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REINFORCING SACCADIC AMPLITUDE VARIABILITY

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Saccadic endpoint variability is often viewed as the outcome of neural noise occurring during sensorimotor processing. However, part of this variability might result from operant learning. We tested this hypothesis by reinforcing dispersions of saccadic amplitude distributions, while maintaining constant their medians. In a first experiment we reinforced the least frequent saccadic amplitudes to increase variability, and then reinforced the central part of the amplitude distributions to reduce variability. The target was placed at a constant distance from the fovea after the saccade to maintain the postsaccadic visual signal constant and an auditory reinforcement was delivered depending on saccadic amplitude. The second experiment tested the effects of the contingency. We reinforced high levels of variability in 4 participants, whereas 4 other participants were assigned to a yoked control group. On average, saccadic amplitude standard deviations were doubled while the medians remained mostly unchanged in the experimental participants in both experiments, and variability returned to baseline level when low variability was reinforced. In the control group no consistent changes in amplitude distributions were observed. These results, showing that variability can be reinforced, challenge the idea of a stochastic neural noise. We instead propose that selection processes constrain saccadic amplitude distributions.

Key words: variability, operant, motor control, shaping, selection, ocular saccade, humans

Ocular saccades are the rapid movements of the eyes that place the retinal image of a target on the fovea (the small high-acuity area of the retina), allowing the visual system to perceive the fine details of the visual environment. Because of the limited span of the fovea, saccades require a fine motor control as well as adaptive properties to maintain accuracy in response to changes occurring in the saccadic system or in the environment. Saccadic amplitude adaptation is a compelling example of the adaptive properties of the primate visuomotor system to changes occurring during the lifespan—such as growing, aging or ocular pathologies (Abel, Schmidt, Dell'Osso, & Daroff, 1978; Kommerell, Olivier, & Theopold, 1976). Saccadic amplitude is the distance traveled by the eye between two fixation points: When making a saccade toward an intended target the amplitude is usually such that the eyes land close to the object. However, pathological saccadic dysmetria is sometimes observed, as in patients suffering from monocular muscular weakness in whom saccades fall short of the intended goal. Amplitude adaptation may then be induced by placing a patch on the nonaffected eye to block vision: Saccadic amplitude progressively recovers (Optican, Zee, & Chu, 1985; Zee, Optican, Cook, Robinson, & Engel, 1976). Saccadic adaptation has also been demonstrated in the laboratory using an intrasaccadic step paradigm (McLaughlin, 1967) consisting in having the target surreptitiously jump backward or forward during the saccade so that the gaze lands away from the target. This induced mismatch between the target displacement and the saccadic amplitude is progressively compensated by the oculomotor system and the eye lands closer and closer to the target's position (see Hopp & Fuchs, 2004; Pelisson, Alahyane, Panouilleres, & Tilikete, 2010, for reviews).

It has been proposed that the postsaccadic error—the retinal eccentricity of the target after the saccade lands—is the feedback used to correct subsequent eye movements, leading to changes in saccadic gain—the ratio of the saccadic amplitude to the displacement of the target (Noto & Robinson, 2001; Robinson,

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Noto, & Bevans, 2003; Robinson, Soetedjo, & Noto, 2006; Seeberger, Noto, & Robinson, 2002; Wallman & Fuchs, 1998). According to Bahcall and Kowler (2000), the oculomotor system might use elaborated comparisons between the actual postsaccadic retinal error and a predicted retinal image. In a similar vein, computational motor control theories postulate a forward model that predicts the outcome of the movement, the result of the comparison with the actual retinal error being used to adjust the motor command when necessary (Chen-Harris, Joiner, Ethier, Zee, & Shadmehr, 2008; Wolpert & Ghahramani, 2000). Saccadic adaptation would therefore imply a servo-mechanism in which the magnitude of the output is capable of controlling the input by way of a hypothetical internal comparator, a proposition that is difficult to probe at the behavioral level (see Ingvaldsen & Whiting, 1997, for a related discussion).

Contrasting with these computational hypotheses, there is support for the idea that saccades have attributes of operant behavior. For instance, when monkeys had to perform a saccade in a direction associated with food, saccade peak velocities were higher, trajectories straighter and latencies shorter than in a nonreinforced direction (Lauwereyns, Watanabe, Coe, & Hikosaka, 2002; Takikawa, Kawagoe, Itoh, Nakahara, & Hikosaka, 2002; Watanabe, Lauwereyns, & Hikosaka, 2003). Activity changes were found in brain structures such as the superior colliculus (Ikeda & Hikosaka, 2003), the substantia nigra pars reticulata (Sato & Hikosaka, 2002) and the caudate nucleus (Lauwereyns et al., 2002; Nakamura & Hikosaka, 2006; Watanabe et al., 2003) depending on the reinforcement condition, a feature shared by other operant behaviors (Schultz, 2000). In humans, Xu-Wilson, Zee, and Shadmehr (2009) found that saccades had higher peak velocities and shorter durations when they were made to a stimulus with a high value (faces) than to a stimulus with a low value (random pixels). Montagnini and Chelazzi (2005) manipulated the difficulty of a letter discrimination task by reducing the delay between saccade initiation and target onset, so that the ability to perform the task (that is, to clearly see the target) required the subjects to reduce their saccade latency. Under these conditions saccadic latencies decreased and peak velocities increased. Moreover, we recently found that saccadic reaction time variability might be controlled by reinforcement (Madelain, Champrenaut, & Chauvin, 2007), further demonstrating that saccadic eye movements are operant behaviors. Interestingly, there is evidence that visual pursuit—another kind of voluntary eye movements—might also be controlled by learned contingencies (Darcheville, Madelain, Buquet, Charlier, & Miossec, 1999; Madelain & Krauzlis, 2003).

