Saccadic latency in deterministic environments: Getting back on track after the unexpected happens

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Saccadic latencies are commonly used to study decision mechanisms. For instance, in a random sequence, saccadic latency to a target depends on how frequently it has recently appeared. However, frequency is not the only factor that determines probability. Here we presented targets to the left or right, either in random sequences or in repeating patterns. Although the frequency of appearing on a given side was identical in each case, latencies for the low-frequency side were significantly shorter for repeating patterns than in random sequences, showing that the system can respond to the deterministic probabilities in such patterns. We then disrupted our patterns episodically, recommencing at a random starting position in the sequence. This significantly increased the latency, which remained high until the low-frequency target in the sequence reappeared, implying that the oculomotor system makes strategic use of low-frequency—but high-information—events to determine the phase of repeating sequences. The deterministic sequences of events in our patterns represent a simple model for the habitual sequences of actions commonly performed in daily life, which, when disrupted, require the engagement of a higher level problem-solving strategy to return us to our previous automated sequence as quickly as possible.

Introduction

Choosing where to look next is the commonest decision we make, and recent behavioral and neurophysiological studies of saccades have proved extraordinarily fruitful in understanding where and how decision-making processes occur in the brain (Carpenter, 2004; Gold & Shadlen, 2007; Reddi & Carpenter, 2000; Sato, Murthy, Thompson, & Schall, 2001; Thompson, Hanes, Bichot, & Schall, 1996). Expectation—the brain's calculation of the prior probability that a target will appear in a particular location—has a significant influence on decision making. In particular, there is a linear relationship between the logarithm of appearance probability for a given location and saccadic latency in experiments using stochastic runs (Carpenter & Williams, 1995), with more probable stimuli producing shorter latencies. Thus, in experiments in which targets are presented randomly to left and right with different frequencies (Carpenter & Williams, 1995), more frequent stimuli produce shorter latencies. More specifically, latency is a linear function of logarithm of the prior probability derived from the frequency.

This inverse relationship between expectation and latency (Carpenter & Williams, 1995) can be readily interpreted using a "race to threshold" model for saccade initiation such as the LATER model (Carpenter, 1981; Reddi, 2001), whose behavior is similar to that of certain neurons in the frontal eye fields of monkeys that demonstrate rise-to-threshold behavior predictive of saccadic latency (Hanes & Schall, 1996; Sato et al., 2001; Thompson et al., 1996). In LATER, the starting level for this race increases with increasing expectation, decreasing the time taken to reach threshold and so shortening saccadic latencies. When the cumulative probability of a response on a probit scale is plotted versus reciprocal time (a reciprobit plot), LATER predicts that distributions will be linear (Carpenter, 1981) and that changes in expectation will result in a "swivel" of the distribution about a fixed point corresponding to infinite time (Carpenter & Williams, 1995). This characteristic signature of expectation can be distinguished from other mechanisms that can alter saccadic latencies, such as changes in the rate of the rise to threshold, which cause a lateral "shift" of data sets on a reciprobit plot, with no change in slope (Sinha, Brown, & Carpenter, 2006). How does the system calculate the expectation signal that gives rise to the characteristic swivel in reciprobit plots? Observed frequency is not the only determinant of probability. In particular, events that occur in patterned sequences may have higher probabilities than their simple frequencies would suggest. For example, in a simple patterned task where targets alternate between left and right, although the overall frequency of appearing on the left is 0.5, on a particular trial the probability may be 1

(i.e., when immediately preceded by a target on the right). Do probabilities of this kind also influence latency? If so, do they—like "ordinary" probabilities with randomly appearing targets—do so by altering the starting level of the LATER model decision signal (Carpenter & Williams, 1995)? In our current study, we compared patterned target sequences and random ones, to see whether the saccadic system's calculation of expectation is influenced by deterministic information.

The importance of understanding how such patterns influence decision-making processes is clear: most of the day-to-day tasks we do involve a habitual sequence of actions (Hikosaka & Masaki, 2008), which may, in the limiting case, be seen as a deterministic sequence. It has been proposed that such habitual sequences are controlled by relatively automatic parts of the motor system (Hikosaka et al., 1999), presumably allowing cognitive processes to be freed to be directed toward other, non-habitual tasks. Given these specialized areas, it may be that expectation based on deterministic events operates via a mechanism different from that underlying expectation based on frequency information (Carpenter & Williams, 1995). In addition, habitual sequences can be episodically disrupted when an unexpected event breaks our routine. In the hope of understanding the dynamics of the mechanisms encoding habitual sequences and those underlying the effects of disruptions of this kind, we also looked at the effect of episodically disrupting our patterned sequences.

Methods

Stimuli

We presented targets on a calibrated computer monitor system (ViSaGe graphics card: Cambridge Research Systems, Kent, UK, and GDM-F520 monitor, Sony, Tokyo, Japan, or Diamond Pro 2070SB monitor, Mitsubishi, Tokyo, Japan; frame rate 100 Hz) in a dimly illuminated room. For Experiments 1–3, targets and fixation markers were 0.25°, 48 cd/m^2 spots, presented on a 24 cd/m^2 gray background (CIE 1931, $x = 0.281$, $y = 0.306$). Subjects sat 1 m from the monitor, which subtended 23 × 17 deg, and viewed the monitor with their habitual spectacle correction. A chin rest stabilized head position.

