Trade-off between spatiotopy and saccadic plasticity

Thérèse Collins  
Laboratoire Psychologie de la Perception, Paris, France

Saccadic eye movements bring objects of interest onto the high-resolution fovea. They also change the retinal location of objects, but our impression of the visual world is stable: We represent our visual world in spatiotopic coordinates. Visual stability could be the result of a null hypothesis that things do not move during a saccade, or of realigning retinal images based on an internal copy of the eye movement (remapping). The current studies disentangled these hypotheses. Subjects saccaded to peripheral targets that were displaced by different amounts during execution, and detected or discriminated displacement direction. Evidence for a null hypothesis was provided by the relatively poor perceptual performance, and evidence for remapping by the independence of performance from saccade endpoint. There was a trade-off between spatiotopic performance and saccadic plasticity: Good performance (perception of displacements) led to small compensative modifications in saccade amplitude on the next trial while poor performance led to larger modifications. Results also showed that variations in saccade amplitude also depended on the size of the retinal error and of the saccade itself. These results suggest that when faced with a discrepancy between the saccade endpoint and visual target, the visual system attributes the discrepancy to object displacement or to saccade error, in which case the subsequent saccade is corrected. This result reconciles the two hypotheses by suggesting that accurate remapping serves oculomotor accuracy but not visual stability. Internal copies of eye movements may thus be used separately to establish spatiotopic representations and to maintain oculomotor accuracy.

Introduction

Saccadic eye movements bring objects of interest onto the high-acuity fovea and support high-level vision. Saccades also pose some problems to visual perception because each movement displaces the retinal location of objects. Nevertheless, our subjective experience of the visual world is stable: We experience the visual world in a spatiotopic reference frame.

One hypothesis as to how spatiotopy emerges from changing retinal input is that the visual system keeps track of eye movements and combines this information with retinal displacements (cancellation hypothesis; Helmholtz, 1925; Sperry, 1950; Von Holst & Mittelstaedt, 1950). Testing trans-saccadic spatiotopic representations can be done in the laboratory by displacing the visual target during the saccade and asking observers to report whether they perceive a displacement. Humans are surprisingly mediocre at this task: Displacements of up to one third the saccade amplitude are often not reported (Bridgeman, Hendry, & Stark, 1975). This level of performance is not the result of a faulty trans-saccadic comparison process, or of low resolution localization capacities, because the displacements are unmasked when a short temporal blank separates the pre- and postsaccadic targets (Deubel, Schneider, & Bridgeman, 1996). It is also not the result of faulty knowledge about the saccade, because performance does not depend on the saccade landing position (Collins, Rolfs, Deubel, & Cavanagh, 2009; Deubel et al., 1996).

The nonperception of trans-saccadic displacements (a.k.a. saccadic suppression of displacement) is often taken as evidence in favor of a null hypothesis of visual stability, because the small discrepancies between the expected and observed postsaccadic target locations, such as those introduced by the experimenter, are not interpreted as object movement (Deubel, 2004). The displacement is seemingly “lost” to perception.

It is not, however, lost to the oculomotor system. Systematically displacing the visual target of a saccade during its execution leads to oculomotor plasticity: Amplitude adapts within minutes, and saccades become appropriate to the displaced location of the target. Displacing the target in the direction of the saccade leads to amplitude increasing adaptation; displacing it against saccade direction leads to amplitude-reducing adaptation (McLaughlin, 1967; Pélisson, Alahyane, Panouillères, & Tilikete, 2010). In fact, minute amplitude adjustments occur after every saccade even when the displacement occurs in random
It therefore appears that while trans-saccadic displacements are not perceived, they nevertheless lead to oculomotor plasticity. The current study measured trans-saccadic spatiotopic performance concurrently with adaptive amplitude changes. Three experiments were performed. In Experiment 1, healthy adult participants performed saccades to a peripheral target and discriminated or detected displacements of the target that occurred during saccade execution. This classic paradigm was used here to obtain concurrent perceptual and saccadic measures on every trial. Experiment 2 examined the effect of inserting a short blank between saccade onset and the appearance of a displaced target, to measure the consequence of improved perceptual performance on saccadic measures. Finally, Experiment 3 examined to what extent targeting errors on a given trial influenced the saccade on the next trial.

**Experiment 1**

**Methods**

**Subjects**

Seventeen healthy human adults (aged 18–35 years) participated in Experiment 1a, 13 in Experiment 1b, and 10 in both, including the author (TC). All had normal or corrected-to-normal vision and no visuomotor impairments. All gave their informed consent prior to starting the experiment, which was carried out according to the ethical standards of the Declaration of Helsinki (2004). All (except the author) received payment of 10€/hour or course credit.

