Exp Brain Res (2009) 192:9–23
DOI 10.1007/s00221-008-1546-y

RESEARCH ARTICLE

Effects of saccadic adaptation on visual localization before and during saccades

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Received: 6 September 2007 / Accepted: 6 August 2008 / Published online: 21 August 2008

Abstract Short-term saccadic adaptation is a mechanism that adjusts saccade amplitude to accurately reach an intended saccade target. Short-term saccadic adaptation induces a shift of perceived localization of objects flashed before the saccade. This shift, being detectable only before an adapted saccade, disappears at some time around saccade onset. Up to now, the exact time course of this effect has remained unknown. In previous experiments, the mislocalization caused by this adaptation-induced shift was overlapping with the mislocalization caused by a different, saccade-related localization error, the peri-saccadic compression. Due to peri-saccadic compression, objects flashed immediately at saccade onset appear compressed towards the saccade target. First, we tested whether the adaptation-induced shift and the peri-saccadic compression were either independent or related processes. We performed experiments with two different luminance-contrast conditions to separate the adaptation-induced shift and the peri-saccadic compression. Human participants had to indicate the perceived location of briefly presented stimuli before, during or after an adapted saccade. Adaptation-induced shift occurred similarly in either contrast condition, with or without peri-saccadic compression. Second, after validating the premise of both processes being independent and superimposing, we aimed at characterizing the time course of the adaptation-induced shift in more detail. Being present up to 1 s before an adapted saccade, the adaptation-induced shift begins to gradually decline from about 150 ms before saccade onset, and ceases during the saccade. A final experiment revealed that visual references make a major contribution to adaptation-induced mislocalization.

Keywords Eye movements · Saccades · Saccadic adaptation · Space perception · Mislocalization · Peri-saccadic compression · Peri-saccadic shift · Adaptation-induced shift

Introduction

Motor actions are continuously being monitored and adjusted by the nervous system. Movements are optimized by constantly comparing and calibrating motor commands and their sensory consequences. This is valid for a range of different types of movements, saccadic eye movements being one of those. Saccades are insofar special as they are 'ballistic' movements, i.e. they cannot be altered during the actual movement. Instead, they have to be programmed completely a certain time before movement onset. If saccades have not reached an intended target accurately for a number of times, saccadic adaptation adjusts the amplitude of the saccade. Saccadic adaptation is thought to counteract changes in saccade gain, i.e. the ratio between intended and executed saccade. Such changes can be caused by growth, injuries, diseases or exhaustion (Vilis et al. 1983; Optican et al. 1985). The error signal driving saccadic adaptation is visual, not motor, and no corrective saccades are necessary for adaptation to occur (Wallman and Fuchs 1998). To be effective, the visual error signal has to be available immediately after the saccade (Shafer et al. 2000).

Saccadic adaptation is investigated in humans and monkeys by means of computer-based psychophysical experiments. Therein, the target of a saccade is displaced during the eye movement. Consequently, the initial saccade target

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visible before the saccade, referred to as T1, is presented at a different location during and after the saccade, referred to as T2. Due to saccadic suppression, this displacement is not noticed by subjects. Instead, the oculomotor system gradually adapts the amplitude of the saccades to the target displacement, such that the displaced target, T2, will be reached even though it does not match the eccentricity of the initially visible target, T1. In the course of this procedure saccade amplitude can be increased or decreased by several degrees, depending on the location of T2 relative to T1. In monkeys, saccadic adaptation develops within 1,000–1,500 trials (Fuchs et al. 1996), with rate constants ranging from several hundred to about a thousand trials (Straube et al. 1997; Scudder et al. 1998). In humans, saccadic adaptation is much faster: About 50 trials are sufficient (Deubel et al. 1986), with rate constants of about 30–60 trials (Deubel et al. 1986; Frens and van Opstal 1994; Albano 1996). Because of this significant difference in adaptation rate the adaptation in humans is named short-term saccadic adaptation.

In humans, several different mechanisms of saccadic adaptation occur at different levels of the oculomotor system (Deubel 1995; Albano 1996; Alahyane and Pelisson 2003; Alahyane et al. 2007). Nerve palsy and prism adaptation, for example, lead to long-term adaptation that is both, disconjugate and dependent on eye position, but not dependent on saccade amplitude (Hopp and Fuchs 2004). In contrast, short-term saccadic adaptation is conjugate (Albano and Marrero 1995; Das et al. 2004), direction- and amplitude-specific (Straube et al. 1997; Watanabe et al. 2003), and dependent on eye displacement vector rather than eye position (Albano 1996). Different types of saccadic adaptation may be driven by different processes, for example a slower, long-lasting motor adaptation, and a faster, short-lasting, possibly higher cognitive or perceptual, process.

The possibility of a cognitive or perceptual process in particular should be taken into account because saccadic adaptation shows perceptual consequences: It affects the localization of stimuli presented in the adapted space. Moidell and Bedell (1988) described that saccadic adaptation-induced apparent shifts of localization targets that were briefly presented near the saccade target. These shifts were small in amplitude (about 0.5°) but occurred during fixation when subjects were previously adapted. Bahcall and Kowler (1999) found a more pronounced effect when comparing the perceived location of stimuli presented at the saccade target before and after an adapted saccade. Awater et al. (2005) reported a large, adaptation-induced localization shift of stimuli presented in the area between T1 and T2. When visible between 300 and 100 ms before the execution of an adapted saccade, stimuli presented in this area were perceived shifted in the direction of adaptation. Stimuli presented after an adapted saccade were localized correctly. Recently, a similar effect of saccadic adaptation has been demonstrated for open-loop pointing movements (Bruno and Morrone 2007). We refer to this error in localization of pre-saccadically presented stimuli, specifically caused by saccadic adaptation, and shifting localization in the direction of adaptation, as *adaptation-induced shift* throughout this article.

In the time range from about 100 ms before the saccade to the end of the saccade, the time course and magnitude of adaptation-induced shift could not be studied in isolation. In this epoch, *peri-saccadic compression* occurred. Peri-saccadic compression is a specific error in the localization of stimuli presented briefly around the time of a saccade. Stimuli are perceived shifted towards the location of the saccade target (Ross et al. 1997; Morrone et al. 1997; Lappe et al. 2000; Kaiser and Lappe 2004). In the experiments of Awater et al. (2005), both effects, the adaptation-induced shift and the peri-saccadic compression, emerge in the temporal vicinity of a saccade and result in a distortion of perceived space. It remains unclear if the adaptation-induced shift and the peri-saccadic compression are entirely different effects or whether they interact with each other.

In addition to the aforementioned peri-saccadic compression, the *peri-saccadic shift* is another mislocalization effect around the time of saccadic eye movements. In contrast to peri-saccadic compression, however, the direction of this mislocalization is uniform throughout the visual field (Honda 1991). Arising about 50 ms before saccade onset, the peri-saccadic shift reaches its maximum around saccade onset and decreases within 50 ms after saccade onset. Stimuli being presented briefly in this epoch are perceived shifted in the direction of the upcoming saccade. Typically, there is a transient undershoot short-time after saccade onset, resulting in a mislocalization in the opposite direction (Honda 1989, 1991). This peri-saccadic shift is independent of stimulus position (Honda 1991) and stimulus contrast (Michels and Lappe 2004).

The experiments described below were designed to separate the adaptation-induced shift from other peri-saccadic localization errors. We aimed at replicating previous experiments (Awater et al. 2005), yet extending the findings over a wider range of presentation times and stimulus positions. Based on these findings, we wanted to address a series of questions:

1. Are the adaptation-induced shift and the peri-saccadic compression either independent or related processes? We varied stimulus contrast, thereby isolating the adaptation-induced shift from peri-saccadic compression, since the strength of the latter depends on stimulus contrast (Michels and Lappe 2004). In the study of

Awater et al. (2005), the adaptation-induced shift could not be examined separately.