Eye movement recording

We recorded eye position with an infrared reflection oculometer (Ober Consulting, Poznan, Poland; Ober et al., 2003) whose characteristics have been described previously (Anderson, Yadav, & Carpenter, 2008). Saccades were automatically detected by the computer (Carpenter, 1994) based on a velocity threshold optimized for each observer. We recorded latencies in 10-ms bins, synchronized by the ViSaGe system exactly to the frame rate of the display, and excluded those less than 80 ms or greater than 790 ms. After an experimental run, all records were checked manually and those containing errors such as blinks, head movements, or other artifacts were excluded. In Experiment 2 (in which all subjects took part), the median percentage of excluded saccades was 1.1% and was not higher than 3.2% for any subject. No subject made more than one error (a non-excluded saccade in a direction opposite to the target) in this experiment.

Experimental protocols

Experiments 1–3 employed a step task (Carpenter & Williams, 1995) in which targets jumped 5° to either the left or the right from a central position. Upon detecting a saccade, fixation returned to the center of the screen and the next target appeared after a randomly selected delay uniformly distributed between 770 ms and 1770 ms. Data were collected in runs of 120 (Experiments 1 and 2) or 110 (Experiment 3) saccades, with only the final 100 saccades in each run being included in our analyses. In order to minimize the level of implicit learning in each task, subjects were explicitly informed as to the structure of each experimental run, including the probabilities involved, and so knew the expected pattern of target appearance.

Experiment 1

In this experiment, we measured saccadic latencies under two conditions: *stochastic* runs and *deterministic* runs (Figure 1). In stochastic runs, left and right targets had a particular frequency (probability of left either 0.25 or 0.75), but each trial was stochastically independent (in control runs, the frequencies were 0.5 and 0.5). In deterministic runs, we used a fixed sequence of targets (either LRRR or LLLR) that continuously repeated. Thus, in both run types, the long-term frequencies to each side were the same (0.25 and 0.75). Therefore, if the oculomotor system forms expectations of what will happen based only on long-term frequencies, latency to the low-probability target should be identical in both the stochastic and deterministic runs. Data were collected in an interleaved and counterbalanced fashion, with a total of 12 stochastic runs (8 runs at $p = 0.5$, plus two at each of $p = 0.25$ and $p = 0.75$) and 4 deterministic runs (2 each of LRRR and RLLL) per subject.

Experiment 2

In this experiment, subjects viewed deterministic sequences similar to those described in Experiment 1, except that occasionally ($p = 0.03$) we presented a target randomly to the left or the right, after which the sequence
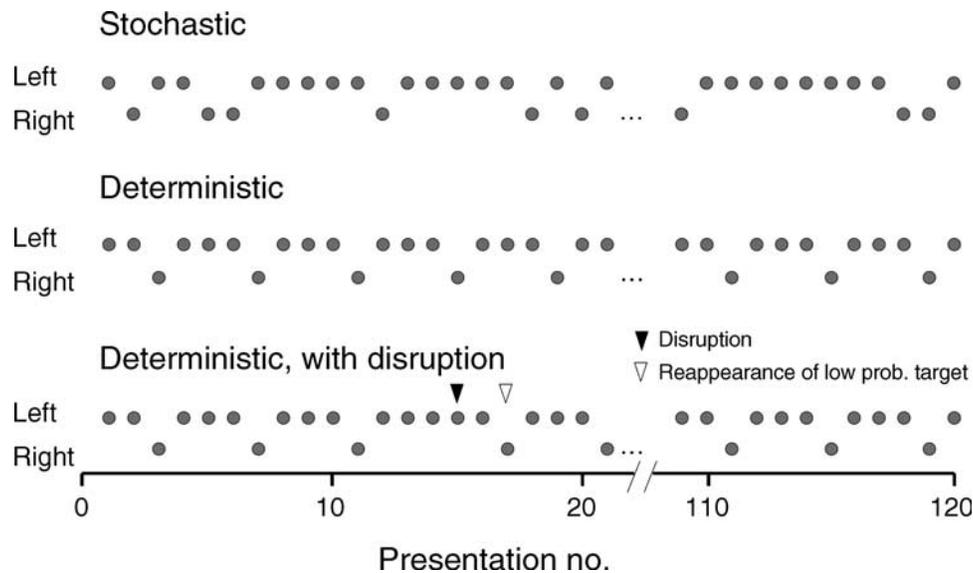
Figure 1. Schematic representation of stochastic and deterministic target presentation (Experiment 1), as well as deterministic presentation with episodic disruption (Experiment 2). Note that the long-term frequency ($p = 0.25$) of a target appearing on the right is equal in the stochastic and deterministic runs. All targets were presented as part of a *step task,* wherein a central fixation marker disappeared when the peripheral target appeared: after a saccade was detected, the target disappeared, the fixation marker reappeared, and the subject returned their gaze to the fixation marker. It should be noted that the long-term frequency ($p = 0.25$) of a target appearing on the right is equal in the stochastic and deterministic runs represented above. In the actual experiments, the low-frequency side was to the left as often as it was to the right.

recommenced at a randomly selected starting point (Figure 1, lower set). This served to create transient disruptions in the sequence. If the effects of deterministic presentation are because the oculomotor system learns the entire pattern, disruptions to the sequence should have a protracted effect on saccadic latency. In particular, the effect of the disruption should last at least until the reappearance of the low probability target, as it is this comparatively rare target that provides the most information about the starting point—or phase—of the recommenced sequence.