**Instruments and stimuli**

Stimuli were 0.5° dots presented on a 22" Formac ProNitron 22,800 screen with a resolution of 1024 × 768 and a refresh rate of 100 Hz. Participants were seated 57 cm from the screen and their heads kept stable by chin- and forehead-rests.

Fixation dots were presented at one of nine possible locations around screen center. Saccade targets were presented 12° to the left or right. During saccade execution, the target dot stepped to one of several equiprobable locations, illustrated for each experiment in Figure 1. The displaced target dot remained on screen until subjects gave a response.

**Eye movement recording and analysis**

Viewing was binocular. Movements of the right eye were monitored with an Eyelink 1k (SR Research, Mississauga, Ontario, Canada) at 1000 Hz sampling rate. At the beginning of a session, the Eyelink was calibrated with the standard nine-point Eyelink procedure. Before each trial, fixation was checked. If the distance between the fixation check and the calibration was greater than 1.5°, a new calibration was initiated. Calibration was also automatically renewed every 50 trials. On-line saccade detection was based on a boundary criterion: Gaze-contingent changes occurred when the eye position crossed one half of the target eccentricity. Eye movement traces were subsequently analyzed offline. Instantaneous velocity and acceleration were computed for each data sample and compared to a threshold (30°/s and 8000°/s²). Saccade onset was defined as two consecutive above-threshold samples for both criteria. Saccade offset was defined as the beginning of the next 20-ms period of below-threshold samples.

**Procedure**

Subjects were instructed to fixate the fixation dot. After 500–1000 ms, the dot disappeared and the saccade target appeared 12° to the left or right (between-subjects design). Subjects were instructed to make a saccade to the target. During saccade execu-
tion, the target stepped to a new location. In Experiment 1a, the displaced location was chosen equiprobably from −3 to 3 in steps of 0.5, and subjects had to discriminate whether the target had stepped to the left or right by pressing one of two buttons. Each subject performed 468 trials in an approximately 1-hr session (36 trials per displacement). In Experiment 1b, the displaced location was chosen equiprobably from 0 to 1 in steps of 0.25 and subjects had to detect whether the target had stepped or not by pressing one of two buttons. The range of displacements was reduced in Experiment 1b to make the detection task more difficult. Each subject performed 459 trials in an approximately 1-hr session (51 trials per displacement).

Results

Saccade latency and amplitude were normal for visually-guided saccades (Experiment 1a latency: 213 ± 25 ms, mean ± standard deviation, and amplitude: 10.8 ± 0.66°; Experiment 1b latency: 209 ± 31 ms, and amplitude: 11.0 ± 0.55°). A single-factor ANOVA revealed no significant effects of target displacement for either latency or amplitude, in either Experiment (Fs < 1).

Perceptual performance in the discrimination task was measured by fitting cumulative gaussians to response data. Figure 2 presents individual data and the mean psychometric function, calculated by fitting averaged individual points. Discrimination performance, defined as the slope of the fitted function, was comparable to previous reports (e.g., Deubel et al., 1996), with relatively high thresholds (1.81 ± 0.92) and a small forwards bias (0.53 ± 1.02). Also consistently with previous reports (Collins et al., 2009; Deubel et al., 1996), performance did not depend on saccade endpoint (Figure 3), meaning that the visual system predicts a discrepancy between saccade endpoint and post-saccadic target, and does not confound this saccade-caused discrepancy with object movement.

Trial-by-trial variations in saccade amplitude correlated with the retinal offset on the previous trial. Retinal offset corresponds to the distance between the saccade endpoint and the visual target (in its post-saccadic, displaced location). When the saccade undershot the target (i.e., retinal offset was positive), the amplitude of the next saccade tended to increase. When the saccade overshot the target (negative retinal offset), the amplitude of the next saccade tended to decrease. Figure 4 illustrates an individual example, and Figure 5 presents mean correlation coefficient and slope over all subjects. The slope of the correlation was 0.11 (bootstrapped mean, 95% confidence intervals = [0.08–0.14]) in the discrimination version (Experiment 1a), and 0.24 [0.19–0.30] in the detection version (Experiment 1b). The same correlation can be calculated between current amplitude change and retinal offset from other trials: Figure 5 shows bootstrapped means and 95% confidence intervals when considering retinal offsets from the three previous trials and three future trials. The correlation was highest between the amplitude change on the current trial and the retinal offset on the previous trial (Trial t−1) (discrimination version: r = 0.23 [0.18–0.28]; detection version: r = 0.33 [0.29–0.38]). No such correlation was found when considering retinal offsets from Trials t−2 or t−3, nor from Trials t+1, t+2, or t+3.

