0.0000	6.84	0.0000
S1 error	6.05	0.0000	12.50	0.0000	13.20	0.0000
Condition	13.60	0.0000	15.20	0.0000	12.10	0.0000
S1 shoot	3.52	0.0113	4.14	0.0011	4.42	0.0003
S1 error: Condition	6.19	0.0000	9.69	0.0000	11.60	0.0000
S1 error: S1 shoot	4.24	0.0007	8.43	0.0000	7.69	0.0000
S1 shoot: Condition	6.37	0.0000	4.96	0.0000	4.78	0.0001

TABLE 5 Additive hazard model output for tests for time-invariant effects for the factors included in the models (S1 error, condition, S1 shoot and interactions) for the three groups (controls, patients, patients excluding outlier). Values of $p < .05$ indicates a significant effect, meaning that the effect of the factor on the generation of secondary saccades varies significantly over time.

	Test for time-invariant effects					
	Controls		Patients		Patients, excl. Outlier	
	Kolmogorov–Smirnov test	p-value H ₀ : Constant effect	Kolmogorov–Smirnov test	p-value H ₀ : Constant effect	Kolmogorov–Smirnov test	p-value H ₀ : Constant effect
(intercept)	0.711	0.0000	0.818	0.0036	0.823	0.0079
S1 error	9.750	0.0000	13.600	0.0000	13.900	0.0000
Condition	0.844	0.0000	1.070	0.0016	1.070	0.0031
S1 shoot	0.238	0.0056	0.611	0.0106	0.604	0.0159
S1 error: Condition	8.030	0.0000	14.000	0.0000	14.400	0.0000
S1 error: S1 shoot	4.040	0.0015	9.130	0.0000	9.530	0.0000
S1 error: Condition	0.380	0.0000	0.922	0.0000	0.967	0.0002