A critical feature of operant behaviors is that their variability might be controlled by reinforcement (Neuringer, 2002). A large body of research has been devoted to studying operant variability in various dimensions of behaviors, such as interresponse times (Blough, 1966), topography (Goetz & Baer, 1973; Pryor, Haag, & O'Reilly, 1969; Stokes, 1995) or response sequences in animals (e.g. Abreu-Rodrigues, Lattal, dos Santos, & Matos, 2005; Machado, 1989, 1992, 1997; Neuringer, 1992; Page & Neuringer, 1985) as well as in humans (e.g. Miller & Neuringer, 2000; Neuringer, 1986; Stokes & Balsam, 2001; see Lee, Sturmey, & Fields, 2007, and Neuringer, 2002, for reviews). We propose that saccadic endpoint variability may be placed under operant control as well. This hypothesis contrasts with the conventional view stating that sensorimotor variability results from an uncontrollable stochastic neural noise that affects each stage between a sensory event and the motor response—sensing, information processing, movement planning and executing (Van Beers, 2007; see Faisal, Selen, & Wolpert, 2008, for a review).

One could hypothesize that, in real life, specific levels of tolerated—or required—saccadic endpoint variability may be reinforced by the consequences of a movement, namely the clear vision of targets. For instance, a low level of variability is helpful when reading: Given that letters in peripheral vision appear too blurred to be distinguished, eye movements must accurately orient the fovea to specific locations in order to see each word of a sentence. On the other hand saccade accuracy is not relevant when facing a blank page: Eye movement consequences are the same whichever location is targeted and here the constraints acting on endpoint variability may be looser such that any level of variability would be equally efficient. To test the hypoth-

esis that part of saccadic variability may result from operant learning, we manipulated the reinforcement contingencies of saccadic amplitude variability and induced changes in the spread of the amplitude distributions.

EXPERIMENT 1: EXTENT OF CONTROL BY REINFORCEMENT CONTINGENCIES

Method

Rationale and Subjects

This experiment aimed at probing the extent of control one can exert on saccadic amplitude variability. The two authors and an undergraduate student (with normal or corrected to normal vision) had first to increase the spread of their amplitude distribution while maintaining the median constant, and then a comparable decrease in variability was required.

Auditory reinforcement was delivered on a trial-to-trial basis depending on the saccadic amplitude, the criteria being continuously updated. It was necessary to control the postsaccadic retinal error so that it remained constant after each saccade and matched the individual baseline saccadic gain (i.e., the ratio of the saccadic amplitude to the target displacement). Four experimental conditions were performed. All experimental procedures were reviewed and approved by the Institutional Review Board and each participant gave informed consent.

Regular Baseline

Four sessions of 200 regular saccades were used to assess the baseline saccadic gain in each participant, that is, the median gain computed over these baseline saccades. Each trial started with a random fixation period of 500–1000 ms during which a white fixation cross was displayed pseudorandomly between 2.4° and 13° to the left or right of the center of the screen against a grey background (luminance 40 cd/m2). When the fixation cross was extinguished the target stimulus, a luminance-defined Gaussian patch (SD=0.93°, maximum luminance 62 cd/m2), appeared between 9.5 deg and 14.2° horizontally from the fixation cross. Subjects were required to make a saccade to this target within 700 ms following target appearance. If the latency was less than 100 ms or longer than 700 ms the trial was canceled and the fixation cross immediately reappeared in place of the target. In a trial, the target was displayed for a total duration of 1200 ms. We usually recorded two sessions per day; participants had about 10 min break between sessions, during which they were free to move.

Stabilized Baseline

For four other sessions of 200 trials, as well as for the subsequent experimental conditions, we stabilized the target image on the fovea at a distance depending on the individual baseline saccadic gain. That is, when the saccade was detected the target was extinguished and it reappeared at a new location relative to the eye position at the end of the movement. This location was obtained by multiplying the landing point of the current saccade (e.g. 12° to the right of the fixation point) by the reciprocal of the baseline gain (e.g., 0.9) obtained over the regular baseline. Therefore, for a participant in whom the baseline saccadic gain was less than 1 the target appeared slightly further from the postsaccadic eye position. In our example the target would be displayed (1/0.9)*12 = 13.33 deg to the right of the fixation point, as illustrated in Figure 1A. This manipulation was designed to avoid the undesired change of gain that might be induced when the target image is stabilized on the fovea without this correction, because of the tendency of the saccadic system to maintain its gain (Havermann & Lappe, 2010; Henson, 1979; Robinson et al., 2003). A new trial began with the reappearance of the fixation cross. The median gain measured during these 800 stabilized trials was then used as the goal gain in the subsequent experimental conditions.

Learning

Increases in saccadic amplitude variability were induced by reinforcing least frequent amplitudes: Saccades of rare amplitudes were reinforced but saccades of frequent amplitudes were not. Reinforcement criteria were computed using 10 gain bins centered on the individual goal gain for each participant. Because of the common tendency of having a saccadic gain slightly less than 1 we used unequal bins: The size of the five lower bins were 0.06 units of gain whereas the size of the five upper bins were 0.03 units of gain.