2. What is the time course of the adaptation-induced shift? After confirming the premise of both processes being independent and superimposing, we aimed at characterizing the time course of the adaptation-induced shift in more detail. Therefore, we conducted an experiment without previous saccadic adaptation, and without peri-saccadic compression. This permits quantifying the impact of the pure peri-saccadic shift. Subsequently, this peri-saccadic shift is subtracted from the data obtained in the adaptation experiment, thus eliminating both, the peri-saccadic compression and the peri-saccadic shift. Isolating the adaptation-induced shift should allow for studying the uncorrupted time course of this very effect, particularly the time course of its disappearance relative to saccade onset. From the data of Awater et al. (2005) this was impossible, since around the time of saccade onset it was intermingled with peri-saccadic shift and peri-saccadic compression. We aimed at differentiating between the possibilities of a pre- or an intra-saccadic disappearance, the former indicating the involvement of pre-saccadic perceptual processes, potentially related to saccade planning, the latter pointing at the relevance of motor processes generated during the execution of an adapted saccade.
3. What is the temporal extent of the adaptation-induced shift? From the experiments of Awater et al. (2005) the adaptation-induced shift was already known to exist some time before the execution of an adapted saccade, and disappearing around saccade onset. Here, we wanted to further specify how long before saccade onset this shift is detectable, and in particular whether it is a transient effect, arising and diminishing before saccade onset. Therefore, we extended our previous experiments by very early pre-saccadic stimulus presentations, up to 1 s before saccade onset. Up to now, no peri-saccadic effect is known to be operative such a long time before a saccade.
4. Further on, we implemented a different, less stereotype way of establishing saccadic adaptation, thereby addressing possible issues of methodological validity and the general applicability of our results.
5. In a final experiment, we examined the emergence of the adaptation-induced shift in the course of developing saccadic adaptation. Comparing changes in saccade amplitude with changes in peri-saccadic localization across successive trials allows for estimating symmetry and synchrony of the two parameters. If saccadic adaptation directly causes the adaptation-induced shift, both effects have to be tightly coupled over time.

Methods

Participants

Twelve participants, five males and seven females, took part in the study. All of them were students at the Department of Psychology and had normal or corrected vision. Their age ranged from 21 to 38 years. One participant was completely naïve to eye movement experiments. One participant was an author. The other participants were experienced psycho-physical observers but naïve to the aims of the experiments. There were no qualitative differences ascertainable in the results of naïve and non-naïve participants. All participants gave informed consent. The experiments were carried out along the principles laid down in the declaration of Helsinki.

Experimental setup

Two personal computers were running the experiments: a Windows PC sampling gaze position throughout the experiments and a Macintosh PC for stimulus presentation. The two computers were connected to each other via a dedicated ethernet connection. Participants were seated about 45 cm distant from the stimulus screen. Their head was supported by a chin rest. The presentation monitor was a 22″ (20″ visible screen diagonal) *iiyama Vision Master Pro 514* with a vertical frequency of 200 Hz at a resolution of 800 × 600 pixels.

Eye movement recording

Eye movements were recorded with the *EyeLink II* system (*SR Research*, *Ltd*, Canada). Gaze positions were sampled and stored on the Windows PC with a frequency of 500 Hz. Eye movement events (saccades, fixations, and blinks) were detected online, stored, and sent to the stimulus computer via the ethernet connection. Saccades were detected when eye velocity and eye acceleration crossed thresholds of 22°/s and $4{,}000°/s^2$, respectively. The eye-tracking computer calculated the gaze position from the camera data every 2 ms, detected saccades by comparing successive samples, and then sent a message to the Macintosh PC via ethernet. The delay of the availability of saccade information on the stimulus computer was in the range between 24 and 32 ms. On the Macintosh PC, the incoming eye movement event triggered the further stimulus presentation within the time of two monitor refreshes (less than 10 ms). At an average saccade duration of about 60 ms this was sufficient for complete intra-saccadic stimulus changes. For off-line analysis the data stored on the recording PC was used. Off-line data analysis confirmed that display changes occurred usually during saccades. Infrequent trials, in which the saccade detection was too slow to adjust the display during the saccade, were omitted from further analysis.

Experiment 1: main experiment

Six participants, two males and four females, took part in Experiment 1. Before beginning the first experimental session, each participant received detailed instructions by the investigator and was allowed to perform some trials to familiarize with the task. At the beginning of each experimental session the eye-tracking system was calibrated and validated, using an automated, manufacturer-provided routine involving successive fixations on the single points of a 3 × 3 grid. The participant's dominant eye was used for tracking gaze position. Throughout the experiment the instructor was present in most of the sessions. Experiment 1 was carried out in two different contrast conditions (high and low luminance-contrast), conducted in separate sessions. Each participant took part in at least four sessions per contrast condition. An experimental session started with five regular saccade trials, in which the saccade target was not displaced intra-saccadically and no localization stimulus was shown. Subsequently, 50 adaptation trials and thereafter 200 localization trials followed.

Adaptation procedure

Each adaptation trial began with the presentation of a fixation point (a square of 1.25° × 1.25° visual angle) on an empty background. The fixation point was vertically centered and horizontally located 12.7° to the left of the center of the screen. After a random time of 1,100–2,100 ms, the fixation point disappeared. At the same time the initial saccade target (T1, same features as fixation point) was presented 25.0° to the right of the fixation point. Participants were required to execute a saccade to T1 as fast as possible after its presentation. While the eyes were moving, T1 was replaced by T2, located 6.2° left of T1. Accordingly, the amplitude of the adapted saccade to T2 was decreased to 18.8°, or about 75% of the initial amplitude to T1. 1,000 ms after the intra-saccadic target displacement, T2 disappeared, and a new trial began with the presentation of the fixation point.

Localization procedure

The localization trials largely resembled adaptation trials. The presentation of the fixation point and T1 were identical, as was the target back-step, i.e. the distance between T1 and T2. In addition, at some predetermined time during a trial a localization stimulus was presented briefly (10 ms). The stimulus was a vertical bar of about 0.4° width, extending the height of the screen (37.5°). The bar was presented at one of seven possible positions (15.8°, 17.8°, 19.9°, 21.9°, 24.0°, 26.1° or 28.1° to the right of the fixation point). Stimulus presentation time ranged from about 200 ms before (referred to as −200 ms) to 200 ms after saccadic onset. After each localization trial, a mouse pointer appeared, and participants had to indicate the perceived horizontal position of the stimulus. If participants did not see the stimulus, they were instructed to click in the outermost right and lower corner of the screen. Such trials were discarded during later analysis. A temporal scheme of the events in single trials can be found in Fig. 1.

Fixation trials

In 10% of the localization trials, pseudo-randomly intermixed, the fixation point did not disappear, no saccade target (neither T1 nor T2) was presented, and the participants were requested not to elicit a saccade until they had indicated the perceived position of the localization stimulus by using the mouse pointer. The maintenance of fixation was monitored off-line. Trials in which fixation was broken were discarded from analysis.

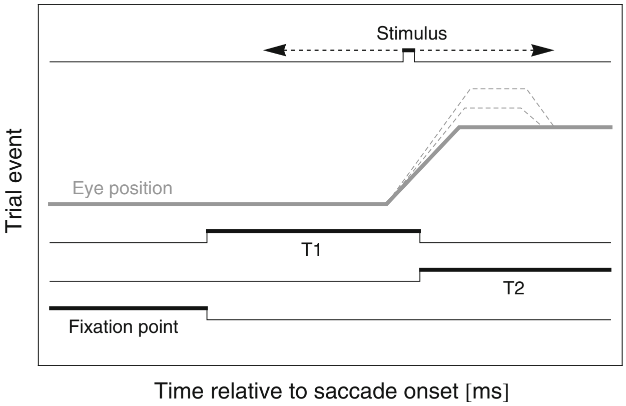

Fig. 1 Temporal scheme of events during exemplary adaptation and localization trial. Initially, only the fixation point (*FP*) was visible and the participant fixated it. After a random period of 1,100–2,100 ms the fixation point disappeared and at the same time the initial saccade target (*T1*) was presented. The participant conducted a saccade as fast as possible after appearance of the saccade target. While the eyes were moving (average saccade duration about 60 ms) T1 disappeared and the adaptation target (*T2*) appeared. Initially, saccades aim at *T1*, gradually adapting to *T2* over successive trials (*dashed gray lines*). The next trial started 1,000 ms later. In localization trials, at some time before, during or after the saccade (from about 200 ms before to 200 ms after saccade start) a stimulus was presented briefly (10 ms). The next trial started after the participant had indicated the perceived position of the flashed stimulus by means of a mouse pointer that appeared after the trial

Low luminance-contrast

In the low luminance-contrast condition, the background of the screen presentation was medium gray. The fixation point, the saccade target, and the localization stimulus were blue. Their brightness was preliminarily adjusted to be roughly equiluminant to the background. Complete equiluminance was neither tested nor necessary, the aim was solely to largely reduce luminance-contrast to result in a considerable amount of peri-saccadic compression. For the same reasons the background as well as the foreground items were structured by pseudo-randomly varying luminance by some amount between −20 and +20% independently for every pixel to increase positional uncertainty. Average luminance of the background and the foreground items was 5.0 and 3.8 cd/m^2, respectively, resulting in a Michelson contrast (C_M) of 0.14. Due to color contrast and the remaining luminance-contrast, the presented items were nevertheless clearly visible on the background. Ambient luminance was about 0.04 cd/m^2.

