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Eye-head coordination in the guinea pig II. Responses to self-generated (voluntary) head movements

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Abstract

Retinal image stability is essential for vision but may be degraded by head movements. The vestibulo-ocular reflex (VOR) compensates for passive perturbations of head position and is usually assumed to be the major neural mechanism for ocular stability. During our recent investigation of vestibular reflexes in guinea pigs free to move their heads (Shanidze et al 2010), we observed compensatory eye movements that could not have been initiated either by vestibular or neck proprioceptive reflexes because they occurred with zero or negative latency with respect to head movement. These movements always occurred in association with self-generated (active) head or body movements and thus *anticipated* a voluntary movement. We found the anticipatory responses to differ from those produced by the VOR in two significant ways. First, anticipatory responses are characterized by temporal synchrony with voluntary head movements (latency ~1 msec versus ~7 msec for the VOR). Second, the anticipatory responses have higher gains (0.80 versus 0.46 for the VOR) and thus more effectively stabilize the retinal image during voluntary head movements. We suggest that anticipatory responses act synergistically with the VOR to stabilize retinal images. Furthermore, they are independent of actual vestibular sensation since they occur in guinea pigs with complete peripheral vestibular lesions. Conceptually, anticipatory responses could be produced by a feed-forward neural controller that transforms efferent motor commands for head movement into estimates of the sensory consequences of those movements.

Keywords

vestibulo-ocular reflex; eye movements; feed-forward models; anticipatory movements

Introduction

Gaze shifts consist of coordinated rapid eye and head movements that shift the line of sight into a new direction and ocular counter-rotations that stabilize the retinal image during the head movement. The vestibulo-ocular reflex (VOR) is usually assumed to produce the ocular counter-rotations during gaze shifts since it is the main mechanism by which retinal image stability is achieved during passive perturbations of head position. Several studies have described coordination of eye and head movements with vestibular reflexes during gaze shifts and passive perturbations of head position (Bizzi et al. 1971; Dichgans, et al. 1973; Dichgans

et al. 1974; Collewyn et al. 1983; Jell et al. 1988; Barnes & Grealy 1992; McCrea & Cullen 1992; Tweed et al. 1995; Crawford et al. 1999; Roy & Cullen 2002; Cullen & Roy 2004; Freedman 2008). During our own recent investigation of vestibular reflexes in guinea pigs that were allowed to move their heads (Shanidze et al. 2010), we observed ocular counter-rotations that were unlikely to have been initiated either by vestibular or neck proprioceptive reflexes as they occurred with zero or negative latency with respect to head movement. These movements always occurred in association with self-generated (voluntary) head or body movements. Because of their short latencies, we refer to them as *anticipatory* eye movements in order to distinguish them from eye movements produced by the VOR in response to passive perturbations (VOR latency ~7 msec, Shanidze et al. 2010).

Eye movements that compensate for self-generated head rotations in guinea pigs have been reported previously (Gresty, 1975) and were thought to be mediated by the VOR. Compensatory eye movements with an extr vestibular origin were also reported by Bizzi, et al. (1971) in monkeys with bilateral vestibular lesions. They interpreted the eye movements to be a learned compensatory mechanism to restore retinal image stability during active gaze shifts. In this report, we systematically describe the anticipatory responses in guinea pigs and supplement that description with lesion studies that can be compared to those described by Bizzi et al. in the monkey. Our results suggest that the anticipatory responses act synergistically with the VOR to stabilize retinal images during voluntary gaze shifts and repetitive head movements induced by passive stimulation. Furthermore, they are independent of actual vestibular sensation since they occur in animals with complete peripheral vestibular lesions. Conceptually, anticipatory responses could be produced by a feed-forward neural controller that transforms efferent motor commands for head movement into estimates of the sensory consequences of those movements.

Finally, an important consideration is the significance of behavioral goals to eye movement patterns either in response to passive perturbations of body position (Shanidze et al. 2010), or as anticipatory components of self-generated head movements. Based on our data, we propose that the guinea pig seeks to resolve two potentially conflicting behavioral goals: (1) to maintain alignment of head and eyes with respect to the body axis (Shanidze et al. 2010) at the possible expense of retinal image stability and (2) to minimize retinal image slip (gaze speed equal to zero). The first behavior is normally seen when an animal sits quietly and is passively perturbed; typical VOR gains are ~0.5 and VCR gains are ~0.2 (Shanidze et al. 2010). The second behavior is seen during self-generated head or body movements during which compensatory eye movements minimize gaze velocity (this report). We hypothesize that the first behavior is consistent with the relatively poor visual acuity of guinea pigs (maximally 2.7 cycles/deg in the visual streak, Buttery et al. 1991) and may reflect what Fuller called “visual inattentiveness” (Fuller 1981), whilst the second behavior is essential for the animal to distinguish object motion in the environment during self-generated movements that reorient the animals’ gaze.