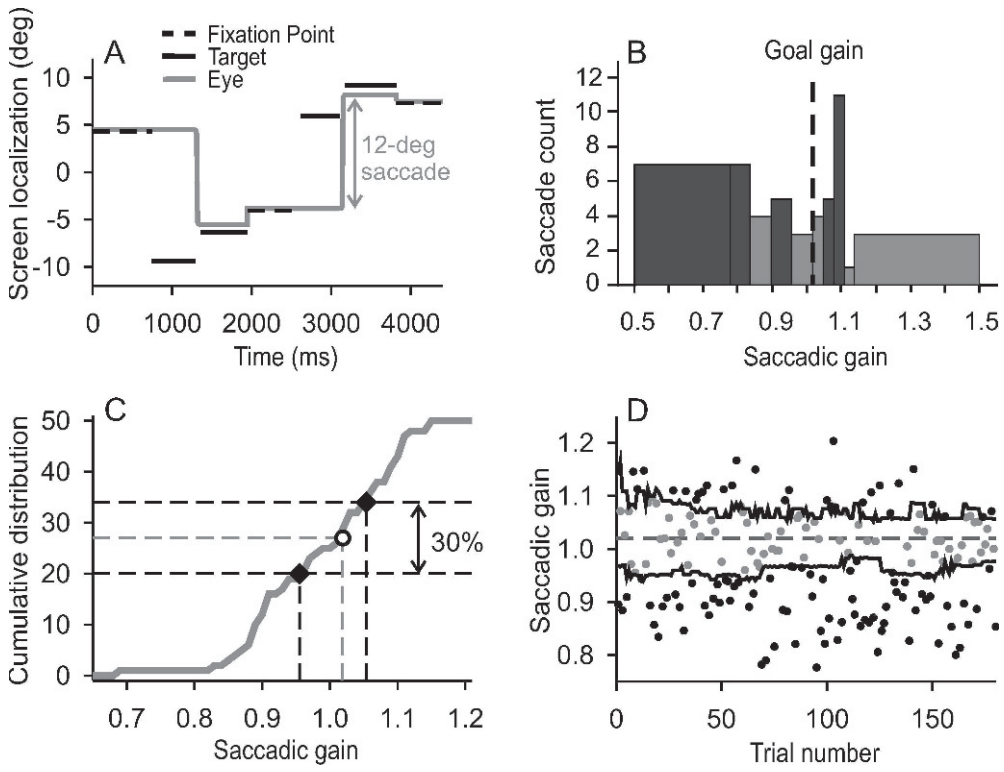

Fig. 1. A: Representative examples of fixation point, target, and eye positions for two trials during stabilized baseline (participant S3, baseline saccadic median gain 0.92). B: Gain bins and reinforcement criteria for a single learning trial (participant S2). A tone was emitted if the current gain fell into one of the five least frequent bins (light grey). C: Computation of reinforcement criteria for a single recovery trial (participant S2). Solid grey line, cumulative gain distribution for the previous 50 trials. White circle, goal gain. Black diamonds, saccadic gain values ± 15% around the goal gain; vertical black dashed lines, reinforcement criteria. D: Example of a recovery session (participant S2): changes in reinforcement criteria (solid black lines), reinforced saccades (grey points) and nonreinforced saccades (black points). Gray dashed line represents goal median gain.

Figure 1B shows an example for a single learning trial of participant S2 whose goal gain was 1.019.

The lowest and highest bins values were set to 0.5 and 1.5. For instance, for participant S2 the bin intervals were [0.5; 0.779], [0.779; 0.839] and so on, the last ones being [1.079; 1.109] and [1.109; 1.5]. For each trial we computed the amplitude frequencies for each bin over the previous 50 saccades. Each new value then replaced the oldest one so that the frequencies were always computed with respect to the 50 most recent trials. For the first trial of a session the frequencies were obtained over 50 trials randomly chosen from the previous session. If the current gain fell into one of the five least frequent bins a tone (a 60 ms 1000 Hz sine wave) was emitted.

Furthermore, whenever three out of five consecutive saccades (including the fifth one) were followed by the tone, another auditory reinforcer was delivered (a synthetic voice saying ''bravo'') indicating that the subject won one point. The number of learning sessions (200 trials) depended on the time needed to double the standard deviations of the amplitude distributions with respect to the stabilized baseline standard deviation (S1, S2 and S3 performed 40, 18 and 32 learning sessions, respectively).

Recovery

During this fourth experimental condition the reinforcement contingencies aimed at decreasing the level of amplitude variability. The differential reinforcement criteria were

systematically changing from trial to trial but they were now computed based on a targeted percentile reinforcement procedure (Galbicka, 1994; Galbicka, Kautz, & Jagers, 1993), similar to the ones we used to shape the distributions of saccadic latencies (Madelain et al., 2007) and to alter saccadic gain (Madelain, Paeye, & Wallman, 2008).

This procedure involved two simultaneous criteria which control the range of reinforced amplitudes below and above the goal gain. Specifically, the cumulative distribution of the 50 most recent saccadic amplitudes was used to compute the range of reinforced saccades: The lower and upper reinforcement criteria corresponded to the gains of the seventh saccades (15% of the previous 50) which were closest (below and above) to the goal gain (the median gain obtained in the stabilized baseline trials). By reinforcing only a fraction of saccades (± 15% in our experiments) closest to the goal gain we expected the amplitude distribution to become sharper and sharper and peak near the goal gain. Figure 1C illustrates how the reinforcement criteria were computed for a single trial: The goal gain corresponded to the stabilized baseline gain (1.019); based on the cumulative gain distribution computed over the last 50 trials, the lower criterion was the gain of the seventh saccade smaller than the goal gain (0.955) and the upper criterion corresponded to the gain of the seventh saccade larger than the goal gain (1.054). On the subsequent trial the saccade would be reinforced if its gain fell between 0.955 and 1.054. Each new value replaced the oldest one. For the first 50 trials of a session the cumulative gain distribution was computed based on 50 saccades randomly extracted from the previous session. Figure 1D illustrates the changes in the reinforcement criteria and in the reinforced gains on a trial-to-trial basis during a recovery session. With this procedure every trial might be reinforced if the gain fell within the reinforcement criteria but the overall probability of reinforcement remained approximately constant across trials. As in the learning sessions, participants were encouraged to maximize their local rate of reinforcement by giving one point signaled by an auditory stimulus as soon as three out of five consecutive trials were reinforced. This aimed at constraining the amplitude criteria around the goal gain, thereby having the variability decrease. The number of recovery sessions (200 trials) necessary to reduce the standard deviations was 18, 22 and 6 for S1, S2 and S3 respectively.

