0270-6474/83/0306-1234$02.00/0

Printed in U.S.A.

The Journal of Neuroscience
Vol. 3, No. 6, pp. 1234–1244
June 1983

FREQUENCY-SELECTIVE ADAPTATION: EVIDENCE FOR CHANNELS IN THE VESTIBULO-OCULAR REFLEX?[1]

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Received February 23, 1982; Revised January 21, 1983; Accepted January 24, 1983

Abstract

The vestibulo-ocular reflex (VOR) is under long-term adaptive regulation to minimize retinal image slip during head movement; normally this process keeps VOR gain (eye velocity divided by head velocity) near 1.0. It has been common to think of the adaptive mechanism as a single pure gain element, although some properties of the system (e.g., frequency-selective changes in the gain of the VOR) argue that it must be more complex. We now report new observations on the frequency selectivity of the adaptive mechanism. Our data suggest a new model in which the VOR operates as a series of parallel, temporal frequency channels, each of which has an independently adjustable gain element.

Adaptive changes were produced by oscillating monkeys sinusoidally at a single temporal frequency (0.2 or 2.0 Hz) in visual conditions that cause either increases (toward two) or decreases (toward zero) in VOR gain. When tested in darkness at the adapting frequency, the VOR showed large changes in gain and little or no change in phase. When tested at frequencies other than the adapting frequency, the VOR showed less pronounced changes in gain and unexpected changes in phase. The phase changes were orderly but depended in a complex way on adapting frequency, testing frequency, and VOR gain.

We have tested the channels concept by calculating the response properties of a mathematical model that processed its inputs in parallel pathways. The model reproduced our data when we assumed that the vestibular primary afferents were distributed in an orderly way to parallel brain channels that had differing dynamics: vestibular inputs with more phase lead projected to higher frequency channels, which themselves had faster dynamics than their low frequency counterparts. Such an organization, when regulated by an adaptive controller that can selectively alter the gain of one channel, could play a key role in establishing and maintaining the frequency-independent performance seen in the adult VOR.

Images must remain relatively stable on the retina if visual processing is to occur effectively. Of the perturbations that might cause images to slip, head movements are the most common; if the eyes remained fixed within the orbit, then each head turn would be associated with rapid slip of retinal images and vision would be poor. This problem is minimized by the vestibulo-ocular reflex (VOR), which operates in light and dark and generates short latency, smooth, compensatory eye movements that are opposite in direction and roughly equal in amplitude to head movement. To maintain appropriate performance and minimize image slip throughout life, the VOR is subject to long-term adaptive regulation by visual inputs (for reviews, Ito, 1975; Melvill Jones, 1977; Collewijn, 1979; Miles and Lisberger, 1981). The existence of an adaptive mechanism has been demonstrated by devising optical means to disturb the normal relationship between visual and vestibular stimuli. In the adapting conditions, each head turn is associated with excessive retinal image slip; depending on the directional relation-

[1] We wish to thank E. V. Evarts for his continued support and encouragement, D. S. Zee for allowing us to use two of his monkeys, and V. Perry and E. Neville for secretarial assistance. We are also grateful to D. A. Robinson for pointing out the "lumped" action of the extraocular motor units on the eyeball. This research was supported in part by National Institute of Health Grant EY03878. S. G. L. is also supported by a fellowship from the Alfred P. Sloan Foundation and a Scholars Award from the McKnight Foundation.

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ship of the visual and vestibular inputs, the adaptive mechanism gradually increases or decreases the amplitude ("gain") of the VOR so that slip is gradually reduced to normal (i.e., minimal) levels.

The mechanisms underlying adaptive gain control in the VOR have usually been represented as a single pure gain element (Ito, 1972; Davies and Melvill Jones, 1976; Lisberger et al., 1981); there have been no overriding reasons for suggesting more complex models. However, we now report observations that could not be reproduced by such minimal models of the adaptive mechanism. When the vestibular input for adaptation was provided by passive, sinusoidal vestibular stimulation, there were frequency-selective effects. First, the gain of the VOR showed larger changes when tested at the frequency used for adaptation than when tested at adjacent frequencies. Second, the phase of the VOR showed changes that depended in a complex way on VOR gain, adapting frequency and testing frequency. Although others (Robinson, 1976; Green and Wallman, 1978; Collewijn and Grootendorst, 1979; Schairer and Bennett, 1981) have mentioned frequency-selective effects on VOR gain, no one has conducted a systematic investigation of the phenomenon. In addition, except for Collewijn and Grootendorst (1979), who suggested that the VOR may utilize some kind of pattern generator to effect adaptive changes, no one has attempted to interpret the frequency-selective effects. Our observations, especially those on changes in the phase of the VOR, lead us to suggest a model that can account for our data by invoking a new structure, but not new functions for the brain pathways that mediate the VOR.

Materials and Methods

Experiments were performed on four rhesus monkeys that had been trained to perform a visual fixation task (after Wurtz, 1969) to obtain water reinforcement. Under Nembutal anesthesia, the animals underwent surgical procedures that have been described elsewhere. Briefly, bolts were implanted in the skull to provide a firm base for head restraint and to serve as a mount for spectacles (Miles and Eighmy, 1980). A coil of wire was implanted on the right eyeball so that we could monitor eye movement with the magnetic search coil technique (Fuchs and Robinson, 1966; Judge et al., 1980).

During experiments, the monkey was seated in a primate chair and its head was secured. A single pair of horizontal field coils was mounted on the chair so that the eye carrying the search coil was centered between them. The output from the search coil was then a sinusoidal current that could be converted electronically to a DC voltage proportional to horizontal eye position within the head. The output from the coil electronics was calibrated by rewarding the monkey for fixating red light-emitting diodes placed at known positions along the horizontal meridian. Unfortunately, the configuration of the primate chair made it impossible to situate a second pair of field coils in an appropriate position to measure vertical eye position. Vestibular stimulation was provided by a servo-controlled DC torque motor that oscillated the chair sinusoidally about the vertical axis at frequencies ranging from 0.1 to 4.0 Hz. The monkey's head was positioned in the stereotaxic plane in order to study the horizontal VOR.

Adapting procedures. To obtain adaptive changes in the VOR, we fitted the monkey with optical devices that have been described elsewhere (Miles and Eighmy, 1980). Briefly, increases in the gain of the VOR were produced by having the monkey view the stationary surroundings through × 2 telescopic spectacles; decreases were obtained by having him wear goggles providing a visual scene that remained fixed with respect to his head (× 0 goggles). In one experiment reported here, the monkey's head was unrestrained so that the vestibular stimulation for adaptation resulted from spontaneous, active head turns. In the remainder of the present experiments, the monkey's head was restrained, and the vestibular stimulation for adaptation was provided by passive, whole body oscillation at a single sinusoidal frequency (either 0.2 Hz, ±20° or 2.0 Hz, ±2°). Throughout adaptation, which lasted 1 day at 2.0 Hz and 3 days at 0.2 Hz, eye movements were monitored on a chart recorder running at very slow speed. Such records have a specific and stereotyped pattern when the monkey is awake and viewing the visual stimulus and were used to estimate the actual duration of exposure to the desired visual-vestibular interaction; these values are given, when appropriate, in the figure legends. Because some of these experiments lasted several days, care was taken to insure that the monkeys were seated comfortably. Throughout their time in the chair, the monkeys were offered fruit, chow, and water at regular intervals, and they ate and drank well.

