Perceptual restoration fails to recover unconscious processing for smooth eye movements after occipital stroke

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Abstract

Visual pathways that guide actions do not necessarily mediate conscious perception. Patients with primary visual cortex (V1) damage lose conscious perception but often retain unconscious abilities (e.g. blindsight). Here, we asked if saccade accuracy and post-saccadic following responses (PFRs) that automatically track target motion upon saccade landing are retained when conscious perception is lost. We contrasted these behaviors in the blind and intact fields of 8 chronic V1-stroke patients, and in 8 visually-intact controls. Saccade accuracy was relatively normal in all cases. Stroke patients also had normal PFR in their intact fields, but no PFR in their blind fields. Thus, V1 damage did not spare the unconscious visual processing necessary for automatic, post-saccadic smooth eye movements. Importantly, visual training that recovered motion perception in the blind field did not restore the PFR, suggesting a clear dissociation between pathways mediating perceptual restoration and automatic actions in the V1-damaged visual system.

Introduction

Human observers use eye movements to bring targets of interest to central vision for detailed inspection. For moving targets, they do so effortlessly, with a combination of rapid saccades and smooth eye movements. When observers acquire a moving target via a saccade, they can continue to track it with smooth eye movements that match the eye's velocity to the motion of the target (Buizza & Schmid, 1986; Lisberger & Westbrook, 1985; Lisberger et al., 1987; Rashbass, 1961; Tychsen & Lisberger, 1986). Those pursuit movements can show accurate velocity tracking, matched to the target from the moment of saccade landing, indicating motion integration and predictive planning for the target prior to the saccade (Gardner & Lisberger, 2001). Other smooth eye movements can occur involuntarily, as in ocular following, where the eyes drift after a saccade in response to the onset of wide-field motion (Gellman et al., 1990; Miles et al., 1986).

Recently, we found that saccade planning to peripheral, static apertures containing motion involuntarily generates predictive, smooth eye movements at the saccade target, even when there are no task demands to follow the target's motion (Kwon et al., 2019). These involuntary smooth eye movements anticipate the post-saccadic motion in the target aperture, generating a low-gain, following response along the target's motion direction, which we named the "post-saccadic following response" (PFR). The PFR appears to reflect automatic, unconscious visual processing that occurs during a saccade target's selection - i.e., during pre-saccadic planning (Kwon et al., 2019). Previous studies of pre-saccadic attention have shown that perceptual enhancements for the saccade target are automatic and obligatory (Deubel & Schneider, 1996; Kowler et al., 1995; Rolfs & Carrasco, 2012; Rolfs et al., 2011), and involve selection of target features such as spatial frequency, orientation (Li et al., 2016) as well as

motion features (White et al., 2013), which can drive smooth eye movements. Thus, the PFR may represent an automatic consequence of attentional selection for the motion target prior to the saccade; alternatively, it may play a role in priming the motor system for subsequent tracking of that target.

Both voluntary and involuntary smooth eye movements are thought to rely on processing of stimulus motion mediated through neural pathways in the middle temporal (MT) area (Bakst et al., 2017; Mustari et al., 2009; Newsome et al., 1985; Nuding et al., 2008). Area MT receives strong cortical inputs, routed through primary visual cortex (area V1), but it also receives direct input from sub-cortical centers, which bypass V1 (Glickstein et al., 1980; Maunsell & van Essen, 1983; Rodman et al., 1989; Sincich et al., 2004; Tamietto & Morrone, 2016; Ungerleider et al., 1984; Van Essen et al., 1981). To what extent these different routes of information that are transferred to MT contribute to voluntary and involuntary, smooth eye movements, and to perception, remains to be fully elucidated. Notably, prior studies have suggested that motion pathways driving involuntary smooth eye movements differ from those mediating perception (Glasser & Tadin, 2014; Price & Blum, 2014; Simoncini et al., 2012; Spering & Carrasco, 2012; Spering et al., 2011). As such, the fact that MT receives inputs from sub-cortical centers and from other cortical areas (via V1) prompted the hypothesis that sub-cortical pathways to MT may support smooth eye movements while conscious visual motion perception relies predominantly on input routed via V1 (Spering et al., 2011). Damage to V1 as a result of unilateral occipital stroke offers a unique opportunity to test this hypothesis in humans. Indeed, unilateral V1-strokes cause a loss of conscious visual perception in the contralateral visual hemifield (Smith, 1962; Teuber et al., 1960), but sub-cortical projections to MT are generally spared. Importantly, these projections are thought to underlie the preservation of unconscious residual abilities such

as blindsight (Mazzi et al., 2019; Sanchez-Lopez et al., 2019; Tamietto & Morrone, 2016; Weiskrantz et al., 1974). A key role of MT in blindsight has also been inferred from the particular stimulus properties needed to elicit blindsight: visual targets presented in the blind-field have to be relatively large, coarse, moving or flickering (Weiskrantz et al., 1995), containing high luminance contrasts, low spatial frequencies and high temporal frequencies (Sahraie et al., 2008) – stimulus properties that elicit strong responses from MT neurons (Born & Bradley, 2005; Movshon & Newsome, 1996). However, the impact of V1 damage on unconscious, motion-dependent visual processes used to guide smooth eye movements, such as the PFR, has not been investigated.

Moreover, although visual training can restore conscious visual motion perception in parts of the blind field of V1-stroke patients (Cavanaugh et al., 2019; Cavanaugh et al., 2015; Das et al., 2014; Elshout et al., 2016; Huxlin et al., 2009; Saionz et al., 2020; Vaina et al., 2014), we do not know how effectively patients can use such restored percepts to guide actions. Here, we used a cued, saccade task to demonstrate remarkably preserved ability of trained, V1-stroke patients to correctly target motion-containing peripheral stimuli presented in both their intact and blind-fields. However, by continuously tracking eye movements, we were also – for the first time – able to capture the impact of V1 damage on the PFR. Our unique data reveal a key role for V1 in this unconscious, automatic, oculomotor behavior. By the same token, they provide new insights into the likely neural pathways mediating restored, conscious, motion perception after V1 damage *versus* those involved in the predictive processing necessary for a normal PFR.

Results

To investigate how V1 damage impacts unconscious motion processing for smooth eye movements, we contrasted the PFR for saccades made to motion stimuli placed in the intact and the blind-fields of eight V1-stroke patients (**Fig. 1**) after visual restoration training on a global motion discrimination task.