High luminance-contrast

In the high luminance-contrast condition, the background was black, the fixation point and saccade targets were dark gray, the localization stimulus medium gray. The monitor screen was covered with a dark car window foil, reducing the total luminance of the presentation by about two log units. This was done to minimize the possible effect of phosphor persistence, and to reduce visible references from the monitor edges. Resulting luminance of the background was below 0.01 cd/m^2. Luminance of the saccade target and the stimuli was 0.08 and 0.4 cd/m^2, respectively, resulting in a Michelson contrast of 0.78 and 0.95. The experimental room was almost completely dark (luminance below 0.01 cd/m^2). To avoid dark adaptation, the screen turned completely white for 150 ms after each trial.

Experiment 2: non-adapted fixation condition

The non-adapted fixation condition served as a baseline for the localization trials described in Experiment 1, because even during fixation, localization of briefly flashed objects is not necessarily veridical (Müsseler et al. 1999).

All six participants from Experiment 1 also took part in Experiment 2. Experiment 2 was carried out on separate days, and in both high and the low luminance-contrast condition. Each participant took part in between one and three sessions per condition. In this experiment, single sessions exclusively consisted of 210 fixation trials without a previous adaptation period. The single trials in Experiment 2 were identical to the intermixed fixation trials in Experiment 1.

Experiment 3: non-adapted saccade condition

The non-adapted saccade condition was conducted to estimate the amount and time course of the isolated peri-saccadic shift under conditions similar to Experiment 1. For this purpose, localization trials without previous adaptation were carried out, so there are no effects of adaptation in Experiment 3. Furthermore, Experiment 3 was conducted in high luminance-contrast condition only, so no peri-saccadic compression occurred.

Five participants, two males and three females, took part in Experiment 3. All of them had also taken part in Experiments 1 and 2. Experiment 3 was executed on separate days, and in separate experimental sessions of 220 trials, consisting of 5 initial saccades and 215 localization trials without a previous adaptation period. The single localization trials resembled the localization trials in Experiment 1, except there was no intra-saccadic target displacement. Instead, T1 was presented at 18.8°, the location of T2, and did not change during the eye movement.

Experiment 4: early pre-saccadic presentation

From the experiments of Awater et al. (2005) the adaptation-induced shift is known to be present at least 200 ms preceding a saccade and disappearing before saccade onset. There are at least two possible explanations for these results. First, the adaptation-induced shift is a transient effect, visible for a certain time before the planning or execution of an adapted saccade; or, second, it is an effect arising from an error in the processing of positional information across saccades, being visible at any time before an adapted saccade. We therefore conducted an experiment with much earlier pre-saccadic stimulus presentations.

Three participants, one male and two females, took part in Experiment 4, all of them had also taken part in Experiments 1 and 2. Experiment 4 was conducted in low luminance-contrast condition only. Each participant took part in between four and six sessions. Experiments were carried out on separate days, and in separate experimental sessions of 255 trials, consisting of 5 initial trials, 50 adaptation trials, and 200 localization trials. The single trials were identical to Experiment 1, with only the timing of the stimulus presentation in localization trials being modified. The localization stimuli were presented either peri-saccadically between −175 and 175 ms or about 400, 700 or 1,000 ms before saccade onset, each in 20% of the trials. Note in the latter three cases the localization stimuli were presented even before presentation of T1. The remaining 20% of the trials were fixation trials, also identical to the fixation trials in Experiment 1.

Experiment 5: alternative adaptation procedure

In Experiments 1 and 4, T1 and T2 always appeared at the same location on the monitor screen. By conducting an experiment with a less monotonic procedure we controlled for the potential impact of this stereotypical setup and participant's potential response strategies.

Four participants, two males and two females, took part in Experiment 5; three of them had also taken part in Experiments 1 and 2. Experiment 5 was conducted in low luminance-contrast condition only. Each participant took part in four sessions. In Experiment 5, single sessions consisted of 260 trials, comprising 10 initial trials, 50 adaptation trials and 200 localization trials. The single trials very much resembled Experiment 1, except the starting point of the experimental sequence being varied in each trial. For this purpose, all presented items, i.e. fixation point, T1, T2, and localization stimuli, were shifted to the left or to the right on the background by a fixed offset. This offset was taken from one of seven values, equally distributed from 0° to 15°, independently in each trial. Thus, the complete layout was centered at one of seven equidistant monitor positions, spanning 15° visual angle. Geometric relationships between the individual items remained unchanged.

Experiment 6: emergence of the adaptation-induced shift during the development of saccadic adaptation

In the previous experiments, we examined the adaptation-induced shift under conditions of well-established short-term adaptation, since a large number of trials are necessary to give a comprehensive picture of this effect. In Experiment 6, we reduced the number of independent variables and simplified the procedure, so that an estimate of the emerging adaptation-induced shift could be derived from each trial. Furthermore, there is no separate adaptation procedure in this experiment. Instead localization stimuli are presented throughout the experiment.

Ten participants, five males and five females, took part in Experiment 6; five of them had also taken part in previous experiments. Experiment 6 was conducted in low luminance-contrast condition only and resembles Experiment 5 insofar as the complete layout of presented items was shifted as a whole across the screen in each trial independently. Each participant took part in one experimental session only. Single sessions consisted of 200 trials, comprising of 50 initial trials, 100 trials with intra-saccadic target displacement, and 50 final trials. In the initial and final trials, T1 was presented without any intra-saccadic displacement. Thus, T1 was still visible post-saccadically. The middle 100 trials resembled the localization trials in Experiment 1, i.e. T1 was intra-saccadically replaced by T2. Thus, the first 50 trials provide a baseline for localization without saccadic adaptation. During the next 50 trials, participants acquired saccadic adaptation. The ensuing 50 trials were used as a baseline for localization after saccadic adaptation. In the final 50 trials, de-adaptation could take place. Note that in all 200 trials localization stimuli were presented. Stimulus presentation was limited to the epoch around 300 ms before saccade onset and to three stimulus positions between T1 and T2 (19.9°, 21.9°, and 24°). In every trial, after the saccade, participants indicated the horizontal position of the stimulus by means of the mouse pointer.

Data analysis

Data analysis was done in *Mathematica 5.2* (*Wolfram Research, Inc.*). For each experimental *session*, adaptation was verified by averaging the amplitudes of the localization trials. Data were discarded if the mean adapted landing position in a single session deviated more than 3.1° from T2 or if the mean landing position of the first ten trials (5 initial trials and the first 5 adaptation trials), did not differ significantly (t test, $\alpha = 0.05$) from the mean landing position after adaptation. On average across participants, adaptation was nearly complete, i.e. the adapted landing position of the eyes was mostly on or around the adaptation target. An example of the course of adaptation in a single experimental session is shown in Fig. 2.