Methods

Experimental and surgical procedures were performed in accordance with the National Institutes of Health *Guide for the Care and Use of Laboratory Animals* and were approved by the University of Michigan’s University Committee on Use and Care of Animals.

Data were obtained from the four guinea pigs (3 pigmented, 1 albino) used in the study of passive responses described in the companion paper (Shanidze et al. 2010). The methods were identical to those of that report, where their descriptions can be found in greater detail. No systematic differences were found between the albino and pigmented animals.

For this study, the trunks of the guinea pigs were restrained within the experimental apparatus. However, the animals' heads were free to move and there were frequent bursts of self-generated head movements. Although some of these movements appeared to be irritative, i.e., rapid shaking of the head, most were exploratory movements during which the animal smelled, chewed or oriented toward some feature of the experimental environment. All analyses of active movements included in this report occurred in the absence of any passive rotational stimulus and all occurred in the dark unless described otherwise.

Two broad strategies were employed to analyze active head movements. First, segments of active head movements (>5 msec in duration) were selected by a software algorithm using two criteria; that no passive stimulus was present and head speed exceeded 5 deg/sec. Each segment was analyzed to compute the gain of the compensatory response by regression of eye-in-head against head-in-space velocity. The latency of the compensatory eye response was determined by cross correlating these variables over the same data segments. The latency was assumed to be the lag associated with the maximum correlation coefficient. Alternatively, some data segments were selected by one of the authors during which discrete head and eye movements occurred and which approximated the speeds and accelerations of the passive whole body transient perturbations analyzed in the previous report (Shanidze et al. 2010). Each of these segments was analyzed for gain and latency using the same approach described above for the automated analysis. For those segments, brief intervals containing rapid eye movements were excluded from the analysis. About 150 minutes total of experimental time in the intact animals was included, from which 31 minutes were selected as being long enough with sufficient head movement activity to warrant analysis; in lesioned animals the corresponding totals were 163 (control) and 250 minutes (post lesion). All of these segments were selected using a computer algorithm and were typical and representative of the animals' behavior.

As described in the companion paper (Shanidze et al. 2010), 6 additional guinea pigs were prepared for recording and data were collected prior and up to 4 months after bilateral transmastoid injections of streptomycin that destroyed virtually all hair cells within the labyrinth. Active head movements were extracted automatically, as described above, at multiple test dates throughout the period of recovery.

Results

Active head movements in normal animals

Fig. 1a illustrates a 2 second-long sequence of self-generated head movements in the dark. During this sequence, the guinea pig made large head turns with speeds that exceeded 400 deg/s (lower gray trace). Eye-in-head velocity (black trace) mirrored the head-in-space, and changes in head speed and direction were matched by changes in eye speed and direction. The upper black trace demonstrates that gaze velocity, the sum of eye-in-head and head-in-space, was nearly zero throughout the sequence. The stability of gaze is more clearly seen in Fig. 1b where the gaze velocity scale is expanded by a factor of ten for the data segment beginning at the arrow. The apparent temporal synchrony of the eye and head data suggests that the eye movement anticipates the head movement. This idea is confirmed by the waveform correlation (Fig. 1c) which demonstrates that the eye-in-head movements led head-in-space movements by ~1 msec. The negative (anticipatory) latency of this response is in contrast to the VOR-initiated compensatory eye movements, for which the latency was ~7 msec (Shanidze et al. 2010).

Fig. 1d shows that the anticipatory eye movement accurately mirrored head velocity; eye-in-head velocity was inversely proportional to head-in space velocity (regression slope = -0.95). The near perfect ocular compensation of the voluntary head turn contrasts with the less than

perfect VOR compensation of passive perturbations (regression slope ~ -0.5 , Shanidze et al. 2010).