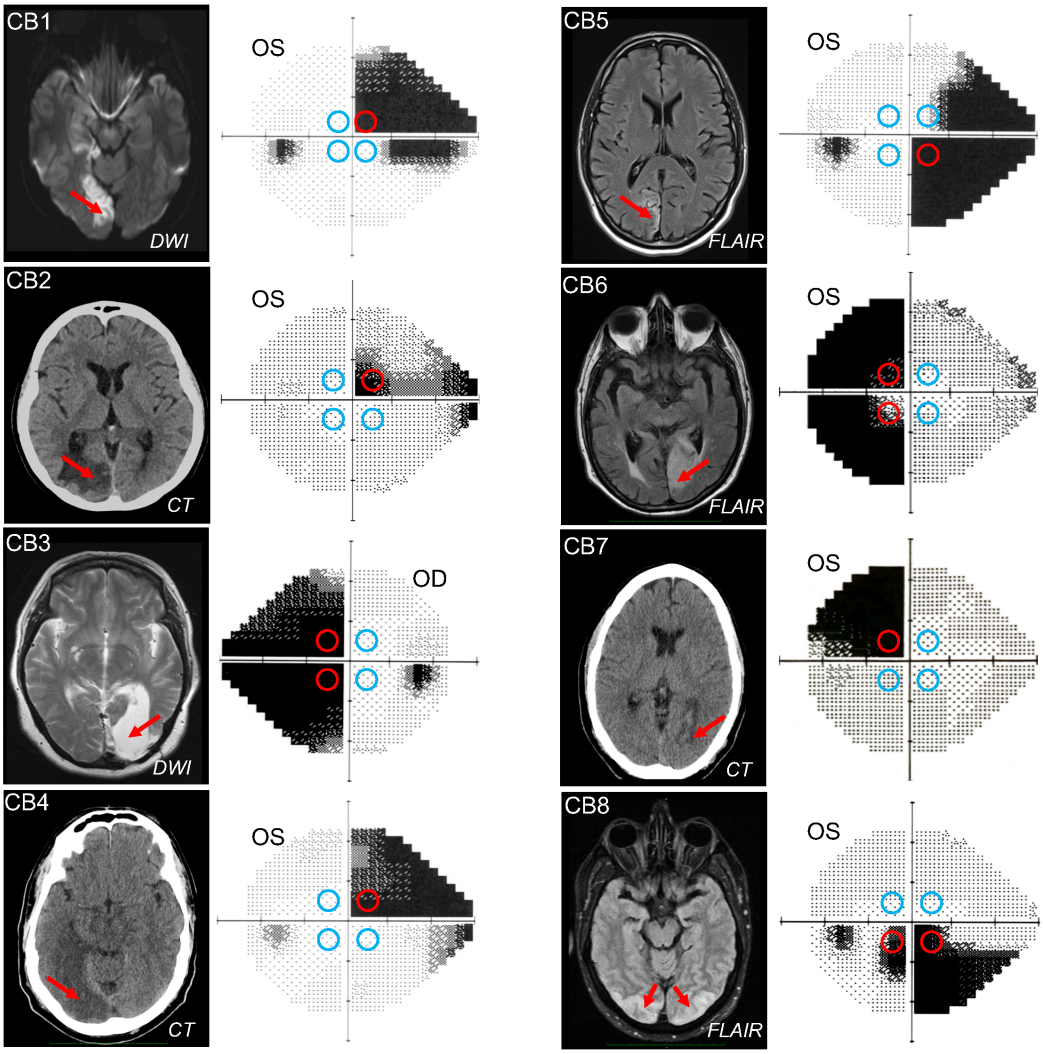


Figure. 1 | Occipital lesions, Humphrey visual field maps and PFR testing locations. Single radiographic images through the brains of each V1-stroke participant, illustrating region(s) of occipital damage (red arrows), shown with right brain hemispheres on image right. The location, size and shape of visual stimuli presented during the PFR testing protocol are indicated by colored circles superimposed on initial 24-2 Humphrey visual field maps acquired for the tested eye in each case. Red circles: blind-field

testing locations; blue circles: intact field locations; OS: left eye; OD: right eye; DWI: diffusion-weighted imaging; FLAIR: T2-weighted fluid-attenuated inversion recovery; CT: computed tomography.

We identified four optimal testing locations in the intact and blind fields of each participant using Humphrey perimetry **(Fig. 1)**, and then measured motion discrimination and integration thresholds using random dot stimuli **(Fig. 2A)** at these locations.

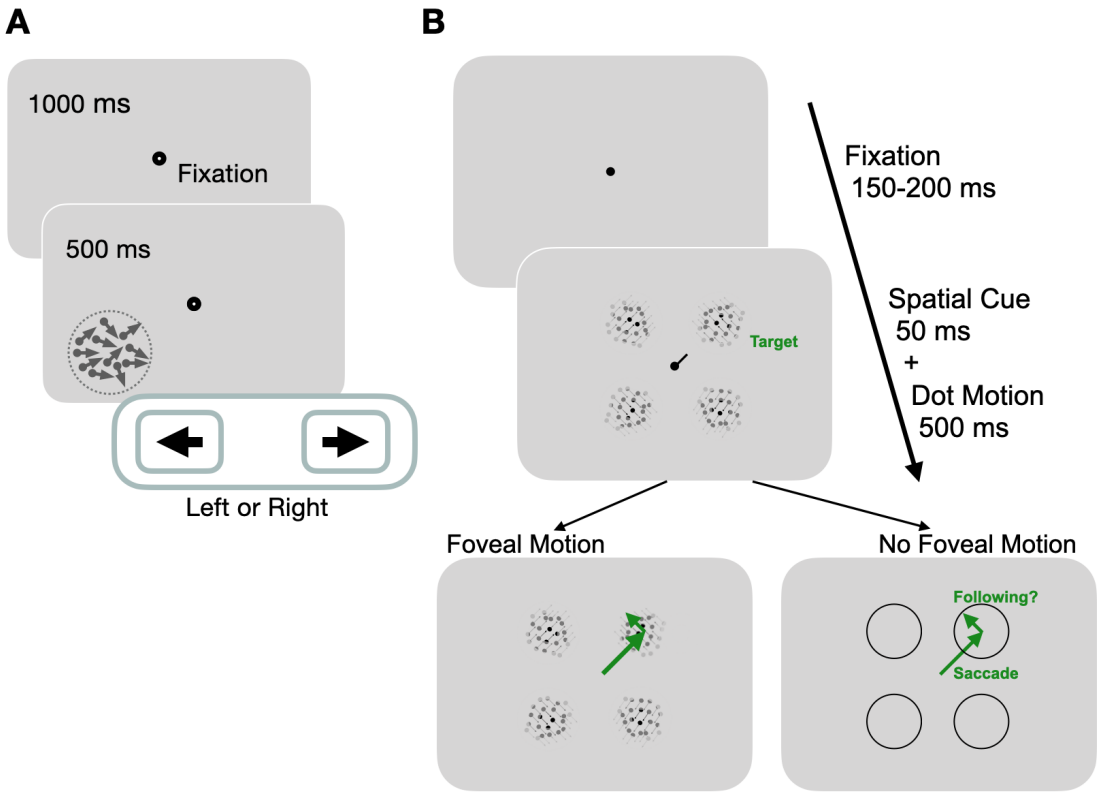


Figure. 2 | Experimental paradigms for measuring motion perception and oculomotor functions. A: Trial sequence for assessing global motion perception: trials started with a fixation period of 1000 ms, followed by appearance of a random dot stimulus either in the blind or intact field for 500 ms. Dots moved globally to the right or left, with a range of directions defined an adaptive staircase. On each trial, subjects were asked to report the stimulus' global direction of motion by pressing the left or right arrow keys on a keyboard. They received auditory feedback on the correctness of each response. **B:** Trial sequence for assessing oculomotor behavior: each trial started with a variable fixation period after which, participants were presented with 4 equi-eccentric motion apertures and a spatial cue at fixation. Dot motion apertures were Gaussian-enveloped and contained 100 % coherent motion along a randomly assigned direction (clockwise or counter-clockwise) in each aperture, that was tangential to the center-out

saccade to the aperture. The spatial cue (50 ms) indicated a peripheral target aperture towards which the participant was instructed to initiate a saccade as fast as possible. In half the trials, the dot motion stimuli persisted for 500 ms, and were thus present upon saccade offset. In remaining trials, stimuli disappeared during saccade flight, such that no stimulus motion was present at the fovea upon saccade landing.

In the blind fields, depending on whether the tested locations overlapped with a previously-trained location, performance was either at chance or had improved to measurable and sometimes near-normal thresholds, assessed in each patient's own intact visual field **(Table 1)**. The end result was a set of 11 blind-field locations across 8 patients, where perceptual thresholds ranged from unmeasurable (reflecting inability to do the task) to near-normal.

Table 1. Demographics and global motion integration thresholds in retrained, V1-stroke participants. M, male; F, female; NDR, normalized direction range (low value=best performance, 1=bad performance). Each NDR threshold denotes performance measured at a single blind- or intact-field location in each patient (see **Fig. 1** for positioning of these locations relative to the pre-training Humphrey visual field).

