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Vestibuloocular Reflex Dynamics During High-Frequency and High-Acceleration Rotations of the Head on Body in Rhesus Monkey

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Huterer, Marko, and Kathleen E. Cullen. Vestibuloocular reflex dynamics during high-frequency and high-acceleration rotations of the head on body in rhesus monkey. *J Neurophysiol* 88: 13–28, 2002; 10.1152/jn.01034.2001. For frequencies >10 Hz, the vestibuloocular reflex (VOR) has been primarily investigated during passive rotations of the head on the body in humans. These prior studies suggest that eye movements lag head movements, as predicted by a 7-ms delay in the VOR reflex pathways. However, Minor and colleagues recently applied whole-body rotations of frequencies ≤15 Hz in monkeys and found that eye movements were nearly in phase with head motion across all frequencies. The goal of the present study was to determine whether VOR response dynamics actually differ significantly for whole-body versus head-on-body rotations. To address this question, we evaluated the gain and phase of the VOR induced by high-frequency oscillations of the head on the body in monkeys by directly measuring both head and eye movements using the magnetic search coil technique. A torque motor was used to rotate the heads of three Rhesus monkeys over the frequency range 5–25 Hz. Peak head velocity was held constant, first at ±50°/s and then ±100°/s. The VOR was found to be essentially compensatory across all frequencies; gains were near unity (1.1 at 5 Hz vs. 1.2 at 25 Hz), and phase lag increased only slightly with frequency (from 2° at 5 Hz to 11° at 25 Hz, a marked contrast to the 63° lag at 25 Hz predicted by a 7-ms VOR latency). Furthermore, VOR response dynamics were comparable in darkness and when viewing a target and did not vary with peak velocity. Although monkeys offered less resistance to the initial cycles of applied head motion, the gain and phase of the VOR did not vary for early versus late cycles, suggesting that an efference copy of the motor command to the neck musculature did not alter VOR response dynamics. In addition, VOR dynamics were also probed by applying transient head perturbations with much greater accelerations (peak acceleration >15,000°/s^2) than have been previously employed. The VOR latency was between 5 and 6 ms, and mean gain was close to unity for two of the three animals tested. A simple linear model well described the VOR responses elicited by sinusoidal and transient head on body rotations. We conclude that the VOR is compensatory over a wide frequency range in monkeys and has similar response dynamics during passive rotation of the head on body as during passive rotation of the whole body in space.

INTRODUCTION

The vestibuloocular reflex (VOR) acts to provide a compensatory eye rotation of equal and opposite magnitude to a head rotation, thus stabilizing the visual axis (gaze) in space and thereby allowing us to move through space and see at the same time. The VOR has been well characterized in humans and monkeys over the frequency range <10 Hz by studies employing passive rotations of the whole body in space (Bohmer and Henn 1983; Demer et al. 1993; Kasteel-Van Linge and Maas 1990; Keller 1978; Mathog 1972; Peterka et al. 1990), passive rotations of the head on the body (Bohmer and Henn 1983; Das et al. 1995; Hanson and Goebel 1998; Hoshowsky et al. 1994), and actively generated rotations of the head on the body (Demer et al. 1993; Hirvonen et al. 1997; Hoshowsky et al. 1994; O'Leary and Davis 1990; Tomlinson et al. 1980). These studies have shown that the eye movements evoked are essentially compensatory, having a gain (eye velocity/head velocity) near unity, and a minimal phase lag (~0°) with respect to head movement.

During natural behaviors such as running, motion of the head in space can have frequency content approaching 20 Hz (Grossman et al. 1988). However, probing the VOR at such high rotational frequencies has proven to be difficult; experimental assemblies capable of providing sinusoidal whole-body rotations at frequencies in excess of 5–10 Hz are not widely available, and subjects have difficulty in consistently generating active periodic rotations of the head on the body >6 Hz (Hoshowsky et al. 1994; Tomlinson 1980). Accordingly, VOR response dynamics for frequencies >10 Hz have been primarily investigated by using passive, sinusoidal rotations of the head on the body (HOB). To date, these studies have been performed in humans and have shown that, over the frequency range 5–20 Hz, the gain of induced eye movements was generally near unity. However, eye movements were found to increasingly lag head movements as the frequency of head rotation increased. For example, Skavenski et al. (1979) characterized the VOR in response to passive horizontal HOB oscillations from 0.1 to 15 Hz and observed that the phase lag increased with frequency reaching values >30° at 15 Hz. Gauthier et al. (1984) generated vertical HOB rotations over the frequency range of 2–30 Hz and found an even more dramatic increase in phase lag (~90° at 20 Hz). More recently, Tabak and colleagues (Tabak and Collewijn 1994, Tabak et al. 1997) characterized VOR dynamics in response to sinusoidal horizontal HOB rotations ≤20 Hz and found eye movements to increasingly lag head motion (approaching a 45° phase lag at 20 Hz). It has been proposed that this trend between increasing phase lag and frequency reflects the fact that the VOR has a

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finite latency; the latency is determined by the physical constraints of the neural circuitry underlying VOR reflex pathways (i.e., synaptic neural delays, neural conduction delays, as well as muscle activation times). Indeed, the phase lags reported in the most recent of these studies (Tabak et al. 1994, 1997) are consistent with a constant 6- to 7-ms transmission latency through the VOR pathways (i.e., 20 Hz corresponds to a period of 50 ms, such that, 6 ms/50 ms*360°/cycle = a phase lag of 43°).

In contrast to the preceding human studies employing high-frequency rotations of the *head on the body*, Minor and colleagues (1999) have most recently used sinusoidal *whole-body rotations* (WBRs) in monkeys to test the VOR at frequencies from 0.5 to 15 Hz. Surprisingly, they reported eye movements that were nearly fully in phase (~0° lag) with respect to head motion across all frequencies tested. There are three possible explanations for the difference in results in humans versus monkeys. First, rotation of the head on the body in the human studies results in both vestibular *and* cervical (neck proprioceptive) stimulation. Therefore activation of the cervicoocular reflex (COR) could have evoked reflexive eye movements in the human studies and have influenced the response dynamics recorded. However, this is unlikely given that the gain of the COR is negligible in normal alert humans and rhesus monkeys (Barlow and Freedman 1980; Bohmer and Henn 1983; Bronstein and Hood 1986; Jurgens and Mergner 1989; Roy and Cullen 2002; Sawyer et al. 1994). Second, a species-specific difference between the neural pathways underlying the reflex might give rise to the disparity observed. Finally, a technical limitation of human studies is that they require coupling the human's head to the experimental torque assembly via a snug-fitting torque helmet, such that obtaining an accurate measurement of true head-in-space motion is difficult. Gauthier et al. (1984) assumed head rotation to be equal to helmet rotation; however, any relative movement between the helmet and the human's head would result in an incorrect characterization of the VOR. Similarly, Skavenski et al. (1979) and Tabak and colleagues (1994, 1996, 1997) measured head rotation with a search coil mounted within a custom bite bar. Any slip between the bite bar and the teeth would have resulted in an erroneous characterization of the induced VOR, although slippage would have mostly likely shifted the response in the opposite direction to the observed disparity (i.e., a relative phase lead rather than lag). On the other hand, obtaining an accurate measurement of head motion was not a problem in the study of Minor et al. (1999) because a search coil was firmly secured to the monkey's skull.

Our primary goal was to characterize high-frequency VOR response dynamics, in the monkey during HOB rotations, and systematically compare our results with those of previous investigations, which have employed rotations of the whole body in space. Additionally, to resolve a current debate in the literature concerning the minimal latency of the VOR, we characterized VOR dynamics using transient perturbations with much greater accelerations than have been previously employed. Actively generated head movements can have accelerations $\leq 25{,}000°/s^2$ (Armand and Minor 2001). However, initial estimates of VOR latency were made based on the eye movements evoked by low acceleration stimuli ($\leq 750°/s^2$), and values in the range of 12–15 ms were reported (Cullen et al. 1991; Lisberger 1984; Minor and Goldberg 1991). More recent studies in humans and monkeys, which used higher acceleration stimuli ($\leq 3{,}000°/s^2$), have reported a much lower values for the VOR latency in the range of 5–10 ms (Aw et al. 1996; Collewijn and Smeets 2000; Crane and Demer 1998; Minor et al. 1999; Tabak and Collewijn 1994; Tabak et al. 1996, 1997). In these prior studies, VOR response latency was typically measured as the time at which the evoked eye velocity crossed a given threshold value. Accordingly, its value depended on the trajectory of the input (head movement) to the system, such that a larger head acceleration input could result in a reduction in the *apparent* latency (see discussion of Cullen et al. 1996). Thus to determine the minimal response time, we characterized VOR response dynamics in response to greater acceleration stimuli than those that have been used to date ($>15{,}000°/s^2$).

METHODS

Surgical preparation

Four healthy monkeys (2 *Macaca mulatta*, 2 *M. fascicularis*) were prepared for chronic behavioral experiments. All procedures were approved by the McGill University Animal Care Committee and were in strict compliance with the guidelines of the Canadian Council on Animal Care.

The surgical preparation has been previously described elsewhere (Sylvestre and Cullen 1999). Briefly, a dental acrylic implant was attached to each animal's skull using stainless steel screws. Within the implant was embedded a stainless steel post that was used to restrain the animal's head during the experiment and to which the head coil and torque motor could be rigidly coupled. Additionally, an 18- to 19-mm-diam eye coil, consisting of three loops of Teflon-coated stainless steel wire, was implanted in the right eye beneath the conjunctiva (Fuchs and Robinson 1966). After the surgery, the animals were administered buprenorphine (0.01 mg/kg im) for postoperative analgesia and the antibiotic Cephalozin (ANCEF; 25 mg/kg im, for 5 days). Animals were given ≥2 wk to recover from the surgery before experiments began.

Data acquisition

During the experiment, animals were comfortably seated in a primate chair. Gaze and head movements were recorded using the magnetic search coil technique (Fuchs and Robinson 1966) with a 1-m-diam magnetic field coil system (CNC Engineering), We placed the head coil as close as possible to the eye coil to minimize their separation within the magnetic field during the experiments. Therefore the head coil was mounted within a plastic mold designed to place the head coil within 5 cm of the eye coil (Fig. 1). Coils were carefully calibrated in each position to verify that the signals were identical. When the entire head coil mold was rigidly attached to the head implant, the monkey's view was unobstructed such that it could fixate on targets within 30° of primary position. In addition, special care was taken to minimize eye and head coil lead motion by embedding the leads in foam close to the monkey's head. Behavioral paradigms, target motion, torque motor triggering, and data storage were controlled by a QNX-based real-time data-acquisition system (REX) (Hayes et al. 1982). Gaze and head position signals were low-pass filtered at 250 Hz (8 pole Bessel filter), sampled at 1,000 Hz, and stored on a hard drive for later analysis. Eye position was calculated as the difference between recorded gaze and recorded head-position signals.