In saccadic localization trials, single trials were discarded when the saccade target was missed by more than 6.2°, the target displacement was not completed before the eyes had landed, or the participants responded that they had not seen a localization stimulus. Fixation trials were discarded in response to a saccade occurring between the

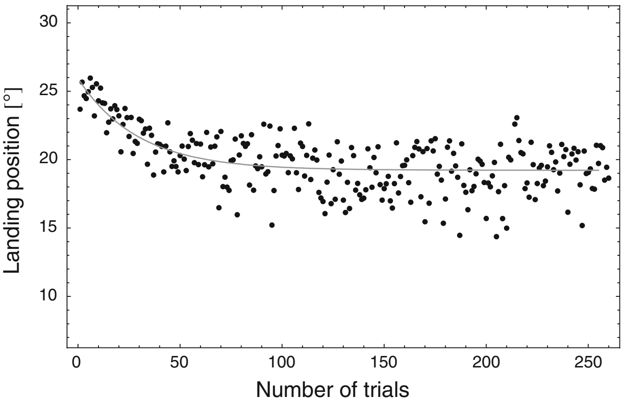

Fig. 2 Time course of saccadic adaptation. Saccade amplitudes against trial numbers for every trial of a single experimental session. The session consists of 255 trials (5 saccade trials without target displacement, 50 adaptation trials, and 200 localization trials). T1 is at 25°, T2 is at 18.8°. *Black dots* represent single trials, the *solid line* an exponential decay curve fitted to the data [$f(x) = 19.2 + 6.7^{-0.049x}$, rate constant 20.3 trials, $r^2 = 0.17$]

beginning of a trial and the mouse response, or participants responding they had not seen a localization stimulus.

Results

Experiments 1 and 2: independence of adaptation-induced shift and peri-saccadic compression

In Experiment 1 we varied stimulus contrast in order to isolate the adaptation-induced shift from peri-saccadic compression. Successful adaptation was verified for each experimental session as described in "Data analysis". Nine of the original 78 experimental sessions were discarded due to insufficient adaptation. Of the remaining sessions, 76% of the saccade trials and only 33% of the fixation trials reached the criteria specified above. The average rate constant of the exponential decay curve was 20 ± 3 trials (mean ± SE) and 23 ± 3 trials in low and high luminance-contrast condition, respectively. The amount of saccadic adaptation, i.e. the difference between mean landing positions of the five initial trials and of the localization trials, averaged 4.8° (SE 0.2°) and 4.3° (SE 0.3°) across participants for the low luminance-contrast condition and the high luminance-contrast condition, respectively. The results for both conditions are shown in Fig. 3. The data was averaged in bins of 25 ms for each of the seven stimulus locations.

The low luminance-contrast condition (Fig. 3a) produced a large amount of peri-saccadic compression. Stimuli presented around the time of the saccade onset were localized incorrectly. The direction of mis-localization depended on the position of the localization stimuli. While stimuli presented between the fixation point and T2 appeared shifted in the direction of the saccade, stimuli presented beyond T2 appeared shifted in the opposite direction. Note that in both cases stimuli appeared shifted towards T2. The focus of compression was the position of T2, from which the actual landing position of the eyes does not differ significantly (T2: 18.8°; mean adapted landing position: 19.4°, SE 0.4°; *t* test, $p > 0.05$). There was no compression towards T1, which was presented during saccade planning and onset (T1: 25.0°; mean initial landing position: 24.2°, SE 0.7°). This reproduced the findings of Awater et al. (2005).

Stimuli presented before saccade onset were perceived systematically shifted in the direction of adaptation, while stimuli presented after saccade end were perceived roughly at the same positions as in the non-adapted fixation condition. The amount of this adaptation-induced shift was clearly smaller than the target back-step. While the target back-step was 6.2°, the mean adaptation-induced shift (well before the saccade compared to after the saccade) was 3.3°. Thus, the adaptation-induced shift was also smaller than the amount of saccadic adaptation, which averaged 4.8° (SE 0.3°).

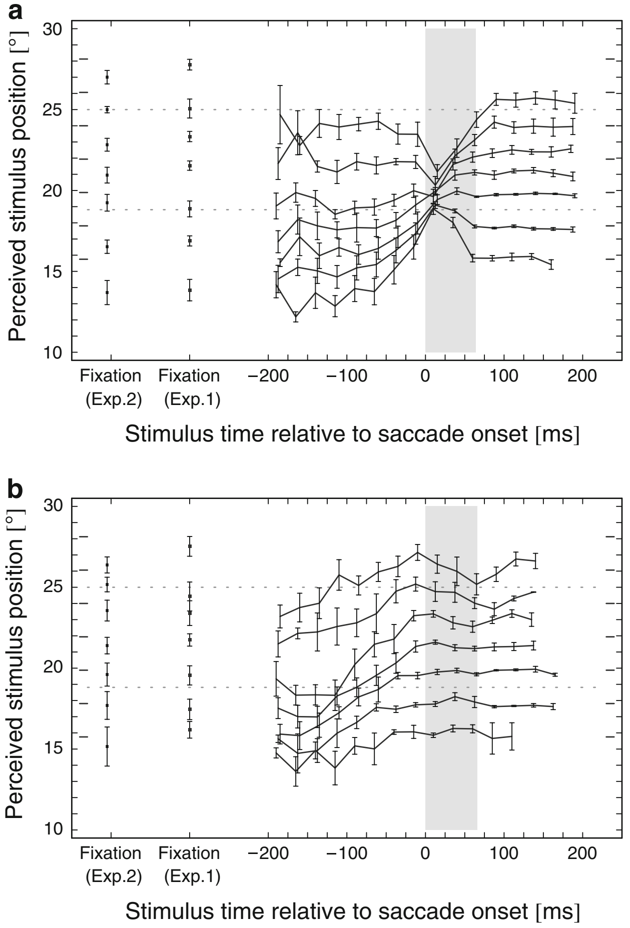

Fig. 3 Peri-saccadic localization after adaptation in Experiment 1. **a**, **b** Low luminance-contrast and high luminance-contrast condition, respectively. The *vertical tick marks* indicate the real stimulus positions, the *horizontal dotted lines* the positions of T1 and T2. The *light gray area* indicates the time of saccade start and average saccade termination. All results are means across participants (*N* = 6). *Error bars* indicate standard error of the mean. The "*Fixation*" columns show the results of the fixation trials intermixed with saccade trials in Experiment 1 and of localization during non-adapted fixation in Experiment 2

Intermixed with saccade trials were trials in which the participants kept fixation, but were in the adapted state. In these trials participants showed very little mislocalization confirming the previous observation that the actual execution of an adapted saccade is mandatory for the appearance of the adaptation-induced shift (Awater et al. 2005).

There was no qualitative difference between the results of the non-adapted fixation condition from Experiment 2, which was recorded in separate sessions, and the fixation trials that were recorded in the adapted state in Experiment 1. Results from Experiment 2 are integrated in Fig. 3.

While the low luminance-contrast condition largely reproduced earlier experiments, the high luminance-contrast condition (Fig. 3b), being described in the following, was a novel approach. In the high luminance-contrast condition, the mean initial and adapted landing positions were 23.2° (SE 0.4°) and 18.9° (SE 0.4°), respectively. The amount of saccadic adaptation was 4.3° (SE 0.3°). No peri-saccadic compression was visible here. This absence of peri-saccadic compression was the incentive for choosing a relatively high luminance-contrast between the stimuli and the background. Nevertheless, the amount of adaptation-induced shift, 3.7°, was qualitatively similar to that in the low luminance-contrast condition. Early pre-saccadic and late post-saccadic, the curves in Fig. 3a, b do not differ significantly.

To assess possible differences between conditions, a two (contrast condition) × four (saccade condition: adapted fixation, non-adapted fixation, pre-saccadic and post-saccadic stimulus presentation) × seven (stimulus position) repeated-measures ANOVA was calculated on the localization judgments. Pre- and post-saccadic stimulus presentation relates to the earliest pre-saccadic and latest post-saccadic time bin, respectively, that was available for each individual participant. ANOVA revealed significant effects of saccade condition and stimulus position ($F = 132.5$ and $F = 404.5$, respectively; $p < 0.01$ in both cases), but no significant effect of contrast condition ($F = 2.2$, $p > 0.1$). Thereby, our premise of peri-saccadic compression but not adaptation-induced shift being affected by varying stimulus contrast was strengthened. Only interaction between saccade condition and stimulus position reached significance ($F = 2.8$, $p < 0.01$), pointing at the fact that stimuli presented between T1 and T2 are affected by the pre-saccadic adaptation-induced shift to a higher degree. Post hoc tests (Tukey's test, $\alpha = 0.01$) revealed significant differences between all seven stimulus positions. Furthermore, the pre-saccadic stimulus presentation differed significantly from all other saccade conditions (adapted fixation, non-adapted fixation, post-saccadic presentation). No other differences reached significance.