Subject	Sex	Age (years)	Time post-stroke (months)	Blind-field NDR thresholds	Intact field NDR thresholds
CB1	F	27	65.0	1	0.3
CB2	F	68	24.8	0.2	0.2
CB3	F	57	48.6	0.7, 0.8	0.3
CB4	M	66	32.2	0.5	0.2, 0.2, 0.2
CB5	M	54	52.2	1	0.3, 0.3
CB6	M	79	22.7	1, 0.1	0.1, 0.2
CB7	M	53	36.8	1	0.3
CB8	M	52	65.5	1, 0.2	0.1, 0.2

Oculomotor behavior was measured using a cued-saccade task for motion stimuli at 4 peripheral locations (colored circles in **Fig. 1; Fig. 2B**). Of particular note, the motion direction inside the apertures was irrelevant to the task and subjects were not asked to track it or report it. Additionally, in half the trials, the motion stimulus disappeared during saccade flight to

disambiguate the contributions of pre-saccadic motion processing to the post-saccadic following response.

Basic saccade behavior of V1-stroke participants – accuracy and latency

Stroke participants were generally able to use central spatial cues at fixation to plan saccades to peripheral aperture locations. Nonetheless, they made slightly more correct saccades to targets in their intact-field (mean 96.2 ± 4.2 %) than to those in their blind-field (mean 86.9 ± 16.7 %), likely reflecting more reliable target identification in the intact field ($t(22)=2.93$, $p=0.008$). However, when correctly selecting targets in their blind-fields, there was high spatial accuracy of saccades to those targets, as measured by the location of the end-points relative to the aperture center. Specifically, stroke participants had a mean absolute landing error relative to the stimulus center of 1.31 ± 0.08 deg (SEM) in the intact field and 1.39 ± 0.16 deg (SEM) in the blind-field, which was not significantly different ($t(7)=-0.703$ $p=0.505$). The latency of saccades was also similar for blind- and intact-field targets (intact-field latencies: 365 ± 49 ms; blind-field latencies: 391 ± 58 ms; $t(30)=1.37$, $p=0.182$).

Finally, we compared saccade accuracy and latency of stroke patients to those of visually-intact controls from a previous study (Kwon et al., 2019). Saccade accuracies in the intact fields of stroke patients were slightly impaired from those of visually-intact controls ($t(14)=-2.16$, $p<0.05$) who exhibited saccade accuracy of 98.7 ± 0.8 % as compared to 96.2 ± 4.2 % for stroke patients. In addition, stroke patients had significantly longer ($t(14)=5.37$, $p<0.0001$) saccade latencies of 365 ± 49 ms in their intact fields, compared to visually-intact controls, whose latencies averaged 260 ± 10 ms (Kwon et al., 2019). We consider several possible causes

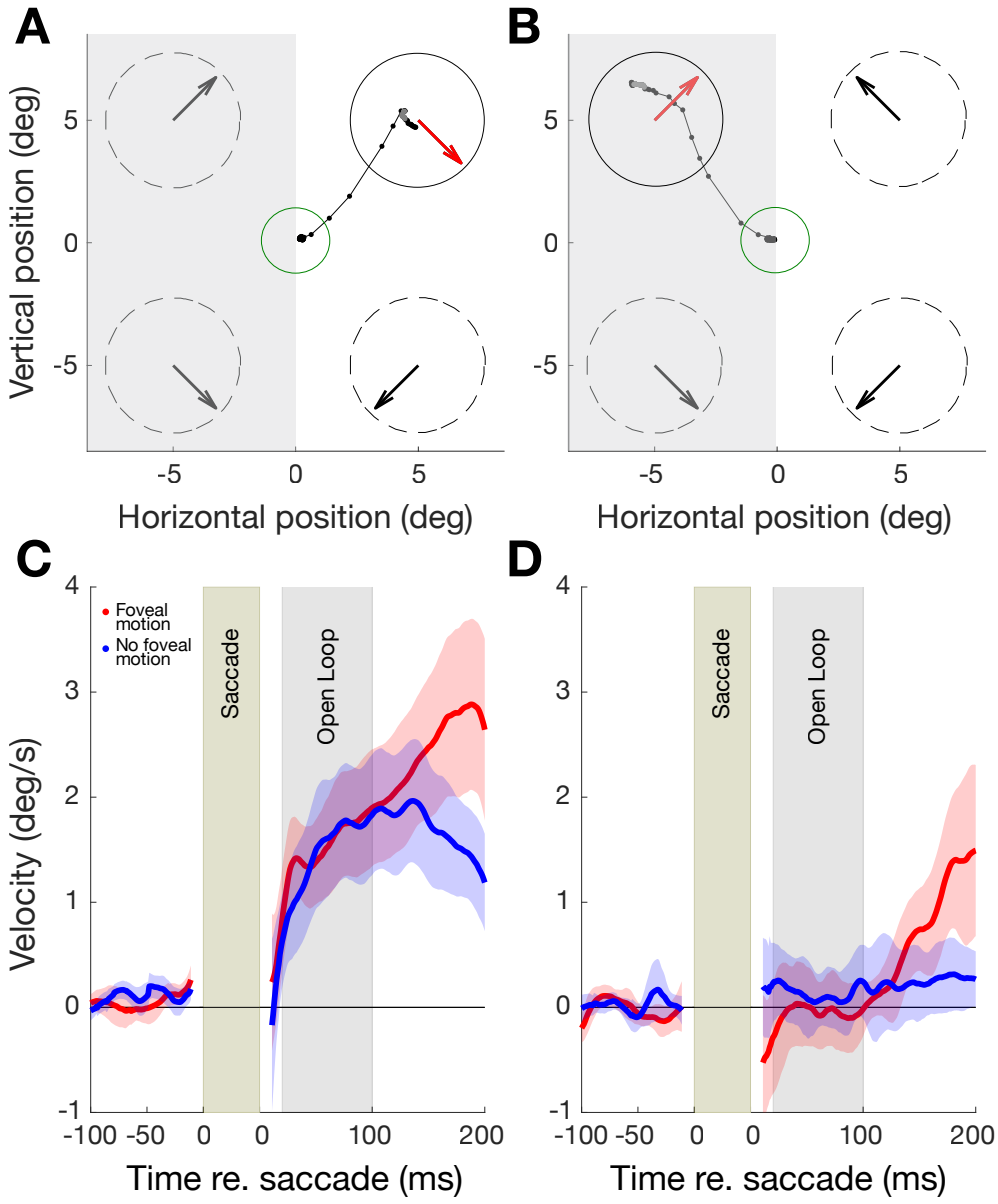
for these differences in the Discussion, including age, and challenges specific to saccade planning in the presence of a blind-field.

Predictive oculomotor behavior in the intact field of V1-stroke participants

In intact portions of their visual fields, stroke patients' post-saccadic smooth eye movements reflected the direction of target motion of the pre-saccadic stimulus immediately upon saccade offset. In a typical trial, a saccade made to a target aperture in the intact field exhibited a smooth drift in eye position from the saccade end-point along the direction of target motion (see example for a single stroke patient in **Fig. 3A**). We quantified the time course of the drift in eye position by computing the eye velocity projected along the direction of target motion, where positive values reflect following of motion. We term this the PFR velocity. Across patients, we observed a net positive PFR velocity (**Fig. 3C**). By including a stimulus manipulation in which the motion target disappeared during saccade flight, we were able to confirm that the PFR velocity was driven exclusively by pre-saccadic motion in the peripheral aperture. Within the first 100 ms after saccade offset (*the "open loop" period*) the PFR velocity did not differ depending on whether the stimulus remained present after the saccade (red trace in **Fig. 3C**) or if it was removed during saccade flight (blue trace in **Fig. 3C**). By including trials where we removed the stimulus in-flight, we could eliminate direct post-saccadic stimulation of motion at the fovea, and thus isolate the predictive, "open loop" component of the PFR velocity. After 100 ms from saccade offset, the presence or absence of foveal motion did influence post-saccadic eye movements. Specifically, the PFR velocity continued to increase along the target motion

194 direction in the foveal-motion-present condition (red trace in **Fig. 3C**), whereas it decreased

195 when no stimulus was present upon saccade landing (blue trace in **Fig. 3C**).