EXPERIMENT 2: IS THE CONTINGENCY BETWEEN EYE MOVEMENTS AND CONSEQUENCES NECESSARY TO CONTROL VARIABILITY? YOKED CONTROL

Subjects

We had 8 naïve participants perform this experiment to ensure that auditory reinforcement contingent on saccades, and not the awareness of the rules or the image stabilization trick, drove the changes in amplitude variability.

The 8 participants had normal or corrected to normal vision. They had no previous experience in oculomotor experiments. To familiarize them with the eye-movement recording apparatus and calibration procedure we first had them make 50 saccades using the same stimuli as in the first regular baseline (we do not report results from these trials). All experimental procedures were reviewed and approved by the Institutional Review Board and each participant gave informed consent.

Procedure

Participants were first instructed to make a saccade to the appearance of the target after fixating the cross. After the two baseline conditions they were told that the tones were emitted depending on the variability of their saccadic amplitude. They were instructed to earn as many points as possible as a game and the winner was rewarded with a prize. Nothing was explained about our expectancy to increase or decrease the variability nor about the target image stabilization.

The design was identical to the one used in Experiment 1 except for a few changes. First, no recovery condition was carried out. Second, during the stabilized baseline trials, saccades were differentially reinforced according to the targeted percentile procedure similar to the one described in Experiment 1, aiming at concentrating saccadic amplitudes around the baseline amplitude. In addition, the fixation cross reappeared pseudorandomly near the target, between $\pm 2.4^\circ$ and $\pm 13^\circ$ from the center of the screen. The median gain

measured during these stabilized trials was then used as the goal gain in the subsequent experimental condition.

Participants were then assigned to two groups of 4. In the experimental group the variability learning procedure lasted until the standard deviations were multiplied by 1.5 with respect to the stabilized baseline standard deviations. The number of learning sessions (200 trials) was 17, 18, 10 and 22 for participants S4 to S7, respectively.

For the yoked control group (S8 to S11) the auditory stimuli were independent of the saccadic amplitude: The tones were programmed in advance and matched the auditory consequences obtained by one of the participants. For example, for S4 the second and fourth trials of the first learning session were reinforced. For the corresponding yoked control participant S8 the second and fourth saccades of the first learning session were followed by the tone regardless of the saccadic amplitudes. Each participant's sequences of reinforcement as well as target presentations were therefore replicated in one of the participants of the yoked control group. The computation of the postsaccadic target position remained linked to the individual baseline saccadic gain. As in the experimental group, trials were aborted when the latency was below 100 ms or above 700 ms. If the instructions, the visual stimuli or the reinforcement intermittency were responsible for increasing amplitude variability, the changes in distributions should be similar in both groups. This would not be the case if the contingency between eye movements and their consequences controlled the saccadic amplitude variability.

Apparatus

Stimuli were generated on a Power Mac G4 using the Psychophysics Toolbox extensions for Matlab (Martinez & Martinez, 2002) and displayed on a video monitor (Iiyama HM204DT, 100 Hz) at a viewing distance of 60 cm. To minimize measurement errors, the subject's head movements were restrained using a dental impression and a forehead rest, so that the eyes in primary gaze position were directed toward the center of the screen. Presentation of stimuli, and acquisition, display, and storage of responses were controlled by a PXI computer using the LabView RealTime software package (National Instruments). Trigger signals from the visual display computer to the LabView computer allowed us to synchronize data collection to stimuli presentation with 1-ms resolution. Eye movements were measured continuously with an infrared video-based eye tracking system (ISCAN, RK-726) at 240 Hz. Before each experimental session we calibrated the eye tracker by having subjects repeatedly fixate a set of seven horizontal locations to generate a linear function for converting raw eye tracker values to horizontal eye positions.

Data Acquisition and Analysis

For on-line saccade detection, a real-time algorithm used a point-to-point velocity criterion to identify the start and end of the first saccade within 700 ms after the target onset. To have the target blank at saccade onset and reappear close to the fovea after the saccade, we computed the median eye position over 12 ms following the saccade, and then restored the target at its new location with respect to the fovea as soon as the position computation was completed. On average the target was blanked for 65 ms from saccade onset. On-line measured amplitudes were used to compute the reinforcement criteria.

For off-line analysis of the eye movement recordings, an interactive analysis program was used to display and analyze the data. Horizontal eye velocities and accelerations were obtained by differentiating the eye position signal over an 8 ms window. Saccades were detected by applying a set of fixed velocity and acceleration criteria. A program written in LabView software presented the start and end of each saccade immediately following the target step to the investigator for confirmation or, if necessary, for correction. Saccadic amplitudes were computed by subtracting the median eye positions across 25 ms windows preceding and following the saccade. We measured only the first saccade within 700 ms after the target onset. We discarded trials in which the gain was below 0.5 or above 1.5 or the latency shorter than 100 ms (on average 3.27% of the saccades were discarded).

Results

The saccadic gain distributions varied according to the reinforcement contingencies.