Experiment 3: time course of the adaptation-induced shift

To investigate the time course of the adaptation-induced shift we calculated at each point in time the difference between the localization in saccade trials from Experiment 1 and the localization in the non-adapted fixation condition from Experiment 2, averaged over the seven stimulus locations and the participants. This was done for both contrast conditions. Figure 4 shows the result. In both conditions, the adaptation-induced shift was about 3°, beginning to decrease between 150 and 100 ms before saccade onset. About 50 ms after saccade onset localization was correct (i.e. shift was zero). In the time around saccade onset, the curves differ because the peri-saccadic compression in the low luminance-contrast condition also influenced the localization (dashed part of the black curve in Fig. 4). From the data in Fig. 4 it remains unclear whether the adaptation-induced shift reached a stable level for stimuli presented earlier than 200–300 ms before saccade onset. This fact is explicitly addressed in Experiment 4.

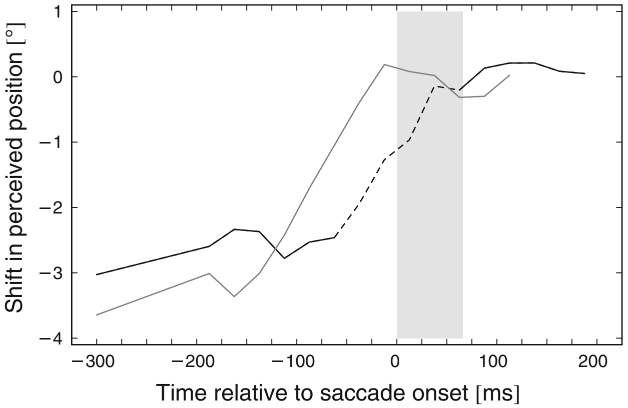

Fig. 4 Time course of mean overall shift in low luminance-contrast condition (*black*) and high luminance-contrast condition (*gray*) from Experiment 1. *x*-axis shows the time of stimulus presentation relative to saccade onset. The overall shift was based on means across participants ($N = 6$) and was computed as the difference between perceived stimulus positions in saccade trials and in non-adapted fixation trials from Experiment 2, averaged across stimulus positions. A value of 0 means no overall shift, negative values mean a shift in the direction of the fixation point. The *light gray area* indicates the times of saccade start and average saccade termination. The *dashed part of the black curve* indicates the epoch in which the peri-saccadic compression occurs

However, the time course of the adaptation-induced shift near saccade onset in the high luminance-contrast condition was likely to be contaminated by a further saccade-related effect, the peri-saccadic shift. To determine the time course of the pure adaptation-induced shift, we aimed at eliminating the putative impact of the peri-saccadic shift. Therefore, in Experiment 3, we measured the isolated peri-saccadic shift in the same condition and with the same participants as in Experiment 1 but without saccadic adaptation. Thus, Experiment 3 was conducted without an intra-saccadic target displacement. The saccade target was presented at the location T2. The difference between these measurements in Experiment 1 and the measurements in Experiment 3 should reveal the time course of the thereby isolated adaptation-induced shift in more detail.

Figure 5 shows the results of this experiment. The data was divided and averaged in bins of 25 ms. Around the time of the saccade, there was a specific mis-localization noticeable. Immediately before saccade onset, participants perceived the location of the stimuli slightly shifted in the

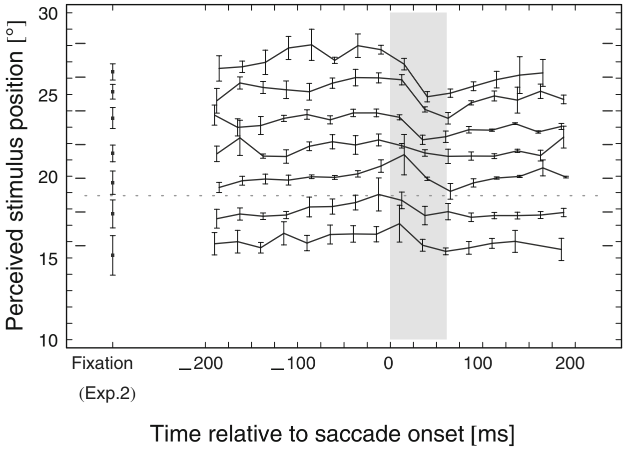

Fig. 5 High luminance-contrast condition without previous adaptation procedure and without intra-saccadic target displacement in Experiment 3. The *x*-axis shows the presentation time of the stimulus relative to saccade onset. The *y*-axis shows the perceived horizontal position of the localization stimuli. The *vertical tick marks* indicate the real stimulus positions, the *horizontal dotted line* the position of the saccade target. The *light gray area* indicates the times of saccade start and average saccade termination (mean saccade duration 62 ms). All results are means across participants (*N* = 5). *Error bars* indicate standard errors. All participants also took part in Experiments 1 and 2. The "*Fixation*" column shows results of non-adapted fixation from Experiment 2

direction of the saccade, while shortly after saccade onset, stimuli were perceived shifted in the opposite direction. This basically agrees with earlier results (Honda 1991; Lappe et al. 2000). As specified for the adaptation-induced shift, the index of the mean peri-saccadic shift was calculated as the mean across stimulus positions of the differences between the localization in saccade trials and the localization in the non-adapted fixation condition in Experiment 2 (Fig. 6a).

Using this isolated peri-saccadic shift to subtract adaptation-independent mislocalization from the adaptation-induced shift obtained by Experiment 1, we were able to obtain the pure adaptation-induced shift around saccade onset (Fig. 6b). The adaptation-induced shift started to diminish about 150 ms before saccade onset, reaching zero shortly after saccade onset. Figure 6b indicates the way the characteristics of the time course changed in comparison to the first experiment. Especially the slope of the decrease was less steep, with the maximum being reached later. Due to the time course of the peri-saccadic shift, it only affected the adaptation-induced shift in the epoch from about 100 ms before to about 100 ms after saccade onset.

Experiment 4: pre-saccadic temporal extent of the adaptation-induced shift

The adaptation-induced shift depends on the execution of a saccade and was not observed in (adapted) fixation trials. Moreover, it was present at least 300 ms preceding a saccade and disappearing before saccade onset (see Fig. 6b). To test whether the adaptation-induced shift is a transient effect, visible for a certain time before the planning and execution of an adapted saccade, or an effect arising from an error in the processing of positional information across saccades, being visible at any time before an adapted saccade, we conducted Experiment 4 with much earlier stimulus presentations. Thereby we were also able to rule out the possibility of the adaptation-induced shift continuously increasing with earlier pre-saccadic stimulus presentations, which could not be addressed in the previous experiments.