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197 **Figure. 3 | Oculomotor behavior in perceptually-trained, V1-stroke participants. A**: Eye-movement

198 traces to a cued target in the intact field (white background) of a single V1-stroke patient in a stimulus

199 absent condition (i.e. with no foveal motion upon saccade landing at the cued target). Small, connected

200 black dots denote the raw eye movement sampled from our eye tracker; the green circle represents the

201 electronic window around the fixation spot; random dot stimuli were presented inside the 4 dashed circles

202 and their global motion direction is indicated by large arrows inside each circle. Note the accurate saccade

to the target center, and how the eye follows the pre-saccadic target motion direction (red arrow) upon saccade landing. **B**: Raw eye movement traces to a cued target in the blind-field (grey background) of a single V1-stroke patient in a stimulus absent condition. Note successful saccade landing onto the cued target but how the eye fails to follow the pre-saccadic target motion direction. Labeling conventions as in A. **C**: Eye velocity traces for saccades to intact portions of the visual field, averaged across all 8 stroke patients. In half the trials, stimuli were present upon saccade landing, resulting in foveal motion (red trace). In the remaining trials, stimuli were absent - i.e. there was no foveal motion upon saccade landing on the target (blue trace). Error bars = 2 SEM across subjects. Average eye velocities were projected along the target motion direction time-locked prior to the saccade onset (-100 to 0 ms) and offset (0 to 200 ms), such that positive values reflected motion consistent with the stimulus, while negative values reflected motion opposite. **D***:* Eye velocity traces for saccades to blind portions of the visual field, averaged across all 8 stroke patients (same conventions as in A). Error bars = 2 SEM across subjects. Note the near-zero eye movement velocity during the open loop period, reflecting the lack of PFR.

For subsequent analyses, we focused on the "open-loop" period (i.e., within 100 ms after saccade offset), as the PFR velocity in this period depends on pre-saccadic motion information accumulated from the peripheral target aperture (Kwon et al., 2019). The average velocity of these open-loop following responses, both for stimulus-present and -absent trials, was small in magnitude relative to the stimulus speed (10 deg/sec), ranging from 5-15 % relative velocity gain. These lower gain responses are consistent with an involuntary following response, such as ocular following (Gellman et al., 1990; Miles et al., 1986), rather than voluntary pursuit of a target. Overall, the pattern of oculomotor behavior in the intact field of stroke patients was highly similar in its time-course and magnitude to that previously measured in visually-intact controls (Kwon et al., 2019).

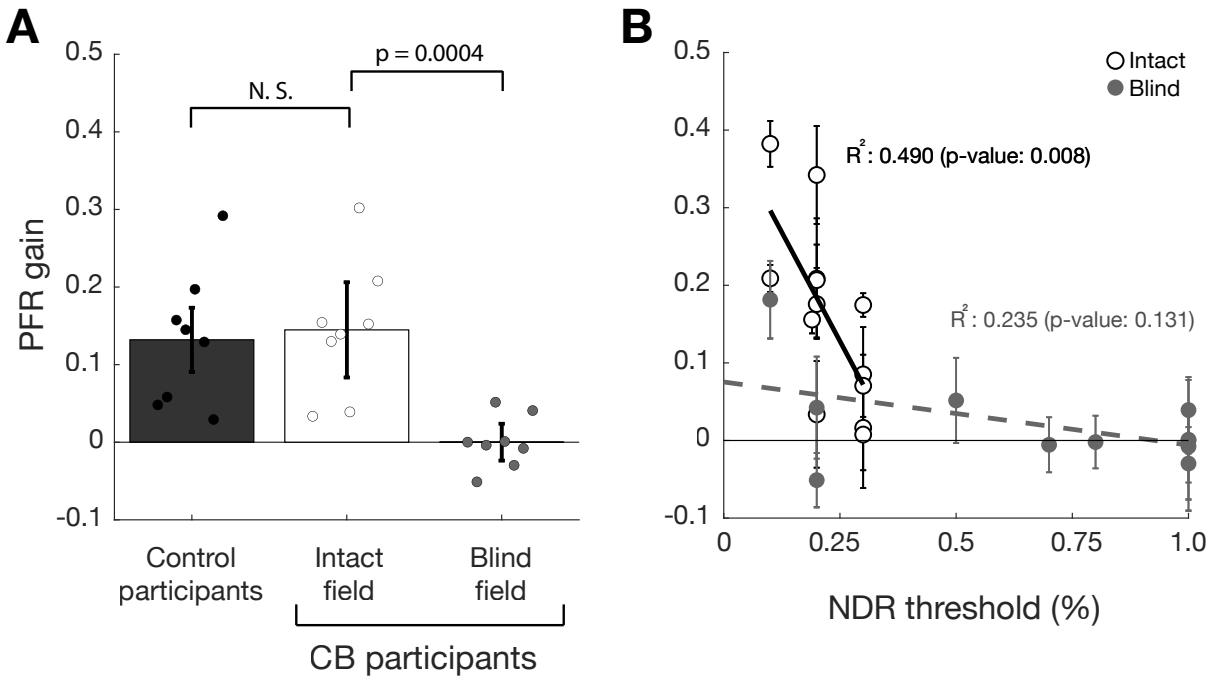

Figure. 4 | PFR gain in the intact and blind-field of V1-stroke participants. A*:* Plot of mean PFR gain in visually-intact controls (Kwon et al., 2019), and in V1-stroke patients' intact and blind-fields during the open-loop period. The PFR gain is here represented as a proportion of pursuit gain along the target motion direction (1 = perfect following, 0 = no following, -1 = following in the opposite direction). Individual dots represent the mean PFR for each participant. There was no significant difference in PFR gain between the intact fields of stroke patients and visually-intact controls. PFR gain approximated 0 for saccades to targets in stroke patients' blind-fields. Error bars represent 2 SEM across subjects. **B***:* PFR gain for individual intact field locations (open circles) and blind-field locations (grey circles) as a function of the normalized direction range (NDR) threshold measured at each location in stroke participants. Within intact regions of the visual field, there was an inverse correlation between this global motion threshold and PFR gain: the lower the threshold (i.e. the better the motion perception), the higher the PFR gain. This relationship was lost in the blind-field of stroke patients, with no significant correlation between NDR thresholds and PFR gain – the latter remaining abnormally low.

Visually-intact controls in our prior study had a net positive PFR gain in the open loop epoch that differed significantly from zero ($t(7)=4.31$, $p=0.004$ – dark grey bar in **Fig. 4A**). PFR gain in the intact field of stroke patients (white bar in **Fig. 4A**) was not significantly different from PFR

gain in our prior, visually-intact controls ($t(14)=-0.29$, $p=0.77$), and also showed a net positive effect that differed significantly from zero ($t(7)=4.72$, $p=0.002$). Finally, we noted a significant correlation ($R^2 = 0.49$, $p=0.008$) between perceptual performance measured by NDR thresholds, and the magnitude of the PFR (white circles, **Fig. 4B**) in the intact field of stroke participants.

Predictive oculomotor behavior in the blind-field of V1-stroke participants

Although saccades landed correctly on target stimuli in the blind-field of our stroke participants ~87 % of the time, post-saccadic eye movements differed dramatically from those in the same participants' intact field. Specifically, in the open loop period, the eyes no longer moved along the direction of motion in the target (see example in **Fig. 3B**) – in other words, there was no positive PFR velocity. This pattern was reflected in the average PFR velocity across all 8 stroke patients' blind-fields (**Fig. 3D**), irrespective of visual rehabilitation training. In contrast to oculomotor behavior when making a saccade to targets in their intact fields, stroke patients did not show any positive PFR velocity within the first 20-100 ms after saccade offset ("open-loop" period), whether the motion stimulus was present (red trace) or absent (blue trace) post-saccadically (**Fig. 3D**).

Beyond 100 ms from the saccade offset, post-saccadic foveal motion – when present – was sufficient to drive an ocular following response (red trace, **Fig. 3D**). This is an important observation, as it confirms that post-saccadic ocular following remained functional in these patients. Only the predictive component during the open-loop period was abnormal, reflected by the absence of the PFR velocity in stimulus-absent trials (blue trace, **Fig. 3D**). Consistent with these observations, PFR gain in the blind-field of our stroke patients (**Fig. 4A**) was significantly

lower than PFR gain in their intact field ($t(7)=4.2$, $p=0.004$), and was not significantly different from zero ($t(7)= 0.01$, $p=0.992$).