Fig. 2 shows two more samples of gaze shifts initiated by head turns that represent the typical pattern of these movements in guinea pigs. Both examples are from the same animal: the records in Fig. 2a were obtained in darkness, those in Fig. 2b in the light. Across all animals, we found no substantive differences between responses that occurred in the light and those that occurred in darkness. Both examples show that a head movement initiates the gaze shift (upper panels, gray traces) and is followed by a rapid eye movement (upper panels, black traces) that orients the eye in the new gaze direction. With the exception of the reversed order of head and eye movement, the pattern resembles that of primates. However, the guinea pig does not have a fovea. Thus, although the rapid eye movement is saccadic in its kinematics, it is analogous to a vestibular quick phase in that it predictively corrects for the change in eye position produced by the image stabilizing slow phase rotation of the eye. The eye-in-head position records (second panel from the top) illustrate the complementary interaction of the slow and rapid components of the ocular response. In Fig. 2a, the final position of the eye-in-head is centered after the ~ 25 degree head turn; in Fig. 2b, the eye is initially off-center and is returned to a central position at the end of the head turn. The VOR is usually assumed to produce the compensatory ocular rotation; in these examples, however, the latency of the response is too short for it to be produced by the VOR. Figs 3a&c show waveform correlations for the initial compensatory eye movements (arrows, Figs 2a&b respectively) that confirm latencies less than zero thus demonstrating the anticipatory nature of these responses. Although the initial compensatory response shown in Fig. 2a (arrow) was anticipatory, the compensatory response following the rapid eye movement had a longer latency (Fig. 3b, 8.2 msec) consistent with this segment having been produced by the VOR. The corresponding data segment in Fig. 2b was interrupted by two small anticomensatory rapid eye movements that caused the intervening compensatory intervals to be too short for the computation of latency using waveform correlation. The lower panels, Figs 3d–f, show regression analyses of eye-in-head relative to head-in-space for the corresponding data segments whose correlations are illustrated in Figs 3a–c; for each data segment, the relationship was linear (Fig. 3d, slope = -0.75 ; Fig. 3e, slope = -0.89 ; Fig. 3f, slope = -0.94).

Fig. 4a shows the distribution of latencies for 74 segments of anticipatory responses to self-generated head movements in 3 animals. To compute lag (or response latency), cross correlations of the eye-in-head and head-in-space velocities were performed. The mean anticipatory latency was 0.0001 ± 0.0025 sec (standard deviation). To ensure that segment lengths were not confounding the results, a regression of segment length to lag was performed and no relationship was found ($r^2 = 0.029$). Additionally, the 74 segments were broken up into 25 msec long intervals, analyzed for latency and binned. The procedure confirmed the results shown in Fig. 4 (mean -0.2 ± 0.27 msec, $n = 47,827$). Most of the computed latencies were less than zero verifying the anticipatory nature of the responses illustrated in Figs 1&2.

We analyzed the 74 data segments further to establish the relationship of head and eye velocity. Eye and head velocity were recorded for multiple segments of active head movement that occurred in the absence of passive rotation. Linear regressions of eye-in-head versus head-in-space velocity were done for consecutive points of each active head movement segment. Segments for which no valid cross correlation value could be found were excluded from the analysis. Fig. 4b shows the resultant distribution of slope values. Statistically, the slopes were normally distributed with a mean of -0.80 , i.e., the anticipatory movements compensated on average for 80% of head velocity. To determine if there was a relationship between response gain and latency, the gain values were re-plotted subject to certain conditions. The data set was divided into two distributions – one with only gain values associated with lags less than 2 msec ($n = 61$) and another with values associated with lags greater than or equal to 2 msec. The mean

of the smaller lag subset was slightly higher (-0.81 ± 0.23) than that of the other subset (mean = -0.74 ± 0.19).