VOR testing procedures. VOR performance was tested during passive sinusoidal oscillation in total darkness with the monkey's head secured to the chair and the spectacles removed. At the beginning and end of each experiment, tests were run over a wide frequency range that always included 0.1, 0.2, 1.0, 2.0, and 4.0 Hz. In addition, the time course of adaptation was determined for two or three frequencies by testing VOR performance at regular intervals during adaptation. At each frequency, the amplitude of oscillation was adjusted so that the peak-to-peak velocity of the stimulus was 50°/sec. Voltages proportional to eye and chair velocity were obtained by electronic differentiation, and, along with voltages proportional to eye and head position, were monitored on a chart recorder. Measures of VOR performance were made by hand from 20 consecutive stimulus cycles at 0.1, 0.2, and 0.4 Hz and from 20 consecutive saccade-free cycles at 1.0, 2.0, and 4.0 Hz. The gain of the VOR was estimated as average peak-to-peak eye velocity divided by peak-to-peak head velocity. The phase of the VOR was estimated by averaging the time difference between the zero crossings of the eye and chair velocity records. Because the averages included 10 zero crossings in each direction, any bias due to slow drift in one direction did not influence the measure of phase. The VOR was defined as having a phase of zero when eye velocity was 180° out of phase with head velocity. Unfortunately, the analysis of phase was an afterthought; data were available only for two monkeys, and then only at testing frequencies for which we routinely ran the chart recorder fast enough to allow good temporal resolution (0.1, 1.0, and 4.0 Hz). Resolution was good enough so that the

standard deviations in Figures 3 and 4 represent biological noise and not measurement uncertainty.

Results

Adaptation with free head. When the vestibular stimulation for adaptation results from active head turns, changes in VOR gain are independent of the sinusoidal testing frequency over the range 0.1 to 1.0 Hz (Miles and Eighmy, 1980). Figure 1 (*top*) extends these observations to show that the changes are frequency independent over the entire range of testing frequencies used in this paper, 0.1 to 4.0 Hz. The three curves in the *upper panel* show VOR gain in darkness as a function of stimulus frequency (*a*) before the monkey had experienced any of our adaptation procedures, (*b*) after 5 days of free-head adaptation to × 2 spectacles, and (*c*) after 5 days of free-head adaptation to the × 0 goggles. All three curves show a tendency for VOR gain to be higher at 4.0 Hz than at other frequencies (cf. Keller, 1978) but no evidence of frequency-selective changes.

Contrary to previous work using free-head adaptation, we did find some small changes in the phase of the VOR. Normally, the VOR showed a phase of zero at 0.1 and 1.0 Hz and approximately 5° of phase lag at 4.0 Hz (Figs. 3 and 4). After adaptation to × 0 goggles (*lower panel* of Fig. 1, *circles*), the VOR showed phase lag at 0.1 Hz and increasing phase lead at the higher test frequencies. After adaptation to × 2 spectacles (*triangles*), the VOR showed virtually no phase shift at low test frequencies but more than 10° of phase lag at 4.0 Hz. Figure 6 will show that these small changes would be expected if the monkey's active head turns provided him with somewhat more experience near 1.0 Hz than near 0.1 or 4.0 Hz.

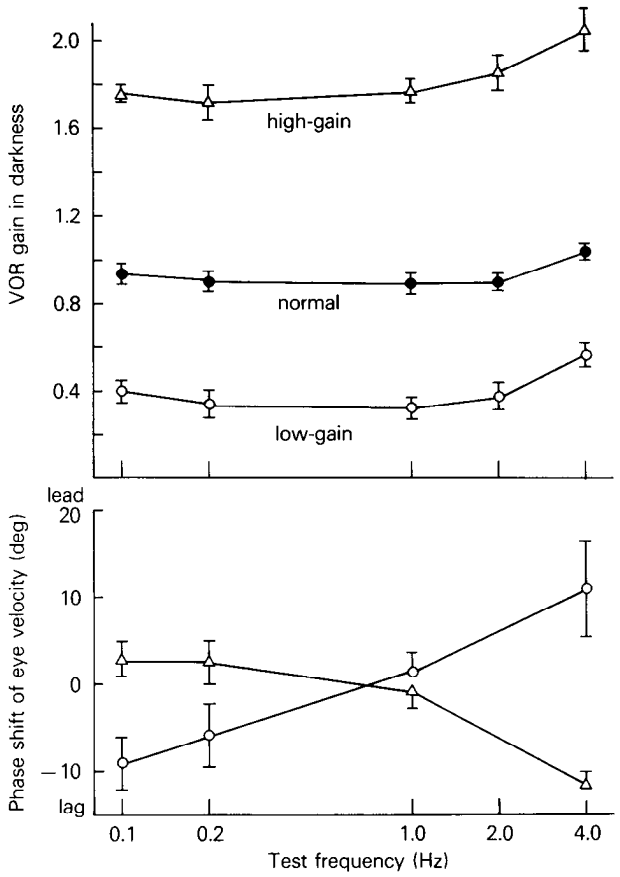

Figure 1. Frequency response of the VOR after spectacle adaptation with free head. The *upper panel* shows VOR gain for one animal, before adaptation (*normal*), after adaptation to the × 2 spectacles (*high-gain*), and after adaptation to the × 0 goggles (*low-gain*). The *lower panel* plots the phase shift of the VOR for the same monkey in the high gain (*triangles*) and low gain (*circles*) states. In this and subsequent figures, 0° of phase shift refers to the ideal situation when eye velocity is 180° out of phase with head velocity.

Adaptation with passive oscillation. Spectacle adaptation during passive, sinusoidal oscillation at a single frequency caused changes in VOR gain that were strongly dependent on the testing frequency. Figure 2*A* shows the result of oscillating a monkey continuously at 0.2 Hz, ±20° while he viewed the stationary environment through × 2 spectacles. At regular intervals VOR gain was tested at 0.1, 0.2, and 1.0 Hz, revealing changes that were largest at the adapting frequency (0.2 Hz), slightly smaller at 0.1 Hz, and markedly smaller at 1.0 Hz. In Figure 2*B*, the monkey underwent spectacle adaptation during oscillation at 2.0 Hz, ±2°, and the analogous result was obtained; the changes in VOR gain were largest at the adapting frequency and much smaller at 0.2 Hz. Within each experiment at a single adapting frequency, the rate of adaptation was similar for all testing frequencies (Fig. 2). Certainly, the smaller changes seen at 1.0 Hz cannot be explained as slower adaptation that, allowed enough time, would have reached the same asymptotic level.