Since portions of the blind fields of stroke patients underwent training that restored global motion perception - indicated by NDR thresholds <1 **(Table 1)** - we next asked if such training restored PFR gains. The answer was no: there was no significant correlation between restored NDR and PFR gains ($R^2 = 0.235$, $p=0.131$) in the blind field of stroke patients (black points, **Fig. 4B**). At blind-field locations where post-training stroke patients attained NDR thresholds <1, the PFR gain was not significantly different from 0 (mean PFR gain = 0.036 ± 0.033, $t(5)=1.10$, $p=0.320$). Even when we isolated blind-field locations where training restored "normal" NDR thresholds (<0.3), contrasting them against blind-field locations where NDR thresholds were >0.3, PFR gain was not significantly different between those groups ($t(4)=0.61$, $p=0.575$). Thus, over several tests, we found no significant recovery of PFR gain with recovery in perceptual motion performance.

Discussion

For the first time, the present study measured the impact of V1 damage and subsequent, training-induced, visual restoration on both voluntary saccadic behavior and a class of unconscious, automatic, post-saccadic smooth eye movements: the PFR. Our findings revealed an unexpected, critical reliance of pre-saccadic visual motion processing on visual pathways that include V1. We confirmed that V1-stroke patients exhibit normal saccade accuracy and normal PFR when saccading to motion targets in intact regions of their visual fields, where vision is mediated by intact V1. However, while the same patients exhibited normal saccade accuracies for targets presented in their blind-fields, they had no measurable PFR, even after visual discrimination training recovered global motion perception at those blind-field locations. Thus, restoration of motion perception did not automatically restore the unconscious visual motion processing necessary for the PFR. This is surprising because traditionally, patients with V1 damage are well-known for having preserved, unconscious visual processing in their blind-fields – under the umbrella of blindsight phenomena (reviewed in Weiskrantz, 1996, 2009). That visual discrimination training can recover the ability to perform the relatively complex computations needed to integrate motion direction into a global percept available to consciousness – even to the extent of attaining normal NDR thresholds - inside chronic blind-fields is remarkable. That this could occur without automatically restoring the unconscious global motion processing necessary for predictive smooth eye movements, was unexpected. Our findings suggest that primary visual cortex (V1) may be key for both conscious visual perception (Tong, 2003) and for unconscious visual processes that influence smooth eye movements induced by peripherally-presented motion targets. They also suggest that visual restoration, after V1 damage, recruits

different neural circuits than are normally used for these processes in the intact visual system; finally, it suggests that these newly-engaged circuits now dissociate conscious and unconscious visual motion processing.

The extrastriate visual area critical for many aspects of visual motion processing - area MT - receives strong inputs from V1 as well as from sub-cortical projections that bypass V1 (Glickstein et al., 1980; Hagan et al., 2019; Maunsell & van Essen, 1983; Rodman et al., 1989; Sincich et al., 2004; Ungerleider et al., 1984; Van Essen et al., 1981). This diversity of inputs to MT likely explains why, after V1 damage, residual visual motion processing persists inside the resulting blind fields (reviewed in Das & Huxlin, 2010; Melnick et al., 2016; Tamietto & Morrone, 2016). Key for rehabilitation efforts, this residual processing can be leveraged by intensive visual training to recover both simple and complex motion perception (Cavanaugh et al., 2019; Das et al., 2014; Huxlin et al., 2009; Saionz et al., 2020).

In non-human primates, activity in area MT has been causally linked to perceptual reports of motion in discrimination and detection tasks (Britten et al., 1992; Newsome et al., 1985; Salzman et al., 1990; Siegel & Andersen, 1986), and to accuracy in pursuit eye movements (Huang & Lisberger, 2009; Newsome et al., 1985; Osborne et al., 2005; Salzman et al., 1990; Siegel & Andersen, 1986). The perception of velocity is also well correlated with velocity gain in voluntary pursuit, supporting the notion that pursuit and perception share common motion processing at the neural level (Gegenfurtner et al., 2003; Spering et al., 2005; Stone & Krauzlis, 2003). Voluntary pursuit and involuntary ocular following responses, such as the PFR, are also thought to rely on motion processing by the dLGN, V1, as well as area MT and the medial superior temporal (MST) area (Bakst et al., 2017; Mustari et al., 2009; Nuding et al., 2008; Takemura et al., 2007). Just as pursuit is modulated by stimulus contrast (Spering &

Gegenfurtner, 2007; Spering et al., 2005), the PFR also exhibits a dependence on stimulus contrast; in visually-intact humans, we saw a steep rise of the PFR contrast response function starting right below 10 % luminance contrast, quickly reaching saturation at or above 15 % contrast (**Supplementary Fig. 1**). This is comparable to the contrast response function of neurons in macaque area MT (Heuer & Britten, 2002; Kohn & Movshon, 2003; Sclar et al., 1990). However, pathways for ocular following are thought to be at least partly non-overlapping with those involved in motion perception (Glasser & Tadin, 2014; Price & Blum, 2014; Simoncini et al., 2012). Other studies in humans also support a distinction between neural circuits underlying smooth eye movements and conscious motion percepts (Spering & Carrasco, 2012; Spering & Gegenfurtner, 2007; Spering et al., 2011). Consistent with those studies, we now find that despite recovery of processing used for accurate, global motion perception in the blind-field of V1-damaged humans, the PFR remains absent at trained, blind-field locations.

One possible explanation for this outcome is that while training post-stroke improved processing for perception, it did not correct problems with pre-saccadic attention and/or other aspects of saccade planning. It is well established that sensory processing among neurons in MT and MST can be strongly influenced by attention (Treue & Maunsell, 1996; Treue & Trujillo, 1999) and also by target selection immediately prior to saccades (Ferrera & Lisberger, 1997; Recanzone & Wurtz, 2000). Recent studies suggest that like target selection in voluntary pursuit, selective attention can modulate ocular following responses (Souto & Kerzel, 2014), and our findings here and previously (Kwon et al., 2019) support this notion. Pre-saccadic attention is thought to operate through feedback from oculomotor planning areas to visual cortex (Moore & Armstrong, 2003; Moore & Fallah, 2004), and while its impact has been studied mainly in visual area V4, it is also thought to occur in MT/MST. Indeed, electrical micro-stimulation in an

oculomotor area, the frontal eye fields (FEF), influences selection of motion signals prior to saccades and can alter subsequent saccade trajectories to favor stimulus motion (Schafer & Moore, 2007). That the FEF and its projections to area MT are intact in V1-stroke patients suggests preservation of pre-saccadic planning and attention selection for the saccade target even when visual input is weak or abnormal in a blind field.

Although the effects of attention have not been studied extensively in V1-damaged patients, work to date suggests that some attentional mechanisms remain functional within cortical blind-fields; as such, they could modulate motion signals at the level of MT/MST in the current behavioral paradigm. For instance, covert spatial attention was reported to improve stimulus detection in the blind field (Poggel et al., 2006) and in a separate study, it was shown to significantly decrease reaction times in V1-stroke patients performing an orientation discrimination task without any speed-accuracy trade-off (Kentridge et al., 2004). Feature-based attention was also able to improve fine direction discrimination training in cortically-blinded fields (Cavanaugh et al., 2019). One piece of evidence suggesting that pre-saccadic attention remains functional in the current experiments is that other aspects of saccade pre-planning related to perceptual shifts in the position of motion targets, remain in the blind-field. Previous studies reported a motion-induced perceptual shift for stimulus location along the direction of stimulus motion (De Valois & De Valois, 1991; Nishida & Johnston, 1999; Ramachandran & Anstis, 1990; Whitney & Cavanagh, 2000), which for saccades is reflected by a shift in their end-points along the direction of target motion (Kosovicheva et al., 2014; Kwon et al., 2019; Schafer & Moore, 2007). For stroke patients, we confirmed similar shifts in saccade end-points in their intact-fields, as well as significant, albeit reduced shifts along the target motion in their blind-fields (**Supplementary Fig. 2**). Thus, it seems unlikely that the lack of PFR reflects an

372 impairment to engage attention in motion processing circuits. Instead, we posit that perceptual
373 recovery through repetitive discrimination training did not entrain the specific motion processing
374 pathways that support post-saccadic following.