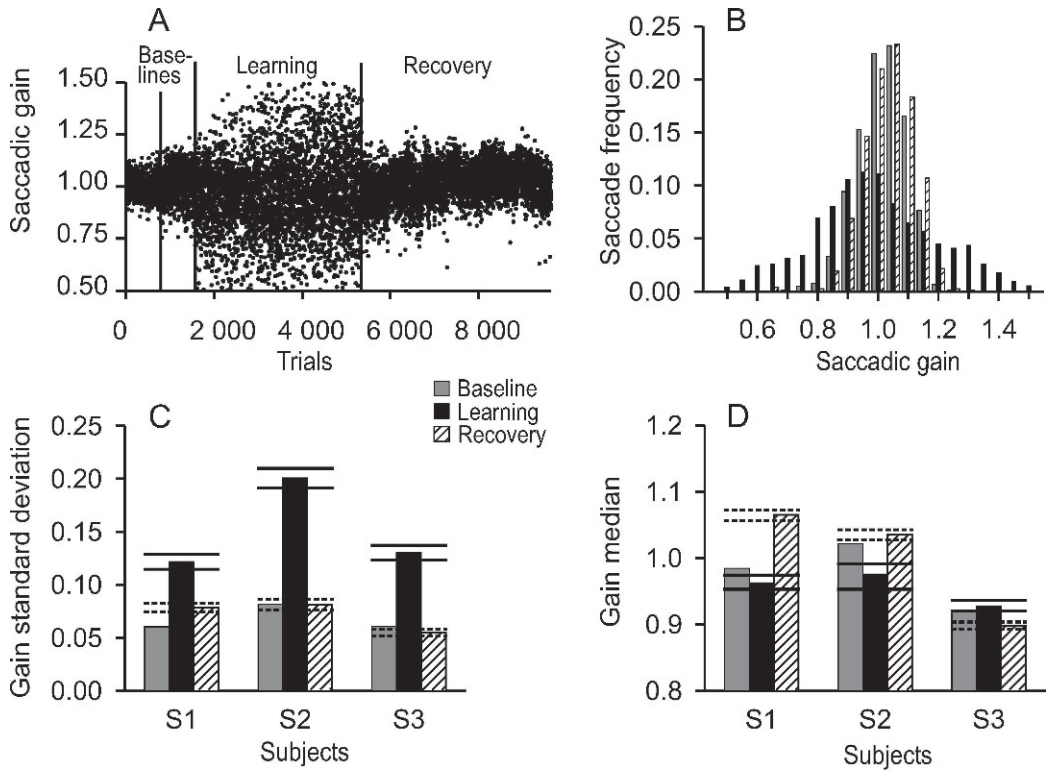

Fig. 2. Results of Experiment 1. A: Saccadic gain in each experimental condition, each data point corresponding to one trial (participant S2). B: Individual frequency distributions (participant S2) of saccadic gains for the stabilized baseline (800 saccades − gray), for the learning trials (last 800 saccades − black) and for the recovery trials (last 800 saccades − hatched). C: Saccadic gain standard deviations for the last four sessions (800 trials) of stabilized baseline (gray), learning (black) and recovery (hatched). 95th bootstrap percentile confidence intervals over the learning and recovery conditions, solid and dashed lines, respectively. D: Corresponding median gains. Bar colors and lines as in panel B.

For all experimental subjects we observed a systematic increase in the dispersion but not in the median gain. In the control group the changes in gain distributions were not consistent across subjects.

Experiment 1

Figure 2A displays the saccadic gain (saccadic amplitude / target displacement amplitude) in each experimental condition for participant S2. Figure 2B summarizes these data by plotting the frequency distributions of saccadic gains for the four last sessions of each experimental condition. The dispersion increased after learning compared to baseline. It was then dramatically reduced at the end of recovery: The frequency distributions of baseline and recovery were almost perfectly superimposed. The median gains remained mostly unchanged throughout the three experimental conditions (1.02, 0.98 and 1.04 for the stabilized baseline, the learning condition and the recovery, respectively). The standard deviation changed from 0.08 in the baseline condition to 0.2 at the end of the learning condition and returned to 0.08 at the end of recovery.

To estimate the changes in the standard deviations we computed the 95th bootstrap percentile confidence intervals (Efron, Jolivet, & Hordan, 1995) over the last four learning sessions (Figure 2C). For participant S2 the standard deviations in the baseline and the recovery were clearly outside this confidence interval revealing that the change was statistically significant. This was also true for partic-