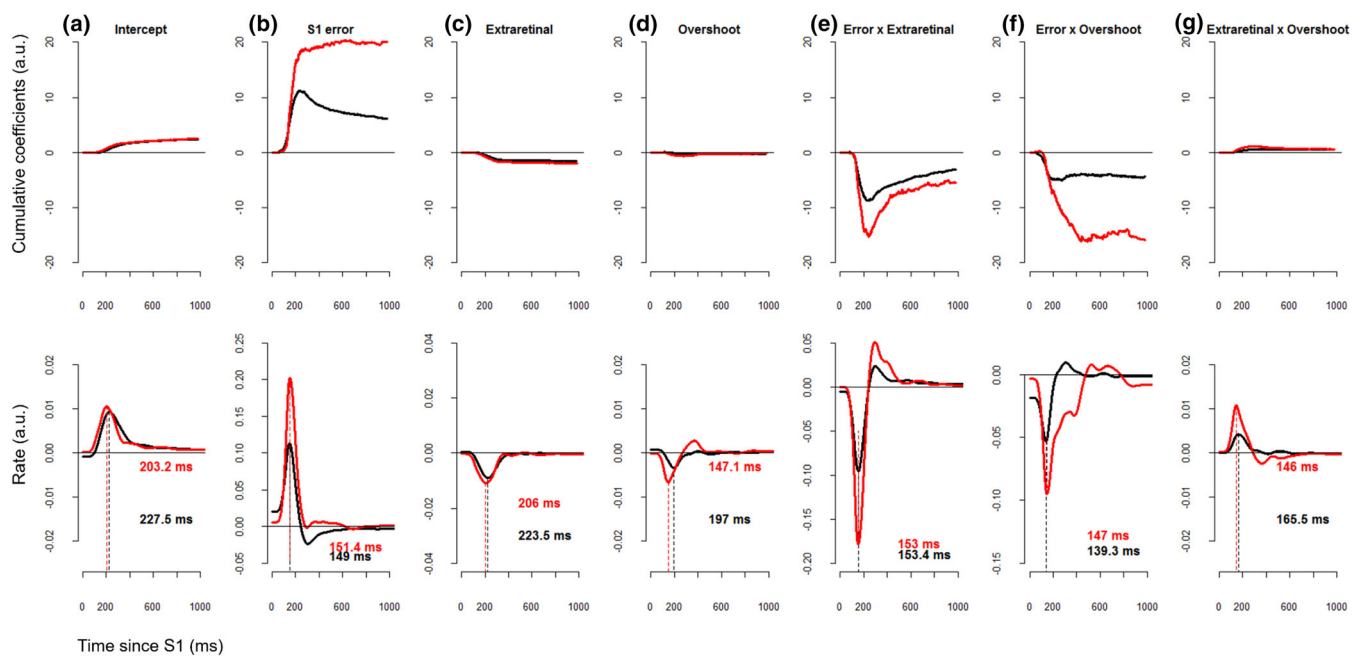


FIGURE 5 Cumulative coefficients (top row) and the estimated first derivative of the cumulative coefficients (bottom row) for all factors (a–g) in the Aalen's additive hazards model for healthy controls (black) and patients with a lesion to the right PPC (red). Intercept refers to trials with a target presented to the left, without retinal feedback. Values >0 indicate that the specific factor increases the likelihood of a secondary saccade (S2) to be generated with respect to the baseline rate. Values <0 indicate that a specific factor decreases the likelihood of an S2 being performed. Peaks and valleys in bottom graphs indicate at which time point the given factor has its most pronounced effect in the generation of an S2 since S1 offset; the most pronounced peak/valley in the graph is indicated with the associated timepoint.

controls and the patient group. The top row of Figure 5 displays the cumulative coefficients over time for the different factors; the bottom row displays a kernel regression smoothing of the first derivative of the cumulative coefficients over time since S1 offset. Values >0 indicate that the specific factor *increases* the likelihood of an S2 to be generated with respect to the baseline

rate. Values <0 indicate that a specific factor *decreases* the likelihood of an S2 being performed. Thereby, the bottom row clearly visualizes the point in time at which a specific factor has its most pronounced effect (its peak or valley) *with respect to the baseline*. Again, like in the S2 presence analysis, estimates need to be combined to come to the final value for a specific condition.

Intercept. The positive values for the Intercept (Figure 5a) indicate an increase in S2 rate in the baseline condition (independent of S1 Error characteristics). This holds for both controls and patients, with the maximum effect being slightly earlier for patients (203 ms) than for controls (227 ms; Figure 5a). This timepoint (Figure 5a, bottom) corresponds to the median S2 latency for this type of trial. The peak timepoints arise somewhat later than observed by Ohl & Kliegl (~174 ms), and could be explained by the higher age of our participants (Irving et al., 2006).

S1 Error. The effect of absolute S1 Error (Figure 5b) is more pronounced for patients than for controls, but it peaks at about the same time for both groups (151 ms and 149 ms after S1 offset, respectively). In other words, with an error of the same size, patients will be more inclined to make an S2 than controls. This early peak effect shows that S2s in response to a primary saccadic error are quicker than S1s themselves (>206 ms in the current study; >203 ms in Tian et al., 2013), advocating the monitoring-based corrective nature of S2s instead of starting a freshly new S1 all the time.

S1 error and the (un)availability of (extra)retinal signals. Condition OFF (Figure 5c) has a decreasing effect on S2 rate across both groups. This means that S2s are initiated to a lesser extent when there is reliance only on extraretinal signals as compared to when both error signals are available (as in baseline). The effect arises at 206 ms for patients and at 224 ms for controls.

When combining this information with the effect of S1 Error (Figure 5e), we see that the effect of S1 error is flipped. The effect of S1 error decreases drastically when there is no retinal feedback as compared to when retinal feedback is available. This holds for both groups; we do not see any differences in secondary saccade generation between patients and controls.

Predicted survival curves (also see Ohl & Kliegl, 2016) are provided in Supplementary Figure S6.

What does all this mean? Taken together, time course analysis revealed that some factors have a different effect at different points in time. Therefore, this analysis brings a more nuanced view to the study of (secondary) saccadic behaviour. Although we did not statistically test the peak timing nor the survival curves of the groups against each other, our results indicate that patients do at least not process extraretinal signals *later* than controls. In sum, patients' initiation of S2s does not seem to be hampered by their PPC lesion in terms of timing.