375 An important consideration for the present experiments was whether failures to elicit
376 PFRs in cortically-blinded portions of the visual field might simply reflect a motor deficit for
377 accurately targeting peripherally-presented motion apertures. Because the PFR requires pre-
378 saccadic attention to select the target motion, any loss in target localization accuracy could
379 impair selection. Previous studies in monkeys with V1 lesions have found reduced spatial
380 accuracy for saccades made into the blind-field with larger end-point errors from 0.2 to 0.6
381 degrees at matched eccentricities for intact and blind-fields (estimated from data in Fig. 2 of
382 Yoshida et al., 2008). In the present study, we observed that stroke patients were less likely to
383 correctly *select* a target aperture in their blind *versus* intact fields when given a central spatial
384 cue (87 % vs. 96 %). However, when the target was correctly selected, spatial accuracy of the
385 saccade end-points was normal (1.31˚ *versus* 1.38˚ absolute error relative to the target's center).
386 A key difference in the prior study is that Yoshida *et al.* (2008) used smaller stimuli, measuring
387 only 0.45 degrees in diameter, while we used large, dot-motion fields (5.5 degrees in diameter,
388 Gaussian enveloped - ***see Methods***). Because spatial accuracy of saccade landings was similar
389 for blind and intact fields of our stroke participants, we conclude that a motor deficit for
390 targeting peripheral motion apertures in cortically-blind regions of the visual field was not a
391 likely explanation for the absence of a PFR.

392 The slightly larger number of errors made by stroke patients when selecting cued targets
393 in their blind-fields could also be consistent with a reduction in their relative, perceived salience.
394 Although all 4 motion apertures appeared simultaneously, iso-eccentrically and had equal

[~100 %] luminance contrast, it is possible that stroke participants required extra effort to ignore blind-field-related perceptual inhomogeneities between the four apertures. Indeed, a prior study in chronic V1-stroke patients showed depressed luminance contrast sensitivity for motion and orientation discriminations at trained, blind-field locations, in spite of normal NDR thresholds at these locations (Das et al., 2014). Thus, when patients are cued to saccade to a presumably less salient target in their blind-field, this may require additional effort to suppress a reflexive saccade to the more salient targets. In an anti-saccade task where a salient target is ignored in order to plan a cued movement to the opposite [but empty] visual field, there is typically a reduction in saccadic reaction time (Hallett, 1978; Munoz & Everling, 2004). If saccadic reaction times slow down by 140 ms or more due to task difficulty, this impacts saccades to both visible and anti-saccade locations due to the extra volitional demands (Hallett & Adams, 1980). In line with this observation, we found that saccade reaction times were slower for stroke patients by roughly 100-130 ms, both for the intact and blind-fields, relative to visually-intact controls. However, controls did differ in age from stroke patients (~20 years old *versus* ~57 years old), and prior work showed a correlation between slower saccade latency and growing age; however, the typical reduction from 20 to 80 years of age was 40-45 ms (Abel et al., 1983; Pirozzolo & Hansch, 1981; Spooner et al., 1980). Therefore, it appears unlikely that age alone would account for the >100 ms reduction in saccadic reaction times in stroke patients. Rather, we posit that patients had to exert greater volitional control to select the cued target inside blind regions of their visual field.

In summary, V1 damage in humans, such as occurs from occipital stroke, causes a dramatic loss of conscious visual perception across large regions of the visual field, impairing most aspects of daily living. Paradoxically, this condition was famously known for its relative

418 preservation of unconscious visual processes, such as those mediating blindsight. With the
419 advent of visual restoration training for this patient population, an important question in the field
420 has been to ascertain what aspects of visual processing can recover, which cannot, and why.
421 Peripheral visual motion processing is key to many aspects of daily living. Not only is it critical
422 for accurate perception and identification of targets, it is also essential for our motor actions and
423 reactions to these targets. Here we show that visual training that can restore perceptual
424 discrimination of peripheral motion does not automatically recover the PFR [or normal saccade
425 targeting to peripheral motion stimuli]. Our findings support a dissociation between smooth eye
426 movements, saccade targeting and perception following V1 damage, and suggest that V1 is
427 critical for driving smooth eye movements such as the PFR. A key realization emerging from
428 these results is that *alternative* pathways, which convey motion information from subcortical
429 centers directly to area MT, are insufficient to support predictive oculomotor behaviors when V1
430 is damaged, even if they are sufficient to mediate recovery of conscious motion perception.
431 A second insight attained presently is that repetitive motion discrimination training in CB
432 fields might only influence circuits and processes that supporting perception, without transfer to
433 those driving motion-dependent behaviors, such as the smooth eye movements involved in the
434 PFR. It remains to be determined if deliberate training on tasks that focus on saccade planning to
435 motion targets might recover predictive motor behaviors. Rehabilitation of predictive ocular
436 behaviors remains an uncharted area of research for V1-stroke patients, even though saccade
437 training is one of the few forms of rehabilitation more readily available to these patients
438 (Kerkhoff, 1999, 2000; Kerkhoff et al., 1992; Mannan et al., 2010; Nelles et al., 2001; Ong et al.,
439 2015; Pambakian et al., 2004; Roth et al., 2009; Sahraie et al., 2016; Spitzyna et al., 2007;
440 Trauzettel-Klosinski, 2010; Weinberg et al., 1977; Zihl, 1980). Of relevance to our observation

441 of an apparent dissociation between perception and eye movements after V1 damage, training
442 patients to saccade to targets in their blind-field does not induce perceptual recovery (Campion et
443 al., 1983; Pollock et al., 2019). Nonetheless, it is conceivable that an approach combining
444 perceptual training with training of ocular behaviors could improve the efficiency with which
445 patients use information from their blind-fields in everyday life.
446
447

Materials and Methods

Participants: eight participants with long-standing cerebral blindness (CB) were recruited 2 to 5 years after a stroke that damaged their V1 unilaterally or in one case, bilaterally (see **Table 1** for details). The location and nature of V1 damage was verified from clinical brain imaging performed as part of each patient's standard of care. Homonymous visual field defects were confirmed using monocular, Humphrey automated perimetry performed at the Flaum Eye Institute of the University of Rochester (**Fig. 1**). Participants suffering from neglect, cognitive impairments or ocular diseases were excluded from enrollment, as were those using psychoactive drugs. All participants had their visual acuity corrected to normal (with glasses or contact lenses) during testing.

Testing of V1-stroke participants occurred following completion of separate, visual restoration studies whereby they underwent visual training at one or more, blind-field locations. Some of these trained, blind-field locations overlapped with testing locations used in the present study. The end result was a set of 11 blind-field locations from 8 patients, where pre-training performance was initially at chance – i.e. participants were unable to discriminate left from right coherent, global motion. Post-training however, depending on whether the tested locations overlapped with a trained location, performance either remained at chance or improved, generating measurable and sometimes near-normal direction integration thresholds (**Table 1**). For comparison we also measured performance at a set of 13, iso-eccentric, intact-field locations (**Table 1;** see below for details of global motion assessment methods).

PFR data from stroke patients were contrasted with a previously published data set obtained from eight, visually-intact controls (18 to 22 years old; 4 females and 4 males) who had normal or corrected-to-normal vision(Kwon et al., 2019).

All procedures were approved by the Institutional Review Board of the University of Rochester and adhered to the tenets of the Declaration of Helsinki. Written, informed consent was obtained from each participant, and participation was at all times completely voluntary.

