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Scott N. J. Watamaniuk  
*Wright State University - Main Campus, scott.watamaniuk@wright.edu*

Japjot Bal

Stephen J. Heinen

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A Subconscious Interaction between Fixation and Anticipatory Pursuit

Scott N.J. Watamaniuk,1 Japjot Bal,2 and Stephen J. Heinen2

1Department of Psychology, Wright State University, Dayton, Ohio 45435, and 2The Smith Kettlewell Eye Research Institute, San Francisco, California 94115

Ocular smooth pursuit and fixation are typically viewed as separate systems, yet there is evidence that the brainstem fixation system inhibits pursuit. Here we present behavioral evidence that the fixation system modulates pursuit behavior outside of conscious awareness. Human observers (male and female) either pursued a small spot that translated across a screen, or fixated it as it remained stationary. As shown previously, pursuit trials potentiated the oculomotor system, producing anticipatory eye velocity on the next trial before the target moved that mimicked the stimulus-driven velocity. Randomly interleaving fixation trials reduced anticipatory pursuit, suggesting that a potentiated fixation system interacted with pursuit to suppress eye velocity in upcoming pursuit trials. The reduction was not due to passive decay of the potentiated pursuit signal because interleaving “blank” trials in which no target appeared did not reduce anticipatory pursuit. Interspersed short fixation trials reduced anticipation on long pursuit trials, suggesting that fixation potentiation was stronger than pursuit potentiation. Furthermore, adding more pursuit trials to a block did not restore anticipatory pursuit, suggesting that fixation potentiation was not overridden by certainty of an imminent pursuit trial but rather was immune to conscious intervention. To directly test whether cognition can override fixation suppression, we alternated pursuit and fixation trials to perfectly specify trial identity. Still, anticipatory pursuit did not rise above that observed with an equal number of random fixation trials. The results suggest that potentiated fixation circuitry interacts with pursuit circuitry at a subconscious level to inhibit pursuit.

Key words: eye movements; human; potentiation; priming; smooth pursuit

Introduction

The smooth pursuit system follows moving objects, whereas the fixation system maintains gaze on stationary ones. The behavior and neural control of smooth pursuit has been extensively studied (Keller and Heinen, 1991; Krauzlis, 2004). However, we know much less about how fixation operates. Motion is thought to be the dominant sensory input to pursuit (Kowler, 1989; de Hemptinne et al., 2006). Anticipatory pursuit is ubiquitous and survives target randomization (Kowler et al., 1984; Heinen et al., 2005), despite that it renders upcoming motion unpredictable. However, it can be overridden by a cognitive expectation of target motion, if observers are cued of the direction (Kowler, 1989; de Hemptinne et al., 2006).

Most literature on the fixation system is concerned with the dynamics of miniature eye movements that occur while the eyes view a small stationary target; a pattern of microsaccades and...
smooth movements (Poletti and Rucci, 2016). Little is known about the physiology of fixation dynamics other than that neurons in the rostral superior colliculus (SC) are active during microsaccades (Hafed et al., 2009). However, there is abundant literature on neurons active when the eye is simply viewing a target, without regard for fixation dynamics. These neurons are usually called fixation neurons, and are found in many structures where pursuit neurons reside including the supplementary eye field (SEF; Bon and Luchetti, 1992), the cerebellar vermis (Kase et al., 1980), and the parietal lobe (Bremmer et al., 1997). They also exist in localized zones that are adjacent to neural regions generating saccades or pursuit, most notably in the rostral SC (Munoz and Wurtz, 1993) and the frontal eye field (FEF; Izawa et al., 2009).

Having pursuit and fixation neurons in close proximity suggests that pursuit and fixation systems share a functional substrate. However they are generally believed to be different systems because ocular dynamics when transitioning from one to the other are different (Luebke and Robinson, 1988). Furthermore, in countermanding experiments when fixation is activated to prevent pursuit from following a moving target, pursuit and fixation processes are modeled as independent (Kornryo et al., 2003). Despite the presumed independence of pursuit and fixation, there is evidence that they interact in the brainstem. A brainstem structure, the interpositis raphe nucleus, has neurons that maintain a tonic level of activation during fixation. However, during a saccade their activity completely quiesces. These omnidirectional “pause” neurons (OPNs) are theorized to function as a “gate” that holds fixation, and releases it to allow a saccade (Van Gisbergen et al., 1981; Scudder et al., 1988). OPN activity only partially subsides during pursuit, evidence that neurons active during fixation interact with pursuit (Missal and Keller, 2002).