The Bode plots in Figures 3 and 4 summarize the frequency-selective changes in the gain and phase of the VOR. Regardless of the conditions employed for adaptation, the changes in VOR gain were always greatest when tested at the adapting frequency, smaller when tested at adjacent frequencies, and smallest when tested at the frequency most remote from that used for adaptation (Figs. 3, *A* and *B* and 4, *A* and *B*). In the gain plots, the data shown with symbols and standard deviations are taken from just one monkey; however, they are representative of the data from all four monkeys, which were averaged to obtain the curves shown without symbols. Although the data in Figures 3 and 4 were taken when the peak-to-peak head velocity was 50°/sec, we did occasionally test the VOR at 1.0 Hz, ±10°. This 2.5-fold increase in stimulus velocity did not alter our estimate of the gain of the VOR in either normal or adapted animals.

Changes in VOR phase were complex but consistent in both monkeys that were studied and showed a property that we will call "phase crossover." For each adapting condition shown in Figures 3 and 4, the shift in phase below the adapting frequency was opposite in direction to that above the adapting frequency. In Figure 4*C*, for example, the adapted VOR shows phase lead when tested above the adapting frequency (0.2 Hz) and phase lag below 0.2 Hz; the crossover of the normal and adapted curves implies that there would have been no phase shift at the adapting frequency. One consequence of the phase crossover is shown in Figure 5, which contains records from two VOR tests at 1.0 Hz, ±4° following adaptation to the × 0 goggles. In both examples, VOR gain was reduced to values between 0.4 and 0.5. However, in the records taken following adaptation at 2.0 Hz (*top*) the

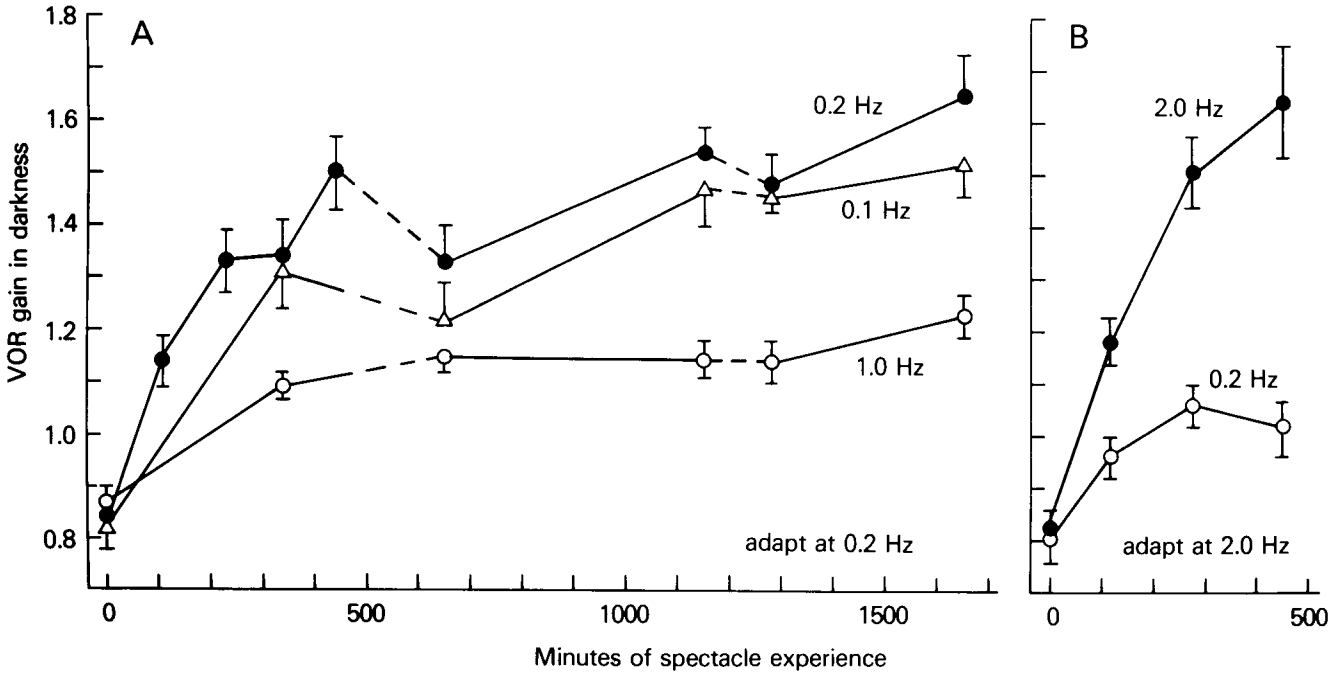

Figure 2. Time course of adaptation to × 2 spectacles during passive oscillation at 0.2 Hz (*A*) and 2.0 Hz (*B*). In each panel, *solid symbols* indicate VOR gain tested in darkness at the frequency used for adaptation; *open symbols* represent VOR gain for test frequencies other than that used for adaptation. The experiment shown in *A* lasted 3 days and 2 nights. *Minutes of spectacle experience* refers to the time during which the monkey was awake and experiencing the desired visual-vestibular interaction; *dashed lines* show the nighttime hours when the monkey slept most of the time, so that the spectacle experience was intermittent.

VOR showed slight phase lag, whereas in those taken after adaptation at 0.2 Hz (*bottom*), the VOR showed slight phase lead.

The phase shifts seen in Figures 3 to 5 result from a consistent dependence of VOR phase on adapting frequency, testing frequency, and VOR gain. In Figure 6 we have replotted the data so that each panel shows the results for a single combination of adapting and testing frequencies. Within a given panel, the phase of the VOR is plotted as a function of VOR gain, the *arrows* indicate the shift from before (*solid symbols*) to after (*open symbols*) adaptation, and the *circles* and *triangles* differentiate between the two monkeys for which we had phase data. Substantial phase shifts occurred only when there were large changes in VOR gain. Tests at 0.1 Hz after adaptation at 2.0 Hz (Fig. 6*B*) and tests at 4.0 Hz after adaptation at 0.2 Hz (Fig. 6*E*) showed relatively little change in either the gain or the phase of the VOR. In all six panels, the direction of the relationship between phase and gain is determined by the combination of adapting and testing frequencies. When the testing frequency was *below* the adapting frequency (Fig. 6, *A*, *B*, and *D*), *increases* in VOR gain caused eye velocity to show phase *lead*, whereas *decreases* caused phase *lag*. When the testing frequency was above the adapting frequency (Fig. 6, *C*, *E*, and *F*), the converse occurred: increases in VOR gain caused eye velocity to show phase lag, whereas decreases caused phase lead. There was a tendency for decreases in VOR gain to produce a slightly steeper relationship than did increases (Fig. 6, *A*, *B*, *E*, and *F*); this indicates that an increase in VOR gain produces somewhat less change in phase than would the same magnitude decrease in VOR gain.