The negative correlation observed between amplitude change and retinal offset on the same trial (Trial t) is an artefact of the design. If the retinal offset observed on Trial t were a consequence of the amplitude change that occurred between Trials t−1 and t, as the significant correlation at Trial t−1 indicates, then one would expect a negative correlation on the next trial (Trial t). Take for example a positive retinal offset on Trial t−1 that leads to increased amplitude on Trial t. Because the average target displacement was 0, this
increased amplitude likely led to an *overshoot* of the target on Trial t, and thus a negative correlation between an amplitude increase and a negative retinal offset. Any factor that influences the correlation between retinal offset on Trial t−1 and amplitude change on Trial t will therefore be passed on to the correlation at Trial t (such as correct vs. incorrect perception of the displacement, as described below).

The degree to which amplitude change correlated with previous retinal offset depended on the perceptual report from the previous trial, that is, whether the previous response was correct or incorrect. When the target displacement on the previous trial was correctly perceived as object displacement, that is, when the subject correctly responded forwards or backwards (in the discrimination version of the task), the correlation between amplitude change and retinal offset was reduced relative to when the target displacement was not perceived. The correlation coefficient was 0.21 [0.12–0.28] for correct trials and 0.32 [0.20–0.41] for incorrect trials; confidence intervals overlap meaning that this difference did not reach significance. The slope difference did reach significance, with slopes of 0.10 [0.05–0.13] for correct trials and 0.23 [0.14–0.293] for incorrect trials.

When subjects responded incorrectly in the discrimination version of the task, they very likely did not perceive the displacement. When they respond correctly, however, either they perceived the displacement and responded accordingly, or did not perceive the displacement and made a lucky guess. Correct responses thus mix trials in which the subject perceived with trials in which she did not. The detection version of the task (Experiment 1b) does not suffer from this problem. Participants responded that a displacement occurred in 50% of trials (range 32%–74%), and the difference between correct and incorrect trials was statistically significant for both correlation coefficient (correct responses: 0.28 [0.23–0.32], incorrect responses 0.39 [0.35–0.42]), and slope (correct responses: 0.19 [0.16–0.23], incorrect responses 0.29 [0.25–0.33]) (Figure 5).

**Figure 4.** Experiment 1a: Individual scatter plots relating amplitude change (the difference in amplitude between the current trial and the previous trial) to retinal offset (the distance between saccade endpoint and target) on previous trials. Each panel corresponds to a particular trial offset, from three trials in the past (i = −3) to three trials in the future (i = 3).
Discussion

Trans-saccadic spatiotopic representations

To discriminate the direction of a trans-saccadic displacement, pre- and postsaccadic targets must be localized in a spatiotopic reference frame. As such, the present results shed light on the mechanisms that may support visual stability. The apparent stability of the phenomenal visual environment could be the result of a null hypothesis that things do not move during a saccade, or of realigning retinal images based on an internal copy of the eye movement (remapping).

Performance in the spatiotopic task was poor, replicating the phenomenon of saccadic suppression of displacement (Bridgeman et al., 1975; Deubel et al., 1996). Poor performance coexisted with accurate information about the saccade that could, in principle, be used to solve the spatiotopic task (Collins et al., 2009). A system with no knowledge about the saccade would confound offsets due to faulty saccades with offsets due to target displacement (each time the saccade undershot the target, this would be taken as evidence in favor of forwards target displacement). Accurate information about the saccade is available, but seems not to be used, suggesting an active null hypothesis (Deubel, 2004; Niemeier, Crawford, & Tweed, 2003). The visual system apparently has a tolerance for small offsets (perhaps, that fall within the range of habitual motor variability). These offsets do not disrupt the construction of a spatiotopic reference frame (Wexler & Collins, 2014).

Relationship between spatiotopic performance and oculomotor plasticity

Experiments 1a and 1b confirmed the existence of single-trial modifications in saccade amplitude to compensate for discrepancies between the saccade endpoint and visual target (i.e., retinal offsets) on
previous trials. In both datasets, on a per trial basis, the difference in saccade amplitude between two subsequent trials depended on the retinal offset observed in the first of those trials: If the saccade undershot the target, subsequent amplitude was likely to increase; whereas if the saccade overshot the target, subsequent amplitude was likely to decrease. The present results extent previous reports (Havermann & Lappe, 2010; Srimal et al., 2008) by showing that the correlation was modulated by the perception of the target displacement. Indeed, correct perception that the retinal offset was due to visual target displacement led to less subsequent single-trial adaptation than following incorrect trials.