In the current study we demonstrate an interaction between the pursuit and fixation systems that appears to be subconscious and beyond direct cognitive intervention. We show that a fixation trial dramatically and actively reduces anticipatory pursuit on an immediately subsequent pursuit trial. Furthermore, the reduction persists even when the observer knows with certainty they should pursue a moving target in that trial.

**Materials and Methods**

**Subjects.** Two of the authors and two other healthy adult volunteer observers, one male and one female, participated in the current study after providing informed consent. One author (S4) and one non-author (S3) were experienced observers, whereas the other two had no previous experimental experience and were naive to the purpose of the study. All observers reported having normal or corrected to normal visual acuity.

**Apparatus.** Visual stimuli were generated using PsychToolbox functions (Brainard, 1997; Pelli, 1997; Kleiner et al., 2007) in MATLAB (MathWorks). Horizontal and vertical eye positions were sampled at 1000 Hz by an EyeLink 1000 eye tracker (SR Research). The EyeLink was calibrated and validated using its resident nine-point method. A chin and forehead rest stabilized the observer’s head and maintained a constant viewing distance. Data were collected at two locations with different monitors and viewing distances. Half of the subjects (S1 and S3) viewed stimuli on a 17 inch high-resolution Nanoa (model T2-17) color monitor (1.76 min arc/pixel) at a rate of 60 Hz from a viewing distance of 48 cm (California laboratory), whereas the other observers (S2 and S4) viewed stimuli on a 23 inch high-resolution Samsung LCD (model SA750) color monitor (1.6 min arc/pixel) at a rate of 120 Hz from a viewing distance of 57 cm (Ohio laboratory). The data collection and analysis software and procedures were standardized across sites.

**Experimental design.** The stimulus was a single dark spot (0.2° diameter, 0.16 cd/m²) presented on a light gray background (19.9 cd/m²). For pursuit trials in the main experiment, the target spot appeared at the far left of the display for a random fixation time (500–1000 ms) and moved horizontally across the display at a speed of 20°/s for 750 ms. For fixation trials, the spot remained stationary at the left edge of the screen for the entire trial duration. In blank trials, the screen simply remained empty at background luminance for the duration. Trials were triggered automatically with an intertrial interval of ~1 s. In different experiments, we manipulated the type of trials interleaved with pursuit (blank or fixation), fixation trial duration, the proportions of fixation trials within a block, and the predictability of fixation and pursuit trials.

**Eye movement data analysis.** Horizontal and vertical eye velocity were calculated offline from the recorded position signals by differentiating and filtering the raw eye position data (2-pole Butterworth noncausal filter; cutoff = 50 Hz). Saccades were detected offline when eye velocity exceeded 50°/s, with manual correction if necessary. Each saccade was removed from the velocity trace and replaced with a line that interpolated eye velocity before and after the saccade. The main metric of interest was anticipatory velocity. A complication when measuring anticipatory velocity is that observers will typically realign their gaze to the target when they become aware that their eye is drifting off of the target. As a result, measuring anticipatory velocity at any particular time point around target motion onset will likely underestimate the true anticipatory effect. We thus measured anticipatory velocity by averaging eye velocity over the time period extending from 50 ms before to 50 ms after target motion onset. For fixation trials, we randomly assigned (without replacement) an analysis period from a pursuit trial to a fixation trial within the same block, since there is otherwise no target motion onset to anchor the anticipatory pursuit analysis period. All statistical tests used an α level of 0.05. Throughout the paper, the adjustment for computing Cohen’s d for paired samples uses Equation 8 from Morris and DeShon (2002).

**Results**

Our primary question was whether the pursuit and fixation movement systems interacted. Thus, in the first experiment we randomly interleaved fixation trials with pursuit trials and measured anticipatory pursuit. Each observer completed a 60 trial block (30 pursuit and 30 fixation trials). They also completed a control block consisting solely of pursuit trials. Figure 1A shows a random selection of raw eye velocity traces of pursuit trials from blocks without (blue traces) and with (red traces) randomly interleaved fixation trials. For fixation trials, the spot remained stationary for 1100 ms and was then extinguished. In pursuit trials, the target moved for 750 ms following the random fixation period. Figure 1B shows summary anticipatory pursuit data (only anticipatory pursuit before a pursuit trial was used in this analysis) for all four observers for both conditions. Both the raw traces and summary data show that randomly interleaving an equal number of fixation trials with pursuit trials dramatically reduced anticipatory pursuit. The effect was verified using a paired t test (two-tailed) performed on anticipatory eye velocity, which showed significantly reduced anticipatory eye velocity (M = 0.33, SEM = 0.11) compared with pursuing every trial (M = 3.705, SEM = 0.40; t(3) = 6.95, p = 0.006, Cohen’s d = 3.56).