Behavioral paradigms

Monkeys were allowed complete freedom of movement in the horizontal plane during experiments. Passive horizontal head pertur-

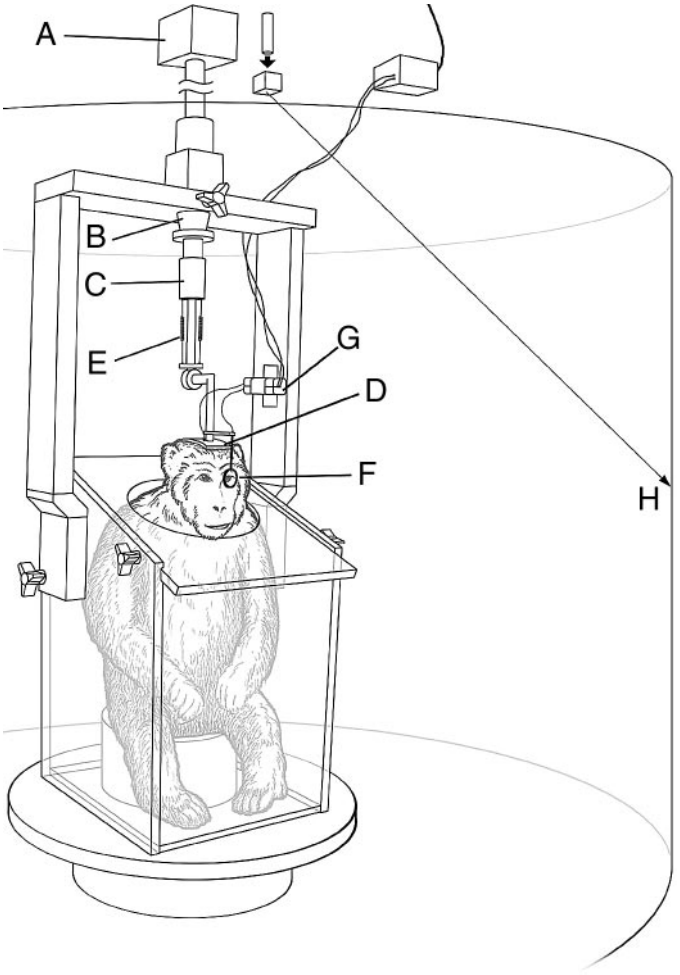


FIG. 1. Experimental setup. Rhesus monkeys were seated in a Plexiglas chair and were allowed complete freedom of head motion in the horizontal plane during experiments. Passive horizontal head perturbations were generated using a torque motor (a) that was attached through precision universal joints (b) and a linear bearing (c) to the post implanted on the monkey's head (d). A spring system offloaded the weight of the apparatus (e). A head coil (f) was mounted within a plastic mold designed to place it as close as possible to the eye coil. Wire motion was minimized by embedding the leads in foam (g). In some experiments (the "laser on" condition), monkeys fixated an earth stationary target (h).

bations were generated using a torque motor (Animatics), which was securely coupled (through precision universal joints) to the post implanted on the monkey's head (Fig. 1, *inset*). Head perturbations were applied during two different viewing conditions: in the laser-on condition, the monkey actively fixated an earth stationary target (HeNe laser projected onto a tangent screen 60 cm from their head, for 400–800 ms before perturbation was triggered); in the laser-off condition, the monkey was in darkness, and no target was present (with head perturbations applied only when eye position was within ±10° of center position).

We used two types of head-velocity stimuli to characterize VOR response dynamics. The first type of head perturbation was sinusoidal rotation of the head in the yaw axis. The head was rotated at frequencies of ~5, 10, 15, 20, 23, and 26 Hz. For the higher frequencies of rotations (23 and 26 Hz), the actual frequency of the resultant head rotation varied by about ±1 Hz, depending on how much resistance the monkey generated in response to the applied motion. The torque applied was adjusted for each animal such that the peak rotational head velocity was relatively constant across all frequencies (±50 and ±100°/s peak velocity). As a result, the peak-to-peak amplitude of the resultant head movements varied 10-fold from 6.4 (5 Hz, 100°/s peak velocity) to 0.61° (26 Hz, 50°/s peak velocity).

The second type of head perturbation used in this study was a transient "bump-like" stimulus. We used two transient stimuli of different durations (30 and 70 ms), which we refer to as the "very short" perturbation and the "short" perturbation, respectively. The very short perturbation had a peak acceleration in excess of 15,000°/s^2, while the short perturbation had a peak acceleration of ~5,000°/s^2. The command to the torque motor was adjusted for each monkey so that peak head velocity was held constant at ~100°/s for each of these transient perturbations. One of the three monkeys studied during passive head oscillations (*monkey T*) was not available for studies with the transient head perturbations. Therefore in addition to *monkeys J* and *C*, a fourth monkey (*monkey G*) was used to characterize the VOR response to short-duration transient stimuli.

Data analysis

To quantify VOR response dynamics, the recorded data were imported into the Matlab (The MathWorks) programming environment for analysis.

Sinusoidal head perturbations

Horizontal gaze, eye, and head-position data obtained during the sinusoidal head oscillations were digitally low-pass filtered using a 51st-order finite-impulse-response (FIR) filter with a Hamming window and cutoff frequency set to 75 Hz ("fir1" function: Matlab Signal Processing Toolbox). Zero-phase forward and reverse digital filtering were employed so that the filtered signal had precisely zero-phase distortion. To differentiate signals, the first-order backward difference was calculated. The gain of the VOR, as well as the dynamic "lag" time of eye motion with respect to head motion, was estimated by least-squares optimization (interior-reflective Newton method) of the equation

$$E'(t) = (\text{gain}) * [H'(t - t_d)] + \text{bias} \quad (1)$$

where $E'(t)$ is eye velocity, $H'(t)$ is head velocity, gain is a constant value, t_d is the dynamic lag time (in ms) of the eye movement with respect to the head movement, and bias is a DC offset, usually minimal. Using the dynamic lag time (t_d), the corresponding phase ($\phi°$) of eye velocity relative to head velocity was calculated ($\phi° = t_d * 360°/T$, where T = 1/frequency of the applied head velocity; negative values indicate a phase lag).

To compare the model's ability to predict eye velocity, the variance-accounted-for (VAF) was determined (Cullen at al. 1996). The VAF was computed as $\{1 - [\text{var}(\text{est} - E')/\text{var}(E')]\}$, where var represents variance, est represents the modeled eye velocity, and E' represents the actual eye velocity. The VAF provides a normalized measure of a model's "goodness of fit." *Equation 1* typically provided VAF values between 0.90 and 1, where a VAF of 1 would indicate a perfect fit to the data. Trials for which the VAF was <0.7 were rare (<2.5% of the trials) and were excluded from the analysis. *Equation 1* was fit to a minimum of three trials at each frequency for each animal to determine how the optimal gain and phase of the VOR varied as a function of frequency. For frequencies of oscillation >10 Hz, 10 consecutive stimulus cycles were applied within each trial. For 5- and 10-Hz oscillations, only five consecutive stimulus cycles were applied within a trial because the monkeys began to resist lower frequency oscillations over the course of longer trials. Additionally, we determined whether the gain and phase of the VOR varied as a function of stimulus cycle at 15 and 26 Hz. For this analysis, the gains and phases for cycles 1, 3, 5, 7, and 9 of the applied head motion were calculated using *Eq. 1*.

Transient head perturbations

Horizontal gaze, eye, and head position data obtained during the transient head perturbations were filtered using a 51st-order FIR filter (see preceding text), with a cutoff frequency of 125 Hz. The frequency content of the transient head perturbations were analyzed using a fast Fourier transform (FFT), using the velocity profiles as described by Harris (1998). The velocity profile was computed by taking the running difference of the position samples for the segment of data beginning at transient onset and ending at transient offset (i.e., velocity began and ended at zero) and padding to 8,192 points.

The torque motor provided reproducible stereotyped head deflections, such that the variability between trials was minimal. Twenty or more saccade-free trials were used to evaluate VOR gain, which was defined as the absolute value of the peak eye velocity divided by that of the peak head velocity. To evaluate the latency of the VOR, two methods were used. In the first method, the mean and SD of the baseline head and eye velocities were calculated over the interval of 40 to 0 ms prior to the perturbation onset. The onset of head and eye movements were defined as the time at which head and eye velocity first exceeded the mean baseline velocity by 4 SDs (4 − SD), respectively. The latency was then determined as the difference between the head and eye motion onset. Note, previous studies in head-restrained subjects have typically used a 3-SD criterion to determine onset latency (Angelaki and McHenry 1999; Crane and Demer 1998; Minor et al. 1999). However, in our study, the head was not restrained and we observed a 4-SD criterion to be more robust. In the second method, the latency was determined, for each individual trial, as the difference between the time of peak head velocity and peak eye velocity. The two methods generally yielded similar latency values. Trials with negative latencies or latencies >30 ms were very infrequent and were excluded from analysis (Crane and Demer 1998).

Statistical analysis

Data are described as means ± SD. A repeated-measures linear regression was used to determine the influence of frequency or cycle number on VOR gain and phase. A Student's *t*-test was used to determine whether the average of two measured parameters differed significantly from each other.

RESULTS

The principal goal of our study was to characterize high-frequency VOR response dynamics induced by perturbations of the head on the body. Recent studies in humans have shown that, during locomotion, rotational head movement can have frequency content ≤20 Hz (Grossman et al. 1988). We first determined whether the voluntarily generated head-on-body movements of our monkeys covered a comparable frequency range. Figure 2*A* illustrates an example of gaze redirection, where the monkey's goal was to redirect his visual axis in space (gaze) using a combined eye and head movement. Figure 2*B* shows an example of gaze stabilization, where the monkey kept his gaze stable in space, despite shaking his head rapidly. Fourier analysis of both head velocity profiles revealed frequency content approaching 20 Hz (Fig. 2, *A* and *B, insets*).

Sinusoidal head rotations

To characterize response dynamics of the VOR for frequencies of head rotation that are much greater than those that are typically studied, we developed a torque motor assembly that enabled us to apply passive rotations of the head on the body over a large frequency range. A schematic of this apparatus was illustrated in the preceding text in Fig. 1. We tested VOR elicited by passive sinusoidal head rotations over a frequency range of 5–26 Hz. Figure 2, *C* and *D* shows gaze-, eye- and head-velocity profiles for three of the six frequencies of head oscillation that were tested within this frequency range (10, 19, and 26 Hz) for *monkeys G* and *T*. In these examples, monkeys viewed earth stationary targets in the light (laser-on condition, see METHODS). Peak head velocity was kept constant at approximately ±50°/s. In this and all following figures, the head-velocity profiles have been inverted to facilitate comparison between head movements and resultant eye movements. For all the frequencies of head rotation illustrated, the eye and inverted head-velocity traces nearly superimposed, demonstrating that the VOR was remarkably compensatory across the entire frequency range tested. This point is further emphasized by the relative stability of the gaze-velocity profile (dotted line = gaze velocity re space) across all frequencies of head rotation.