Discussion

Passive, sinusoidal oscillation with altered visual conditions produces large changes in VOR gain at the frequency used for adaptation and smaller changes at adjacent frequencies. In addition, there are small but orderly changes in the phase of the VOR at frequencies other than that used for adaptation. It is not possible to account for our data in terms of a velocity-selective mechanism because testing stimuli at all frequencies had the same peak-to-peak velocity. Furthermore, we cannot have been studying acceleration-selective mechanisms because, over the frequency range we used, most information about the magnitude of angular accelerations is lost in the sensory transduction processes in the labyrinth. Therefore, we feel that our results reflect a genuine frequency-selective mechanism in the VOR.

Frequency-selective gain changes have been noted previously in chickens (Green and Wallman, 1978), goldfish (Schairer and Bennett, 1981), Dutch-belted rabbits (Collewijn and Grootendorst, 1979), and cats (Robinson, 1976). However, this feature of the VOR has gone largely uninterpreted because frequency selectivity is not seen in the discharge of individual neurons in the VOR pathways of normal animals. Only Collewijn and Grootendorst (1979) have proffered an explanation. These authors suggested that the adaptive mechanism is able to entrain central neurons so that they become preferentially sensitive to the adapting frequency, and that this is accomplished by memorizing specific motor patterns and recalling them when the requisite stimulus conditions recur. Their suggestion seems appropriate for the rabbit, which showed very sharply tuned frequency se-

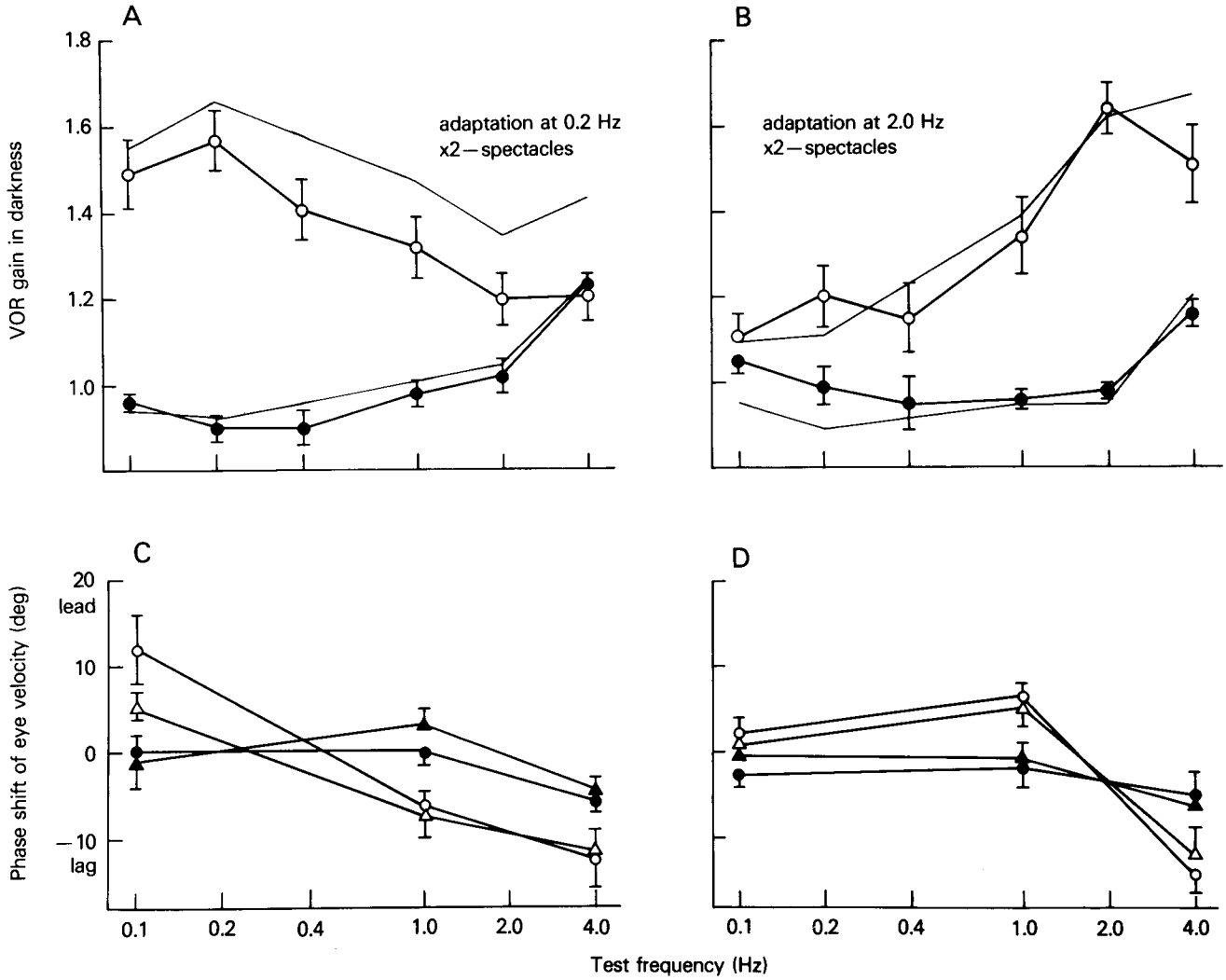

Figure 3. Frequency response of the VOR after adaptation to the × 2 spectacles with passive oscillation. In *A* and *C*, the adapting frequency was 0.2 Hz; in *B* and *D*, the adapting frequency was 2.0 Hz. In all the panels, the *solid symbols* represent data recorded before adaptation, and the *open symbols* represent measurements made after VOR gain had reached asymptotic levels. In the gain plots (*A* and *B*), the *symbols* and *error bars* show the means and standard deviations for one monkey and the *fine lines* show the averages of the responses for all four monkeys. In the phase plots (*C* and *D*), data are shown by *triangles* and *circles* for two monkeys that are included in the averages for *A* and *B*; *error bars* are standard deviations. For adaptation at 0.2 Hz (*A*), the four monkeys were alert and experienced the combined visual-vestibular stimulation for 1355, 1650, 1870, and 1950 min. For adaptation at 2.0 Hz (*B*), the monkeys were alert and experienced the adapting conditions for 265, 310, 450, and 460 min.

lectivity and occasional spontaneous oscillations of the eyes at the adapting frequency, when the animal was stationary and in total darkness. In the monkey, however, we find that frequency selectivity is quite broad, and we have never observed spontaneous oscillations of the eyes following our adaptation procedures (unpublished observations). In addition, the monkey exhibits changes in the phase of the VOR, in the form of phase crossover; this would not be expected of an adaptive mechanism that depended on pattern storage and recall. Therefore, we favor another class of explanation in which frequency selectivity is a regular feature of the VOR pathways but is revealed only when the vestibular stimulation for VOR adaptation is restricted to a single sinusoidal frequency.