Updating future motor plans is only useful if the source of the discrepancy is the motor system, and not the outside world. The current results are therefore compatible with the idea that estimating the sources of error is a key component for motor learning generalization. The classic example is throwing a stone that falls closer than intended. The error can come from the fact that either the stone was heavier than initially estimated, or the arm is weaker. If the arm is weaker, then future movements of that arm should be adjusted. If the stone is heavier, then future throws of that stone should be adjusted, whatever the effector. Attributing the source of the motor error to the outside world (the stone is heavier) should lead to a generalization of learning to other effectors, and such generalization should not occur when the error is attributed to a change in a specific effector (Berniker & Kording, 2008; Wei & Körding, 2009). Taking this reasoning to the oculomotor domain, if the saccade falls short because the object moved forwards, there is no need to adapt future saccades, but if it fell short because its amplitude was inappropriate to achieve the target, then future saccades should be adapted.

Perceptual performance in the spatiotopic task is poor, suggesting that small retinal offsets are not perceived. Performance is not, however, dependent on saccade accuracy, showing that accurate metric information, possibly via efference copy signals, is available to the visual system. Additionally, these same non-perceived retinal offsets lead to small changes in future saccades. As such the present results suggest a trade-off between performance in the spatiotopic task and saccadic adaptation. When a discrepancy between saccade endpoint and expected target position occurs, its source can be either saccadic error or object movement. When subjects correctly perceive the discrepancy to be due to object movement, the amplitude of the following saccade is only slightly modified. When the subjects do not perceive the object movement, the amplitude of the following saccade is modified such that the discrepancy is reduced. This suggests that the discrepancy may be attributed to the saccade.

One of the key characteristics of saccadic suppression of displacement is the increase in performance with a temporal blank (Deubel et al., 1996). This simple experimental manipulation is crucial in demonstrating that saccadic suppression of displacement is not the result of poor trans-saccadic encoding or memory, but may be the result of an active suppression of otherwise discernible object displacements. If single-trial saccadic adaptation and saccadic suppression of displacement are indeed flip sides of a coin, as the results of Experiment 1 suggest, an experimental manipulation that influences suppression should also influence single-trial adaptation. The goal of Experiment 2 was thus to examine whether the blanking effect influenced single-trial adaptation.

### Experiment 2

#### Methods

##### Subjects

Fifteen healthy human adults (aged 18–35 years) participated in the experiments, including one author (TC). All had normal or corrected-to-normal vision and no visuomotor impairments, and gave their informed consent prior to starting the experiment, which was carried out according to the ethical standards of the Declaration of Helsinki (2004). All (except the author) received payment of 10€/hour or course credit.

Instruments and stimuli and Eye movement recording and analysis were identical to those in Experiment 1.

##### Procedure

The procedure was identical to that of Experiment 1, except that upon saccade onset detection, the target disappeared for a random duration between 0 and 250 ms (Figure 6). After this blank, it reappeared in its displaced location, chosen equiprobably from the set $[-2, -1.5$ to 1.5 in steps of 0.25, 2], and subjects had to discriminate the direction of displacement (left or right). Each subject performed 2400–2700 trials in three sessions of equal duration (about 1.5 hr each).

#### Results

Saccade latency and amplitude were normal for visually-guided saccades (latency: $200 \pm 19$ ms, mean $\pm$ standard deviation, and amplitude: $10.1^\circ \pm 1.5^\circ$). A
single-factor ANOVA revealed no significant effects of target displacement on either latency or amplitude ($F < 1$).

To characterize the effect of blank duration on perceptual performance, trials were binned into 20-ms windows and psychometric functions fit to the data in each window. Each bin comprised approximately 200 observations. Figure 7 shows thresholds (slopes of the psychometric functions) as a function of blank duration. As expected from previous reports (Deubel et al., 1996), performance improved with blank duration (Figure 7). The correlation between amplitude change on Trial $t$ and retinal offset on Trial $t$ was also apparent in this data set, but did not depend on blank duration (Figure 7).

The measurement of saccadic adaptation was obtained, as in Experiment 1, by the correlation between previous retinal offset and current amplitude change. Replicating what was observed in Experiments 1a and 1b, the correlation differed significantly depending on whether the previous trial was correct or incorrect: the correlation was smaller after a correct trial ($r = 0.50$ [0.47–0.55]; slope = 0.53 [0.45–0.62]) relative to an incorrect trial ($r = 0.64$ [0.59–0.67]; slope = 0.78 [0.70–0.84]).

Discussion

Displacements between pre- and postsaccadic targets that usually go unseen are unmasked when a short temporal blank separates the pre- and postsaccadic targets, replicating previous reports (Deubel et al., 1996; Wexler & Collins, 2014). However, there was no independent effect of blank duration on single-trial adaptation. Single-trial adaptation remained similar whatever the target blank, but was influenced, as in Experiment 1, by whether the previous trial was correct or incorrect. Therefore, short blanks that nevertheless resulted in perception of the displacement led to less adaptation than blanks that did not result in perception of the displacement.