The results of Experiment 4 are shown in Fig. 7. Mean initial and adapted landing positions were 24.2° (SE 1.0°)

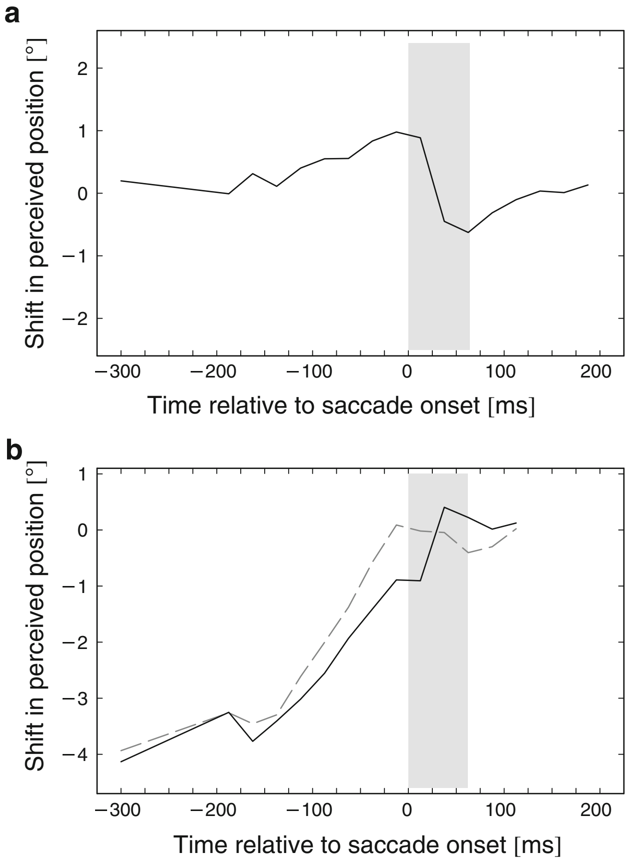

Fig. 6 Time-courses of peri-saccadic and adaptation-induced shift. *X*-axes show the time of stimulus presentation relative to saccade onset. The *light gray area* indicates the times of saccade start and average saccade termination. All indices are based on mean results across identical participants (*N* = 5), computed as the difference between perceived stimulus positions in saccade trials and in non-adapted fixation trials from Experiment 2, averaged across stimulus positions. **a** Mean peri-saccadic shift from Experiment 3. *Negative values* indicate a shift in the direction of the fixation point, *positive values* in saccade direction. **b** Direct comparison of the pure adaptation-induced shift (*solid black line*), computed as the difference of the overall shift of Experiment 1 and the perisaccadic shift from **a**, and the overall shift from Fig. 4 (*dashed gray line*)

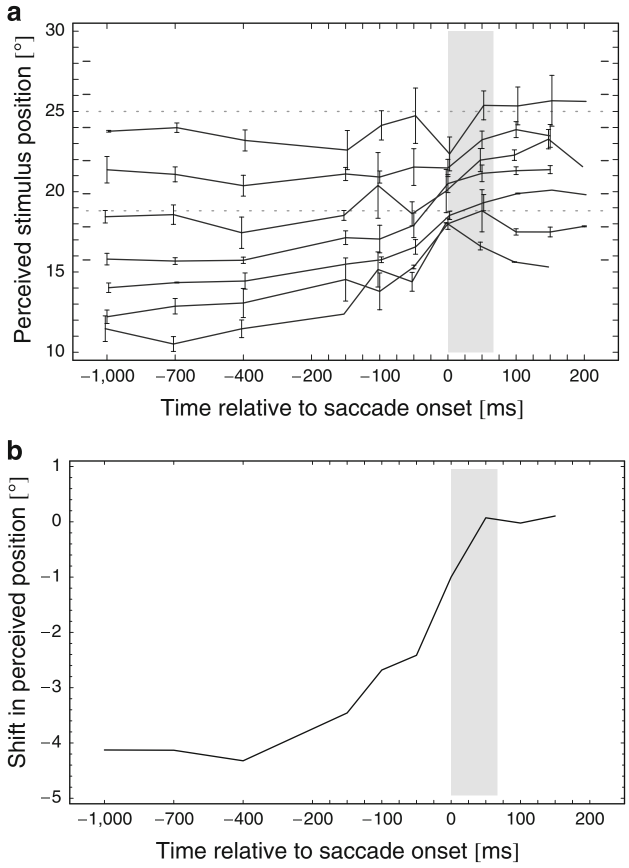

Fig. 7 Results from Experiment 4 with early pre-saccadic stimulus presentation. a Perisaccadic localization. **b** Adaptation-induced shift. The *x*-axis shows the presentation time of the stimulus relative to saccade onset, the *y*-axis the perceived horizontal position of the localization stimuli. Note that the *x*-axis is compressed for earlier presaccadic presentation times. The *vertical tick marks* indicate the real stimulus positions, the *dotted lines* the positions of T1 and T2. The *light gray area* indicates the times of saccade start and average saccade termination (mean saccade duration 67 ms). All results are means and SEs across participants ($N = 3$)

and 19.1° (SE 0.5°), respectively, the resulting amount of adaptation being 5.1° (SE 0.6°). The adaptation-induced shift occurred similarly in all the early pre-saccadic presentations (−400 to −1,000 ms) of the localization stimuli. If the stimuli were presented up to 1 s before saccade onset, there was still the qualitatively equal amount of shift. This adaptation-induced shift is not a transient effect of saccade execution or planning, only arising in a certain time window before the saccade. It rather is a stable effect that appears whenever a stimulus location is presented before an adapted saccade but the response is retrieved after saccade execution.

Experiment 5: validation of the adaptation procedure

In Experiment 5, a different stimulus procedure was utilized, not only in adaptation trials but throughout the experiment. We aimed at testing whether participants show a comparable amount of adaptation-induced shift in this less monotonous protocol where the fixation point location varied across trials.

Results were qualitatively similar to Experiment 1, and are not shown here. In Experiment 5, the adaptation-induced shift, i.e. the difference between pre-saccadic and post-saccadic localization, averaged 2.8° (SE 0.6°). The amount of saccadic adaptation, i.e. the difference between initial and adapted landing position, averaged 4.6° (SE 0.5°). A two (pre- or post-saccadic presentation time) × seven (stimulus position) repeated-measures ANOVA revealed significant differences for presentation time and stimulus position ($F = 76.9$ and $F = 77.9$, respectively; $p < 0.01$ in both cases) and no significant interaction ($F = 0.4$, $p > 0.8$). Pre- and post-saccadic stimulus presentation relates to each participant's earliest pre-saccadic and latest post-saccadic time bin, respectively.

The results of Experiment 5 were in many aspects comparable to the results of previous experiments. In particular, a pronounced amount of saccadic adaptation had been established and likewise adaptation-induced shift was visible.

Experiment 6: emergence of the adaptation-induced shift during the development of saccadic adaptation

In our previous experiments, participants underwent 50 separate adaptation trials before the localization stimuli were presented. In Experiment 6, localization stimuli were presented throughout the experiment. In the first 50 trials, only T1 was continuously visible, without an intra-saccadic target displacement. Landing position and localization performance during this period were used as a baseline and are referred to as pre-adaptation. In the next 100 trials, T1 was intra-saccadically replaced by T2. The first half of these trials (trials 51–100) are referred to as adaptation phase, the second half (trials 101–150) as post-adaptation. In trials 151–200, again only T1 was presented, referred to as de-adaptation phase. Dependent variables in each trial were the deviation of saccade landing position from T1 and the error in localization. Data from three of the ten participants had to be discarded from further analysis due to their inability to systematically localize the stimuli (errors > 5°). In the pre-adaptation phase, landing positions showed a considerable undershoot relative to T1 (average landing position across participants −2.5°, SE 0.6°), whereas localization was closer to veridical values (mean localization error −1.6°, SE 0.7°; negative errors indicate mislocalization in the direction of fixation point and T2). In the post-adaptation phase, this difference was less pronounced (average landing position −5.0°, SE 0.6°; mean localization error −4.4°, SE 0.4°). For further analysis, data was normalized to individual pre-adaptation and post-adaptation levels. This allows for directly comparing the relative time course of changes in landing position and localization judgments.

Results of Experiment 6 are shown in Fig. 8. Data was divided and averaged in bins of ten trials. In the adaptation phase, localization errors converged faster towards post-adapted level than deviations in landing position. More precisely, the transition of localization errors was abrupt, reaching values below 0.3 already in the first ten adaptation trials. In contrast, transition of landing positions was smooth, showing a roughly exponential time course. The de-adaptation phase yielded comparable but inverted results, even though not all participants showed complete de-adaptation of the landing position and variability is higher across participants. These results are confirmed with a two (modality: landing position vs. localization error) × five (trial bins) ANOVA per experimental phase (adaptation and de-adaptation). For the adaptation phase, ANOVA revealed significant effects of modality and of trial number ($F = 4.4$ and $F = 7.8$, $p < 0.04$ and $p < 0.01$, respectively), and no significant interaction ($F = 1.7$, $p > 0.1$). For the de-adaptation phase, only modality reached significance ($F = 19.9$, $p < 0.01$).