Active head movements in animals with bilateral vestibular lesions

None of the guinea pigs with bilateral peripheral vestibular lesions were able to produce eye movements that could compensate for unpredictable, passively-induced head movements; this deficit persisted for the entire post-lesion survival time of 4 months. However, within one week post-lesion, the same animals were able to effectively compensate for self-generated head movements. Fig. 5 shows 2 representative samples of anticipatory responses in one animal 4 months post-lesion. Fig. 5a illustrates the animal's initial lack of response to a passive perturbation followed by a robust anticipatory response to a self-generated head turn. Initial eye-in-head velocity (black trace, 3rd panel from top) was persistently zero immediately after the abrupt onset of head and body rotation (arrow). As a result, initial eye-in-space (gaze) velocity (arrow, lower panel) tracked head velocity. In this data segment, ~150 msec after the passive perturbation, the animal actively counter-rotated its head (gray traces, upper & 3rd panel). The head turn was accompanied by an anticipatory ocular counter-rotation (3rd panel, black trace, latency = -2 msec) that produced a gaze velocity of close to zero deg/s during the head movement (regression slope = -0.86 , eye relative to head). Fig. 5b illustrates another example of a post-lesion anticipatory response. In this record, the guinea pig actively rotated its head counterclockwise through an angle of nearly 50 degrees (gray trace, upper panel). In temporal synchrony with the voluntary head turn, the eye-in-head counter-rotated (2nd panel from top) and gaze velocity (bottom panel) was zero prior to the occurrence of the rapid eye movement. One noteworthy feature of the post-lesion data can be seen in this Figure: deviations of eye-in-head position were frequently greater than in the intact animal because rapid anticomensatory eye movements either failed to occur (Fig. 5a) or compensated inaccurately for the ocular counter-rotation (Fig. 5b).

To compare anticipatory eye movements in lesioned animals to those in intact animals, voluntary head movement segments were selected from control and 2 week post-lesion recordings. Fig. 6 shows distributions of latency (a) and gain (b) for anticipatory responses in control and 2-week post lesion animals. Two weeks post-lesion the mean lag of the anticipatory eye movements in responses to head movements was -0.001 ± 0.001 sec ($n = 371$), and was indistinguishable (t-test, $\alpha=0.05$) from the control responses of the same animals (-0.001 ± 0.001 sec, $n = 242$). Fig. 6b shows the distributions of response gains. Two weeks post lesion the mean response gain was -1.11 ± 1.22 , greater than that measured in the control condition (-0.80 ± 0.30). The pre- and post-lesion distributions were statistically different (t-test, $\alpha=0.05$).

Discussion

We describe, for the first time, a novel compensatory and *anticipatory* ocular response that occurs in conjunction with self-generated head movements. Gresty's (1975) pioneering study of the unrestrained guinea pig described the pattern of head and eye movements associated with voluntary gaze shifts, but because of technical limitations, he could not measure the latency of compensatory eye movements. However, he concluded that the "vestibular-ocular reflex is utilized in a frequency range in which it produces perfect compensation for fast programmed head movements". In this study, we find that the anticipatory eye movements are not dependent on vestibular sensory inflow since they occur in animals with complete bilateral vestibular lesions (Fig. 5). Although the extravestibular origin of these responses could not be determined with certainty by our experiments, we believe that neck muscle proprioception or other sensory afferents are unlikely origins for anticipatory responses that precede any detectable movement of the head (Fig. 3). However, we cannot exclude the possibility of EMG

activity in neck musculature that precedes actual movement of the head and modulates the discharge of secondary vestibular neurons (Vibert et al. 1999). Alternatively, the anticipatory responses may be dependent on motor efference (“efference copy”, von Holst & Mittelstaedt, 1950; Mittelstaedt 1971). Bizzi et al. (1971) described compensatory eye movements in nonhuman primates that were associated with self-generated head movements and were not dependent on vestibular sensation since they were observed to occur in animals with bilateral labyrinthectomies. Furthermore, the compensatory responses were still present after surgical lesions interrupted proprioceptive input from the cervical spinal cord. Newlands et al. (1999, 2001) reported similar findings in monkeys with bilateral canal plugs or unilateral vestibular lesions. Although these authors believed the compensatory eye movements developed as an adaptive response to vestibular lesions, Zhou et al. (2010) recently reported zero latency compensatory eye movements in intact monkeys during voluntary head turns, suggesting that anticipatory responses are part of an animal’s normal behavioral repertoire. This behavior may be more common in more species than previously suspected. In swimming *Xenopus* tadpoles (Combes et al. 2008) and in lamprey during fictive swimming (Grillner 2008), similar patterns of anticipatory ocular responses have been described.