Bode plots quantifying the frequency response of the VOR during sinusoidal oscillations are shown in Fig. 3 for each of the three monkeys tested. For two of the monkeys tested (*J* and *T*), the gain of evoked eye movements (Fig. 3*A*) typically remained constant across all frequencies (H_0: slope = 0 vs. H_1: slope ≠ 0; $P > 0.15$), having values near unity. The gain of *monkey C*'s VOR was qualitatively similar but increased slightly as a function of frequency; VOR gain was ~1.1 at 5 Hz and approached 1.5 at 26 Hz ($P < 0.001$). For all three animals, the phase of the VOR (Fig. 3*B*) was much more compensatory across the frequencies tested than has been previously described. For example, in marked contrast to the 66° phase lag at 26 Hz that has been reported in previous studies of HOB rotation at high frequencies (Tabak and Collewijn 1994; Tabak et al. 1997) and is predicted by a 7-ms VOR latency (dotted lines in Fig. 3*B*), we found the VOR response lag to be ≤25°. Specifically, for *monkey J*, the phase of the VOR response decreased only slightly with frequency ($P < 0.05$), from ~0° at 5 Hz to a 5° phase lag at 26 Hz, whereas in *monkey T*, the phase of the response decreased with frequency ($P < 0.001$), approaching a 25° lag at 26 Hz. The VOR phase of *monkey C*'s response increased slightly with frequency ($P < 0.05$), from ~0° at 5 Hz to a 5° phase lead at 26 Hz.

With a few exceptions, gains measured in the laser-on viewing condition were not significantly different from those measured in the dark with no-laser target (Fig. 3*A*, compare black and gray curves; Student's *t*-test: $P > 0.05$). The gain for the laser-on condition was only significantly ($P < 0.05$) greater than when the experiments were performed in darkness for rotations of 23 and 26 Hz at ±50°/s for *monkey J* and of 5 and 23 Hz at ±50°/s for *monkey C*. These increased gains occurred primarily at frequencies which were above the range in which visual feedback could have contributed to fixating the earth stationary target (Carl and Gellman 1987; Fuchs 1967; Schwartz and Lisberger 1994; St-Cyr and Fender 1969; Stark et al. 1962) and thus most likely resulted from a decrease in attention during rotation in the dark (Barr et al. 1976). Similarly, phase values did not differ consistently in the laser-on, or laser-off viewing conditions (Fig. 3*B*, compare black and gray curves; $P < 0.05$), with only a few isolated exceptions for *monkey T* (5 and 19 Hz at ±50°/s and 16 Hz at ±100°/s, $P < 0.05$).

VOR response dynamics also did not vary systematically as

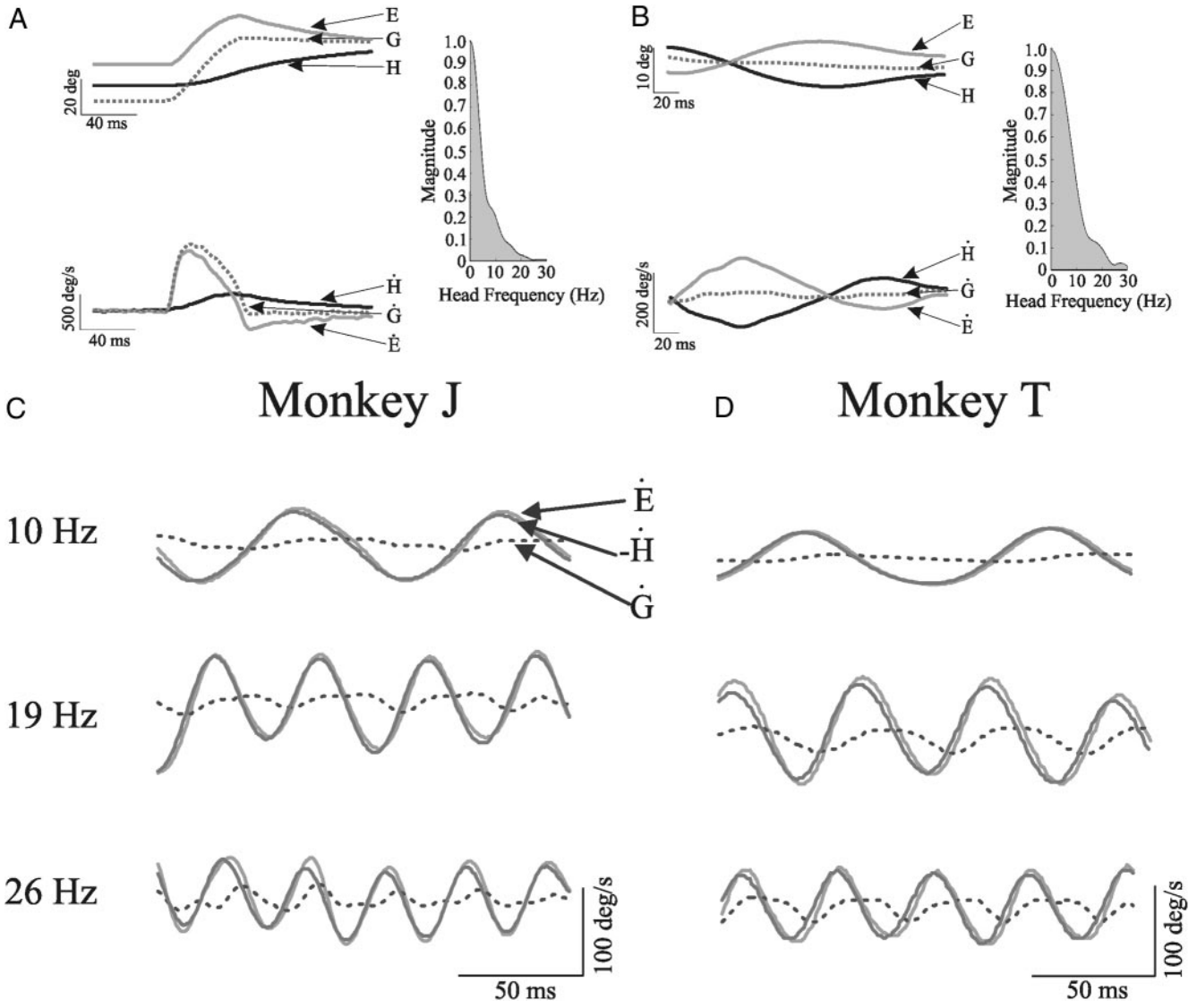


FIG. 2. *A* and *B*: 2 examples of voluntary head motion. The monkey redirected its gaze in space using a combined eye and head movement (*A*). The monkey kept his gaze stable in space, despite shaking his head rapidly (*B*). Fourier analysis of both head-velocity profiles reveals frequency content approaching 20 Hz are illustrated in the *insets. C* and *D*: sample eye (E′), head (H′), and gaze (G′) velocity traces, during sinusoidal rotations of the head-on-body in the dark at 10, 19, and 26 Hz for *monkeys J* (*C*) and *G* (*D*). In this and following figures, the head profile is inverted to facilitate comparison between head movements and induced eye movements.

a function of velocity, when the peak velocity of the head rotations was increased from ±50 to ±100°/s. This result is also illustrated in Fig. 3, where the solid lines (for ±100°/s rotations) superimpose on the dashed lines (for ±50°/s rotations) for both gain and phase plots of all three animals. Statistical analysis confirmed that neither gain nor phase changed significantly as a function of velocity ($P > 0.05$), with the one exception of *monkey T;* this monkey's phase lag was greater for ±100 than ±50°/s rotations for rotational frequencies >20 Hz ($P < 0.05$).

The negligible effects of increasing velocity and visual viewing condition are summarized in Fig. 4, where the averaged data from all three animals are plotted. Overall, mean gains and phases were not affected by viewing condition ($P > 0.05$, compare dark vs. light lines) or doubling peak head velocity ($P > 0.05$, solid vs. dashed lines). In addition for all conditions tested, the average gain and phase increased significantly with frequency ($P < 0.05$). However, the average gain increased only slightly with frequency from 1.1 at 5 Hz to ~1.2 at 25 Hz (Fig. 4*A*) and average phase decreased minimally with frequency, reaching a phase lag of only ~9° at 26 Hz (Fig. 4*B*). Because the VOR response differed more between monkeys for higher frequencies of head rotation (see Fig. 3), the variability of response gain and phase increased with frequency (see the SD bars in Fig. 4, *A* and *B*).

During a single experimental session, *monkey J*'s head was oscillated at peak velocities greater than ±100°/s (±271°/s at 5 Hz, ±233°/s at 10 Hz, ±178°/s at 16 Hz, ±154°/s at 20 Hz, and ±150°/s at 24 Hz). Note that the use of such stimuli was limited to preserve the integrity of the monkey's implant. Although the highest velocity stimuli was more than five times greater than our original test stimulus (±50°/s), the eye movements induced by the VOR were similarly compensatory. Figure 5 quantifies the results obtained during this recording