Channels in the VOR. In thinking about how the VOR pathways might be organized so that they were capable of frequency-selective adaptation, we were influenced by the work of visual psychophysicists, who observed that perceptual thresholds could be adapted selectively within a narrow range of spatial frequencies. These authors concluded that different spatial frequencies were processed in separate parallel "spatial frequency channels" (Campbell and Robson, 1968). The analogy between our results and theirs leads us to suggest that the pathways mediating the VOR can be viewed as a series of parallel, overlapping temporal frequency channels. To achieve frequency-selective changes in the *gain* of the VOR, each channel should have its own modifiable gain element that can be accessed selectively when the VOR is in error over the range of temporal frequencies transmitted by that channel. To achieve phase crossover in the adapted VOR, the phase contributed by a channel must be related to the frequencies it transmits, with high frequency channels providing more phase lead.

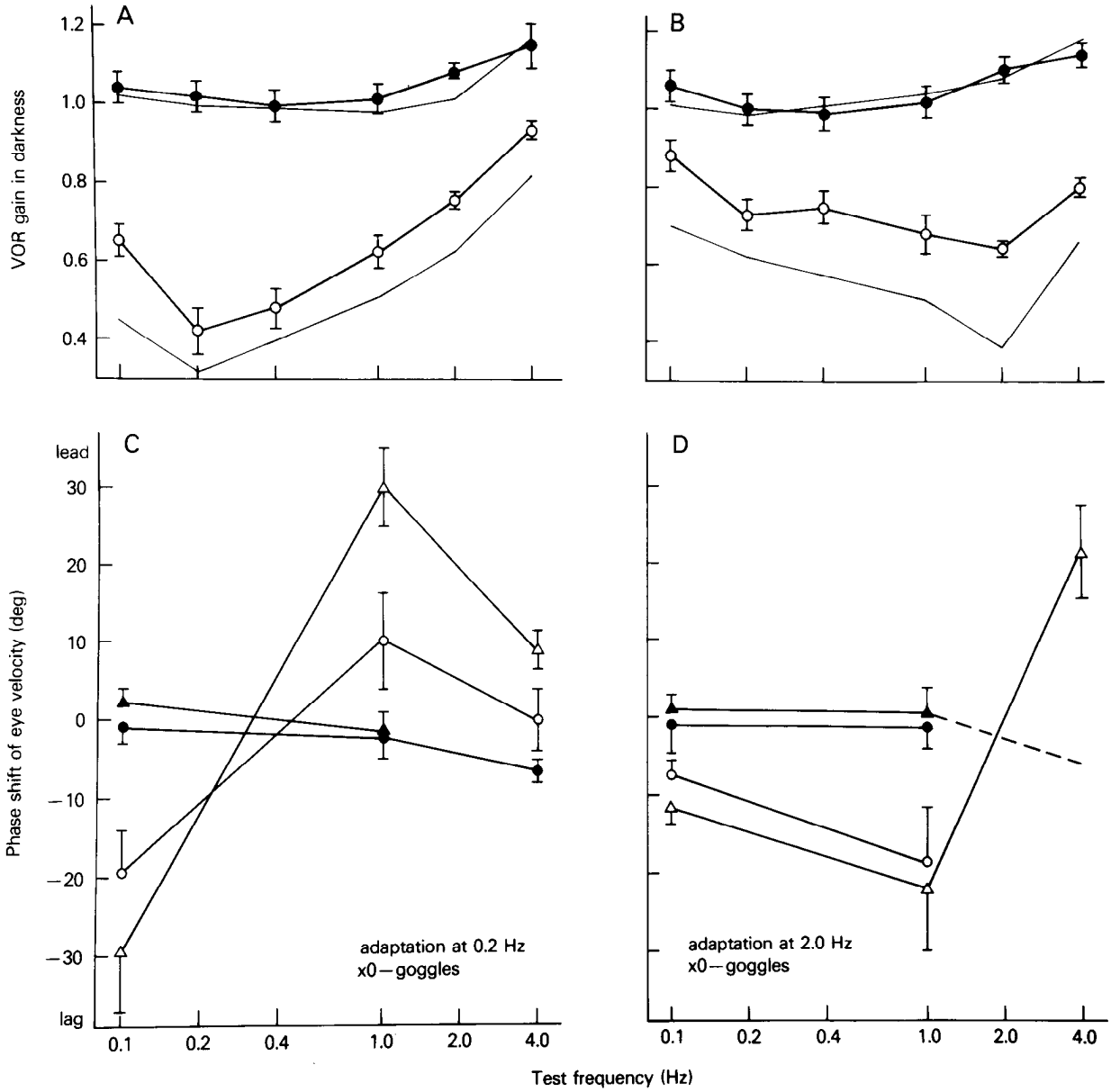

Figure 4. Frequency response of the VOR after adaptation to the × 0 goggles with passive oscillation. The meanings of *symbols* and *lines* are the same as in the preceding figure. In *A* and *C* the adapting frequency was 0.2 Hz; the four monkeys were alert and experienced the adapting conditions for 1510, 1740, 2210, and 2210 min. In *B* and *D*, the adapting frequency was 2.0 Hz; the monkeys were alert and experienced the adapting conditions for 365, 450, 460, and 650 min. In *D*, phase data were not available for 4.0 Hz tests before adaptation; the *dashed line* indicates the 5° phase lag seen at 4.0 Hz at other times when those two monkeys had normal VOR gains.

Separately adjustable temporal frequency channels would afford the adaptive mechanism considerable flexibility in the adjustment of the VOR's overall performance. One function of the VOR pathways is to compensate for the gain and phase of vestibular inputs so that the signals delivered to motoneurons will produce a VOR with a gain of 1.0 and a phase of zero. The magnitude of the compensations is large and must vary with frequency, inasmuch as the afferent and efferent organs are both mechanical systems that have frequency-dependent performances (Skavenski and Robinson, 1973). It seems unlikely to us that the genetic code would specify the details of a neural interface with the correct gains and phase shifts at each frequency. Instead, we assume that rudimentary neural pathways are shaped by experience to meet the specific needs of each individual's mechanical organs. An adaptive mechanism that operates selectively on the channels proposed here could accomplish this by tuning the developing VOR over the appropriate frequency ranges. This mechanism would also help to maintain the VOR's performance throughout life.