Previous studies examined the influence of the timing of the post-saccadic visual error on saccadic adaptation, in the classic systematic step paradigm (i.e., the visual target is always displaced in the same direction). For example, the amount of adaptation decreases when the postsaccadic visual information is delayed for about 150 ms or when it is available for less than 80 ms (Bahcall & Kowler, 2000; Shafer et al., 2000). The current results suggest decreased adaptation with delayed postsaccadic visual feedback may result from the fact that subjects saw the target displacement more often in the temporal blank condition, leading overall to less adaptation.

To sum up, single-trial adaptation is mediated by visual perception of the displacement, whatever the temporal characteristics of the postsaccadic visual information. The final question addressed in this study was about the influence of the spatial characteristics of the post-saccadic visual information on single-trial adaptation.

Figure 6. (a) Experiment 2: Stimuli. Fixation dot and possible displaced target locations (b) Procedure. The fixation dot appeared for 500–1000 ms until the saccade target was displayed 12° to the left or right. Upon saccade detection, the screen was blanked for 0 to 250 ms. After this delay, the target stepped to a new location drawn equiprobably from the positions illustrated on the left.

Experiment 3

Methods

Subjects

Twelve healthy human adults (aged 18–35 years) participated in the experiment, including one author (TC). All had normal or corrected-to-normal vision and no visuomotor impairments and gave their informed consent prior to starting the experiment, which was carried out according to the ethical standards of the Declaration of Helsinki (2004). All (except the author) received payment of 10€/hour or course credit.

Instruments and stimuli

Instruments and stimuli were identical to Experiment 1 except for the following: Fixation dots were presented at a random location within a 2° × 3° zone.
centered on half of the subsequent target eccentricity to the left of screen center. Saccade targets were presented 12° to the right. During saccade execution, the target dot stepped to a location drawn from a Gaussian distribution with average $-3^\circ$ or $-1^\circ$ and variability (sigma of the Gaussian) of $0^\circ$ (constant step size), $1^\circ$ or $3^\circ$ in a blocked design (see Figure 8). The stepped target dot remained on screen for 500 ms.

Eye movement recording and analysis were identical to Experiments 1 and 2.

Procedure

The fixation dot appeared on the left side of the screen. 500–1000 ms later, the fixation dot disappeared and a target appeared horizontally $5^\circ$, $10^\circ$, or $20^\circ$ to the right of the screen (between-subjects design). Subjects were instructed to saccade towards it. During the saccade, depending on epoch, the visual target remained on at the same position, stepped to a new location or disappeared (see Figure 1b). About 500 ms after the primary saccade, any remaining target was extinguished and the next trial began. Each session contained four successive epochs: baseline (100 trials), adaptation (300 trials), posttest (100 trials), and recovery (100 trials). In the baseline epoch, the visual target randomly remained on or was extinguished during the primary saccade. In the adaptation epoch, the visual target stepped to a new location during the primary saccade. Excessively large steps were avoided in the $1^\circ$ and $3^\circ$ variability conditions by clipping the distributions at $6^\circ$ standard deviations. In the posttest epoch, the target was extinguished, and not redrawn during the saccade. These trials assessed the final adaptation level while minimizing the decay of adaptation (Ethier, Zee, & Shadmehr, 2008). Finally, 100 recovery trials in which the target always reappeared were run, whose function was to return amplitude to baseline. Each subject completed nine approximately 1-hr sessions resulting from the combination of three target eccentricities ($5^\circ$, $10^\circ$, $20^\circ$) and three step variabilities (low: $0^\circ$, medium: $1^\circ$, high: $3^\circ$), with only one average step size ($-3^\circ$ or $-1^\circ$) (i.e., six subjects got the $-3^\circ$ average step size; six subjects got the $-1^\circ$ average step size). The order of the nine sessions was random and different for each subject. To avoid interference from adaptation of the previous session (Alahyane & Pélisson, 2005), in addition to the 100 recovery trials at the end of each session, at least 24 hr occurred between two successive sessions.
Results

Saccade latency and amplitude were analyzed with a 3 × 3 × 2 × 2 ANOVA with within-subject factors eccentricity (5°, 10°, 20°), step variability (low, medium, high) and epoch (baseline, posttest) and between-subject factor average step size (–1°, –3°).

Saccade latency was normal for visually-guided saccades (169 ± 57 ms, mean± standard deviation) and was not affected by eccentricity, step variability, epoch, or average step size.