We found the anticipatory responses to differ from those produced by the VOR in two significant ways. First, anticipatory responses are characterized by temporal synchrony with voluntary head movements (~1 msec versus ~7 msec for the VOR). Second, the anticipatory responses have higher gains (0.80 versus 0.46 for the VOR, Shanidze et al. 2010). Although it is obvious that higher gain should produce better compensation, it is less obvious that temporal synchrony might be behaviorally significant. Fig. 7 illustrates the effect of simulating a delay of eye-in-head velocity equal to the average latency of the VOR. The upper panel of Fig. 7a shows a representative segment of a compensated active head movement. The anticipatory eye movement (black trace) was temporally synchronized with the head movement (gray trace, latency = 0 msec) and compensatory eye velocity was linearly related to head velocity (regression slope = -0.97 , Fig. 7b). The black trace in the lower panel of Fig. 7a shows that the guinea pig’s eye-in-space (gaze) velocity was near zero during most of the active head movement segment. However, if the anticipatory eye movement were delayed by the latency of the VOR (~7 msec), then retinal image stability would be significantly worsened as indicated by the larger and more variable gaze velocity (dotted trace, Fig. 7b). During the active head movement, the short delay associated with the VOR is sufficient to disrupt the correlation between eye and head velocities (Fig. 7b, gray data points). Although it is unlikely that the VOR would produce errors of this magnitude, this example suggests that even a small temporal lag during a self-generated rapid head movement has the potential to produce large image slip velocities across the retina. Although a lack of temporal synchrony may only briefly degrade the guinea pig’s already poor visual acuity, more significantly, it may hinder the animal’s ability to detect movement of an object in its environment (Land, 1999). Self-generated movements are purposeful – for example, the guinea pig may shift its gaze toward a sound or odor that might signal the presence of a predator. A heightened ability to detect movement in the environment would be critical at such times.

Anticipatory movements are typically associated with voluntary head movements; however, a similar mechanism could assist the VOR when the head is passively perturbed so as to produce repetitive and predictable head movements. For example, the guinea pig’s head oscillates in response to abrupt acceleration transients with a natural frequency of 12–14 Hz (Shanidze et al. 2010). In this frequency range a 7 msec time lag would be expected to produce up to 36 degrees of phase lag, enough to disrupt image stability unless otherwise compensated. Previous studies of the monkey’s VOR (Huterer & Cullen 2002; Minor et al. 1999; Ramachandran & Lisberger 2005) have shown that VOR responses to periodic stimuli up to 50 Hz exhibit less phase shift than would be predicted by the 7 ms latency of the reflex. In order to account for their results, Ramachandran & Lisberger proposed a model with negative latencies. The

negative latency pathway was suggested as a computational “placeholder” for primary afferent fibers (e.g., Huller & Minor 1995) with sufficient phase leads to account for the required “negative” latency at high frequencies. Alternatively, we propose that the VOR may be assisted by a feed-forward predictive mechanism (see below) that senses vestibular and proprioceptive feedback during periodic (predictable) head motion. Consistent with this idea, we observed synchronous compensatory responses during many data segments with high frequency head oscillations induced by passive perturbations. Such a mechanism would be useful since the head plant has been shown to exhibit instability in many species (humans, Keshner et al. 1995; Peng et al. 1999; cats, Peterson et al. 1981; guinea pig, Shanidze et al. 2010). We suggest that the proposed anticipatory mechanism that stabilizes retinal images during self-generated head movements might also play a role in stabilizing gaze during passive head perturbations that induce head oscillations.