A model with multiple channels departs from the current concept of the VOR as a lumped system; only Baker et al. (1981) have argued for a distributed model, and then in a form and for reasons quite different from ours. We will show in the remainder of the "Discussion" that

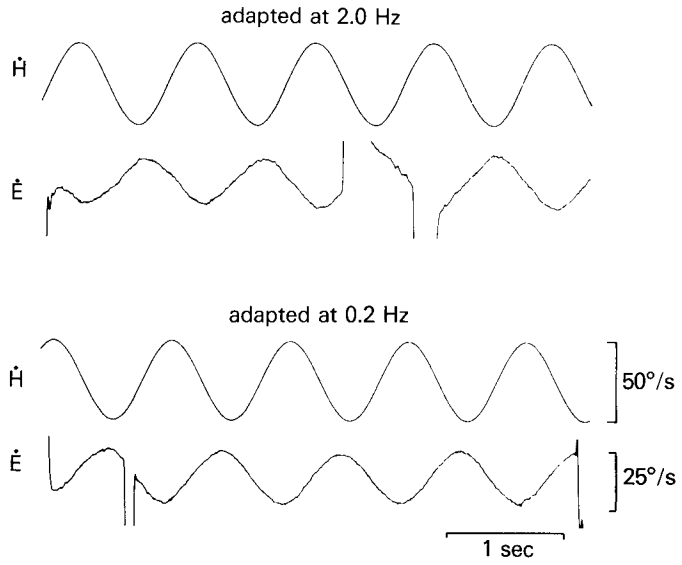

Figure 5. Changes in the phase shift of the VOR after adaptation to the × 0 goggles with passive oscillation. Both sets of records show the VOR elicited in darkness by oscillation at 1.0 Hz ±4°. In the *upper records*, the vestibular stimulation for adaptation was provided by passive oscillation at 2.0 Hz; in the *lower records*, the vestibular stimulation was provided by passive oscillation at 0.2 Hz. Note that the amplitude calibrations are different for eye and head velocity. Upward deflections of the traces represent rightward head ($\dot{H}$) or eye ($\dot{E}$) velocities.

a distributed model can provide a simple explanation for our data without invoking any new functions for the brain pathways that mediate the VOR. To keep the problem manageable, we will treat the VOR pathways as though they were divided into a finite number of channels. However, we are aware that this is just a convenient representation for a much more complicated biological system which we envisage as a continuum of overlapping parallel pathways. It is important to note that the broad tuning and overlap of the channels in our model would not be compatible with a suggestion that the VOR or its adaptive mechanism attempts to do a Fourier analysis of vestibular inputs.

A specific model with multiple channels. We have formulated a quantitative model to address two questions. First, would a model with multiple channels require any elements that were biologically unfeasible? Second, what elements would have to be organized in parallel, and which could be lumped together without affecting the model's frequency-selective performance? We have used only a simple model that could reproduce the essential features of our data: frequency-selective gain changes and phase crossover at a single adapting frequency. No attempt was made to reproduce in detail the quantitative behavior of the normal VOR.

Figure 7*A* shows the basic structure of our model; equations describing each element ("transfer functions") are given in the "Appendix." We have assumed that the dynamics of the eyeball and orbital tissues (*effector*) are lumped, because all motor units pull on a common tendon; the effector's transfer function was taken from Robinson (1981). We have configured the neural part of the model as three channels, where each channel consists of a subpopulation of primary afferents, a modifiable gain element (G) and a network (*phase shifter*) whose function is to incorporate phase lag into the channel's vestibular signals. The transfer functions of the primary afferents were taken from the data of Fernandez and Goldberg (1971), with their average high lead afferent providing the inputs for our high frequency channel, their average low lead afferent providing the inputs for our low frequency channel, and the average of these two extremes providing the inputs to our middle channel. The transfer functions of the phase shifters were taken initially from the model suggested by Skavenski and Robinson (1973). These workers recorded the discharge of extraocular motoneurons during the VOR and calculated the amount of phase shift that must be provided by the brain to convert vestibular inputs into motoneuronal discharge. They represented the brain by the two pathways shown in Figure 7*B*, one of which passed the vestibular signals directly (T_I) to motoneurons, while the other transmitted a signal that had been subjected to mathematical integration (l/s). We assumed that the relative weights of the direct and integrated pathways varied across channels and distributed this parameter so that our high frequency channel emphasized the direct pathway most heavily. This weighting is represented by the values of T_I and will determine the ratio of sensitivities to eye velocity (r) and eye position (k) in neurons downstream from each phase shifter. Therefore, we have taken the range of values of T_I from the range of r/k values in the motoneuronal recordings reported by Skavenski and Robinson (1973) and Robinson (1970). We found that frequency-selective adaptation could be reproduced only if our high and middle channels were modified so that they introduced less phase lag and gain reduction into the vestibular inputs at low frequencies. This was accomplished (Fig. 7*C*) by assuming that the integrating pathways functioned imperfectly ("leaked") and that the time constant of leakiness was shortest in our high frequency integrator and very long in our low frequency integrator.

The Bode plots in Figure 8 show the performance of our model. The model's normal gain (Fig. 8*A*) and phase (Fig. 8*C*) resemble those of the VOR, which shows phase lead and low gain at low frequencies, a gain of 1.0 and zero phase between 0.1 and 1.0 Hz, and an increase in gain with some phase lag at higher frequencies. If the adaptive mechanism can adjust selectively the gain of the middle channel, the model in Figure 7*A* will yield frequency-selective changes at an adapting frequency determined by the leakiness of the middle channel's integrator. Figure 8, *B* and *D* compares the effects of doubling or zeroing the gain of the middle channel when its integrator is adjusted to an adapting frequency of 0.25 Hz; the *smooth curves* show the difference in the performance of the model between these two states. For comparison, the *lines* with *solid symbols* show the difference in the performance of the monkey between the increased and decreased gain states after adaptation at 0.2 Hz. Clearly, the model reproduces the qualitative features of the data: the change in gain is frequency selective with a peak near 0.25 Hz; an increase in gain causes phase lead below 0.25 Hz, phase lag above 0.25