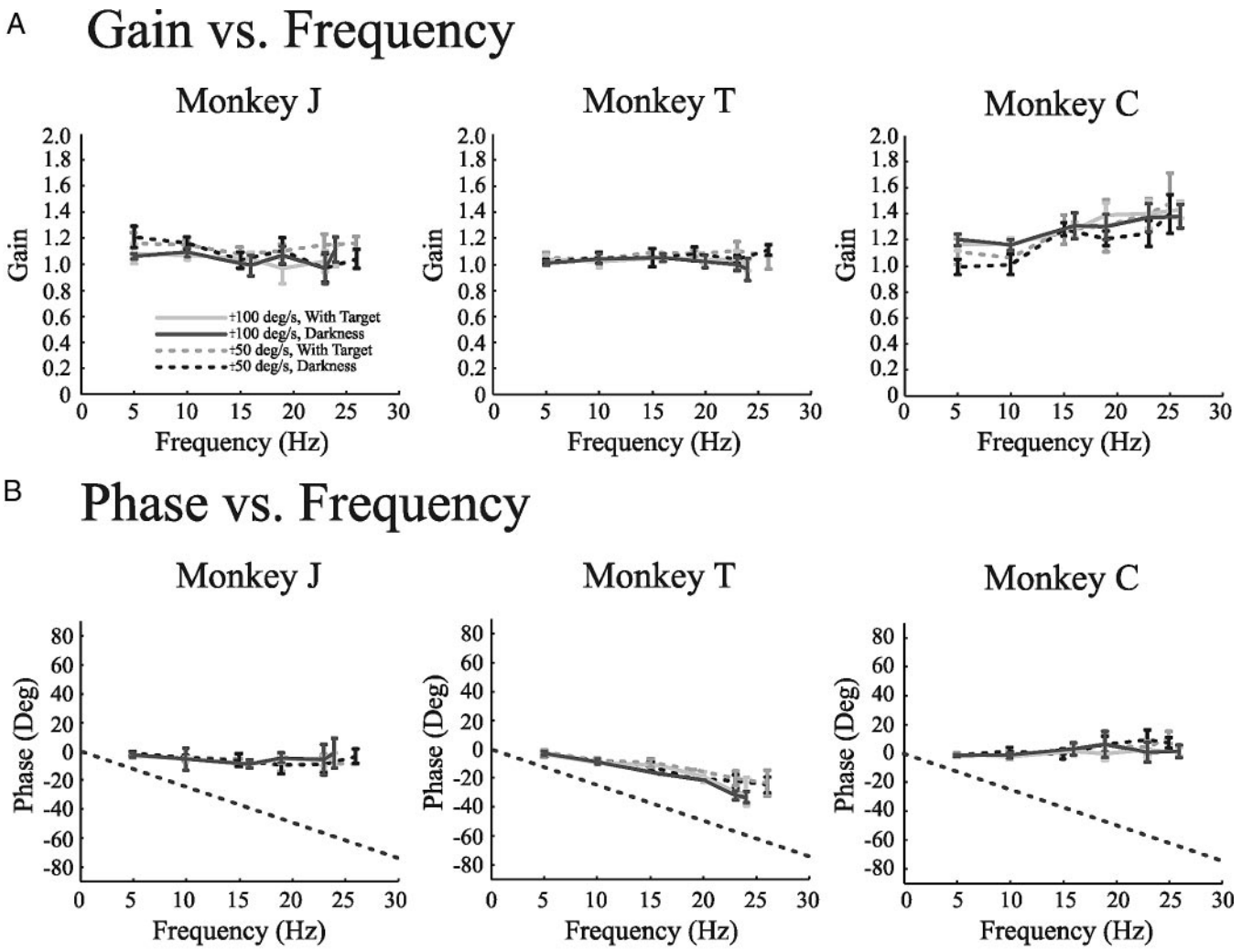


FIG. 3. Gain and phase plots of responses to 5.0- to 26-Hz rotations. *A*: in 2 of the 3 monkeys, gain remained relatively constant with increasing frequency ($P > 0.15$), while in the 3rd monkey, gain increased with frequency ($P < 0.001$). *B*: VOR phase was essentially compensatory across all frequencies tested. In contrast, eye movements would have been expected to increasingly lag head movements (approaching a 65° phase lag at 25 Hz) if based strictly on a VOR pathway latency of 7 ms (see dotted line). Doubling the peak velocity of the oscillation (black vs. gray lines) and/or differences between viewing conditions (solid vs. dashed lines) had no significant effect on the gain and phase of the induced VOR ($P > 0.05$).

session. The response gain at each frequency (Fig. 5*A*, ○, with the peak head velocity at each frequency indicated) is superimposed on the response generated by ±50 and ±100°/s sinusoidal head rotations (—: data taken from Fig. 3 for *monkey J* with SD bars omitted for clarity). The gains for >±100°/s oscillations were compared with the gains induced by ±50 and ±100°/s rotations at each frequency (unpaired *t*-test), and no consistent difference was observed ($P > 0.05$). Likewise, VOR phase did not vary systematically with peak head velocity across all frequencies tested ($P > 0.05$, Fig. 5*B*). Thus VOR gain and phase remained relatively constant at a given frequency for a wide range (from ±50 to approximately ±300°/s) of rotational head velocities.

The torque motor was programmed to deliver a constant torque profile. However, we observed that on the onset of the passive head oscillation, the initial one to two cycles of head rotation generally had greater peak velocities than those that followed. For example, during 16-Hz head oscillations where the motor was programmed to rotate the head at ±50°/s, the mean velocity of the first cycle was >150°/s, while the latter cycles had velocities of ~50°/s. This result indicated that during the first one to two cycles of head rotation, the monkeys offered less resistance to the imposed head motion. To ascertain whether the VOR gain and phase varied with stimulus cycle (and therefore with the amount of resistance imposed by the monkeys), we determined the optimal gain and phase for the first and subsequent cycles of head rotation during ~15- and 23-Hz head oscillations with peak velocities of ±50 and ±100°/s (see METHODS).

Figure 6 shows examples of the head movements that were initially elicited by the torque motor for 16- and 23-Hz head rotations, *top* and *bottom panels*, respectively. Figure 7 illustrates the relationship between gain and cycle number (*A*), and phase and cycle number (*B*) for the three animals tested at these same two frequencies. VOR gain and phase did not vary for early versus later cycles in any animal, regardless of stimulus velocity (compare gray and black lines for ±50 and ±100°/s, respectively; $P > 0.05$) or viewing condition (viewing target in

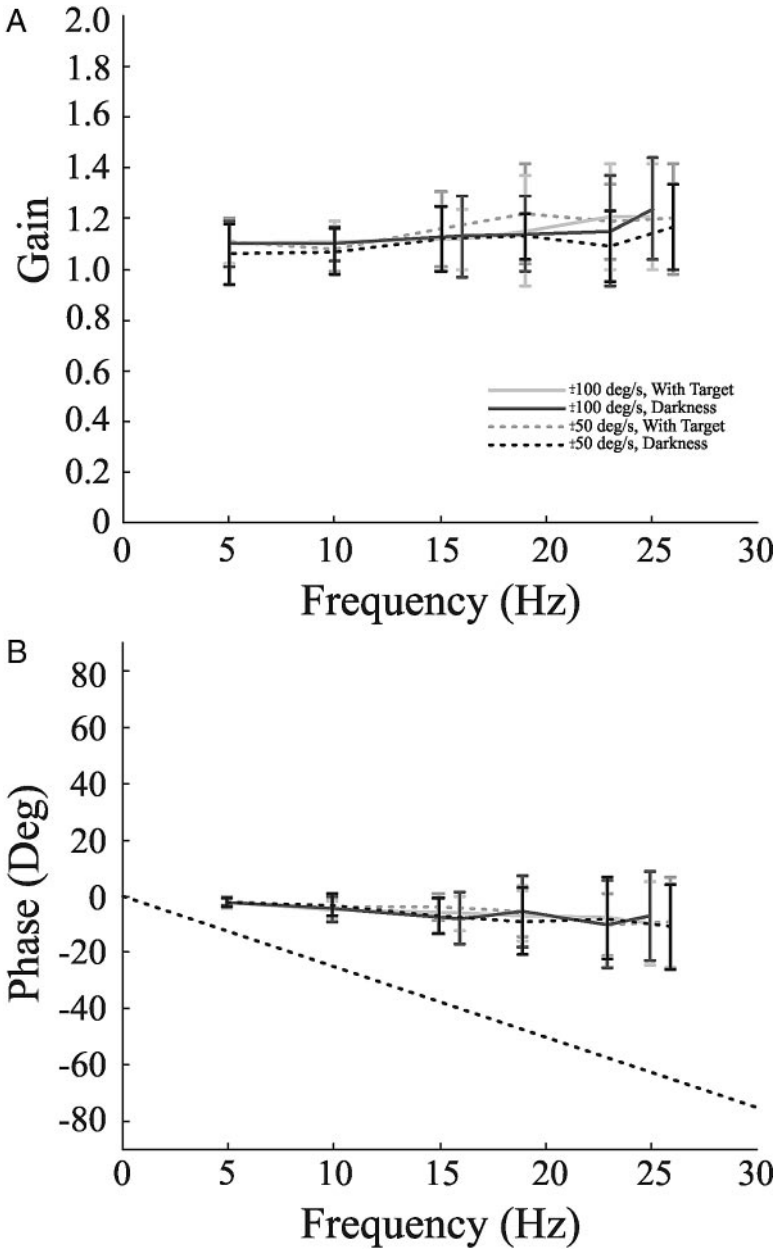


FIG. 4. Gain and phase plots of responses to 5.0- to 26-Hz rotations when data across all 3 monkeys was averaged. Gain (*A*) increased slightly with frequency ($P < 0.05$), and phase (*B*) decreased with frequency, reaching a phase lag of ~9° at 26 Hz ($P < 0.05$). Both gain and phase did not vary consistently with viewing condition or with peak head velocity ($P > 0.05$).

light vs. darkness (not shown); $P > 0.05$). Therefore although there was more resistance to the applied head motion during later cycles of the trial, the gain and phase of the eye movements were not affected. The implications of this finding are addressed in DISCUSSION.

Transient head perturbations

Figure 8 illustrates the two different transient head perturbations, which were used to further characterize the response dynamics of the VOR (labeled "very short" and "short" perturbations). The very short perturbation (Fig. 8*A*, *left*) had a peak acceleration in excess of 15,000°/s^2 and was completed within 20–30 ms. The short perturbation (Fig. 8*B*, *left*) had a peak acceleration of ~5,000°/s^2 and duration of ~70 ms. The torque motor command was adjusted for each monkey so that both perturbations had a peak velocity of ~100°/s.