Saccade amplitude increased with eccentricity (on average, 4.8° ± 0.3°, 9.4° ± 0.4° and 18.9° ± 0.6° for 5°, 10°, and 20° targets in the baseline). Amplitude decreased between baseline and posttest, F(1, 10) = 136.0, p < 0.001. This effect of epoch depended on average step size: Amplitude decreased by 0.61° ± 0.3° when average step size was –1° and by 1.13° ± 0.5° when average step size was –3°. No other interaction with step size was significant. Although there was a decrease for all eccentricities, the magnitude of that decrease depended on eccentricity, F(2, 20) = 34.4, p < 0.001: the greater the eccentricity, the greater the difference between baseline and posttest. The difference between baseline and posttest also depended on step variability, but differently depending on eccentricity, significant triple interaction between epoch, eccentricity and step variability, F(4, 40) = 10.1, p < 0.001. For the 5° and 10° eccentricities, adaptation decreased with increasing step variability (5° targets: 0.68 ± 0.8, 0.63 ± 0.20, 0.66 ± 0.18; 10° targets: 1.10 ± 0.51, 0.92 ± 0.47, 0.50 ± 0.48 for low, medium, and high variability respectively) whereas for the 20° eccentricity, the effect was reversed, with greater adaptation with increasing variability (1.04 ± 0.89°, 1.35 ± 0.68°, 1.58 ± 0.69°). These effects are illustrated in Figure 9.

Amplitude changes are often expressed as a percent change between baseline and posttest epochs: (baseline – post-test)/baseline. This measure normalizes over different eccentricities. Despite an increase in the absolute difference in amplitude between baseline and posttest epochs, as described above, there was a slight decline in the % adaptation with increasing eccentricity, 9.4 ± 6%, 8.8 ± 4%, 7.0 ± 3% respectively for 5°, 10°, and 20°; F(2, 20) = 3.9, p < 0.04, that did not vary with average step size (F < 1). Similarly to the pattern found for the amplitude analysis, the effect of step variability depended on eccentricity, F(4, 40) = 20.5, p < 0.001: Increased step variability led to a decrease in adaptation for the 5° and 10° eccentricities, and an increase for the 20° eccentricity.

There was evidence of adaptation of saccade amplitude not only building over time between baseline and posttest epochs but also on a trial-to-trial basis during the adaptation epoch. Similarly to results observed in Experiments 1 and 2, there was a positive correlation between retinal offset on the previous trial (Trial t − 1) and the amplitude change on the current trial (Trial t). The slope of the correlation was 0.38 [0.32–0.44]. Figure 10a shows bootstrapped means and 95% confidence intervals: The correlation was highest between the amplitude change on the current trial and the retinal offset on the previous trial (Trial t − 1), (r = 0.39 [0.34-0.43]). The correlation was significant for Trial t − 1 (and, as before, artifactual for Trial t), but not for any other trials, suggesting that adaptation is a one-trial phenomenon. To further test whether adaptation did indeed occur within one trial, the correlation between amplitude change and mean retinal offset for windows of previous trials of different sizes was calculated. Figure 10b shows the correlation when taking the mean retinal offset from 1 to 50 previous trials into account. The correlation decreases rapidly when taking more than one previous trial into account. A final analysis checked for cumulative effects. Retinal offsets that systematically indicate that saccade amplitude was too small or too large may lead to greater single-trial adaptation. In other words, when the subject has experienced several undershooting saccades in a row (negative retinal offsets), amplitude-increasing adaptation in response to another undershoot (negative retinal offset) might be bigger relative to amplitude-increasing adaptation in response to an undershoot that is not preceded by several undershooting trials. To test this hypothesis, the proportion of previous positive
or negative retinal offsets in past trials was calculated for each trial. Figure 10c shows the per trial amplitude change for increasing proportions of same-direction retinal offsets in windows of 10, 20, 30, 40, or 50 trials in the past. Cumulative effects would appear as cold to hot colors from left to right; no such effects can be seen.