Importantly, for every pursuit trial the target moved from the same side of the display, at the same speed and direction. Even under these conditions, which typically produce substantial anticipatory pursuit, inserting fixation trials effectively silenced it. It is possible that interleaving fixation trials reduces anticipatory eye velocity because the putative stored velocity signal underlying its generation (Barnes and Asselman, 1991, 1992) passively decays over time, and the fixation trials allow sufficient time for this stored velocity signal to decay to near zero. To test this, we created a paradigm with an identical temporal structure as in the first experiment, but replaced the fixation trials with trials where the fixation point never appeared. To an observer
Figure 3. Anticipatory pursuit velocity for the 1100 and 500 ms duration fixation trials, as well as the 20% fixation trial condition. In all conditions, fixation trials were randomly interleaved with pursuit trials and the pursuit target moved for 750 ms. Individual performance and the average anticipatory eye velocity for the pursue-every-trial condition is shown for comparison (the black line and shaded area shows the average ± 1 SEM). Even when fixation trials were only 500 ms, anticipatory velocity was dramatically curtailed. Moreover, including just 20% fixation trials effectively reduced anticipatory pursuit. Error bars are ± 1 SEM.

Figure 1. A, Representative raw eye velocity traces for one observer (S3) taken from trial blocks in which she pursued rightward from the left screen edge on every trial (blue traces), or had 50% fixation trials randomly interleaved (red traces). B, Summary anticipatory eye velocity data for all four observers for both conditions. Error bars represent ± 1 SEM.

A

B

Figure 2. Anticipatory eye velocity when an equal number of blank or fixation trials were randomly interleaved with pursuit trials. Equivalent duration blank trials (black bars) are ineffective at reducing anticipatory eye velocity compared with fixation trials (red bars). Error bars are ± 1 SEM.

These “blank” trials would appear as an extended intertrial interval. The blank trials had a duration of 1100 ms. As in the experiment above, only anticipatory pursuit before a pursuit trial was used in the analysis. Figure 2 shows summary anticipatory pursuit data for all four observers for pursuit blocks with interleaved blank trials (black bars) or interleaved fixation trials (red bars) replotted from Figure 1. Interleaving fixation trials produced significantly lower anticipatory eye velocity (see above) than interleaved blank trials (M = 2.478, SEM = 0.34; paired, two-tailed t test: \( t_{(3)} = -6.57, p = 0.007 \), Cohen’s \( d = 3.99 \)). Therefore, passive decay of a stored velocity signal is not responsible for the anticipatory velocity decrease observed when fixation trials are interleaved with pursuit trials.

Thus far, the data show that randomly interleaving fixation trials virtually eliminates anticipatory pursuit, and that the effect is not due to passive decay but rather an active interaction between the fixation and pursuit systems. What is the nature of this proposed interaction? Specifically, how does the interaction depend upon the duration during which the fixation and pursuit systems are active? The first experiment roughly equated the duration of the fixation and pursuit trials, but the pursuit target only moved for 750 ms, whereas the fixation stimulus was 1100 ms, engaging the fixation system ~1.5 times longer than the pursuit system. To test whether the duration of system activation played a critical role in the interaction, we maintained 50% fixation and pursuit trials within a block but reduced the fixation trial duration to 500 ms, less than the random fixation period preceding each pursuit trial (500–1000 ms) and 0.67 times the duration of pursuit target motion (750 ms). Figure 3 shows data for all observers for the 500 ms fixation trials along with data from the 1100 ms fixation duration and the pursue-every-trial conditions (replotted from Fig. 1). The data clearly show that even fixation trials 500 ms in duration significantly reduce anticipatory pursuit (M = 0.52, SEM = 0.187) compared with the pursue-every-trial baseline (paired \( t_{(3)} = 6.76, p = 0.007 \), Cohen’s \( d = 3.51 \)), and were as effective as the 1100 ms fixation trials (paired \( t_{(3)} = 0.75, p = 0.51 \)). This suggests that equivalent trial duration is not necessary for fixation to reduce anticipatory pursuit.