Apparatus & eye tracking for assessing global motion perception: participants were asked to perform 100 trials of a 2-alternative, forced-choice, left-*versus*-right, global direction discrimination task at 2 to 4, equi-eccentric, peripheral visual field locations chosen for testing of predictive oculomotor behavior (circles superimposed on Humphrey visual fields in **Fig. 1**; red: blind-field locations, blue: intact field locations). All blind-field locations were tested in each patient (red circles in **Fig. 1**). Time limitations restricted our ability to measure performance at every intact-field location (blue circles in **Fig. 1**), but at least one intact-field location was assessed in each participant. Across intact-field locations tested, we saw normal NDR thresholds that varied from 0.1-0.3 (**Table 1**). Percent correct and direction range thresholds were measured during in-lab testing, with central fixation enforced using an Eyelink 1000 eye tracker (SR Research, Mississagua, Ontario, Canada). Tracking was binocular for all participants except for CB3, who was tested monocularly because she exhibited convergence issues. As such, she had her dominant (right) eye tracked and the non-dominant eye patched both for motion perception and PFR testing. Stimuli were presented in a gaze-contingent manner in either intact or blind regions of the visual field. Viewing distance to a luminance-calibrated CRT monitor (HP 7217 A, 48.5 x 31.5 cm, 1024x640p, refresh rate 120 Hz) was 42 cm, enforced by a chin/forehead rest.

Experiments were conducted using MATLAB (The MathWorks, Natick, MA, USA) and the Psychophysics toolbox (Brainard, 1997; Kleiner et al., 2007; Pelli, 1997). At the start of each trial, subjects were asked to fixate a small target at the center of the CRT monitor. The Eyelink 1000 eye tracker was accurate to within 0.25^{o}, with a sampling frequency of 1000 Hz. Subjects were allowed a fixation window of only ± 1^{o} around the fixation spot. If gaze moved outside this window during stimulus presentation, the trial was aborted, reshuffled and patients received a noxious auditory tone as feedback, reminding them to improve their fixation accuracy.

Following accurate fixation of the central spot for 1000ms, a random dot stimulus appeared in a 5^{o} diameter circular aperture, at one of the pre-determined locations in the peripheral visual field (see colored circles in **Fig. 1**; NDR thresholds in **Table 1**). Black dots moved on a mid-grey background with a 250 ms lifetime, a speed of 10 deg/s, and with a density of 3 dots/deg^2. Stimuli were presented for 500 ms, accompanied by a tone to indicate stimulus onset. Dots moved globally with a variable range of directions, uniformly distributed around the left- or rightward vectors (Das et al., 2014; Huxlin et al., 2009; Saionz et al., 2020). On each trial, subjects were asked to report the stimulus' global direction of motion by pressing the left or right arrow keys on a keyboard (**Fig. 2A**). Task difficulty was adjusted using an adaptive staircase (Levitt, 1971), which increased the range of dot directions from 0^{o} to 360^{o} in 40^{o} steps after each set of 3 consecutive, correct responses; direction range was decreased by one 40^{o} step for every incorrect response (Das et al., 2014; Huxlin et al., 2009; Saionz et al., 2020). Auditory feedback was provided on each trial, indicating the correctness of each response. For each session, we fit a Weibull function to the data to generate a direction range threshold representing the direction range at which performance reached 75 % correct. Direction range thresholds were

then normalized to the maximum possible range of dot directions (360°), generating a normalized direction range (NDR) threshold, defined as:

NDR threshold (%) = (360°-Weibull-fitted direction range threshold)/360° x 100

Apparatus & eye tracking for PFR measurements: stimuli were generated using the Psychophysics toolbox in MATLAB 2015b on a PC computer (Intel i7 CPU, Windows 7, 8 GB RAM, GeForce Ti graphics card). They were presented on a gamma corrected display (BenQ X2411z LED Monitor, resolution: 1920x1080p, refresh rate: 120 Hz, gamma correction: 2.2) which had a dynamic luminance range from 0.5 to 230 cd/m^2, at a distance of 95.25 cm in a dark room. Brightness on the display was set to 100 % and contrast to 50 %, and additional visual features of the monitor such as blur reduction and low blue light were turned off. Gamma corrections were verified with measurement by a photometer. Position of the left eye was recorded continuously in all participants except for CB3, who had her right eye tracked (see above). Eye position was recorded at 220 Hz using an infrared eye tracker (USB-220, Arrington Research, Scottsdale, AZ, USA). The accuracy of the Arrington Eye Tracking system was 0.25˚, with a precision of 0.15˚. To minimize potential head movements, participants performed the task using a bite bar.

PFR stimulus and task: CB patients performed a centrally-cued saccade task towards peripheral motion apertures (**Fig. 2B**) as previously described in visually-intact controls (Kwon et al., 2019). In brief, and as schematically illustrated in **Fig. 2B,** trials were initiated by fixation of a small, dark, fixation spot presented on a gray background. After a variable fixation period of 150-200 ms, a saccade cue appeared at fixation together with four dot-motion apertures in a

square configuration (colored circles, **Fig. 1**). The cue (dark bar, 1˚ in length, extending from fixation) was used to indicate the target aperture to which the participant should saccade. Each target aperture was 5.5˚ in diameter and centered at (±5˚, ±5˚), with the exception of CB1, for whom the apertures were centered at (±3˚, ±5˚). There were 180 dots total in each aperture, with dot luminance set to 0.5 cd/m^2 (100 % contrast) and dot velocity fixed at 10 deg/s. Following parameters from our previous study (Kwon et al., 2019), a Gaussian envelope was applied to each dot-motion aperture to create a gradient in dot contrast from the center of the aperture (sigma= 1˚).

To avoid stereotyped eye movements, we varied saccade directions across trials. Thus, the spatially cued motion aperture could appear in the intact or blind-field of a given participant, on any given trial. Of particular note, *the motion itself or its direction were irrelevant to the task*. The motion within the aperture was 100 % coherent and ran along a direction that was tangential to an imaginary line from the fixation point to the aperture. For each aperture, the motion was selected independent of the other apertures in one of the two tangential directions relative to the center out saccade, either clockwise or counter-clockwise relative to the screen center.

We first compared eye movements in which the peripheral motion aperture was either present or absent upon saccade offset. Participants were instructed to make a saccade to the peripheral aperture as quickly as possible following the movement cue. A saccadic grace period (i.e., a maximum latency) was allowed for participants to initiate the saccade. In half the trials, selected at random, the stimulus motion remained present in all four apertures for 300 ms following detection of the eye landing within 3.5 visual degrees from the center of an aperture. In the other half of trials, the stimulus was removed as soon as the eye had been detected leaving the fixation window, thus leaving a blank screen through the post-saccadic period. A saccade

was labelled "correct" when it fell at least 3.5˚ from the saccade target center within 90 ms of the eye leaving the fixation window.

Eye movement recordings and PFR analysis: eye position data were collected as participants performed saccades from fixation to the peripheral target. Eye tracking and saccade detection procedures were identical to those previously published (Kwon et al., 2019). We sub-sampled eye position using the ViewPoint Matlab toolbox (Arrington Research) at the display refresh rate (120 Hz) to initiate gaze-contingent task events. For offline detection of saccadic eye movements, we used the full eye position data recorded at 220 hz and applied an automatic procedure that detected deviations in 2D horizontal and vertical eye velocity space (Engbert & Mergenthaler, 2006; Kwon et al., 2019). Only the trials where the saccade was labelled "correct" were included in the PFR analysis. We then focused our analysis by time locking eye velocity traces on intervals 200 ms prior to saccade onset and 200 ms following saccade offset. Details for eye position filtering, smoothing, and saccade detection were as previously described (Kwon et al., 2019). In brief, the 2D eye velocity was computed from smoothed eye position traces and then projected onto the motion vector in the target aperture on each trial. These projected velocity traces were then aligned to saccade onset or offset, and averaged across trials for each participant.