Experiment 3 confirms the effect of previous trial retinal offset on saccade amplitude of the observed in the Experiments 1 and 2. The goal of Experiment 3 was to take this analysis a step further by examining whether all retinal errors were as good at driving subsequent amplitude changes. To do so, per trial amplitude change was measured for retinal offsets of different sizes. For each subject and target eccentricity, the mean amplitude change for 0.5° bins of previous retinal offset was calculated. Figure 11 presents the results. Per trial amplitude change was best for small or intermediate retinal offsets, and dropped off for larger offsets. The influence of a given retinal offset on subsequent saccade amplitude depended on eccentricity: The larger the eccentricity, the more the subsequent saccade was influenced by that offset. This was the case when the retinal offset was considered as an absolute value, and when it was considered a proportion of the initial saccade amplitude (i.e., gain). The responsiveness of the saccadic system to retinal offsets is not constant for a given proportional error; rather, as saccade amplitude increases, so does responsiveness to error. Take for example a retinal offset of −2° (the saccade overshot the target). For all eccentricities, this retinal offset led to a decrease in amplitude on the next trial, of −0.16° for the 5° eccentricity, −0.31° for the 10° eccentricity, and −1.16° for the 20° eccentricity. A retinal offset of 0 (the postsaccadic target was exactly on the fovea) also led to different amounts of adaptation for different eccentricities: a 0.33° increase in amplitude for 5°, no change for 10° (0.14°, not significantly different from 0), and a −0.38° decrease for 20°. The point at which per trial amplitude change switches from amplitude-decreasing to amplitude-increasing depends on eccentricity and is not necessarily zero: approximately −1°, −0.5°, and 0.5° (for 5°, 10°, and 20° respectively). For the 5° eccentricity, small negative offsets (saccade overshoots) did not lead to subsequent amplitude changes. Zero retinal offset (the eye landed exactly on the target) led to small increases of subsequent amplitude. For the 10° eccentricity, small negative or zero offsets did not lead to subsequent amplitude changes. For the 20° eccentricity, small positive offsets (saccade undershoots) did not lead to
subsequent amplitude changes. Zero retinal offset led to amplitude-decreasing adaptation. The switch from amplitude-decreasing to amplitude-increasing may be related to the predicted landing position during the baseline epoch. Indeed, if the saccadic system aims for a particular landing position which is not necessarily the target itself, saccadic adaptation should maintain this nonzero goal, which should be the retinal offset at which the switch occurs. To test this hypothesis, the individual saccade landing site distributions were fit with Gaussians. The switch from amplitude-decreasing to amplitude-increasing may have occurred around the mean baseline landing site. Converted into mean retinal offset, baseline landing sites are 0.02° ± 0.26°, 0.4° ± 0.41° and 0.86 ± 0.56° (for 5°, 10°, and 20° eccentricities respectively): Saccades tended to undershoot. The switch (−1°, −0.5°, and 0.5° for 5°, 10°, and 20° respectively) therefore occurred to the left of the predicted retinal offset. Although the quality of the data may not be sufficient to fully support this claim, such a result would be expected if the efference copy of the saccade were slightly smaller than the saccade.

Discussion

The degree to which retinal offsets drove subsequent saccade amplitude depended on the amplitude of the saccade (as shown for monkeys by Robinson, Noto, & Bevans, 2003). There was a window of offsets that drove adaptation best, and offsets outside that window drove subsequent amplitude less. The size of the offset was not the only factor determining to what degree it drove the next saccade amplitude, because a given offset drove subsequent changes more when the saccade that led to that offset was large. This result suggests that the visuomotor system has some knowledge about the range of expected offsets for each eccentricity. When a retinal offset is observed on a particular trial, to what extent it drives subsequent amplitude changes depends on amplitude: Large saccades are more adaptable than small saccades.

Havermann and Lappe (2010) measured adaptation of a 15° saccade as a function of the variability of the target step and found less adaptation with more variability, just like in the 5° and 10° eccentricities tested here, suggesting that the large steps in the most variable conditions impeded adaptation. The present data shows a different pattern for 20° saccades: Adaptation increased with step variability. The relevant parameter may not be the variability of the error as defined by the experimenter, because when the eye lands, the subject does not know whether the retinal offset she experiences is due to the experimenter’s manipulation or to her own saccade. Expected offsets and their variability may be more relevant than experimenter-defined variability. The visual system expects some degree of variability and may use this knowledge to estimate whether offsets are due to the saccade or to object displacement (Collins et al., 2009). Part of this knowledge about variability is the expectation that larger saccades are accompanied by a wider distribution of retinal offsets. Thus, when the distribution of retinal offsets falls within the expected range, they are more likely attributed to saccade miscalibration and may constitute a signal that the saccadic system needs to adapt. When the retinal offsets fall outside this range, they may less likely be

Figure 11. Per trial amplitude change for retinal offsets of different sizes. Each point represents the bootstrapped mean over all subjects, collapsed over step size and variability. Filled symbols represent data points significantly different from 0; open symbols points that did not (bootstrap 95% confidence intervals). Left panel: absolute retinal offsets binned into 0.5° windows. Right panel: relative retinal offsets expressed as a proportion of initial target eccentricity.
attributed to a faulty saccade and lead to less adaptation. Although participants' perception of the target steps was not measured in Experiment 3, the larger offsets resulting from larger target steps were probably perceived as such, while the smaller ones were not. The criterion for attributing offsets to one's own saccade appears to depend on initial saccade amplitude. In the case of 5° and 10° targets, adaptation decreased with wider distributions of retinal offset, suggesting that the larger-than-expected retinal offsets were not attributed to the saccade but rather to target displacement and therefore did not cause any adaptation. In the case of the 20° target, however, the wider distribution of retinal offsets caused an increase in adaptation, suggesting that most of the large offsets may have been attributed to the saccade.