On every trial, there was a 1/31 probability that the pattern would be replaced with a target presented randomly to either the left or the right. After such a replacement, normally the sequence was reentered albeit at a random starting position, although there was a small probability ($1/31 = 0.03$) that a further randomly selected replacement target was presented. Because of their random nature, replacement targets did not necessarily cause a disruption to the sequence as in 50% of cases they were of the type required next by the sequence. Equally, returning to the sequence after a replacement target may not have disrupted the sequence either, although the probability of this was lower ($0.5 * 0.25 = 0.125$). We ignored such cases in our analysis and defined a disruption as the appearance of an unexpected target, irrespective of its cause. Although all subjects were told in advance the probabilistic structure of the task, as this experiment was particularly demanding the first two runs were excluded to minimize any residual effects of implicit

learning: to maintain counterbalancing, the two final runs were also excluded. Twenty runs per subject were included in the analysis (10 each of LLLR and LRRR), representing 2000 trials.

Experiment 3

This experiment was used to check that any prolonged effect of disruption seen in Experiment 2 related specifically to learning an entire sequence and was not simply an artifact of the disruption itself. Targets appeared as an alternating sequence (e.g., LRLR), which sporadically ($p = 0.03$) reversed direction, producing a disruption in the expected pattern similar to that in Experiment 2 above. However, here the target to appear after the disruption is highly predictable. A mechanism sensitive to patterns should therefore only show a change in latency at the moment of the unexpected disruption event, in contrast to the prolonged effect hypothesized in Experiment 2. Each subject performed 14 runs.

Experiment 4

When a disruption occurs in Experiment 2, the oculomotor system must withhold the response expected from the previous pattern and instead make a different response. Does the need to withhold one type of response in a task (e.g., a leftward saccade) influence the latency of other, non-withheld responses (e.g., rightward saccades)? Experiment 4 was designed to investigate this question. In

this experiment, targets (0.25° black spots, appearing 3° to either the left or right of a constantly present central 0.25° black fixation spot, background 40 cd/m^2) appeared randomly but with a fixed probability (p(R) = 0.1, 0.5, or 0.9) within a run of 100 presentations. Subjects always made saccades toward targets appearing on the right but, for some runs, were instructed to withhold saccades toward targets appearing on the left. Subjects performed two sequential training runs (200 trials) at each probability and task instruction (withhold/do not withhold) before collecting two further runs for analysis.

Analysis

For Experiments 2 and 3, we calculated average latencies for each subject at the moment of the sequence disruption and up to five stimulus presentations after the disruption (averages were calculated as harmonic means—the reciprocal of the arithmetic mean of the reciprocals; Anderson & Carpenter, 2006). We compared these latencies to those during uninterrupted portions of the sequence. Only those portions of the sequence six or more presentations after a disruption were deemed to be in the uninterrupted portion of the sequence, and only those disruptions that were at least seven trials away from a previous disruption were analyzed. As noted above, the moment of disruption may or may not correspond with the trial in which a randomly selected target was presented.

To allow evidence to be summed across observers, we used a log-likelihood fitting and analysis procedure to compare a swivel fit to one where data sets had a common slope on a reciprobit plot (Sinha et al., 2006), this latter model corresponding to a change in the rate of rise in the LATER model.

Subjects

We tested seven subjects, with a subset of five, seven, and six of these performing Experiments 1, 2, and 3, respectively. All subjects were required to have fewer than 10% direction errors during a preliminary training period in order to be included. Both authors were subjects in each experiment and were the only subjects for Experiment 4. All procedures were in accordance with the 1964 Declaration of Helsinki and were approved by our respective institutional ethics committees. Subjects gave their informed consent prior to inclusion in the study.

Results

Figure 2 shows the average latencies from the five observers who performed Experiment 1, expressed relative to a control condition in which targets appeared randomly
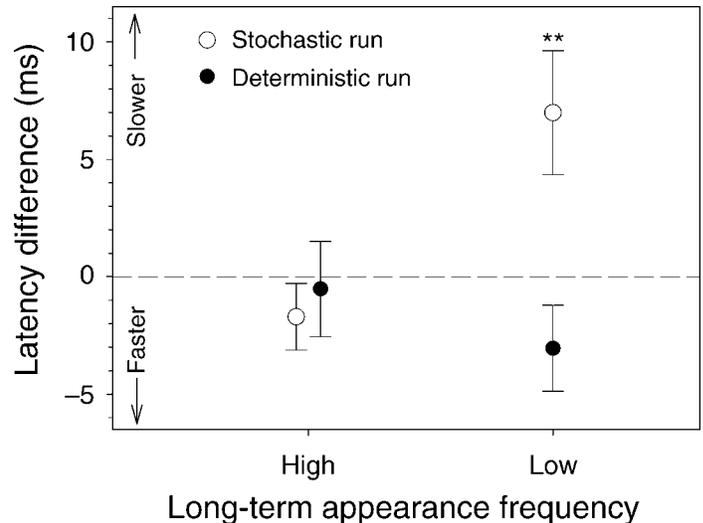