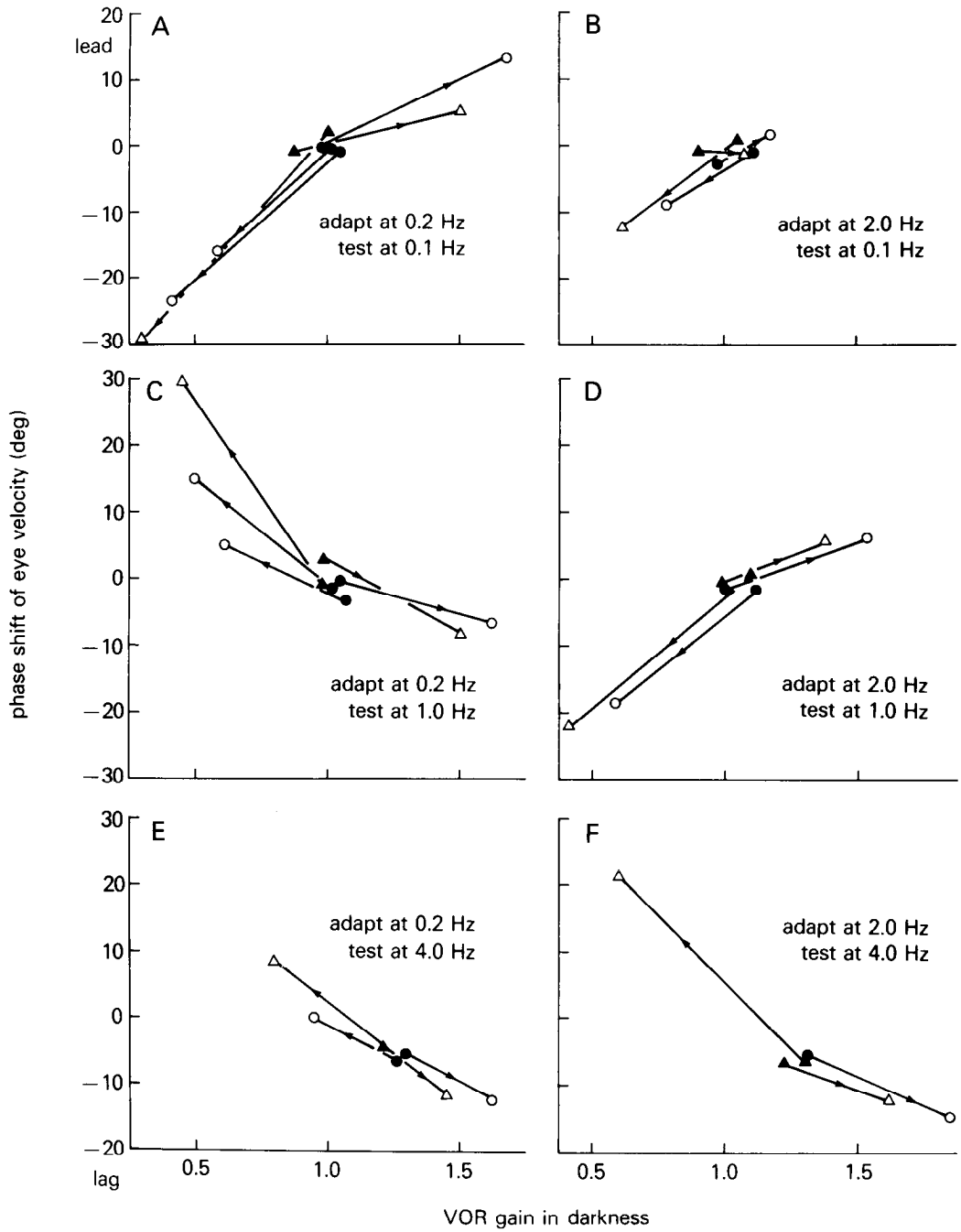

Figure 6. Factors determining the direction and magnitude of changes in the phase shift of the VOR. Phase data from Figures 3 and 4 (two monkeys shown by *circles* and *triangles*) are replotted as a function of VOR gain at the testing frequency. Each panel shows the effect of both increases and decreases in VOR gain for a single combination of adapting and test frequencies. VOR gain and phase shift before adaptation are shown by *solid symbols*; measurements made after adaptation are shown by *open symbols*. The *arrows* show the transition from before to after adaptation in each individual experiment.

Hz, and no change at 0.25 Hz. Minor changes in any of the parameters of the model had virtually no effect on its frequency selectivity. However, lumping any of the elements now distributed across channels had some effect on the model's performance. For example, the magnitude of the frequency selectivity and of the phase changes decreased above the adapting frequency if (*a*) all the channels received vestibular inputs from the same "average" afferent or (*b*) all the phase shifters had the same "average" weighting of the direct and indirect pathways. Frequency selectivity and phase crossover were lost completely if all the channels had perfect integrators or had the same "average" imperfect integrator.

The model in Figure 7*A* has the virtue of being a simple, linear representation that includes the features needed to produce phase crossover and frequency-selective gain changes. Moreover, it does not require any elements not already included in the model proposed by Skavenski and Robinson (1973). We have described a model with three channels because it is the minimum

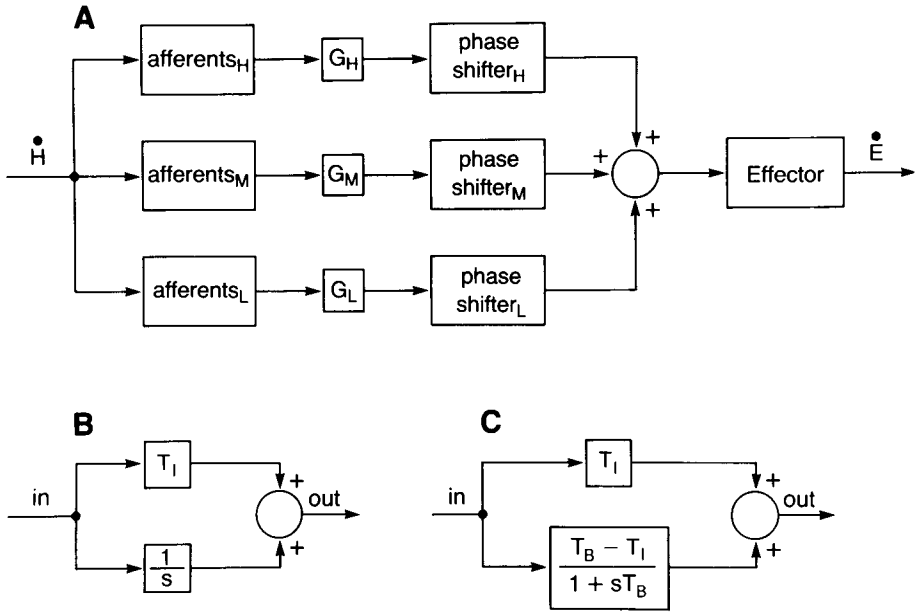

Figure 7. A quantitative model of the VOR with temporal frequency channels. *A*, A block diagram representation of the full model with, from *top* to *bottom*, high, middle, and low frequency channels. The model takes head velocity ($\dot{H}$) as its input and produces eye velocity ($\dot{E}$) as its output. *Boxes* indicate individual elements of the model, for which transfer functions are given in the "Appendix." *Circles* represent summing junctions, which perform a mathematical addition of all their inputs. *Arrows* indicate the direction of signal flow. Each channel consists of a subpopulation of the vestibular primary afferents, a separately adjustable gain element (G), and a phase-shifting network. The outputs of all three channels are added together before they reach the eyeball and orbital tissues (*Effector*). *B*, A detailed representation of the phase shifter suggested by Skavenski and Robinson (1973), containing a direct (T_I) and an integrating ($1/s$) pathway. *C*, A detailed representation of the phase shifter used in our model. Note that the structure of the phase shifter is the same as that in *B* and that the only differences are in the details of the integrating pathway.

configuration that reproduced qualitatively our data: to demonstrate frequency-selective gain changes with phase crossover, there must be at least one channel above and one below the adapted channel. As it stands, however, the model has a number of quantitative deficiencies. First, the model's normal performance shows a small phase lead between 0.1 and 1.0 Hz, whereas the monkey's VOR does not. Second, the model shows a sharp increase in gain at high frequencies that, in contrast to our data, cannot be overcome when gain is increased through adaptation at 0.2 Hz. Finally, the model is frequency selective at only one adapting frequency, and adjustments in the leakiness of the middle channel cannot push the adapting frequency above 0.7 Hz. At least the last, and probably all three, of these quantitative shortcomings could be overcome by increasing the number of channels in the model. Although our model works, other models could also reproduce our data. For example, a lumped model could produce frequency-selective adaptation, although with a smaller phase change, if four elements (imperfect differentiators and integrators) were added in series with a phase-shifting network like the one in Figure 7*B*.