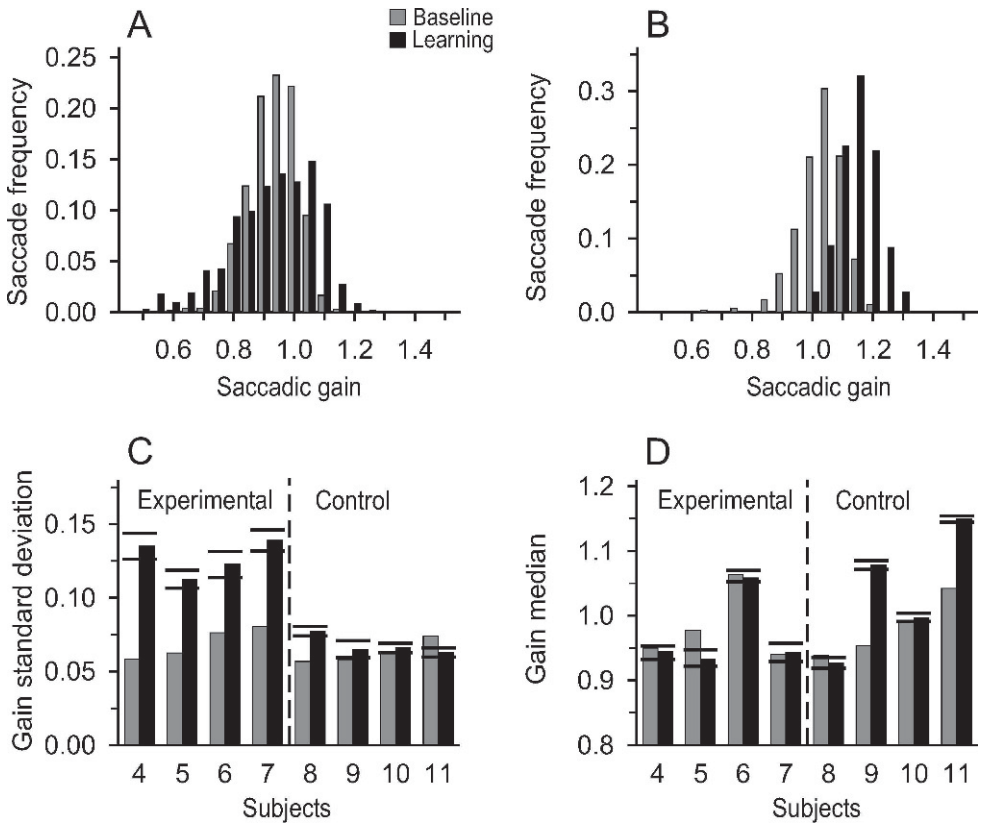

Fig. 3. Results of Experiment 2. A: Individual frequency distributions (experimental participant S7) of saccadic gains for the stabilized baseline (800 saccades − gray) and for the learning trials (last 800 saccades − black). B: Same as A for the control participant S11, yoked with participant S7. C: Saccadic gain standard deviations for the last four sessions (800 trials) of stabilized baseline (gray) and learning (black). 95% bootstrap percentile confidence intervals over the learning condition, solid lines. D: Corresponding median gains. Bar colors and lines as in panel C.

ipants S1 and S3. On average the standard deviations increased by a factor of 2.21 between the baseline and the end of learning. Interestingly, between the baseline and recovery conditions, standard deviations increased by only a factor of 1.06, indicating that the spread of the distribution almost perfectly recovered. The median gains were also altered but the changes were not consistent across participants (Figure 2D). For participants S1 and S2 the medians were significantly lower in the learning sessions whereas for participant S3 it remained unchanged during learning but was slightly reduced at the end of recovery. On average the median changed by a factor of 0.98 from the baseline condition to the learning condition and by a factor of 1.02 from baseline to recovery.

Experiment 2

For the experimental participants the changes in the saccadic gain distributions were analogous to the ones obtained in Experiment 1. As exemplified in Figure 3A for participant S7, the dispersion increased after learning (from 0.08 to 0.14) whereas the median was not altered (0.94 before and at the end of learning). This is not the case for the yoked control participant S11 (figure 3B): The gain standard deviation slightly decreased (from 0.07 to 0.06), but the median increased (from 1.04 to 1.15) after being exposed to the exact same saccades' consequences as participant S7.

The bootstrap percentile confidence intervals presented in Figure 3C reveal that for each experimental participant the gain stan-

dard deviation after learning was significantly higher than the one from baseline, increasing by a factor of 1.86 on average. The control participants also exhibited a change in gain standard deviation but these changes were not consistent: It increased in S8, S9 and S10 but decreased in S11. Moreover these changes were much smaller than those observed in the experimental group: On average standard deviations increased by a factor of 1.1 from the baseline to the end of learning.

Figure 3D plots the median gains as well as the confidence intervals computed over the four last sessions of learning. For 3 out of 4 experimental participants there were no statistical differences while there was a decrease in 1 participant (S5, from 0.98 to 0.93). For the control group the median gain significantly increased in participants S9, S10 and S11 and decreased in participant S8. It should be noted that these changes were large in 2 participants (more than 10% for S9 and S11).

U Values

In addition to the standard deviation, we examined another measure of dispersion, the ''uncertainty'' or U value. This statistic is commonly used to evaluate behavioral variability (e.g. Page & Neuringer, 1985) and does not require assumptions upon the distributions. We computed U values using the following formula:

$$U = \frac{-\sum_1^N (p_n \log_2 (p_n))}{\log_2 (N)}$$

where p represents the relative frequency of a bin n and N the number of bins. Here we used 10 equal gain bins of 0.1, from 0.5 to 1.5. U value reflects the likelihood that the gain of a trial falls in each bin. If the 10 bins contain equal number of trials, then U equals 1. Conversely, if all saccadic amplitudes fall within one single gain bin, U equals 0. We computed the U values over the last four sessions of each experimental condition. We also computed 95th bootstrap percentile confidence intervals for the U values obtained at the end of learning.

Figure 4A (squares and circles) plots the U values at the end of learning as a function of the U values obtained during baseline in both experiments. All baseline U values were close to each other—ranging from 0.406 to 0.551. The data scattered into two groups: On the one hand, data from the experimental participants (squares and filled circles) are all clearly above equality line, indicating a large increase in the spread of the distribution after learning. On average the U values were multiplied by a factor of 1.61 (Experiment 1) and 1.49 (Experiment 2, experimental group). On the other hand, data from the yoked control group (unfilled circles) are close to or even below the equality line, indicating small changes with respect to baseline: U values increased by a factor of 1.08 on average after noncontingent reinforcement, but remained unchanged for participant S10 and significantly decreased for S11. Moreover, data from the recovery sessions (down triangles) also plot along the unity line—the U values changed only by a factor of 1.05. In 2 out of the 3 subjects the U values were lower in the recovery than in the baseline (as confirmed by the 95th bootstrap percentile confidence intervals).

DISCUSSION

This study demonstrates that part of saccadic amplitude variability can be manipulated through learned contingencies. The first experiment shows that reinforcement can induce large changes in saccadic amplitude variability while maintaining the median gains globally unaltered: Compared to stabilized baseline, the standard deviations after learning were multiplied by an average factor of 2.21. Furthermore, at the end of the recovery sessions variability decreased to near baseline level. Therefore, these effects may not be attributed to the target image stabilization.