Fig. 8 presents a conceptual model of how this proposed mechanism and the VOR might interact. The model hypothesizes feed-forward control of the head for voluntary movements (Frens & Donchin 2009; McNeilage et al. 2008; Shadmehr et al. 2010; Wolpert & Miall 1996). If an animal is passively perturbed so as to produce an unexpected head movement, the vestibular system will sense that movement and produce compensatory eye movements via the VOR pathways. For self-generated (voluntary) head movements, the feed-forward controller produces the command to move the head. In a feed-forward model, neural circuits that implement an internal model of head/neck plant dynamics and the vestibular sensory apparatus are presumed to exist. These neural circuits generate both a motor command that is appropriate to move the head (but inappropriate to move the eyes) and an estimate of the sensory response. The anticipated sensory estimate of the impending head movement must be correctly scaled and transformed into a vestibular coordinate frame so as to be directly comparable to an actual vestibular sensory signal. One purpose of this signal is to enable the brain to distinguish vestibular sensation resultant from an active head movement from that caused by external perturbations that also produce head movement. In the model this task is accomplished by comparison (at the summing junction) of the sensory estimate to the actual sensory signal. We propose that the difference of these two signals ($H_{est} - H_{ves}$) is used to produce the anticipatory response. If it were added to the vestibular signal in the central VOR pathways, the net input to the VOR would be H_{est} . If the motor controller were accurate and there were no external perturbations, then this estimate would faithfully mimic the actual vestibular sensory inflow and thus would produce an accurate anticipatory eye movement. If either the estimate were in error, or if there were unexpected perturbations of head movement, then this signal could be used centrally to modify the ongoing head movement and the ocular response (Lehnen et al. 2009). The switch labeled “behavioral goal” allows for this possibility; if $H_{est} - H_{ves}$ is not zero, then the best strategy might be to rely more heavily on the actual vestibular inflow rather than the estimate. In a bilaterally lesioned animal, the pathways associated with the “vestibular labyrinth” are destroyed and there is no passive VOR. However, the sensory estimate may be used directly to drive compensatory eye movements such as those shown in Fig. 6a. This model is consistent with our findings and previous studies (e.g., Bizzi et al. 1971; Newlands et al. 2001; Lehnen et al. 2009) but remains hypothetical until tested by future experiments. One aspect of the model merits attention: if the sensory estimate is encoded in vestibular coordinates, then it may be difficult to distinguish it using single unit recordings from an actual sensory response unless the head trajectory is perturbed in an unpredictable manner.

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Fig. 1.

Anticipatory eye movements that preserve retinal image stability occur in temporal synchrony with head movements. **A.** Example of self-generated head movements and eye movement responses. Upper black trace: eye-in-space (gaze) velocity; lower black trace: eye-in-head velocity; lower gray trace: head-in-space velocity. **B.** Portion of record shown in **A** (marked by arrow) with expanded gaze velocity scale. **C.** Waveform cross correlation of the data segment shown in **A**. The anticipatory response latency is the lag (-1 msec) at the maximum correlation. **D.** Linear regression analysis of eye-in-head and head-in-space velocities for the data segment shown in **A**. The regression slope is -0.95

Fig. 2.

Two examples of anticipatory eye movements during self-generated rightward head movements. In both panels the uppermost traces are head (gray) and eye (black) position in space; 2nd panel from top shows eye-in-head position (black); 3rd panel from top shows head (gray) velocity in space and eye (black) velocity in relative to the head; 4th panel from top shows eye velocity in space (black). The arrows indicate the anticipatory eye movement that precedes the rapid eye movement

Fig. 3.

Cross correlation and regression analyses of the data segments illustrated in Fig. 2. **A.** Waveform correlation for the segment indicated by the arrow in Fig. 2a. The latency is -2 msec. **B.** Waveform correlation for the data segment that follows the rapid eye movement in Fig. 2A. The latency is 8 msec. **C.** Waveform correlation for the segment indicated by the arrow in Fig. 2B. The latency is -1 msec. **D.** Regression analysis for the segment indicated by the arrow in Fig. 2A. The regression slope is -0.75. **E.** Linear regression analysis for the segment that follows the rapid eye movement in Fig. 2A. The regression slope is -0.89. **F.** Regression analysis for the segment indicated by the arrow in Fig. 2B. The regression slope is -0.94

Fig. 4.

A. Distribution of eye movement latencies (lags) associated with self-generated head movements. **B.** Distribution of regression slopes (compensatory gain) of eye versus head velocity associated with self-generated head movements. The “normalized count” is the count of items in each bin divided by the total number of counts

Fig. 5.

Two examples of anticipatory eye movements in an animal 4 months after a complete bilateral vestibular lesion. **A.** Passive perturbation followed by an active head movement. **B.** Active head movement. Traces are ordered as in Fig. 2

Fig. 6. Distribution of anticipatory eye movement latencies (**A**) and regression slopes (gain, **B**) in 5 animals recorded 2 weeks after bilateral vestibular lesions. Upper half of each panel shows data from lesioned animals; lower half is control data

Fig. 7.

Temporal synchrony of anticipatory eye movements with head movement improves retinal stability. **A.** Upper panel: head-in-space velocity (gray); eye-in-head velocity (black). Lower panel: eye-in-space (gaze) velocity (black) and simulated eye-in-space if the anticipatory response were delayed 7 msec (dotted trace). **B.** Regression analysis of eye and head velocity. Black dots, actual data; gray dots, delayed data

Fig. 8. Conceptual feed-forward model of proposed anticipatory eye movement mechanism. Details in text