It has been suggested that anticipatory pursuit optimizes eye velocity to a level between the current target velocity and that of the target on an upcoming trial (Heinen et al., 2005). If the optimization process is privy to conscious intervention, observers...
should generate more anticipatory pursuit if they are more certain that an upcoming trial will be a pursuit trial rather than a fixation trial. Nonetheless, the results from the 500 ms fixation trial condition show it is unlikely that trial certainty can fully explain anticipatory pursuit suppression. Every trial where the fixation period exceeded 500 ms was instantly identifiable as a pursuit trial, yet observers still did not generate appreciable anticipatory pursuit. To test directly the effect of certainty on anticipatory pursuit, we constructed blocks of trials in which there were 80% pursuit trials and 20% standard fixation trials (1100 ms duration). If uncertainty is the reason that anticipatory pursuit decreases, there should be greater anticipatory eye velocity in the 20% fixation condition than the random condition because pursuit trials are four times more likely. Data from pursuit trials for the 20% fixation trial condition are plotted in Figure 3, for comparison with the other conditions. Surprisingly, having as few as 20% fixation trials in a block (M = 0.73, SEM = 0.26) reduced anticipatory pursuit as much as did 50% fixation trials (paired t(3) = 1.37, p = 0.26). Therefore, certainty that an upcoming trial will be a pursuit trial does not appear to override the fixation system’s inhibitory effect on anticipatory pursuit.

Even in the 20% case, fixation trials are still possible. If the penalty for generating anticipatory pursuit is perceived as high during a fixation trial, anticipatory pursuit might be consciously suppressed on every trial to avoid the penalty even when fixation trials are rare. To test this, we used a paradigm in which observers knew in advance the type of every trial, and could therefore generate anticipatory pursuit on pursuit trials if conscious intervention against fixation influence were possible. In the paradigm, fixation and pursuit trials were alternated predictably on every trial (alternation probability 1.0), rendering their identity explicit and thereby maximizing their expectancy (Maljkovic and Nakayama, 1994). If observers can consciously override the inhibition imposed by fixation trials, high anticipatory eye velocity should return for the pursuit trials. Alternatively, if the suppressive effect of fixation on pursuit is subconscious and operates when fixation circuitry is potentiated at a low-level, anticipatory pursuit on the following trial should continue to be inhibited. Low-level potentiation, sometimes referred to as priming, is commonly observed in visual (Maljkovic and Nakayama, 1994) and pursuit (Kowler et al., 1984; Heinen et al., 2005) behavior. The paradigm included two other conditions for comparison. A pursue-every-trial condition (alternation probability 0.0) in which no fixation trials were present removed their influence on anticipatory pursuit at either a conscious or subconscious level, thereby also maximizing pursuit expectancy. In the final condition, pursuit and fixation trials were randomly interleaved (alternation probability 0.5). This minimizes the expectancy of pursuit trials and thereby the possibility that anticipatory pursuit is consciously produced, while still allowing subconscious pursuit and fixation interactions.

Figure 4A shows anticipatory eye velocity data for the four observers plotted as a function of the probability of task alternation, along with two theoretical curves corresponding to the three alternatives above. The red dotted curve is the U-shaped function predicted by the cognitive expectancy hypothesis (Attneave, 1959), normalized to average anticipatory pursuit velocity from the pursue-every-trial baseline condition. The black dashed line represents a prediction based upon potentiation from the previous trial, again normalized to baseline. In fact, the data appear not to follow either prediction. Anticipatory eye velocity for alternation probabilities of 1.0 and 0.5 is lower than what the red expectancy curve predicts, and anticipation for the 0.5 alternation probability is also lower than potentiation predicts. A paired t test confirms that the data at the 0.5 alternation probability (M = 0.33, SEM = 0.11) are not significantly different from those at the 1.0 alternation probability (M = 0.91, SEM = 0.30; paired t(3) = 2.34, p = 0.101).