Instructions could have played a role in increasing variability, as observed elsewhere for interresponse times (Joyce & Chase, 1990), topography (Eisenberger, Armeli, & Pretz, 1998) or response sequences (Hopkinson & Neuringer, 2003; Neuringer, 1986). However, in our second experiment the yoked control participants' data show that instructions alone could not account for the increase in variability because when reinforcement was not contingent on saccadic amplitude variability we did not observe a systematic increase in the distributions spread. Our results extend the findings of Madelain et al. (2007) who

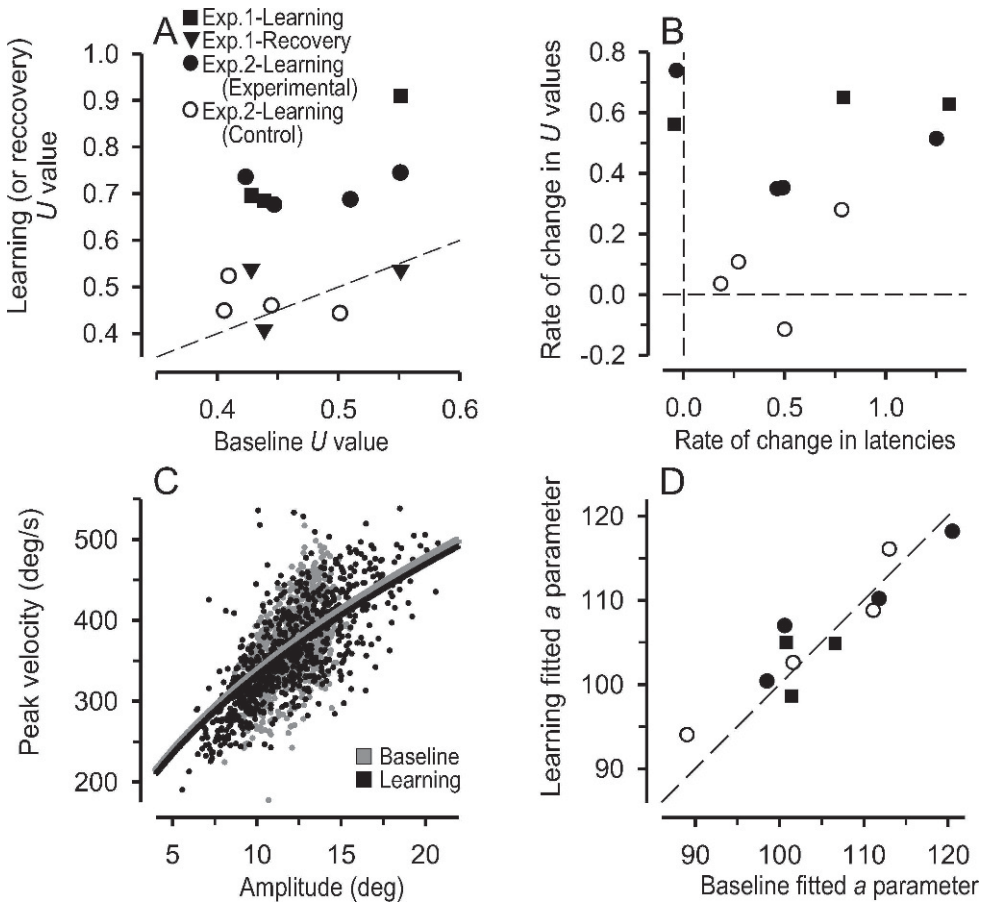

Fig. 4. A: *U* values during baseline with respect to *U* values during learning (circles and squares) or recovery (down triangles). Circles, data from Experiment 2. Filled circles, experimental participants. Unfilled circles, control participants. B: Rates of change (end of learning with respect to end of stabilized baseline) in *U* values as a function of rates of change in latencies. Symbols as in panel A. C: Example of the relationships between saccadic amplitude and peak velocity (participant S2). Gray dots, baseline regular saccades. Black dots, stabilized saccades of the last four learning sessions. Solid lines indicate the corresponding fits. D: Main sequence fitted *a* parameters at the end of learning as a function of the *a* parameters during regular baseline. Symbols as in panel A.

increased and then reduced dispersions of saccadic and manual latencies distributions: In a discrimination task, correct responses (saccades or button presses) were reinforced depending on latency variability criteria. Together with our present study, these experiments suggest that variability levels of various saccadic dimensions can be independently controlled by reinforcement contingencies. An important feature of these results is that they do not support the classical view stating that sensorimotor variability originates exclusively from some uncontrollable stochastic internal noise (Faisal et al., 2008; Van Beers, 2007, 2008).

One could argue that the increased oculomotor variability might be achieved through specific strategies—that is, (conscious or not) cognitive processes involved in visual processing, target selection and motor programming depending on stimulus properties and stored information (Glimcher, 2003)—that would affect various response parameters. Saccadic latencies are often viewed as reflecting such decision-making strategies (Reddi & Carpenter, 2000). To probe possible effects of our procedure on saccadic reaction time, we computed the rates of change in latencies ([median obtained over the last learning session - median obtained over the last

stabilized baseline session] / median obtained over the last stabilized baseline session). Figure 4B plots these rates of change against those of U values, revealing that latencies increased after learning (from 179 to 277 ms on average over the last sessions, across all participants; Wilcoxon signed-rank test, $p = .005$). Nevertheless, for the experimental participants the correlation coefficient between these measures was -0.057, revealing that the changes in amplitude variability are not correlated to the changes in latencies ($p = .903$). Moreover, it can be seen that the experimental and control groups differed in the rates of change in their U values but not in those of their latencies (Figure 4B). Even though the possible use of strategies cannot be entirely ruled out, we did not find any consistent changes in latencies.