Figure 2. Average change in latency across all subjects as a function of long-term frequency for stochastic and deterministic runs (unfilled and filled circles, respectively) in Experiment 1, across all subjects. Latencies are expressed relative to a control experiment in which we presented targets stochastically with equal frequency to the left and the right (control latencies, per subject, in ascending order: 144, 168, 171, 174, and 210 ms; average = 174 ms). Latencies to the low-frequency target were significantly longer under stochastic conditions (asterisks: repeated measures ANOVA ($p < 0.01$), Tukey's Multiple Comparison Test, latency compared with all other conditions). No other conditions significantly differed from each other ($p > 0.05$). Error bars show ±1 *SE*.

to either side with equal probability. Latencies to the low probability stimulus were significantly longer in stochastic compared to deterministic runs despite both stimuli having the same long-term frequency of 0.25. Furthermore, latencies for left- and right-going saccades in our deterministic experiment did not differ significantly (Figure 2), even though one was three times more frequent than the other. In other words, the oculomotor system is influenced not only by long-term frequencies but also by the rapid changes in probability accompanying deterministic sequences, which can alter on a trial-by-trial basis. We also analyzed the distributions to confirm that their changes conformed to what would be expected from a change in probability. Our likelihood analysis failed to show any strong support for either the shift or swivel model (log$_e$ likelihood ratio favoring shift, LLR = 0.25; low appearance frequency data for stochastic versus deterministic conditions, ratios summed across all observers).

Figure 3 (upper panel) shows the influence of episodic disruptions to an otherwise deterministic pattern of targets, as investigated in Experiment 2. A disruption (abscissa = 0) produces a significant increase in average latency in comparison to latencies either before it or substantially after (abscissa ≤ -1 or $\geq +6$). Subsequent to this disruption, if we divide the trials into those in which the low-frequency
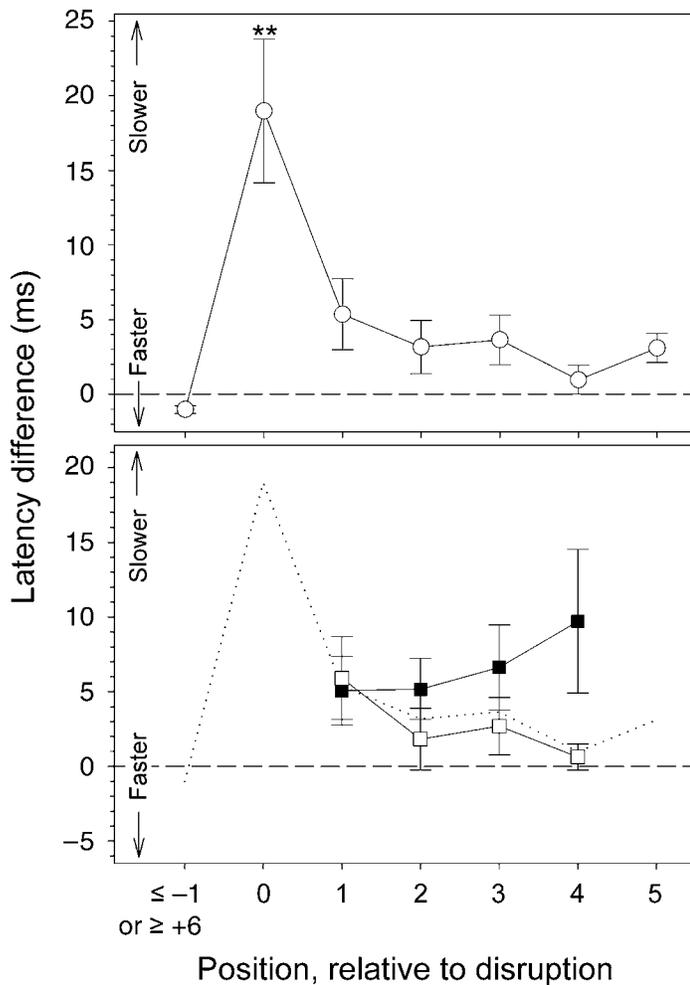
Figure 3. Average change in latency before and after a disruption (Experiment 2, all subjects: the disruption is 0 on the abscissa: symbols show mean differences, ±1 *SE*. Latency differences were relative to the mean latency for each observer, determined over the entire experiment: mean latencies, per observer, in ascending order = 149, 164, 167, 174, 174, 189, and 206 ms). Upper panel: Disrupted latency is greatly increased ($p < 0.01$, one-way repeated measures ANOVA and a Dunnett's Multiple Comparison test compared to trials away from the disruption ("≤−1 or ≥6" on abscissa)) and remains elevated for a period. Lower panel: Latency remains elevated after disruption until the low-frequency stimulus appears. Differences were calculated separately for trials where the low-frequency stimulus had (unfilled squares) or had not (filled squares) yet reappeared after the disruption. Symbols show mean differences, ±1 *SE*. The dotted line reproduces the average latency differences from the upper panel, for comparison. A two-way repeated measures ANOVA found a significant main effect for whether a latency was pre- or post-disruption [$F(1,6) = 7.67$, $p = 0.03$] but no main effect for position relative to disruption [$F(3,18) = 0.18$, $p = 0.91$]: interaction was not significant [$F(3,18) = 1.78$, $p = 0.19$]. The assumption of sphericity was satisfied (Mauchly's Test: $p > 0.05$).