To determine the frequency content of each perturbation, two methods were used. First, a Fourier analysis was done on the head-velocity profiles. As can be seen in Fig. 8, *A* and *B* (*top right insets*), the very short and short perturbations had frequency content up to ~80 and 40 Hz, respectively. Second, the head-velocity profiles were filtered using a Hamming-windowed 1,000th-order finite response filter with a cutoff frequency (Fc) set to 20, 40, 60, 80, or 100 Hz (Minor et al. 1999). By identifying the Fc at which the filtered head-velocity profile was substantially affected, we obtained a second estimate of the frequency content of these perturbations. The results of this analysis (Fig. 8, *A* and *B*, *bottom right insets*) confirm those of the Fourier analysis and show that the very short and short perturbations demonstrated frequency content approaching 80 and 40 Hz, respectively. For example, because increasing the Fc from 80 to 100 Hz did not appreciably affect the head-velocity profile of the very short perturbation, we

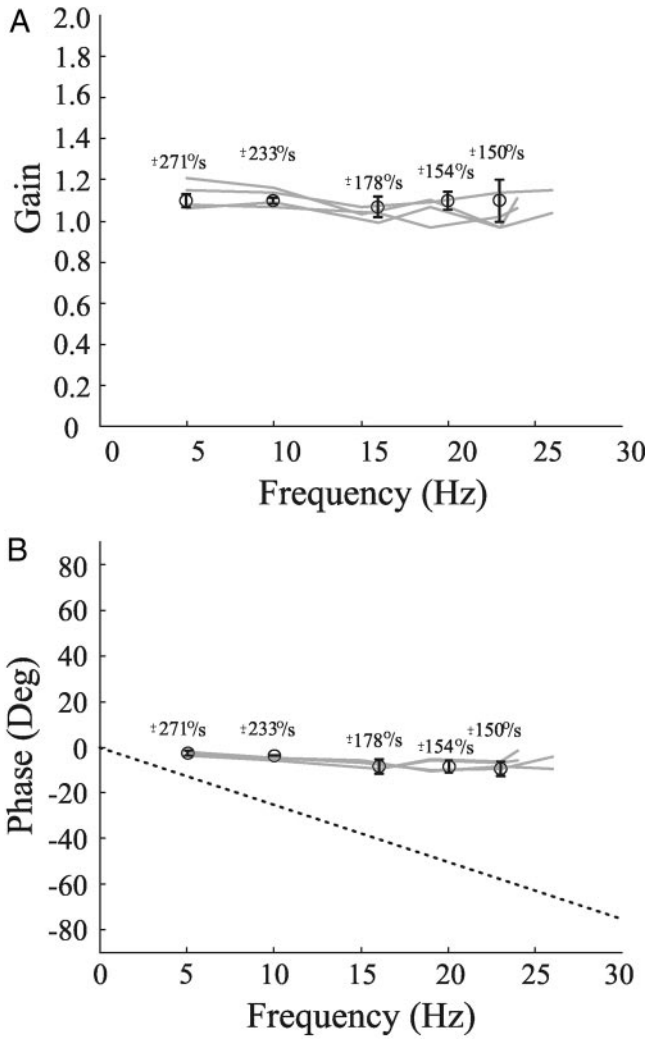


FIG. 5. During 1 recording session, *monkey J*'s head was oscillated at peak velocities of rotation greater than ±100°/s. The gain determined at each frequency (*, with peak velocity at each frequency indicated) during rotation at these higher velocities is superimposed on those induced by ±50 and ±100°/s head rotations (—). Neither gain nor phase varied with peak head velocity for head velocities ±100°/s for the frequencies tested ($P > 0.05$).

concluded that movement has little or no frequency content exceeding 80 Hz. Thus our analysis indicates that both transient perturbations had higher frequency content than did the sinusoidal head-on-body rotations applied in our first series of experiments.

The average gaze-, eye-, and head-velocity trajectories elicited by the very short perturbation are shown in Fig. 9, *left*, for three different monkeys. Each average trace consisted of a minimum of 20 trials. The stereotyped velocity profiles superimposed well on the respective average responses (Fig. 9, *right*), showing that the variability of the head-velocity stimulus as well as that of the elicited eye-movement response was minimal. Following the onset of head rotation, the eyes began to counter rotate after a latency between 5 and 6 ms. In the interval 0–5 ms after the onset of the transient head perturbation, we saw no evidence of an anti-compensatory eye movement (i.e., an eye movement in same direction as the on-going head rotation) in any animal (Fig. 9). Note, this finding is in contrast to that of a recent study, which reported that perturbations of the head produce an anti-compensatory eye movement with zero latency (Collewijn and Smeets 2000). Table 1 summarizes the VOR gain induced by both the very short and short perturbations for all three monkeys. The average gain in response to both perturbations was close to unity for all three animals tested, with the exception of *monkey C*, who, in response to the very short perturbation, had mean gains of 1.6 and 2.0 (for right vs. left perturbations, respectively). These results are consistent with the trends illustrated in Fig. 3 for the two monkeys whose VOR responses to sinusoidal rotation were also characterized: VOR gain remained constant and near unity across frequencies for *monkey J*, whereas VOR gain increased with increasing frequency for *monkey C*.

The VOR latencies to both perturbations were evaluated for each of the three monkeys (see METHODS) and found to be between 4 and 7 ms. These results are summarized in Table 2. Latencies were determined using the SD technique in all three animals with the exception of the VOR latency to the very short perturbation in *monkey G*. In this latter case, the SD method provided unrealistic values of 2–3 ms. Angelaki and McHenry (1999) recently also found that the SD technique can provide physiologically unrealistic latency values; in the analysis of the translational VOR in rhesus monkeys, this method systematically yielded negative latency values for one monkey (i.e., the eye began to move *prior* to the head movement). Accordingly, the latency of *monkey G*'s response to the very short perturbation was estimated using the peak-to-peak method (see METHODS), which provided physiologically realistic latency values. Averaging across animals for each direction of perturbation, the mean latency was 5.23 ± 3.17 ms (rightward), and 5.44 ± 3.71 ms (leftward). Furthermore, there was no significant difference in the latency estimates for the very short and short perturbations in monkeys C and J. Thus our results confirm and complement recent studies, which have reported VOR latencies as short as 5 ms (see DISCUSSION).

DISCUSSION

The purpose of the present study was to characterize the dynamics of the VOR in response to high-frequency head perturbations. The principal findings were that the VOR is remarkably compensatory across a wide range of frequencies and velocities of passive head on the body rotations, the VOR induced by high-frequency passive head-on-body rotations has response dynamics that are comparable to those induced by passive whole-body rotations (Minor et al. 1999), and even when the head is accelerated in excess of 15,000°/s^2, the VOR operates with a minimum fixed latency of 5–6 ms.

Negligible role for neck proprioceptive inputs

Previous characterizations of high-frequency (>10 Hz) VOR dynamics induced by passive head-on-body rotations have been predominantly performed in *humans*. The results of these studies have suggested that eye movements increasingly lag head movements as the frequency of passive head oscillation increases as would be predicted based on a fixed delay (of ~7 ms) being transmitted through reflex pathways [see black dotted line in Fig. 10*B* (Tabak et al. 1994, 1997); gray dotted line: (Gauthier et al. 1984)]. In contrast, Minor and colleagues (1999) have recently shown that passive whole-body rotation,

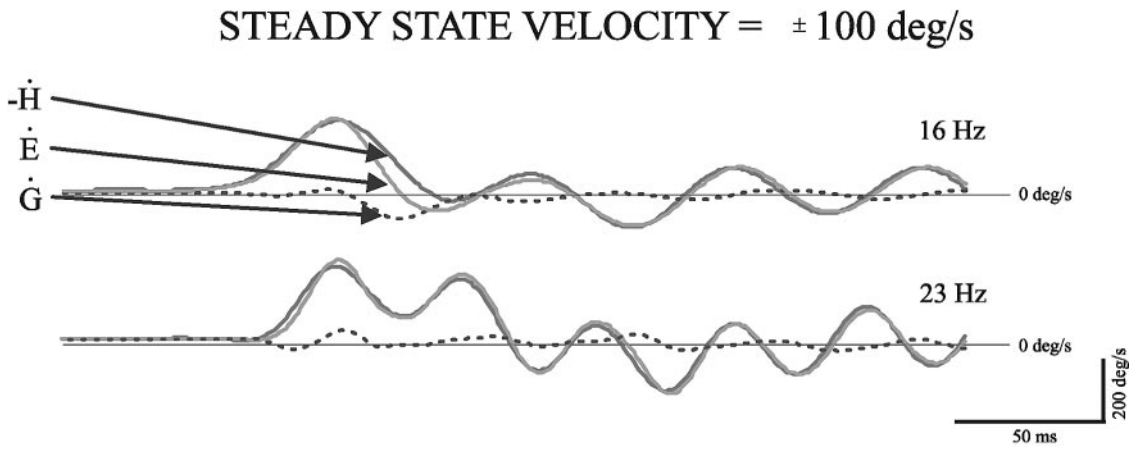


FIG. 6. Example gaze-, eye-, and head-velocity profiles, for 15- and 23-Hz head rotations in *monkey J*. Head and eye velocities were similarly well matched throughout the duration of the trial even though the monkey offered less resistance to head movement during the initial segment of the trial.

in *monkeys*, results in a VOR in which the eye movements are nearly in-phase (~0° lag) with head motion across all frequencies tested (≤15 Hz; light gray line, Fig. 10*B*). A fundamental difference between these experiments is that rotation of the head on the body activates cervical (i.e., neck) proprioceptors as well as the vestibular end organs. Thus a possible explanation for the apparent discrepancy between these findings is that, during passive head-on-body rotations, the resultant eye-movement response is modified by COR pathways, such that eye velocity increasingly lags head velocity for higher frequency head rotations.

In contrast, the results of the present study demonstrate that the VOR has similar response dynamics for rotations of the head on the body and for rotations of the whole body in space. The results of our experiments are largely in agreement with the results of Minor and colleagues; not only was the VOR essentially compensatory across all frequencies tested but also, and importantly, phase lag minimally increased with frequency. Furthermore, the VOR is much more compensatory than would be predicted even using the lower estimate of VOR latency that was obtained in the present study (e.g., a 5 ms would result in a 47° lag at 26 Hz). We conclude that the COR pathways have a negligible influence on VOR response dynamics during high-frequency passive head-on-body rotations in rhesus monkey. One possible explanation for the discrepancy between our data and that of previous human studies is that in the latter, the rotation of a snug-fitting torque helmet or bite bar rather than head rotation was directly measured. Accordingly, any slip between the helmet (or bite bar) and the human's head (or teeth) would have resulted in an erroneous characterization of the VOR dynamics. However, it seems most likely this would have resulted in shifting the phase of the response in the direction opposite to that of the observed discrepancy.

Indeed, our results are consistent with accrued behavioral and neuronal experimental evidence that shows that the COR is functionally insignificant in alert, healthy humans and rhesus monkeys. It operates with a minimal gain (<0.1) at frequencies >0.1 Hz (Barlow and Freedman 1980; Bohmer and Henn 1983; Bronstein and Hood 1986; Jurgens and Mergner 1989; Sawyer et al. 1994; Roy and Cullen 2002). Furthermore, while it has been shown that neck muscle spindle afferents influence the activity of vestibular nuclei neurons in decerebrate animals (Anastasopoulos and Mergner 1982; Boyle and Pompeiano 1981; Wilson 1991), in alert vestibularly intact rhesus monkeys, the activation of neck proprioceptors does not significantly influence the firing patterns of VOR interneurons (Cullen et al. 2001; Roy and Cullen 2002). It is interesting to note that the squirrel monkey may be an exception to this general rule. McCrea and colleagues have recently reported that the COR is far more robust in the alert squirrel monkey (gains approaching 0.3–0.4) (Gdowski and McCrea 2000; McCrea et al. 1999). Since squirrel monkeys have a relatively small oculomotor range (about ±20°) compared with humans and rhesus monkeys (about ±50°), it is conceivable that neck proprioceptive information is processed differently (see Cullen et al. 2001; Roy and Cullen 2001). However, whether the dynamics of the VOR in squirrel monkeys differs during passive rotations of the head on the body versus passive rotations of the whole body in space remains to be determined.