Neural correlates of channels. To account for frequency-selective adaptation, we have made changes in the structure of previous models of the VOR. To what extent is the new structure proposed here consistent with existing data? To create channels, we have assumed that vestibular primary afferents with differing dynamics project to different cells in the central VOR pathways and that this separation is preserved as the signals are processed by the brain's phase shifters. Work by Goldberg et al. (1981), Shimazu and Precht (1965), and ourselves (Lisberger and Miles, 1980) supports this suggestion, at least for projections onto the vestibular nuclei. We have also assumed that the phase shifters contain integrators which function imperfectly to a degree that is distributed across channels. Skavenski and Robinson (1973) demonstrated that a phase shifter must exist and configured it with a lumped neural integrator. Our assumption about the integrator's distributed structure and leakiness do not conflict with their data.

In our model, channels differ only in the dynamic response properties of the primary afferents and the phase shifters. Thus, the neural correlate of temporal frequency channels would be the phase shift of the signals they transmit. It follows that phase shift must play a key role in identifying sites that will undergo gain changes. Specifically, our model will produce frequency-selective changes in VOR gain only if the adaptive mechanism is able to recognize by their phase shifts the neural elements that contribute to the VOR at the adapting frequency. It follows that adaptation may depend upon a precise phase coherence between the vestibular and error inputs to the

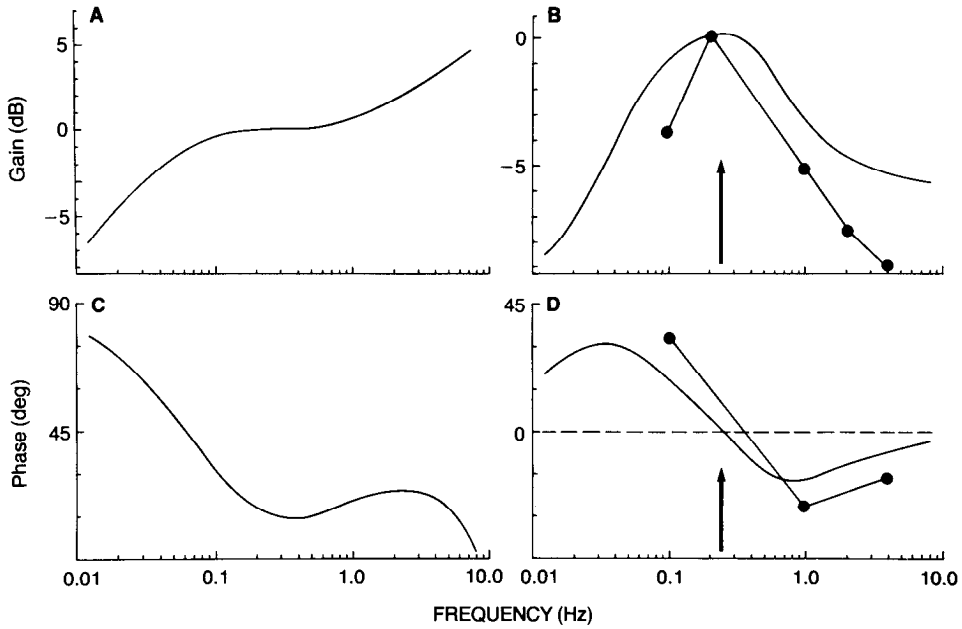

Figure 8. Performance of the model. *A* and *C* show the gain and phase of the model as a function of sinusoidal testing frequency, with all three channels set to have gains of 1.0. Note that we calculated the performance of the model over a wider range of frequencies that we used to test our monkeys. *B* and *D* compare the frequency selectivity and phase crossover of the model with the average performance of our monkeys. The *smooth curves* show the difference between the performance of the model with $G_M = 2.0$ and $G_M = 0.0$. To demonstrate the qualitative similarity of the model's and monkey's performance, we have performed the same calculations on our data and shown with *solid symbols* the difference between the data obtained with the monkeys in the high and low gain states after adaptation at 0.2 Hz. *Arrows* indicate the adapting frequency.

site of changes. In the time domain, this phase relationship would be manifest as a precise timing alignment of vestibular and error inputs; such temporal coincidence is an implicit feature of most models of the adaptive mechanism. Interestingly, the existence of a phase-sensitive adaptive mechanism might predispose the developing VOR pathways to organize themselves automatically along the lines of the distributed model presented here.

Appendix

All of the elements in our model have simple, linear transfer functions that can be described in the Laplace operator notation:

$$\text{effector(s)} = \frac{s \cdot e - 0.008s}{(1 + 0.24s)(1 + 0.016s)}$$

$$\text{afferent(s)} = \frac{s^2 T_A\,(1 + sT_L)}{(1 + sT_A)(1 + 5.7s)(1 + 0.003s)}$$

$$\text{phase shifter(s)} = \frac{T_B(1 + sT_I)}{1 + sT_B}$$

For the high frequency channel: $T_A = 30$, $T_L = 0.063$, $T_B = 0.25$, $T_I = 0.392$; for the middle channel: $T_A = 75$, $T_L = 0.04$, $T_B = 2.5$, $T_I = 0.159$; for the low frequency channel: $T_A = 1000$, $T_L = 0.015$, $T_B = 1000$, $T_I = .078$. Normal performance was assessed with G_L, G_M, and G_H all 1.0.

The factor of s in the numerator of the effector's transfer function provides an extra differentiation, so that the model's output (eye velocity) has the same dimensions as its input, head velocity. The gain and phase of the phase shifters are determined by the relative values of T_I and T_B. If $T_B \gg T_I$, as it is in our low frequency channel, the transfer function of the phase shifter approaches $\frac{1 + sT_I}{s}$, which describes the phase shifter proposed by Skavenski and Robinson (1973). In our middle channel, $T_B > T_I$, and the phase shifter introduces less phase lag than that in the low frequency channel. In our high frequency channel, $T_B < T_I$, so the phase shifter actually introduces a small amount of phase lead. Modifying this channel so that $T_B = T_I$ had no effect on the model's performance.

Because the model is linear, the gain and phase at any frequency can be determined analytically. We used a PDP 11/44 computer to perform the calculations at a number of discrete frequencies ranging between 0.0125 and 8.0 Hz. The Bode plots shown in Figure 8 were obtained by drawing lines through the values provided by the computer. In Figure 8*B*, the difference in gains was converted to dB by the equation: gain (dB) $= 20 \log_{10} (g_H/g_L)$, where g_H and g_L represent the model's or monkey's performance after adaptation to the high or low gain state.

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