3.3.7 | Conclusion on secondary saccades

We performed two extensive analyses on the generation of S2s, one in terms of quantity and one in terms of timing. For both controls and patients there was a clear relation between the S1 error and S2 occurrence; the bigger the error, the more likely it is that an S2 will be executed. Especially when the target remained visible, facilitating an externally driven error signal, the S2 probability was high. This finding aligns with the idea of a varying error threshold depending on whether or not retinal feedback is available (Tian et al., 2013). We found that, indeed, most S2s were of corrective nature, aiming to minimize the distance to the target in order to perceive the finest spatial details.

Our results show that patients were well-equipped to perform S2s. In fact, patients were even more inclined to perform S2s than controls, not only when retinal feedback is available, but also when there were only extraretinal signals to rely on. Given the equal corrective nature of S2s of the control and the patient group, we can reasonably argue that patients are still able to use extraretinal error signals in the generation of sequential saccades. In terms of timing, we see that patients did not exhibit a delayed factor processing, but were still able to quickly process information to perform S2s. With this study, we support the hypothesis that the PPC is *not* indispensable for executing (contralesional) secondary saccades when there are only extraretinal signals to rely on.

4 | DISCUSSION

Classical studies (Duhamel et al., 1992; Heide et al., 1995) proposed that a lesion to the PPC compromises neurons that are critically involved in carrying and processing extraretinal information needed to perform accurate sequential saccades. Is a lesion to the PPC indeed disrupting processing extraretinal signals, or are patients with damage to this area still able to use these signals similarly as controls? And if so, is functioning of the oculomotor system in any other way impaired? Although damage to the PPC is often strongly associated with impairments in the spatial remapping that contributes to the employment of accurate consecutive saccades after a primary saccade mislocalization (Colby & Goldberg, 1999; Duhamel et al., 1992; Guthrie et al., 1983), Rath-Wilson and Guitton (2015) found that patients with a lesion to the PPC were actually still able to perform quite accurate multiple-step saccades. Extending to this insight, our

recent study (Fabius et al., 2020) revealed that patients with substantial chronic lesions to the PPC were able to use *some* form of extraretinal feedback in *perceptual* judgements. These findings suggest that the PPC is not so critical after all, and that, if any, a PPC lesion might result in a suboptimal rather than a distorted performance. We investigated whether the same idea held in the action-domain, by studying the oculomotor consequences of a lesion to the PPC.

Healthy controls and patients in the chronic phase after a cerebrovascular accident (CVA) in the PPC completed a visual localization task (prosaccades) with targets that either remained visible or were removed from the screen after primary saccade onset. Continuous visibility of the target elicited both retinal and extraretinal signals, but only extraretinal signals were available in the condition in which the target was removed. If the PPC would be critically involved in processing extraretinal primary saccade error signals, patients would not be able to use them to make a (corrective) consecutive saccade (S2). If the PPC is not the critical hub, but would be involved in some other way, we might expect a suboptimal rather than a distorted use of extraretinal signals. This suboptimality could be reflected in a decreased or delayed generation of S2s. Basic primary and secondary saccade characteristics, and secondary saccade generation in terms of quantity and timing were analysed to identify possible impairments after a PPC lesion.

Our results show that patients' initial attempt to foveate a visual target was impaired in both accuracy and speed when compared to healthy controls. Although patients were less accurate than controls for both contralesional and ipsilesional target presentation, accuracy was particularly decreased when targets were presented in the contralesional hemifield. A larger inaccuracy reflects a smaller probability that the primary saccade (S1) will result in foveating the target, meaning that an S2 would be required for foveation. Our study found that both groups showed the ability to employ S2s to overcome the initial inaccuracy: similar to controls, patients corrected the error of the S1 by making S2s. Remarkably, although we did not find differences across groups in S2 latency nor accuracy, our results indicate that patients were actually more inclined than controls to execute S2s, not only when relying on retinal signals, but also when there were only extraretinal signals to rely on (i.e. when the target was removed during the S1). Interestingly, when specified on contralesional/ipsilesional target presentation, we observed that patients performed even *more* S2s when targets were presented contralesionally as compared to presentation ipsilesionally. This imbalance between the two hemifields could not be attributed to a larger S1 error alone. Given the equal corrective nature

of S2s for the control and the patient group, we can reasonably argue that patients were still able to use extraretinal error signals in the generation of S2s after a lesion to the PPC.