Activation of neck musculature and efference copy

We observed that following the onset of passive horizontal head oscillation, the initial one to two cycles of head rotation had greater peak velocities than those that followed (see Fig. 6). Because the torque motor was programmed to output a constant torque profile, this result indicates that there was less resistance to imposed head motion during the initial cycles of head rotation. Nevertheless, the gain and phase of the VOR were comparable for early versus later cycles regardless of stimulus velocity (±50 vs. ±100°/s) or viewing condition (Fig. 7). Therefore although the monkeys appeared to resist head motion more during the later cycles (>2nd cycle) of head oscillation, there was no evidence that the dynamics of the VOR response were altered by the generation of a neck motor command to resist the applied head motion. This result suggests that the dynamics of the VOR pathways are not modified by an efference copy of the motor command to the neck musculature. This finding complements and confirms that of our recent study of VOR interneuron discharges during active versus passive head movements [i.e., position-vestibular-pause (PVP) neurons] (Roy and Cullen 2002). In this previous study, we showed

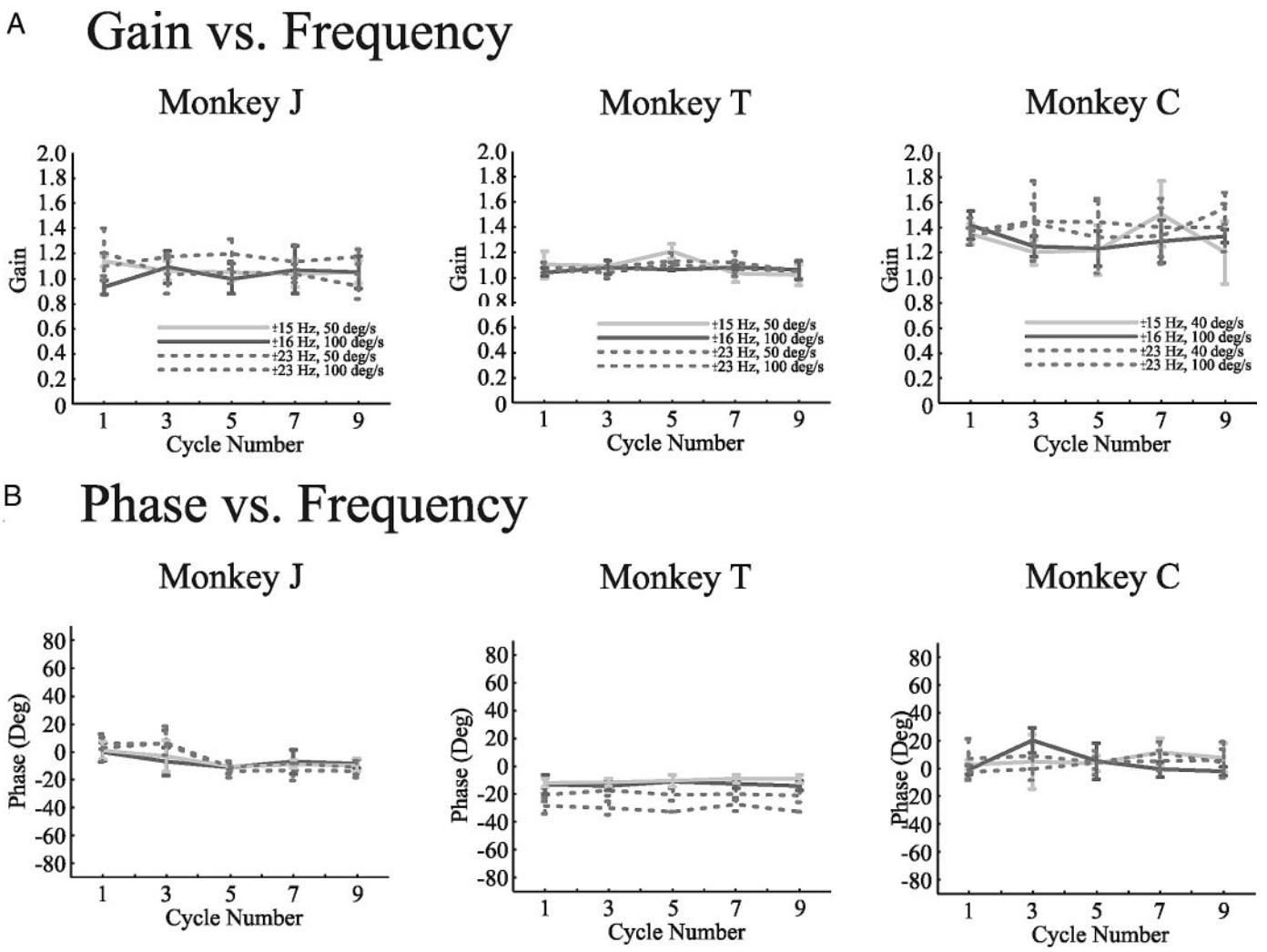


FIG. 7. Gain (*A*) and phase (*B*) plots of responses to 15-and 23-Hz rotations of the head-on-body for *monkeys J, T,* and *C.* The average gain and phase of cycles 1, 3, 5, 7, and 9 was determined for each frequency of head rotation. Gray and black lines show data for rotations of ±50 and ±100°/s, respectively. The gain and phase did not systematically vary for early vs. late cycles in any animal, regardless of stimulus velocity or viewing condition ($P > 0.05$).

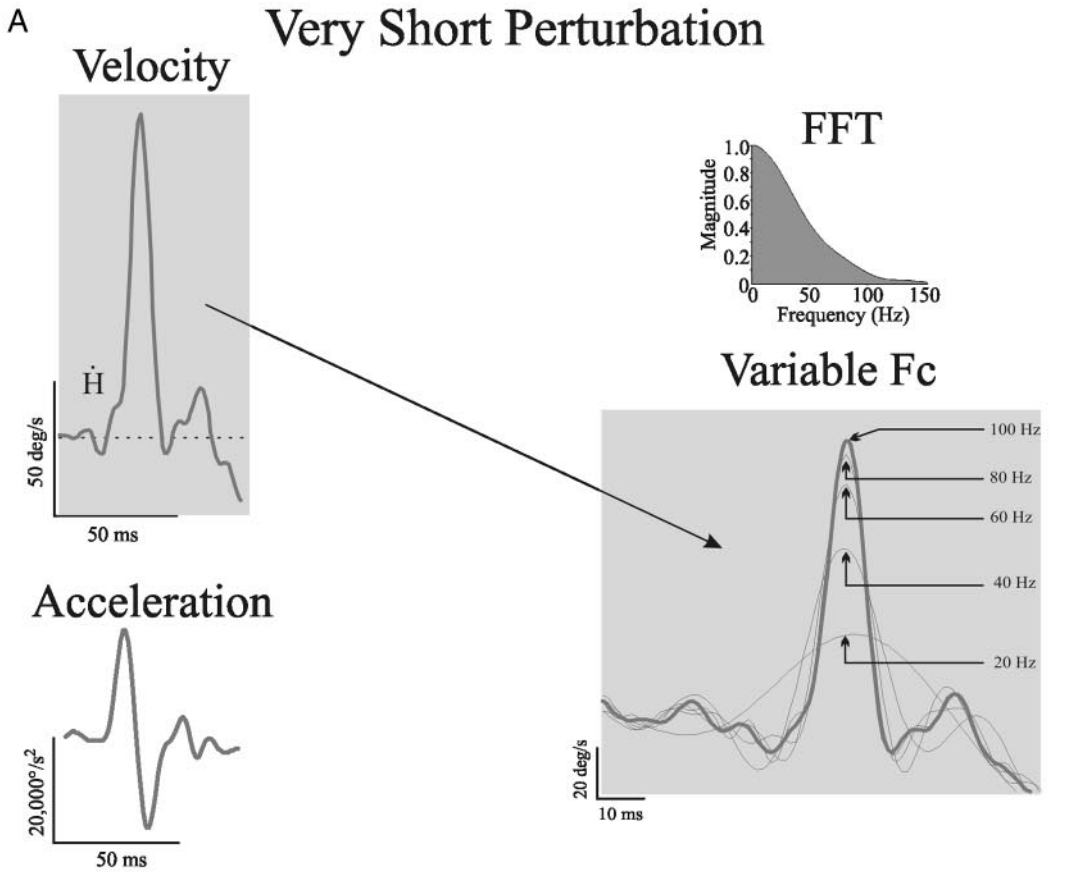
that neurons carry comparable head velocity information during passive rotations and self-generated head-on-body rotations.

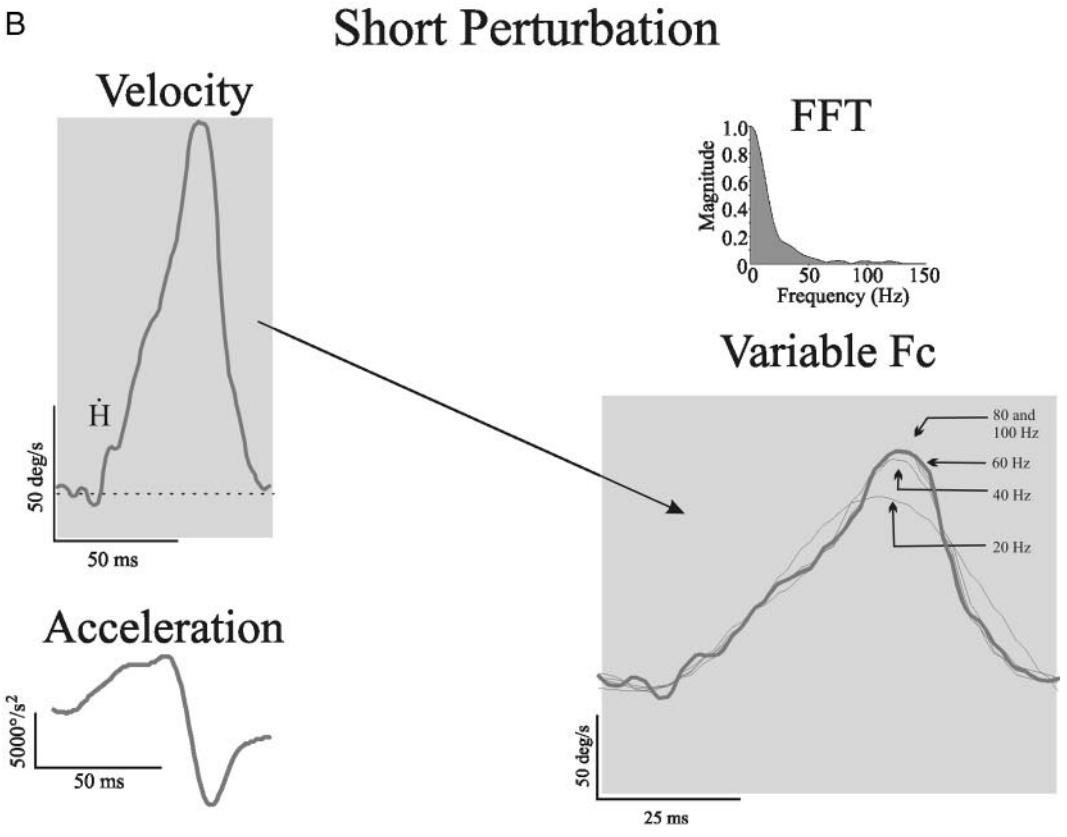
Transient responses: latency and implications