Potentiation and expectancy, if present, should also modulate anticipatory pursuit during fixation trials. Because anticipatory pursuit occurs before target motion and, in the case of the 0.5 alternation probability, before the observer knows whether the trial is a pursuit or fixation trial, the effects of potentiation and expectancy should be measurable but inverted from those observed in pursuit trials. Figure 4B shows anticipatory eye velocity for the four observers for fixation trials, along with the expectancy (red dotted curve) and potentiation (black dashed line) predictions. Note that we did not conduct a fixate-every-trial condition (0.0 alternation probability) but with no pursuit trials, no anticipatory pursuit is expected. Similar to the pursuit trial analysis, the data do not fit either prediction well: the 0.5 data are lower than the expectancy curve prediction, whereas both the 0.5 and 1.0 data are lower than the priming prediction. A paired t test shows that anticipatory pursuit at the 0.5 transition probability (M = 0.13, SEM = 0.07) is not significantly different from that observed for the 1.0 transition probability (M = 0.08, SEM = 0.05; paired t(3) = 0.44, p = 0.69). Moreover, there was no significant difference in anticipatory pursuit when comparing pursuit versus fixation trials at the 1.0 transition probability (t(3) = 2.71, p = 0.073). Thus, even when observers could expect with certainty that the current trial was a pursuit trial, they exhibited no greater anticipatory pursuit than when they could expect a fixation trial.

It could be that microsaccades that occurred during the interleaved fixation trials contributed to the reduced anticipatory pursuit that we observed because merely engaging another oculomotor system interferes with impending anticipatory pursuit. If so, engaging that system to a greater degree should diminish anticipatory pursuit more. To explore this possibility, we assessed
whether the frequency and amplitude of microsaccades (saccades with magnitudes ≤0.5°; Poletti and Rucci, 2016) in fixation trials were correlated with the subsequent anticipatory pursuit (Fig. 5). First we computed the correlation between microsaccade frequency in each fixation trial and the magnitude of anticipatory pursuit in the subsequent pursuit trial for each observer, and tested whether their correlation was different from zero (two-tailed $t$ test). None of the correlations reached significance ($p > 0.05$). We then tested the correlation between microsaccade magnitude and anticipatory pursuit magnitude in subsequent pursuit trials for each observer, also using a two-tailed $t$ test. Again, none of the correlations were significantly different from zero ($p > 0.05$).

**Discussion**

We found that interleaving an equal number of fixation and pursuit trials resulted in less anticipatory pursuit than did pursuing a target that moved with the same direction and speed on every trial. Interleaving blank trials was less effective, indicating that passive decay of the anticipatory signal cannot account for the anticipatory pursuit reduction. Therefore, it appears that fixation trials actively suppress anticipatory pursuit. Moreover, fixation trials did not lose their suppression potency when they were shorter than pursuit trials, or when they were relatively infrequent. Finally, rendering pursuit trials completely predictable by alternating them with fixation trials resulted in the same amount of anticipatory pursuit suppression as found when pursuit and fixation trials were randomly interleaved. Thus cognitive expectation does not appear to significantly alter the suppressive effect of fixation.

**The nature of the fixation–pursuit interactions**

Previous work investigated whether anticipatory pursuit and fixation interact (Kowler and Steinman, 1979). In that study, observers suppressed anticipatory eye movements only when making auditory-cued saccades to static targets. All other conditions, including being instructed to fixate while a target stepped between positions, evoked anticipatory smooth velocity. In another study, the effect on anticipatory pursuit of maintaining fixation within a trial was investigated (Barnes and Donelan, 1999). In one condition, observers began a trial by holding gaze between two fixation targets that were presented on the midline above and below the pursuit target’s starting location. The pursuit target was then illuminated, and simultaneously moved across the screen. In another condition, the fixation stimuli were not presented, and observers merely pursued the target when it appeared and moved. No difference was found between anticipatory eye velocity in the fixation and no-fixation conditions, and it was concluded that fixation did not affect the anticipatory response. Although both prior studies found that anticipatory pursuit was unaffected by fixation, it may be because observers expected the target to always move immediately following the fixation period.

Although Barnes and Donelan’s (1999) results appear to contradict ours, the two studies differ in a key fashion. In their study, observers fixated in the same trial before the pursuit target moved. In our paradigm, observers fixated in discrete trials in which the target remained stationary. Furthermore, the fixation trials, like the pursuit trials, were delineated by intertrial intervals. It is known that cognitive expectations can influence the direction of anticipatory pursuit (Kowler and Steinman, 1979, 1981; Kowler et al., 1984; Kowler, 1989), and therefore the discrete fixation trials in our experiment might have suppressed anticipatory pursuit because they increased the expectation that the next trial would be a fixation trial. However, data from the alternating condition (probability of alternation = 1.0; Fig. 4) show that even when observers should expect the next trial to be a pursuit trial, anticipatory pursuit is still suppressed. Thus it appears to be critical that introducing discrete fixation trials, not just extended fixation periods before pursuit, is necessary to reduce anticipatory pursuit. A potential explanation for this is that the gain on the fixation system is maximal during discrete fixation trials, but reduced during the fixation periods preceding pursuit to allow more rapid pursuit initiation. A higher gain on the fixation system could lead to greater potentiation of it, and hence cause it to influence subsequent pursuit trials.