To put these findings into perspective, it is necessary to address a variation in terminology and paradigm between other studies and the current. Previous studies used double-step paradigms to make inferences about the extraretinal signal processing mechanism (Duhamel et al., 1992; Heide et al., 1995; Rath-Wilson & Guitton, 2015). In double-step paradigms, the subject is asked to consecutively foveate two different locations in the visual field in the order they appeared. To make an accurate S2 towards the second target, the visual system needs to memorize this location. The location then needs to be computed in extraretinal coordinates, as the retinal coordinates of the second location are not useful anymore. There might be a subtle difference in our use of the term 'secondary' in this regard. The double-step studies used this term to define the saccade that is directed to the second target (Duhamel et al., 1992; Rath-Wilson & Guitton, 2015). In our paradigm, however, the S2 was not a saccade that was aimed at a second target, but rather an extension of the S1 in order to reach a single target. Although saccades are visually triggered in both the double-step paradigm and our paradigm, in our study S2s are more of reflexive nature as they do not need to follow a top-down regulated order of appearance. It is therefore likely that different oculomotor circuits are triggered; PPC involvement might be more pronounced in double-step paradigms, given that top-down visual processing is required to a relatively larger extent as subjects need to decide what target to saccade to first (Machado & Rafal, 2004; Paré & Dorris, 2012; Ptak & Müri, 2013).

Additionally, our paradigm might have triggered two different kinds of saccades in terms of predictability. In trials starting from the middle of the screen, participants could not foresee the location of the target. On the contrary, when starting from the sides, participant were able to predict to some extent (with a jitter of 1°) the location of the target. As the predicted location might fall onto a relatively high acuity area of the retina, this could have influenced our results in terms of accuracy (and therefore the urgency of correction; Gaymard et al., 2003). Nonetheless, we chose not to account for predictability; if we wanted to include this factor, we needed to perform triple the amount of trials. This would probably have induced (severe) fatigue, leading to a lesser performance (Kris, as cited in Becker & Fuchs, 1969, p.1248) and a higher dropout rate.

Apart from these considerations, our study clearly shows that patients with PPC lesions do not show deficits in generating (corrective) consecutive saccades PPC in

the 'classical' view of impairments, e.g., where one would expect no S2 generation at all, reduced S2 generation, or a delay in processing factors that urge S2 initiation. Instead, whereas patients were expected to make fewer S2s when they could only rely on extraretinal signals, levels of S2 generation were higher in patients than in controls in either condition, and patients were even more inclined to perform S2s when only extraretinal signals were available to urge a correction. Once initiated, patients' S2s decreased the endpoint error of the S1 (i.e., seem to be corrective in nature) to the same extent as those of controls. This finding is puzzling at first sight. With these, what can be said about the role of the PPC in oculomotor and spatial localization behaviour with and without visual feedback?

Of course, the PPC is associated with a large variety of functions, ranging from spatial perception to higher order executive functions (Whitlock, 2017). Of specific interest here is that the PPC has been strongly associated with spatial remapping (Colby & Goldberg, 1999; Guthrie et al., 1983), where the receptive fields of the neurons in the PPC are aligned to the foreseen situation after saccade execution already before a primary saccade is initiated. Presumably, this extraretinal signal is put to use when visually triggered primary saccades miss the no longer visible target and should be corrected with a newly computed vector. If this function were to be specific to the PPC, we would expect decreased employment of corrective consecutive saccades for patients with PPC lesions specifically when no visual feedback is present, which is clearly not what we find. One explanation for the found accurate use of sequential saccades relates to the specificity of the lesions in our patient sample; although lesions can have a profound and wide-spread effect on functional connectivity, this effect is largely dependent on the lesion location and centrality of the node (Alstott et al., 2009). Possibly, damaged nodes in our sample were not 'central' enough to have a profound effect on functional connectivity of the oculomotor network subserving secondary saccade generation, nor were they homogenous enough to show profound perturbations in (i.e. the absence of) secondary saccade generation on a group level. Alternatively, our results steer in the direction of the right PPC not being the crucial hub in extraretinal remapping for corrective consecutive saccades, which aligns with earlier findings on transsaccadic memory relying on the same mechanism (Fabius et al., 2020; Ten Brink et al., 2019).