target in the sequence has already reappeared versus those in which it has not, we find (Figure 3, lower panel) that latencies remain significantly longer until the low-frequency target reappears. This provides strong evidence that the subjects' strategy for determining the phase of the repeated pattern when it returns is critically dependant upon this low-frequency—but highly informative—target. It also shows that subjects faced with deterministic patterns do not base their expectation on simple first-order conditional probabilities (e.g., the probability that the next target will be on the left, given the current target on the right, or $p(L|R)$) as we would expect saccadic latencies to return to normal shortly after the disruption, irrespective of whether or not the low-frequency target had reappeared.

We compared the distribution of saccades remote from a disruption (grouped under the "≤−1 or ≥+6" category in Figure 3) with those at the moment of disruption and found that for the six subjects in which convergent fits were obtained in our likelihood analysis all favored the shift model, with the summed log-likelihood ratios strongly favoring the shift model (LLR = 22.3). We performed a similar likelihood analysis to compare distributions pre- and post- the reappearance of the low-frequency target (Figure 3, lower panel, filled and unfilled symbols, respectively) by simultaneously fitting the distribution data for each subject for the four trials after the disruption. Log-likelihood ratios were approximately evenly divided between favoring a swivel or a shift in distributions, with no subject's results strongly favoring either model (LLR ≤ 1.2 for all subjects). Overall, there was only weak support in favor of a shift (summed log-likelihood ratios = 1.6 $\log_e$ units). Data in which the low-frequency stimulus had not yet reappeared four trials after the disruption (Figure 3, abscissa = 4) were not included in the analysis as no subject had more than three latency measurements to define the distribution.

Experiment 3 confirmed that the prolonged effect after the disruption seen in Figure 3 relates specifically to learning the entire sequence and is not simply an artifact of the disruption itself. Here, targets alternated from left to right on successive trials, but occasionally ($p = 0.03$) the sequence reversed. A mechanism sensitive to patterns should only show a change in latency at the moment of the unexpected sequence reversal, as the target to appear after this disruption was predictable with a high confidence. Figure 4 shows this is indeed the case: the significant elevation in latencies is confined to the disruption event itself. For each subject, we compared the distribution of saccades remote from a disruption (grouped under the "≤−1 or ≥+6" category in Figure 4) with those at the moment of disruption and found that for the four subjects in which convergent fits were obtained three favored the shift model (one subject weakly favored a swivel, LLR = 0.62). The summed log-likelihood ratios strongly favored the shift model (LLR = 24.1).

Figure 5 shows the results of Experiment 4, in which the latency for rightward saccades was examined when
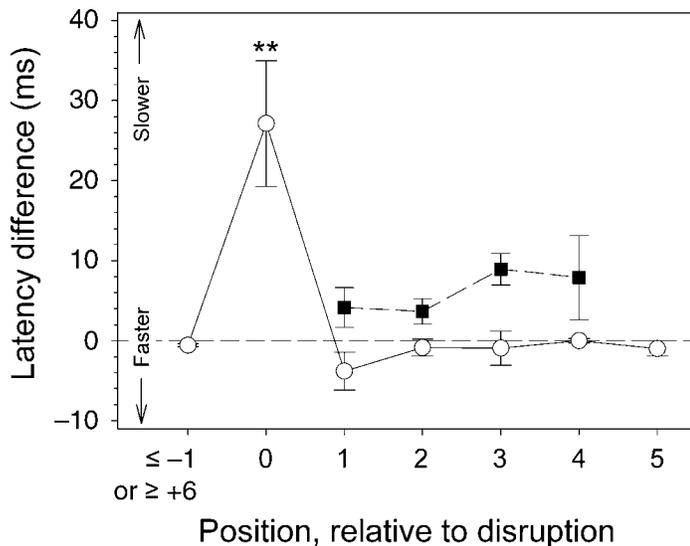
Figure 4. Transient increase in latency when a simple alternating sequence is disrupted by reversing direction (abscissa = 0), as investigated in Experiment 3. Latency differences were relative to the mean latency for each observer, determined over the entire experiment: mean latencies, per observer, in ascending order = 143, 166, 174, 175, 176, and 206 ms. Symbols show mean differences, ±1 *SE*. For comparison, data from Experiment 2 (filled squares) showing post-disruption latencies prior to the reemergence of the low-frequency target (see Figure 3, filled squares) are also shown, recalculated to exclude one subject who did not participate in Experiment 2. Asterisks show values that are significantly ($p < 0.05$) different from trials remote from the disruption ("≤−1 or ≥6" on abscissa): one-way repeated measures and a Dunnett's Multiple Comparison test.

leftward saccades in the same run were either withheld (squares) or allowed to proceed (circles). Despite identical target frequencies, rightward saccades had higher latencies when made in a run where leftward saccades were withheld, compared to a run when leftward saccades proceeded. The sum of the log-likelihood ratios weakly favored a shift (LLR = 0.71) as the cause of this latency change.