We subsequently calculated paired t tests across participants for all individual bins of trials, separately for each phase. In both adaptation and de-adaptation phase, differences between landing position and localization error were significant only for the very first bin of each phase ($\alpha = 0.05$, Bonferroni-corrected for multiple comparisons).

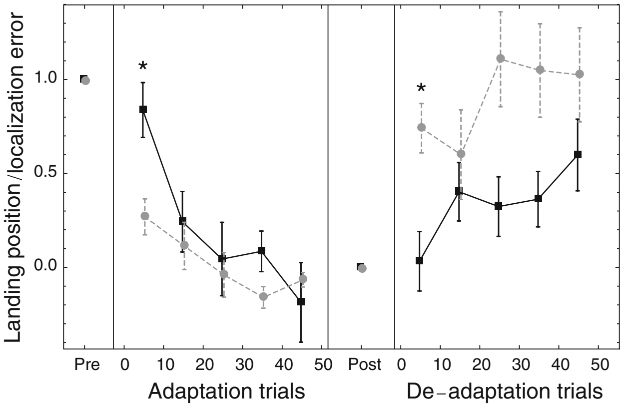

Fig. 8 Results from Experiment 6. The x-axis shows the trial number within the current phase (adaptation or de-adaptation), the y-axis the saccade landing position and the error in localization, respectively. "*Pre*" and "*Post*" columns indicate pre-adaptation and post-adaptation values, respectively. Pre-adaptation values are normalized to 1.0, post-adaptation values to 0.0. *Black lines* indicate saccade landing position, the *dashed gray lines* indicate localization error. Participant's data have been normalized to individual participant's average landing position and localization error in pre-adaptation phase and post-adaptation phase, respectively. All results are means and SEs across participants ($N = 7$). *Asterisks* indicate significant differences between landing position and localization error for the first bin of trials (paired t test)

This means that landing position and localization error particularly differed from each other in the first few trials of the adaptation and de-adaptation phase. In the adaptation phase, the normalized values for landing position and localization error dropped from 1.0 to 0.84 and 0.27, respectively, during the first bin of trials. Thus, localization error instantaneously and closely approached post-adaptation level. However, it still differed significantly ($p < 0.02$ and $p < 0.01$, respectively) from post-adaptation level. In de-adaptation phase, the difference between landing position and localization error became even more apparent. The normalized values increased from 0.0 to 0.03 and 0.74 for landing position and localization error, respectively, within the first bin of trials. As before, localization error as well differed from pre-adaptation level ($p < 0.05$).

Discussion

In the first three experiments, we demonstrated that the adaptation-induced shift and the peri-saccadic compression are independent, whereby the two effects can be evoked separately and superimpose if occurring together. The adaptation-induced shift did not differ significantly in high and low luminance-contrast conditions, whereas peri-saccadic compression varied with luminance-contrast. The focus of peri-saccadic compression, if occurring, was close to T2. This basically agrees with findings of Awater et al. (2005).

Having quantified the peri-saccadic shift under the same stimulus conditions as the adaptation-induced shift, but with non-adapted saccades, we were able to estimate the time course of the pure adaptation-induced shift by subtracting the data gained under non-adapted conditions from the data gained under adapted conditions, where both shifts occured together. By isolating the adaptation-induced shift in this way we were able to specify its time course in much detail. This revealed several findings. First, the adaptation-induced shift begins to diminish about 100–150 ms before the onset of an adapted saccade. At this time the eyes had not started moving yet and the (adapted) saccade has not been executed. Note that the actual execution of an adapted saccade is nevertheless necessary, given that there was no adaptation-induced shift in adapted fixation trials. Second, the decrease of this effect is smooth and gradual and spans about 200 ms. Third, it became obvious that the offset of the adaptation-induced shift occurs sometime during the saccade. This offset time is similar to the offset time of peri-saccadic compression and peri-saccadic shift. This suggests that there is a common time—late during the execution of the saccade—until which the spatial localization is derived from the processing of visual input together with eye movement signals and from which on exclusive retinal signals are used for localization. While the offset time of the adaptation-induced shift and other peri-saccadic effects is similar, the time course before saccade start proved to be very different. While the adaptation-induced shift was visible long before an adapted saccade and began to decrease 100–150 ms before its start, peri-saccadic compression, peri-saccadic shift, and saccadic suppression all emerge temporally closer to saccade start. Consequently, the origin of the adaptation-induced shift is likely to be different from the origin of the peri-saccadic compression.

Isolating the time course of the pure adaptation-induced shift also enabled us to verify the post-saccadically visible target, T2, as the focus of peri-saccadic compression. Initially, this becomes apparent from Fig. 3a, corroborating findings from Awater et al. (2005). However, they had further hypothesized that subtracting the adaptation-induced shift from their data could reveal T1 as the real focus of compression. From our data, we can now exclude this possibility, since the adaptation-induced shift has already disappeared at the time of maximum compression (see Fig. 6b).

Awater and Lappe (2006) have suggested that the peri-saccadic compression can be understood as a combination of a pre-saccadic compression of the distance between the flashed object and the saccade target by an oculomotor feedback signal that modulates responses to flashes near the saccade target (Hamker et al. 2008), and a post-saccadic localization procedure in which the saccade target is used as a reference, whereby the object location is estimated from its (compressed) distance to the saccade target. The combination of these two processes explains why the magnitude of peri-saccadic compression depends on the presence of post-saccadic references (Lappe et al. 2000) while compression of the distances between flash and target can also be observed in the absence of post-saccadic references (Morrone et al. 2005; Awater and Lappe 2006; Georg et al. 2008). The post-saccadic localization procedure becomes obsolete if the stimulus is presented late during the saccade, because it will still be visible after the saccade. In this case, pure retinal localization is sufficient. A trans-saccadic localization procedure involving post-saccadic references may also underly in part the adaptation-induced shift. The localization of the stimulus may be encoded relative to the pre-saccadic target position and read-out after the saccade with respect to the post-saccadic target position. Since the target moved during the saccade, the localization of the stimulus shifts in the same direction as the target movement. This is also in line with T2 being the focus of compression in our experiments.

Compression towards T2 thus emphasizes the role of T2 as a post-saccadic reference and a potential anchor point for post-saccadic retrieval of positional information. In our experiments, however, the magnitude of the adaptation-induced shift was considerably smaller than the target displacement. The adaptation-induced shift in the amplitude-decreasing condition was only about 3°–4°, compared to the size of the intra-saccadic target back-step of 6.2°, and the actual reduction of the saccade amplitude of about 4°–5°. Therefore, the adaptation-induced shift is not a simple one-to-one transformation of neither the target displacement nor the amplitude reduction. It is thus unlikely that the trans-saccadic localization procedure outlined above is the sole reason for the adaptation-induced shift. It might at least partially be delivering the basis for spatial coding and decoding across an adapted saccade in general.

Experiment 4, with much earlier pre-saccadic stimulus presentations, proved the adaptation-induced shift not to be a transient effect that is visible only for a certain time during the planning or execution of an adapted saccade. This applies at least for stimulus onsets up to about 1,000 ms before saccade onset. To our knowledge, there is no peri-saccadic effect that is operative that far pre-saccadically. Instead, the adaptation-induced shift can be expected to arise from a systematic error in the processing of positional information across adapted saccades, being visible for stimuli presented at any time before such a saccade, provided that the localization response is given post-saccadically. Moreover, results of Experiment 4 also limit the potential impact of memory loss on our data. If the adaptation-induced shift was generated by a loss of positional information over time, the adaptation-induced shift was expected to continuously evolve for more pre-saccadic presentation times. Given that there is a reliable positional reference available for localization, a less reliable stimulus is biased towards this reference. The fixation point or one of the saccade targets could serve as this positional reference (Deubel et al. 1996, 1998; Müsseler et al. 1999; Deubel 2004). This bias would be expected to increase with increasing reliability of the references and with decreasing reliability of the stimulus (Niemeier et al. 2003, 2007).