To quantify the net target-related eye velocity in each trial, we used a second measure of eye velocity that did not involve any filtering or smoothing of eye position. We computed a vector for the PFR in units of velocity (deg/sec) as the 2D vector difference in the raw (non-smoothed) eye position from 20 to 100 ms after saccade offset normalized by that time interval. Excluding the first 20ms after saccade offset from this analysis interval reduced the influence of

583 saccade related effects to instead focus on post-saccadic smooth movements. Like velocity
584 traces, we projected this 2D vector onto the vector of the target's motion to produce a single
585 velocity value along the axis of stimulus motion, which we term the 'open-loop' PFR (Kwon et
586 al., 2019). To assess the average PFR across trials, we computed each CB patients' eye
587 movements relative to the target motion direction so that positive average eye velocities meant
588 that the eye was moving along the target motion direction, and negative average eye velocities
589 meant that the eye was moving opposite to the target motion direction.

590 Finally, we considered to what extent the post-saccadic following response tracked target
591 velocity by quantifying the PFR gain: the eye velocity computed from the open-loop PFR
592 normalized to the target velocity, with +1 indicating a perfect match of eye velocity to the target
593 motion, and negative values indicating eye velocity in the opposite direction.

594

595 **Statistics:** to evaluate the significance of PFR gain we computed the one-sample t-test to verify
596 it was greater than zero and we also computed the two-sample t-test to compare whether the PFR
597 gains differed between conditions comparing either intact *versus* blind fields for CB participants,
598 or intact fields for CB participants *versus* normal controls. We used the Pearson correlation to
599 assess the relationship between the PFR gains and NDR thresholds within each stroke patient for
600 intact and blind-field visual locations.

601

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Author Contributions

SK, KRH, and JFM designed the study; SK collected and analyzed the data with inputs from KRH and JFM; SK, KRH and JFM wrote the manuscript and all authors commented on it.

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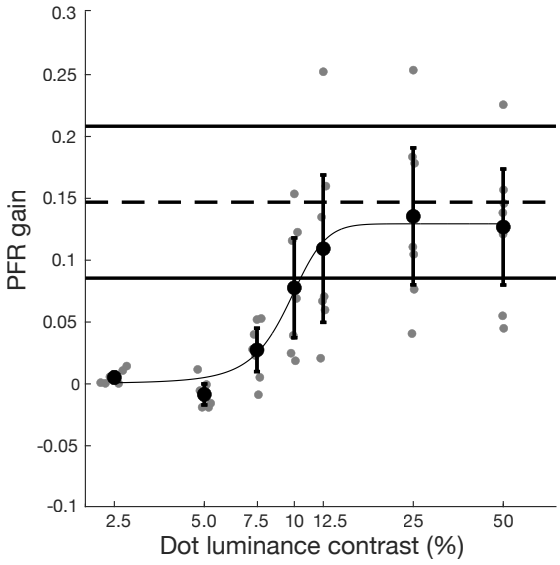
Competing interests

KRH is co-inventor on US Patent No. 7,549,743. The remaining authors have no competing interests.

Supplementary Material

Luminance contrast-dependence of the PFR. A separate group of 7 visually-intact controls were tested to determine how PFR varies with luminance contrast of the motion stimulus. Participants performed the same task as described in the main PFR experiment except that in each session, the target aperture was presented with a fixed stimulus luminance contrast between 2.5% and 50%. As shown in **Supplementary Fig. 1,** PFR gain increased steeply with stimulus contrast, starting around 5-7.5% contrast and reaching saturation quickly between 12.5-25% contrast. This is comparable to neuronal responses in cortical area MT which are sensitive to low stimulus contrasts and saturate in response at roughly 10% or higher contrasts (Heuer & Britten, 2002; Kohn & Movshon, 2003; Sclar et al., 1990). Next, we considered to what extent behavior of CB patients in the blind field might reflect a response to a stimulus of effectively reduced contrast. For comparison, when CB patients performed the same test in their intact-field with 100% contrast, the observed mean PFR was 0.1448, a value that matched the performance of intact controls for contrasts $\geq$ 25% (**Supplementary Fig. 1**). The lowest contrast for which the PFR was significantly different from zero in controls was 7.5% ($t(6)=0.3150$, $p=0.0202$). Thus, if recovered motion perception in the blind field resembles a lower-contrast stimulus representation, then we would estimate its contrast to be less than 7.5%.



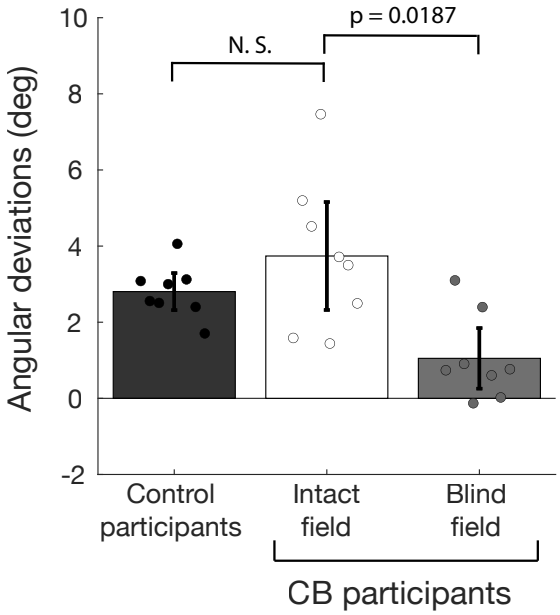
Supplementary Fig. 1 | PFR gain as a function of dot luminance contrast in visually-intact controls. Large black symbols and error bars denote the mean ± 2SEM of PFR gain across 7 visually-intact participants. Small grey dots represent individual participant PFR gains at each luminance level. The dashed black line indicates the mean PFR gain for 100% contrast stimuli in the intact field of our 8 CB patients, bracketed with ± 2SEM lines. A logistic function (sigmoid fit) was used to represent the best fit to the average PFR data across contrasts ($R^2 = 0.9862$).

Occipital stroke does not abolish motion-induced perceptual shifts reflected by saccade targeting. Previous studies showed that location of an aperture is perceived as shifted along the direction of target motion contained in the aperture (De Valois & De Valois, 1991; Kwon et al., 2015; Ramachandran & Anstis, 1990). This reflects a perceptual mis-localization error along the target motion direction that also influences saccade programming by causing saccade end-points to be shifted along the target motion (Kosovicheva et al., 2014). Consistent with previous studies, our earlier study in visually-intact controls found the position of saccade end-points to be displaced along the direction of dot motion contained in a peripheral target aperture (Kwon et al.,

2019). Thus, like the PFR, saccade end-points are also influenced by pre-saccadic selection of target motion, providing another measure of the predictive influence of target motion. However, unlike the PFR, saccade end-points appear to provide a measure that correlates well with perception (Kosovicheva et al., 2014).

Here, we asked if deviations of saccade end-points were biased by the direction of motion in both the blind and intact fields of our CB participants, and whether restoration of motion perception in the blind field influenced these saccade parameters. For each saccade, we computed the angle of the line from fixation to the saccade end-point relative to the line from fixation to the center of the target aperture. Positive angular deviations were interpreted to reflect a bias along the target motion.

As shown in **Supplementary Fig. 2**, visually-intact controls (from Kwon et al., 2019) showed a net positive saccade angular deviations that differed significantly from 0 ($t(7)=11.53$, $p<0.001$ – leftmost grey bar). This was also observed in CB patients, in intact portions of their visual fields ($t(7)=5.28$, $p=0.001$) - white bar in **Supplementary Fig. 2**). In fact, there were no significant differences between saccade end-point deviations between the two groups ($t(14)=-1.25$ $p=0.233$). In the blind-field of CB participants, saccade angular deviations were smaller than in their intact fields ($t(7)=3.05$, $p=0.019$), but unlike the PFRs, they were greater than 0 ($t(7)= 2.64$, $p=0.033$), providing positive evidence of pre-saccadic motion integration within the blind field.



Supplementary Fig. 2 | Saccade angular deviations in the intact and blind field of CB participants. Plot of mean saccade angular deviations in visually-intact controls (from Kwon et al., 2019), and for motion targets presented in CB patients' intact and blind fields. Individual dots represent the mean saccade angular deviations for each participant. Error bars represent 2 SEM across subjects.

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