Discussion

We found that deterministic patterns (Experiment 1) produce similar changes in average latency as do high-frequency probabilities (Carpenter & Williams, 1995). While it could be that deterministic patterns and high-frequency probabilities therefore might act through a common mechanism—in particular, by elevating the starting point from which rise-to-threshold decision signal proceeds—our likelihood analysis of saccadic distributions could not satisfactorily distinguish between this option and the alternative model considered in this paper (a change in the rate of rise of the decision signal). The

elevation of latencies after a disruption to a sequence yet before the phase of the pattern could be reestablished (Figure 3, filled squares) was similarly equivocal in distinguishing between these two potential mechanisms. However, disrupting deterministic patterns caused a prominent and immediate delay both in short (Figure 5) and longer (Figure 4) sequences that provided strong evidence for a shift in the underlying latency distributions, providing strong evidence that latencies were not elevated simply because the event was unexpected.

Encoding of saccadic sequences

Given that our results suggest the oculomotor system is sensitive to deterministic probabilities (Experiment 1) and that this occurs through the learning of the saccadic sequence (Experiments 2 and 3), it is worth considering where this encoding of a sequence may occur. There is evidence that certain neurons in the supplementary motor area code for specific memory-guided sequences of movements (Tanji & Shima, 1994) and that lesions to this area, or the adjacent pre-supplementary motor area, prevent monkeys from performing this task (Shima & Tanji, 1998). Lesions to the supplementary motor areas in humans similarly impair memory-guided sequences of saccades (Gaymard, Rivaud, & Pierrot-Deseilligny, 1993). Neurons in the supplementary eye fields of primates also show pre-saccadic activity related to a particular sequence (Isoda & Tanji, 2002; Lu, Matsuzawa, & Hikosaka, 2002) as well as activity related to the position within a sequence (Isoda & Tanji, 2002). Cells in the prefrontal cortex are also able to encode general types of sequence classes (e.g., alternating) that are independent of the specific sequence (Shima, Isoda, Mushiake, & Tanji, 2007). It would therefore appear that this general area of the frontal lobe is a likely candidate for encoding deterministic sequences of the sort investigated in this paper. Direction expectation arising from pre-target cuing is similarly reflected in the activity of certain neurons within the SEF from smooth pursuit eye movements (de Hemptinne, Lefèvre, & Missal, 2008).

Disruption detection and inhibition of habituated actions

When disruptions episodically occurred in our experiment (Experiments 2 and 3), a conflict would have arisen between the target anticipated by the observer and the one actually presented. There is evidence that the anterior cingulate cortex is involved in detecting such information processing conflicts (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999, although see Mayr, Awh, & Laurey, 2003 for an alternative interpretation), and that neurons in the pre-supplementary motor area (Isoda & Hikosaka, 2007) and the supplementary eye fields (Stuphorn, Taylor, & Schall, 2000; Sumner et al., 2007) may perform similar
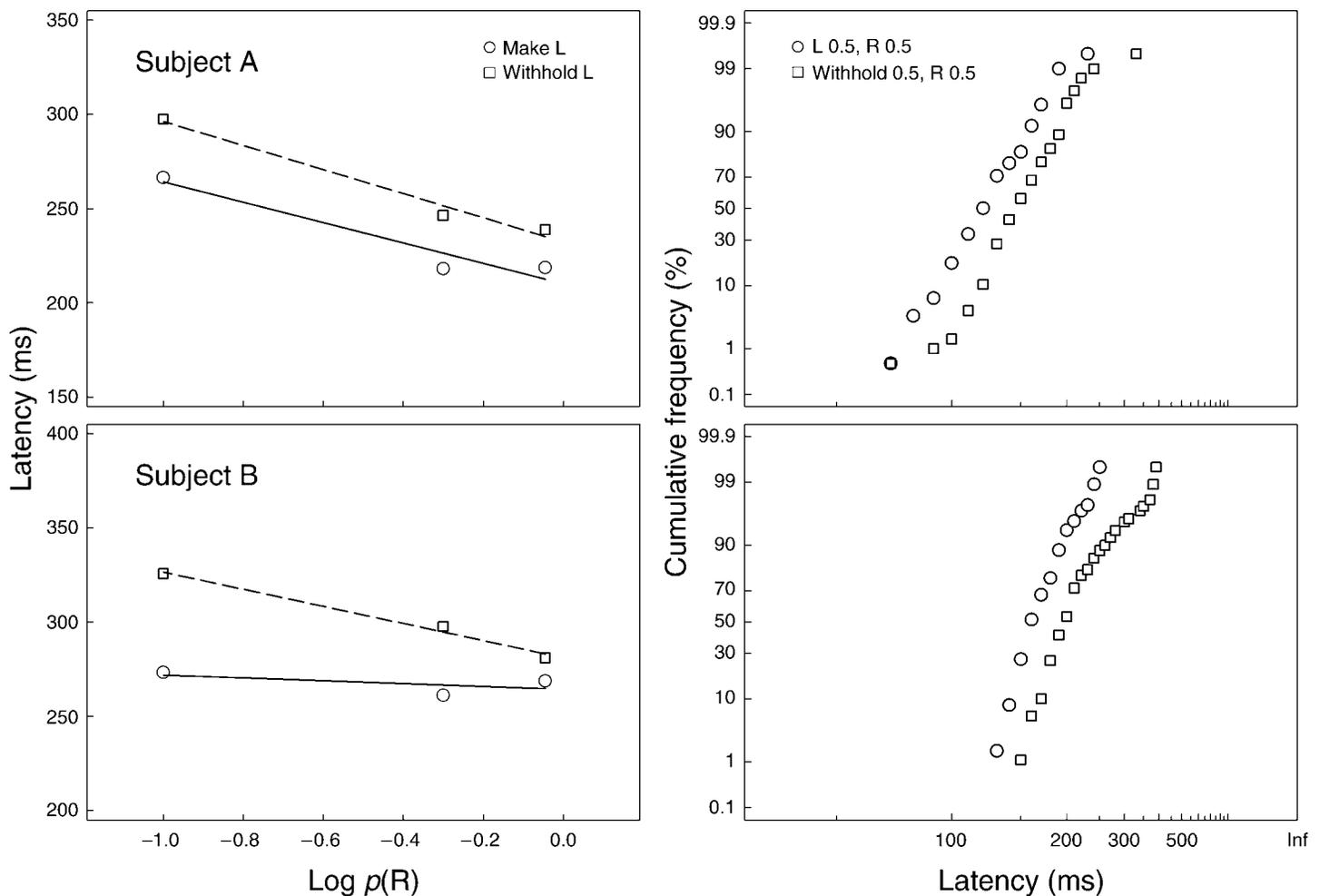
Figure 5. Latencies for the appearance task used in Experiment 4, in which subjects either withheld (squares) or did not withhold (circles) left-going saccades. Left panels give average latencies for each condition, with straight lines giving linear regression through the data. Latency distributions on recinormal plots for the condition where the probability of a right-going saccade was 0.5 are shown in the right panels.