Microsaccades are miniature eye movements that occur during fixation (Poletti and Rucci, 2016). It might be that engaging the microsaccade mechanism interferes with anticipatory pursuit generation and causes its reduction. If so, larger microsaccades or a greater number of them in fixation trials might be expected to cause a greater reduction in the magnitude of anticipatory pursuit observed in subsequent trials. We therefore examined whether the amplitude or frequency of microsaccades during fixation trials in the alternating condition were correlated with the magnitude of anticipatory pursuit in the immediately following
pursuit trial, and found no evidence of a link between them. This suggests that merely engaging a different oculomotor system does not cause a reduction in anticipatory pursuit, although our results do not conclusively rule out this possibility. Instead, we think that the potentiation is specific to the fixation system, and that fixation and pursuit are normally maintained in a balance that prevents the eyes from moving, or allows them to pursue given situational demands. Our results suggest that potentiating the fixation system tips the balance toward fixation dominating the response.

What might be the neural circuitry that underlies fixation’s interaction with pursuit? OPNs, located in the raphe nucleus and thought to maintain fixation between saccades (Raybourn and Keller, 1977), might participate in suppressing anticipatory pursuit. OPNs inhibit premotor saccade neurons during fixation, and must silence for a saccade to be executed (Raybourn and Keller, 1977). These neurons also reduce their activity, though not completely, during pursuit (Missal and Keller, 2002). It is possible that a fixation trial potentiates the OPNs, which then suppress anticipatory pursuit on the next trial. Modulation of OPN activity might occur via the rostral SC, which has excitatory projections to the raphe nucleus that could increase neural activity there and potentiate fixation (Gandhi and Keller, 1997). Alternatively, frontal cortical regions might also be involved in implementing fixation. Stimulation of the foveal representation of the FEF can suppress saccades (Burman and Bruce, 1997), and the FEF has also been implicated in a type of behavioral potentiation known as priming during an oculomotor pop-out task (Bichot and Schall, 2002). The SEF might be involved in suppressing anticipation as well, because microstimulation there during pursuit decreases eye velocity (Missal and Heinen, 2017). The fixation–pursuit interaction could even be manifest within circuitry internal to the SEF, because this area is also involved in anticipatory pursuit generation (Heinen and Liu, 1997; Missal and Heinen, 2004).

Implications for pursuit research
Anticipatory pursuit has been studied to understand how stimulus predictability contributes to pursuit (Becker and Fuchs, 1985; Barnes et al., 1987; Boman and Hotson, 1988, 1989; Knox, 1996, 1998; Barnes and Donelan, 1999; Blohm et al., 2003a,b; Collins and Barnes, 2005; Heinen et al., 2005; Barnes, 2008). However, anticipation makes it difficult to study how visual signals contribute to pursuit initiation because it not only generates pursuit before the target moves, but also modulates eye velocity during the “open-loop” initiation period (Kowler and McKee, 1987; Kao and Morrow, 1994; Heinen et al., 2005), and the open-loop period has been characterized as being driven solely by visual motion (Lisberger and Westbrook, 1985; Keller and Khan, 1986). Stimulus randomization is used in the attempt to reduce anticipatory pursuit, as randomization eliminates certainty about the identity of an upcoming stimulus (Dallos and Melville-Jones, 1963). Nonetheless, anticipatory pursuit persists despite randomization, because eye velocity is generated as a consequence of potentiation in the pursuit system from the previous trial or sequence of trials (Kowler et al., 1984; Heinen et al., 2005). We found that interleafing fixation trials with pursuit trials virtually eliminated anticipatory pursuit (Fig. 1). Furthermore, even short duration (~500 ms) or infrequent (~20%; Fig. 3) fixation trials can achieve the effect. Therefore, it appears that interleafing fixation trials is an effective method for reducing anticipatory pursuit, and may therefore permit more direct study of visually guided pursuit behavior and physiology.

References
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