The maximum frequency of sinusoidal head perturbation that our experimental torque assembly was capable of consistently delivering was ≤26 Hz. However, by applying abrupt "bump-like" transient head perturbations, we were able to probe the VOR in response to a stimulation profile that contained frequencies approaching 80 Hz. The two different transient head perturbations that were used in the study had frequency content approaching 80 and 40 Hz, and peak acceleration in excess of 15,000 and 5000°/s^2, respectively (Fig. 8). For the two monkeys whose VOR was tested in response to both transient and sinusoidal stimulation (i.e., *monkeys J* and *C*), the gain of eye movements evoked by the short-duration perturbations qualitatively corresponded to the gains that would be predicted based the VOR response dynamics during sinusoidal stimulation (Fig. 3).

Averaging across animals and perturbations, we measured the latency of the VOR to be ~5–6 ms. Because our estimate is comparable to other recent estimates obtained using stimuli having accelerations more than fivefold lower (<3,000°/s^2) (Collewijn and Smeets 2000; Crane and Demer 1998; Minor et al. 1999; Tabak and Collewijn 1994; Tabak et al. 1996, 1997) we conclude that 5–6 ms accurately reflects the minimal latency of the VOR. This estimate is in accordance with the known mechanical and signal transduction delays that constitute the reflex: the delay from vestibular hair cell stereocilia deflection to afferent spike train initiation was recently estimated to have an irreducible value of ~0.7 ms (Rabbitt et al. 1996), the combined neural transduction and synaptic trans-

FIG. 8. *Left*: head velocity (*top*) and acceleration (*bottom*) profiles induced by the very short (*A*) and short (*B*) perturbations, respectively. *Right*: velocity profiles were subjected to Fourier analysis (*top*) and variable cutoff analysis (*bottom*) to determine at what frequencies the perturbations have significant frequency content.





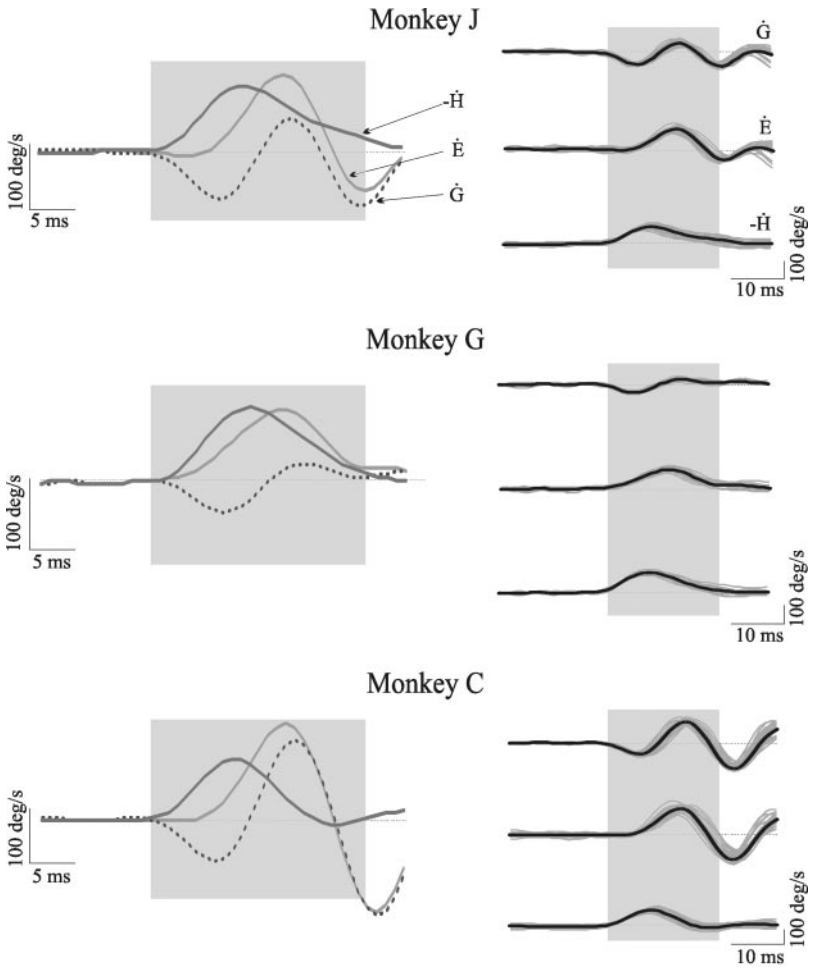


FIG. 9. Gaze-, eye-, and head-velocity responses following very short head perturbations. Each average trace (*left*) consists of a minimum 20 trials (gray traces: *right*). Following the onset of head rotation, the eye begans to counter-rotate after a latency of ~5–6 ms. No anti-compensatory eye movements were ever observed in the interval immediately following onset of head rotation.

mission delay for each leg of the three neuron VOR pathway is between 0.and 1.0 ms (Goldberg et al. 1987), and the latency from shock stimulation of motor neurons in the abducens nucleus to onset of contraction in lateral rectus muscles of the eye plant is 2.8 ms (Fuchs and Luschei 1971); the sum of the component delays is ~5 ms.

In contrast to the recent report by Collewijn and Smeets (2000), we observed no evidence of a short-latency anti-compensatory eye movement in any animal during the interval 0–5 ms after the onset of passive head rotation, regardless of the frequency content of head perturbation. As in our experiments, Collewijn and colleagues imposed passive rotations of the head on the body about a head-centered axis, which results in an eccentric rotation of the eyes. They interpreted the anti-compensatory eye movements as a passive mechanical response to the *linear acceleration* of orbital tissues caused by eccentric rotation of the eye. Although we used accelerations over an order of magnitude greater than those used by Collewijn and colleagues, which would presumably serve to further unmask the presence of such eye movements, we saw no evidence of an early anti-compensatory ocular response to passive head rotation in any of our animals. Instead our results are consistent with previous studies of transient head perturbations in which early anti-compensatory responses were not observed (rotational VOR: Halmagyi et al. 1990;

TABLE 1. *VOR gains*

	Very Short Perturbation	Short Perturbation
Monkey J		
Right	1.05 ± 0.07	1.06 ± 0.08
Left	1.17 ± 0.15	1.06 ± 0.08
Monkey G		
Right	0.88 ± 0.09	1.04 ± 0.04
Left	0.98 ± 0.10	1.13 ± 0.14
Monkey C		
Right	1.64 ± 0.25	1.05 ± 0.05
Left	1.98 ± 0.36	1.23 ± 0.14

Values are means ± SD. VOR, vestibuloocular reflex.

TABLE 2. *VOR latencies*

	Very Short Perturbation	Short Perturbation
Monkey J		
Right	5.10 ± 0.96	6.49 ± 4.51
Left	5.74 ± 1.00	6.36 ± 4.67
Monkey G		
Right	5.72 ± 0.80	4.16 ± 3.39
Left	5.65 ± 0.78	3.89 ± 2.88
Monkey C		
Right	5.59 ± 0.95	4.11 ± 3.64
Left	5.22 ± 0.90	5.22 ± 4.72

Values are means ± SD in milliseconds.

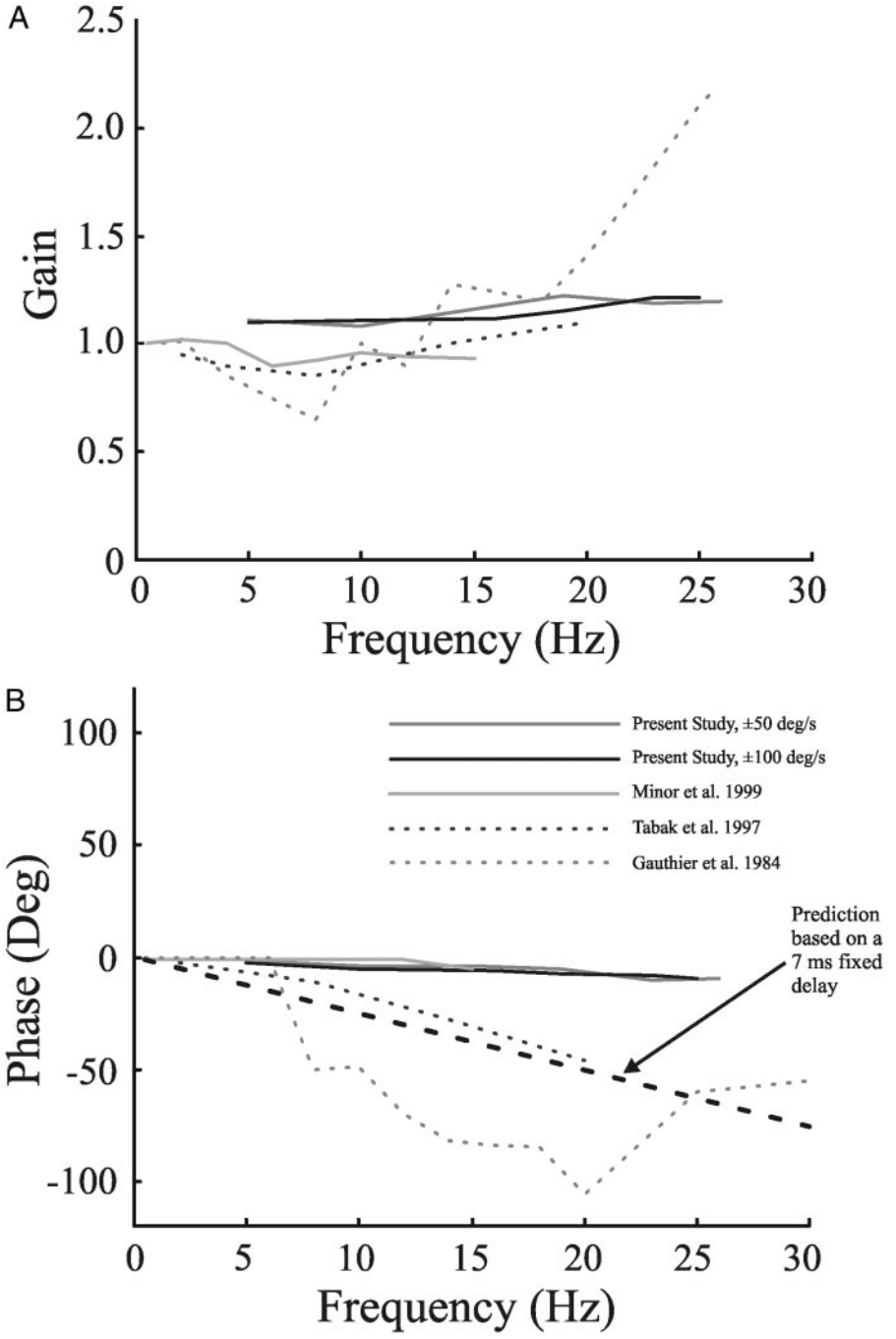


FIG. 10. Comparison of the gain (*A*) and phase (*B*) data obtained in the present study with those obtained in other studies of high-frequency VOR dynamics. Note that in contrast to previous experiments in humans, we observed a minimal phase lag throughout all frequencies tested during head-on-body rotations. In contrast, our data are in excellent agreement with the results of recent experiments performed using whole-body rotations in monkeys (Minor et al. 1999).