roles. After a disruption, but before the reappearance of the low-frequency target, average latencies were significantly increased (Experiment 2). This suggests that latencies did not simply default to a 50:50 expectation level once the pattern sequence was lost, but rather were actively delayed, presumably reflecting inhibition of this habituated response that is no longer appropriate once the sequence was disrupted. Indeed, recent evidence shows that neurons within the pre-supplementary motor area in monkeys are involved in actively suppressing a habituated response that is no longer appropriate (Isoda & Hikosaka, 2007) in a task-switching paradigm: such experiments are probably most analogous to our Experiment 3 in which the task required was known with a high degree of confidence on both sides of a disruption. Our results for this experiment showed a transient increase only at the time of the disruption (Figure 4), with latency distributions appearing shifted relative to saccades away from the disruption. Similar task-switching experiments, also showing a lengthening of latencies at the time of the switch, have been performed in human observers (Sinha et al.,

2006). Sinha et al. (2006) have shown that the transient latency increase in task switching is compatible with the operation of two LATER units in series: the first to interpret the instruction for a given trial prior to a second LATER unit deciding the appropriate response. It is therefore not altogether surprising that such a compound mechanism does not produce a simple swivel in distributions. More recent work has suggested that task-switching costs only occur when an instruction alters a response programmed by the appearance of a stimulus (Cameron, Watanabe, & Munoz, 2007), in concordance with the fact that no switching cost was found by Sinha et al. when the switching instruction preceded the target onset by a criterion amount. Viewed within the context of a two-stage LATER model, deciding the task instruction is already completed by the first stage and so no additional delay occurs when, upon appearance of the stimulus, the second stage must decide upon an appropriate motor response. However, Cameron et al. (2007) found that merely being able to predict where a target would appear was insufficient to program a response and so generate a

switching cost, which appears at odds with our results. There are, however, several important differences between these two studies. First, the bias used by Cameron et al. to allow the location of the target to be predicted was relatively low at 75% and so is unlikely to generate the sorts of habituated responses seen in our experiments. Second, switching occurred at a dramatically higher rate than in our experiment (50% of the time, as opposed to our 3%) and so their subjects' expectation that a switch would occur would be dramatically increased.