However, the increased amount of S2 generation might still be an expression of a suboptimal sensory decision-making system, e.g., when there is an increase of noise in the oculomotor system after a PPC lesion. Even in intact sensory systems, noise is inherent to sensory-motor pathways and introduces sensorimotor

uncertainty about saccade landing points (Lisi et al., 2019). When aiming directly at the target, chances are that an S1 overshoots the target due to this noise. Saccading back from overshoot to target could be more time-consuming than generating a second saccade into the same hemifield as the S1 (i.e. when undershooting the target; Henson, 1978; Robinson, 1973; Ohl et al., 2011). In order to arrive at the most time-efficient manner to reach a target *given motor noise and sensory uncertainty* it would be most beneficial to systematically undershoot a target and make successive saccades to the target's location (Lisi et al., 2019). Although the patients' damaged PPC nodes may not be that 'central', it is reasonable that sensorimotor noise is bigger in the patient group than in healthy controls due to the lesion, resulting in a higher sensory uncertainty. This idea of increased sensory noise seems to be substantiated by the observation that patients are relatively impaired in primary saccade latency and accuracy. The system might have readjusted to this suboptimum by accounting for this primary saccade noise by building in the tendency to employ more (corrective) consecutive saccades. Therefore, increased levels of S2 generation might be seen as a novel optimal equilibrium given the effects of the lesion on S1, regardless of whether visual feedback is available. We therefore argue that damage to the PPC caused more general deficits in saccadic spatial orientation and/or attention, rather than specific impairments in spatial remapping associated with employing corrective consecutive saccades on the basis of extraretinal signals solely. We speculate that our patients showed this new optimum in the form of a multiple-step (≥ 2 saccades) trajectory to the target with shorter S1s and more successive saccades, where healthy controls might suffice with one. As compared to healthy controls, therefore, this can be seen as suboptimal saccadic behaviour due to the damage to the PPC. Whether this system adjustment follows an optimal equilibrium *given the lesion in the PPC* remains elusive.

5 | CONCLUSION

Patients with substantial chronic lesions to the PPC show slight impairments in accuracy and speed in their first attempt to fixate a visual target location. Nevertheless, they show the ability to overcome this initial impairment by employing a (corrective) consecutive saccade (S2). Whereas retinal feedback may be the most prominent force to drive the initiation of an S2, it is no requirement per se; when patients could only rely on extraretinal signals to initiate the S2, they still showed the ability to do so. Although earlier studies claimed profound impairments in employing secondary saccades, we show that patients with a lesion to

the PPC are still able to rely on solely extraretinal signals to overcome primary saccade inaccuracy.

AUTHOR CONTRIBUTIONS

Sanne Böing: Data curation; formal analysis; investigation; methodology; project administration; software; visualization; writing—original draft; writing—review and editing. **Jasper Fabius:** Conceptualization; data curation; formal analysis; project administration; software; supervision; writing—review and editing. **Marjoleine Hakkenberg:** Conceptualization; investigation; methodology; writing—review and editing. **Tanja C. W. Nijboer:** Conceptualization; funding acquisition; resources; supervision; writing—review and editing. **Stefan Van der Stigchel:** Conceptualization; funding acquisition; resources; supervision; validation; writing—review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare no competing financial interests.

PEER REVIEW

The peer review history for this article is available at <https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/ejn.16121>.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available in Open Science Framework at <https://osf.io/hua6t/>.

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