Saccadic duration, average velocity, and peak velocity all increase as the size of the saccade increases, a relationship known as the ''main sequence'' (Bahill, Clark & Stark, 1975). Given the results obtained in studies in which saccades were reinforced (Takikawa et al., 2002; Xu-Wilson et al., 2009), one could also expect a change in peak velocities (the maximum eye velocities during saccades) induced by our procedure. We examined the relationships between amplitude and peak velocity: Saccades recorded during the first regular baseline were compared to saccades from the last four learning sessions. Figure 4C depicts an example of these comparisons for participant S2. The relationships were fitted using the following equation (Lebedev, Van Gelder, & Tsui, 1996):

$$a = \frac{\text{Peak Velocity}}{\sqrt{\text{Saccade Amplitude}}}$$

The a parameters before and after learning were similar (106.6 and 104.9, respectively). This lack of change in the a value was true for all participants, as shown Figure 4D (104.9 and 105.9 on average over the first baseline and the last four learning sessions, respectively; Wilcoxon signed-rank test, $p = .401$), indicating that our reinforcement procedures did not alter saccade dynamics even though variability changed.

Optimal control theory of motor control predicts that specific cost functions are minimized by the central nervous system to generate movements in the most efficient way (Bays & Wolpert, 2007; Todorov, 2004; Wolpert & Ghahramani, 2000). These theoretical propositions have been applied to the saccadic system (Harris, 1995; Harris & Wolpert, 1998, 2006; Van Beers, 2008). Harris (1995) focused particularly on two parameters to account for saccadic control. The first cost is related to vision impairments during saccadic movement: Saccades landing beyond targets must be avoided because they increase the total eye displacement and therefore saccade durations, in which case vision is lost for a period of time longer than necessary. The other cost is due to saccades' inaccuracy: Gain must be as close to unity as possible to place the retinal image of the target on the high-acuity area of the retina. Harris' saccadic flight time minimization model predicts that the visual system would reduce the overall saccadic gain given the presence of endpoint variability in order to minimize the proportion of saccades landing beyond the target. There would be a mechanical relation between gain and variability such that when variability is high, the gain must be lower than when the spread of saccadic error is low. In our experiments we found that median gains significantly decreased in 3 experimental participants (S1, S2 and S5), tended to decrease in 2 (S4 and S6) and to increase in 2 others (S3 and S7). By contrast, variability significantly increased in all 7 experimental participants. In the control experiment we observed a decrease in gain in 1 participant (S8) and an increase in the other 3 while variability decreased in 1 participant (S11) and increased in the other 3. Harris's prediction was observed in 5 participants (S1, S2, S5, S8 and S11) but not in the other 6. In 2 control participants (S9 and S10) the opposite was true: The median and standard deviation both increased. It is, however, noteworthy that when large changes in variability are induced such as in the experimental participants, the median gains did not decrease in 4 out of the 7 cases. In these participants we did not find a correlation between the rates of change in median gains and in U values (obtained for each participant over the last four learning sessions with respect to the stabilized baseline) ($r = -0.322$, $p = 0.481$, NS). This lack of consistent relationship between changes in

gain and variability confirms that we were able to manipulate each parameter independently, a result that challenges Harris' optimization model.

Variability in saccadic amplitude is thought to strongly affect saccadic control in infants. Harris (1995) suggests that during the first months of life cortical structures responsible for the mapping between target eccentricity and saccadic motor output would not be functional. In fact, infants' saccadic gain has been reported to be extremely low (Aslin & Salapatek, 1975). As a consequence, only the cost related to the saccadic duration would be relevant and priority would first be given to minimize saccadic duration. Only after sufficient maturation of these structures could the saccadic system establish the correspondence between visual signals and motor output. The costs associated with saccadic accuracy would then play a role in the saccadic control which would explain the decreased variability and increased gain observed during development. In other words, the change in gain associated with a decreased variability would be driven by cortical maturation. However, our results, which provide further evidence that eye movements have attributes of operant behavior, suggest that the increase in gain and the reduction in variability would be controlled by a single reinforcement process. Saccadic control might be in part learned through the progressive modification of the visual consequences that exert selective pressure on eye movements. Because infants' retinas are far from mature (Yuodelis & Hendrickson, 1986) and visual interactions are limited, the oculomotor system would first tolerate large endpoint errors. During the first months of life most eye movements would therefore not be differentially selected. Progressively, clearer and clearer vision might extinguish the saccades that land away from visual objects and select the saccades that lead to a better visual perception. We propose that both the spread and median of saccadic amplitude distributions develop by means of selection processes: Eye movements allowing sufficiently efficient vision to favor visually driven interactions with the environment would be selected, while less efficient movements would be extinguished.

The operant behavior selection theory postulates that variability is necessary for adaptation (Donahoe, Burgos, & Palmer, 1993; Skinner, 1981). This hypothesis has been probed in experiments on response sequences showing that only after reinforcement contingent on variability can animals emit difficult-to-learn target sequences, unlike subjects in whom variability had not been reinforced (Grunow & Neuringer, 2002; Neuringer, 1993; Neuringer, Deiss, & Olson, 2000; but see Maes & Van der Goot, 2006, and Neuringer, 2009, for a discussion). One may postulate that some variation in saccadic amplitude is necessary to adapt to changes affecting the oculomotor system. For instance, we experimentally reinforced modifications in saccadic gain (Madelain et al., 2008) and suggested that, in real life, saccades that lead to a clear vision of the target are selected by their consequences. This adaptation would require some variability as a basis for differential reinforcement. That environmental contingencies control amplitude variability might thus be critical to maintain saccadic accuracy during the lifespan.

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