Another result observed in Experiment 3 is the short time constant for the influence of retinal offsets on subsequent amplitude. (A short time constant was also observed in Experiments 1 and 2, but the increased number of trials in Experiment 3 allowed a more thorough analysis.) Saccade amplitude was influenced by the previous retinal offset, but not by offsets farther into the past. Furthermore, error signals building up over more than one trial had no effect. Models of saccadic adaptation propose that amplitude adapts as the result of a change in the forward model that predicts the sensory consequence of the saccade. When the sensory consequence is not met, as when the target is in an unexpected location, internal feedback modifies the forward model such that subsequent saccades deliver expected sensory consequences (Ethier, Zee, & Shadmehr, 2008; Miall & Wolpert, 1996). The current results suggest that this internal feedback occurs for every saccade, and that learning does not accumulate over several trials.

To sum up Experiment 3, unexpected offsets are not systematically attributed to object displacement (Bridgeman et al., 1975) and lead to saccadic adaptation (Collins & Wallman, 2012; Wong & Shelhamer, 2011). Experiment 3 shows the limits of adaptation-evoking offsets, and that their capacity to evoke adaptation depends on saccade amplitude. Thus, the saccadic system tolerates some unexpected offset and even changes future behavior to reduce the offset: Observers may attribute more than their share of error to themselves.

### General discussion

The experiments reported here examined perceptual performance in a spatiotopic task and single-trial saccadic adaptation. They addressed fundamental questions about how a spatiotopic visual representation is established despite saccade-induced retinal displacements, and how this relates to oculomotor plasticity, in the form of single-trial fluctuations in saccade amplitude.

The construction of a spatiotopic visual representation seems to involve an active null hypothesis that things do not move just during a saccade, leading small discrepancies on the spatiotopic map not to be registered as object displacements (Deubel, 2004; Niemeier et al., 2003). The current results do not add anything new to this story, but confirm the fact that perceptual performance is worse than the accuracy of efference copy would predict.

The novelty of the current result resides in the observation of single-trial modifications of saccade amplitude that compensate for discrepancies between the saccade endpoint and visual target (retinal offsets) on previous trials. In all datasets, on a per trial basis, the difference in saccade amplitude between two subsequent trials depended on the retinal offset observed in the first of those trials: If the saccade undershot the target, subsequent amplitude was likely to increase; whereas if the saccade overshot the target, subsequent amplitude was likely to decrease. The present results extent previous reports (Havermann & Lappe, 2010; Srimal et al., 2008) by showing that the correlation was modulated by the perception of the target displacement: Unperceived displacements drove subsequent single-trial adaptation more than perceived displacements.

Taken together, the results suggest that accurate information about the saccade does not inform perception—visual stability does depend, at least in part, on the hypothesis that things do not move during saccades, and that small displacements are to be ignored as likely due to our own saccades. Accurate information about the saccade, however, does inform the oculomotor system. Remapping therefore does occur and serves the saccadic system. As such the present results suggest a trade-off between performance in the spatiotopic task and saccadic adaptation. When a discrepancy between saccade endpoint and expected target position occurs, its source can be either saccadic error or object movement. When subjects correctly perceive the discrepancy to be due to object movement, the amplitude of the following saccade is only slightly modified. When the subjects do not perceive the object movement, the amplitude of the following saccade is modified such that the discrepancy is reduced, suggesting that the discrepancy may be attributed to the saccade. Blanking the target before displaying it in its displaced location unmasked the displacements but did not have an independent effect on single-trial amplitude changes.

In conclusion, these experiments suggest that remapping may serve perception and action in two
complementary ways. Remapping is the realignment of object locations in a spatiotopic reference frame, based on saccadic eye movement information. When faced with a discrepancy between the saccade endpoint and visual target, the visual system attributes the discrepancy to object displacement or to saccade error, in which case the subsequent saccade is corrected. When remapping indicates a discrepancy outside of the range of habitual offsets, object displacement is seen, and subsequent saccade amplitude is only minimally affected. When remapping indicates that an offset between saccade endpoint and expected target location is within the habitual range, it is attributed to an internal source. No object displacement is perceived, which allows for the construction of a robust, stable spatiotopic representation of the visual world despite oculomotor variability. Offsets in this habitual range do influence the oculomotor system, as expected for a system able to maintain very accurate saccades. Internal copies of eye movements may thus be used differently to establish spatiotopic representations and to maintain oculomotor accuracy.

Keywords: visual stability, spatiotopy, saccadic adaptation

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Corresponding author: Thérèse Collins.
Email: collins.th@gmail.com.
Address: Laboratoire Psychologie de la Perception, Paris, France.

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