The adaptation-induced shift has also been found in trials in which the target was post-saccadically not available as a visual reference (Awater et al. 2005; Collins et al. 2007). Collins et al. directly compared the spatial layout of the localization shift with that of the motor adaptation. Both were similar in shape if the saccade target was extinguished during the saccade. We also conducted a control experiment with intra-saccadic target extinction that confirmed these findings (data not shown). If the adaptation-induced shift occurred without visual feedback of the target displacement, it must, at least in part, result from motor signals. Naturally, the actual availability of visual references is the crucial factor in these experiments. Since the experiments of Awater et al. (2005) and Collins et al. (2007) were not conducted in complete darkness, the availability of external, visual references cannot be ruled out.

Different possible origins of the adaptation-induced shift have been proposed, one of them the idea of perceptual remapping (Bahcall and Kowler 1999). Short-term saccadic adaptation causes changes in saccade gain, i.e. the ratio between the intended and the executed saccade. After amplitude-decreasing adaptation—also referred to as gain-decreasing adaptation—this gain is smaller than 1. This gain change might cause localization errors, if the intended eye movement is used for converting pre-saccadic to post-saccadic positions, but the actual eye movement differs from that (see Bahcall and Kowler 1999). Without post-saccadic visual references, the location of a pre-saccadically presented object must be derived from trans-saccadic memory of the retinal location of the object, and a motor estimate of the eye displacement, i.e. an efference copy signal (see Honda 1989, 1991), or some measure of post-saccadic orbital position, such as proprioception of tonic rate of motoneuronal firing. Either of these would be adequate given that the pre-saccadic eye position was constant. Thus, in the case of saccadic adaptation changing the gain of the saccade but neither efference copy signal nor the proprioceptive signal, the post-saccadic localization of the pre-saccadic object would be distorted. This model is implausible for several reasons. First, if the altered gain of the eye movement drives the perceptual effects, the magnitude of the resulting error—the adaptation-induced shift—should equal the amount of saccadic adaptation. However, the observed adaptation-induced shift is somewhat smaller than the gain change of the saccade. Second, the localization error predicted from the difference between intended and actual saccade is supposed to be mostly uniform across the visual field, which is clearly not the case (Awater et al. 2005; Collins et al. 2007; Bruno and Morrone 2007). In our experiments, stimuli between T1 and T2 were also shifted significantly further in the direction of adaptation than stimuli before or beyond these targets.

A different approach was recently discussed by Collins et al. (2007). Initially, they established the characteristics of a human adaptation field, i.e. the spatial window around the adapted site in which adaptation transfers to saccades of differing vectors (Frens and van Opstal 1994; Alahyane et al. 2007), analog to the monkey adaptation field (Noto et al. 1999). Furthermore, they directly compared the human adaptation field with the pattern of adaptation-induced mislocalization. The stimulus was presented pre-saccadically but had to be localized after the saccade. In conditions with and without post-saccadically visible saccade target—and thus complete or reduced post-saccadic visual references—two distinct and potentially superimposing patterns of localization errors became identifiable. The authors attributed these different patterns to the use of two different sources of positional information: (1) when post-saccadic, exocentric information, i.e. T2, is available, it is used for localization; (2) when no or insufficient post-saccadic references are available, extraretinal information is used for localization. So far, this has been described in the reference object theory of Deubel and colleagues (Deubel et al. 1996, 1998, 2002; Deubel 2004). Collins et al. (2007) applied this theory to the integration of positional information across adapted saccades. Available visual references induced a uniform shift in the direction of adaptation. This shift was larger around T1 and T2, decreasing with increasing distance. With reduced post-saccadic references, the pattern of mislocalization was spatially nonuniform, resembling the adaptation field previously established. The authors thus hypothesize that the metrics of a saccade required to acquire a certain position contribute to the localization at that position. The results of Bruno and Morrone (2007) also indicate that saccadic adaptation affects both the action map and the perception map. In their experiments, a spatially restricted shift in localization occurred for localization by verbal reports as well as for open-loop pointing movements.

Collins et al. (2007) gave evidence of the involvement of two independent mechanisms that may superimpose, interact, or alternate in generating adaptation-induced mislocalization. Following this approach, we aimed at estimating the weight of both sources of information—exocentric visual references, and egocentric extraretinal information—for localization in our experiments. In our experiments, the saccade target was always visible after the saccade. Therefore, visual references should be predominantly used for localization. The object reference theory also postulates that extraretinal information is of no importance when immediate post-saccadic visual references are available (Deubel et al. 1998). Hence, the impact of extraretinal information remains unclear from our Experiments 1–5. Moreover, participants were completely adapted in these experiments. Thus, any observed effects cannot unambiguously be attributed to the use of either visual references or extraretinal signals.

However, Experiment 6 allows for disentangling, at least to some extent, the potential impact of these different sources of information that are potentially used for the retrieval of positional information across adapted saccades. Although usually unnoticed by participants, the positions of the pre-saccadic target T1 and of the post-saccadic target T2 were not identical. If a stimulus position was pre-saccadically encoded relative to T1 and post-saccadically decoded relative to T2, a constant error of the size of the target displacement (6.2°) was introduced. Nevertheless, the expected size of the localization error resulting from this landmark effect was smaller than the size of the target displacement (Deubel 2004). The target was displaced by the same amount in each adaptation trial. In contrast, saccadic adaptation is a gradual process and can be described with an exponential decay curve. Especially in the early trials of the adaptation phase, there is still a considerable mismatch between the actual landing position and the post-saccadic target position. Thus, there is also a considerable mismatch between extraretinal information and post-saccadic visual references. In Experiment 6, the localization error predominantly followed the displacement of the post-saccadic visual reference (see Fig. 8). From trials with a continuously visible target to trials in which it was displaced during the saccade, the transition of the localization error was quite abrupt, compared to the more gradual adaptation of the saccade amplitude. Consequently, a large part of the localization error is likely to rely on visual references or landmarks.

A landmark effect forming the basis of the observed mislocalization is also in line with the notion of the adaptation-induced shift as a non-transient effect, being detectable as long as 1,000 ms before the execution of an adapted saccade. If the position of a stimulus is encoded before an adapted saccade but decoded after that saccade, the subsequent localization is affected no matter how long before the saccade the stimulus was presented—given that the position is still stored in memory. In our results, there is no evidence for a noticeable memory loss. Neither localization judgments nor variability systematically change for stimulus presentations between 400 and 1,000 ms before the saccade. This could be explained by the relatively simple and reduced experimental setup, containing only few potentially competing visual stimuli. Note that before the saccade, stimulus position is not necessarily encoded directly relative to T1. For early pre-saccadic presentation times only the fixation point is visible. Rather, the whole reference system has to be realigned to the post-saccadic reference.

Regarding the use of pre- and post-saccadic visual references, also the time course of the adaptation-induced shift relative to saccade onset has to be reassessed. The adaptation-induced shift gradually decreases between about 150 ms before and 50 ms after saccade onset. This decrease might well reflect the changeover between the pre-saccadic and the post-saccadic reference, relative to which positional information is encoded. Due to latencies and filtering operations in the visual system (see Pola 2004), stimuli presented shortly before saccade onset might already interact with post-saccadic available references. If a stimulus position is already encoded relative to the displaced, post-saccadically visible target, no adaptation-induced shift becomes apparent. Moreover, the time at which the adaptation-induced shift had completely disappeared, about two-thirds through the saccade, is almost exactly the time at which T1 was replaced by T2.

Visual references are not the only source of information used for the integration of positional information across adapted saccades. From Experiment 6 it becomes apparent that, additionally, a smaller component is involved that develops slower, potentially along with the motor adaptation. However, our methods used here are not sensitive enough to further specify the characteristics of this component in more detail. In particular, we could not distinguish between a perceptual remapping as proposed by Bahcall and Kowler (1999) or the restructuring of perceptual space as proposed by Collins et al. (2007). More specialized experiments are necessary to clarify this.

Acknowledgments M. L. is supported by the German Research Foundation DFG LA-952/2 and LA-952/3, the German Federal Ministry of Education and Research project Visuo-spatial Cognition, and the EC Project Eyeshots. The authors want to thank the anonymous reviewers for helpful comments and suggestions on previous versions of the manuscript.

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