Minor et al. 1999; translational VOR: Angelaki and McHenry 1999).

The VOR has a finite latency that is determined by the physical constraints of the neural circuitry underlying VOR reflex pathways (i.e., synaptic neural delays, neural conduction delays, as well as muscle activation times). Thus the question arises: how does the neural circuitry that mediates the VOR compensate for the phase lag predicted by the fixed latency of the system? Recent work by Minor and colleagues suggests that the responses of vestibular afferents may compensate, at least in part, for the predicted phase lag. In particular, the phase lead of regular afferents appears to be appropriate for compensating for most of the phase lag that would be expected from a 5-ms response latency (Hullar and Minor 1999). For example, at 20 Hz, a 5-ms latency would produce a 36° lag if there was no compensatory phase lead in the system. However, the responses of regular afferents lead head velocity on average by 30° at this frequency.

Linearity of the VOR

Unlike in humans, where eye movements evoked by the VOR have been shown to display a saturating nonlinearity at velocities >350°/s (Pulaski et al. 1981), the VOR in monkeys has generally been assumed to be linear over a wide range of

head velocities. Using whole-body rotations in squirrel monkeys, Paige (1983) varied peak head velocity over the range 40–360°/s at 0.2 Hz and reported that gain and phase of the induced VOR was essentially constant across all velocities tested. This was furthered by Tomlinson (1990), who evaluated the eye velocities induced by quasi-sinusoidal whole body rotation (frequency, ~1.5 Hz) with velocities varied from 20 to 850°/s. Using linear regression analysis, he determined that the gain of the VOR was constant and near unity (0.9) for all velocities of head rotation. Recent investigations by Minor and colleagues have revealed two pathways involved in control of the reflex in squirrel monkey (Clendaniel et al. 2001; Lasker et al. 1999, 2000; Minor et al. 1999). Using high-frequency (0.5–15 Hz) whole-body rotations with velocities from 20 to 100°/s and steps of acceleration in darkness (3,000°/s^2 reaching a velocity of 150°/s), they described a linear pathway that has a constant gain and phase lead across higher frequencies and a nonlinear pathway whose gain increased with velocity at higher frequencies. Experimental limitations prevented them from testing their entire frequency series at higher velocities; nevertheless the response of the latter pathway increased with peak rotational velocity for frequencies >4 Hz. With respect to the current study, it is important to note that in the normal squirrel monkey, the nonlinear pathway makes only a small (10–15%) contribution to the overall VOR response. The influence of this pathway, however, increases significantly following adaptation to unilateral vestibular damage or motor learning magnifying spectacles, suggesting it plays an important role in VOR adaptation (Lasker et al. 1999, 2000).

In contrast to experiments in which the whole body is rotated in space, imposing passive rotations of the head on the body does not necessitate moving the combined mass of the entire monkey as well as an appreciable portion of the experimental superstructure. Consequently, by passively rotating only the head on the body, we were able to impose high velocities of head rotation (velocities from ±50 to approaching ±300°/s) across a wide range of rotational frequencies (from 5 to 26 Hz). In all three animals tested, doubling peak head velocity from ±50 to ±100°/s had no consistent effect on the gain and phase of the VOR across all six frequencies of head rotation ($P >$ 0.05, see Fig. 4). Remarkably, even when peak head velocity was increased to values approaching ±300°/s (in *monkey J*, see Fig. 5), the gain and phase of the VOR did not differ from that evoked by ±50°/s head oscillations, regardless of the frequency of head rotation. Thus in contrast to the findings of Minor and colleagues in *squirrel monkey*, our results suggest that *rhesus monkeys* display negligible velocity dependent nonlinearities in VOR gain and phase during ±50–±300°/s head oscillations over a wide frequency range (5–26 Hz).

Modeling the VOR response to head perturbations

To more formally characterize the dynamics of the VOR, we determined the simplest (lowest order) model that could fit the data obtained using sinusoidal head rotations and accurately predict the eye-movement response obtained when the head was perturbed by both the short and very short head perturbations. For this analysis, we took advantage of data from the two monkeys whose VORs was characterized in response to both sinusoidal and transient head perturbations.

A simple model relating eye velocity to head velocity that contained only a fixed latency and a gain term poorly predicted (VAF <0.2) the eye movements evoked by the transient head perturbations used in this study—as expected based on the frequency dependence of VOR gain and phase illustrated by the bode plots in Fig. 3. Consequently, we systematically increased the complexity of the model simulations to obtain the lowest order transfer function which would predict the VOR response that was evoked by both types of passive head perturbation used in this study. We began by attempting to derive a transfer function with a single zero and no poles. For each animal, transfer function coefficients were estimated by using a least squares fit to the VOR Bode plots (Fig. 3). A value of 5.5 ms was assumed to be the transduction latency of the VOR reflex pathways, and accordingly the model included a fixed delay set to this value.

For each monkey tested, we found that a transfer function with a single zero and no poles, derived to fit the sinusoidal head rotation frequency response data, well predicted the VOR response to both transient head perturbations used in this study [*monkey C*: $(0.0068s + 1)/1$, *monkey J*: $(0.0048s + 1)/1$, where s is the Laplace operator]. Figure 11*A* shows the Bode plots of the derived transfer function superimposed on the data obtained from *monkey C* (see Fig. 3). For both the actual and simulated responses, the gain characteristically increased with frequency (from ~1.1 at 5 Hz to ~1.4 at 26 Hz), and the phase increasingly lagged head velocity. Figure 11*B* compares the actual VOR response to head perturbation (*left*) to the modeled VOR response to head perturbation (*right*). The similarity between the actual and predicted eye velocity traces is striking, and indeed quantification of the model's "goodness of fit" for the two transient perturbations generally yielded VAF values >0.7 and frequently approaching 0.95 in both animals. Further increasing the transfer function order generally did not substantially improve the VAF of the simulated response to transient perturbations.

The results of our model simulations confirm that the VOR is linear over the entire frequency range of head velocities and frequencies that were encompassed by the stimuli used in this study. Thus the dynamics of the horizontal VOR in the rhesus monkey are similar to those of the linear pathway recently described by Minor et al. (1999). However, we did not observe an increase in gain with velocity at higher frequencies of rotation, which would be compatible with the nonlinear pathway described by these investigators, although it is possible that subtle nonlinearities might exist for velocities of rotation below those tested in the present study (<50°/s). Hence, we conclude that the VOR in rhesus monkeys does not display the same frequency and velocity-dependent nonlinearities as do squirrel monkeys. Because squirrel monkeys are New World monkeys, which phylogenetically diverged from Old World monkeys (e.g., rhesus) ~35 million years ago, it is conceivable that these two species have evolved subtle differences in their response dynamics. Indeed the results of a recent study suggest that human VOR response dynamics during high-acceleration head-on-body rotations are more similar to those reported for rhesus monkeys in the present study than squirrel monkey (Della Santina et al. 2001). In Old Word monkeys (i.e., human and rhesus monkey), the contribution of the nonlinear pathway may only become significant following adaptation to unilateral vestibular damage or motor learning magnifying spectacles.

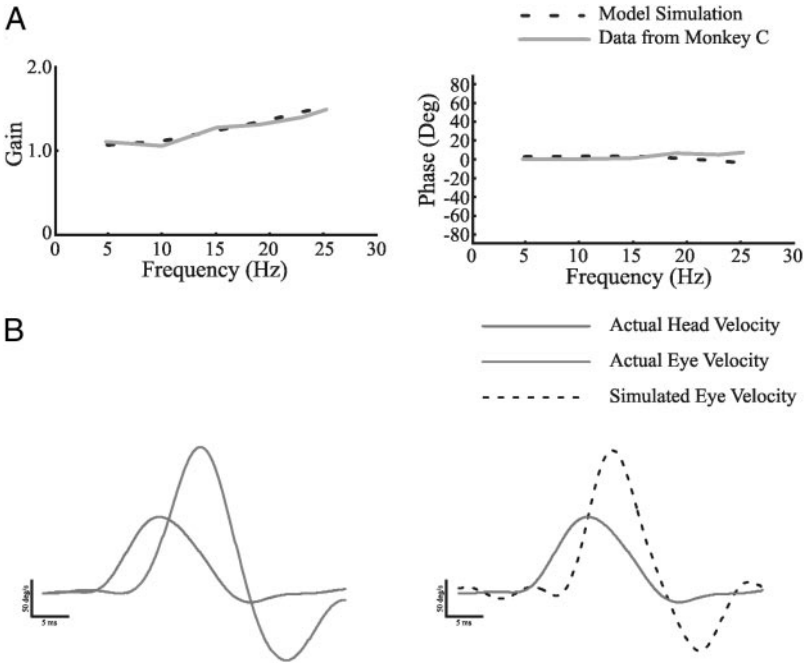


FIG. 11. For both *monkeys C* and *J*, a low-order transfer function with a single 0 and no poles, well predicted the VOR response to the head perturbations used in this study. *A*: Bode plots illustrate the frequency response dynamics of the derived transfer function used in the simulation. Note that plots derived from simulations superimpose on experimental data. *B*: simulated response to higher frequency content perturbation very similar to the actual average eye-velocity profile induced by the perturbation.

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