Our experiments also share some similarities with other experimental paradigms in which responses are withheld. In Experiment 2, the presence of a target other than that predicted by the previous pattern may create a conflict that generates something analogous to a countermanding signal in order to suppress an inappropriate, habituated saccade. Previous modeling (Asrress & Carpenter, 2001; Hanes & Carpenter, 1999) suggests that this suppression occurs by decreasing the rate of rise of the decision signal to be suppressed, which could explain the shift in the latency distributions observed if such a suppression inhibited the rate of rise of all saccadic LATER units data indiscriminately. Withholding is also clearly seen in go/no-go paradigms when a particular cue is present. A delay in pro-saccades has been noted after responses were withheld on the immediately previous trial in such a paradigm (Barton, Raoof, Jameel, & Manoach, 2006), suggesting that the suppressive effect of withholding a response persists for some time. It has also been suggested that an anti-saccade, which requires a reflexive pro-saccade to be withheld, produces a saccadic inhibition on a subsequent trial (Cherkasova, Manoach, Intriligator, & Barton, 2002). Although the results of our Experiment 2 (Figure 3) might appear consistent with such a persistence, our Experiment 3 (Figure 4) shows that this is not the case: withholding an anticipated response influenced only the trial in which a disruption occurred (abscissa = 0), with latencies returning to normal immediately afterward. The reason for the discrepancy between our results and those of Barton et al. (2006) may relate to the high frequency (0.5) of no-go trials in the latter's experiment, making the likelihood that any given no-go trial would be followed by another high (0.5). In comparison, the probability of a further no-go trial was more than tenfold lower (0.03) in Experiment 3. This is also consistent with our findings in Experiment 4 (Figure 5) where withholding one type of response increased the latency for non-withheld responses, given the comparatively high probability that a response would need to be withheld on any trial (0.1 to 0.9).

Strategies for reestablishing patterned actions

In contrast to the acute increase in latency seen in Experiment 3, our paradigm in Experiment 2 showed a more protracted increase in latencies after a disruption,

consistent with a habituated response needing to be inhibited until the phase of the sequence could be reestablished. Our results also showed that low-frequency events are used to establish the phase of a repeating sequence (Figure 3, lower panel) triggering the decision to cease inhibition and to return to the previous habituated actions. How might such a decision occur? There is evidence that neurons in monkey prefrontal cortex can code simple abstract rules to determine saccadic location (e.g., same/different matching; Wallis, Anderson, & Miller, 2001), and that this activity is preceded by similar activity in the pre-motor cortex in certain circumstances (Muhammad, Wallis, & Miller, 2006). Neurons encoding such rules appear to show a steady increase in firing rate when the conditions for the rule is met (Wallis et al., 2001), and so it would be possible that decisions between competing sets of rules proceed in a LATER-like way, with the rule being used at a given time being the one that rises to threshold the fastest, similar to the two-stage model proposed by Sinha et al. (2006). The observation that differing strategies for solving arithmetical tasks produce differing growth of event-related brain potentials and correspondingly different reaction times (small reaction times for larger potentials; El Yagoubi, Lemaire, & Besson, 2003) may perhaps suggest something like a race-to-threshold process that selects competing strategies.

Flexibility in the mechanisms calculating expectation

Overall, our results demonstrate that the saccadic system's calculation of prior probability—the expectation that a target will appear at a particular position—is more complex than has generally been assumed. We have shown previously (Anderson & Carpenter, 2006) that slow changes in expectation—and, correspondingly, latency—occur in stochastic runs following abrupt changes to the probability of a target appearing at a particular spatial location. This suggests the operation of a mechanism that integrates information over an extended series of events. When stochastic runs containing multiple locations are randomly interleaved with runs containing a single location (the simplest type of deterministic pattern), latencies also progressively alter as the subject implicitly determines what type of sequence is being presented: in such situations, expectation appears to change in accordance with Bayes' theorem (Sharma, Dragoi, Tenenbaum, Miller, & Sur, 2003). Such integrative mechanisms are appropriate in experiments where a long-term frequency at a location must be estimated from observing frequencies in a stochastic sequence but may be less so in other situations. Indeed, there is evidence that they do not operate in situations where they would be of no advantage, such as when the overall frequency is 0.5 and so no useful prediction about the location of the next target in a sequence can be made (Anderson et al., 2008). The results of the

current paper show that under appropriate circumstances expectation at a given location can change rapidly and dramatically on a trial-by-trial basis, as would be appropriate when the viewed environment is more deterministic than random. While these changes in latency are clear (Figure 2), we failed to differentiate between two potential mechanisms underlying these changes (shift versus swivel) considered here and so we can neither confirm or refute that latency changes in deterministic patterns have their effect via the same mechanism as stochastic frequencies (Carpenter & Williams, 1995). The comparatively small-magnitude trial-by-trial modifications of expectation we have described previously in stochastic sequences (Anderson & Carpenter, 2006) differ from the dramatic changes in expectation seen in deterministic sequences however, where the probability of appearing in a location can change from 0 to 1 on a trial-by-trial basis. Based on our current findings, and our previous investigations of expectation development (Anderson & Carpenter, 2006; Anderson et al., 2008), a picture emerges that the calculation of expectation is remarkably flexible, reflecting what is most appropriate to suit the observed properties of the environment actually being viewed.

Conclusions

In summary, our results demonstrate that deterministic patterns can result in large and rapid changes in expectation that can influence the LATER decision-making process. We believe the paradigm used in our Experiment 2 is distinct from a simple task-switching procedure in which habituated actions must be inhibited for a single trial by employing a simple rule (Isoda & Hikosaka, 2007), as it involves inhibition of habituated actions over a more protracted series of events and the simultaneous engagement of relatively more complex, strategy-dependent controls. As such, our paradigm may be somewhat closer to real-world situations in which the disruption of an automated series of actions typically involves engaging a higher level problem-solving strategy to allow us to eventually return us to our previous automated sequence, rather than immediately switching to a different one.

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