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# Vestibular Signals Carried by Pathways Subserving Plasticity of the Vestibulo-ocular Reflex in Monkeys

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**The vestibulo-ocular reflex (VOR) is subject to long-term adaptive changes that minimize retinal image slip and keep eye movement equal to and opposite head movement. As a step toward identifying the site of neural changes, we have used a transient vestibular stimulus to study the dynamic response properties of the vestibular signals carried by the modifiable pathways.**

**In normal monkeys, "rapid changes in head velocity" (30°/sec in 50 msec) evoke a VOR that has a slight overshoot and reaches a steady-state gain (eye velocity divided by head velocity) of 1.0. Adaptation to magnifying spectacles causes changes in both the steady-state gain and the degree of overshoot in the eye velocity of the VOR. When the steady-state gain is decreased, the transient overshoot increases, so that peak eye velocity is twice steady-state. When the steady-state gain is increased, the overshoot decreases, so that peak eye velocity is nearly equal to steady-state. The discharge of vestibular primary afferents suggests an explanation for the inverse relationship between the transient overshoot and the steady-state gain of the VOR. In normal monkeys, 73 afferents showed a range of transient responses during rapid changes in head velocity. The afferents with the most regular spontaneous discharge had little overshoot in firing rate. Afferents with less regular discharge had large overshoots in firing; the peak change in firing was 2-6× the steady-state change.**

**We suggest that the large overshoot in eye velocity when VOR gain is low represents the contribution of vestibular signals from afferents with large transient responses. The decrease in overshoot in higher gains represents an increased contribution from afferents with small transient responses. Thus, our data imply that the modifiable VOR pathways receive inputs only from afferents with regular firing and that other, fixed-gain VOR pathways transmit signals from afferents with less regular discharge.**

The vestibulo-ocular reflex (VOR) stabilizes the visual surroundings during head turns by generating smooth eye movements that are opposite in direction and nearly equal in amplitude to head movement (Fuchs and Kimm, 1975; Keller, 1978). Although many details are still missing, it is known that the VOR is mediated by several parallel pathways in the brainstem (Baker and Berthoz, 1975; Highstein, 1973; Ito et al., 1976; Precht and Baker, 1972) and cerebellum (Baker et al., 1972; Highstein, 1973; Ito et al., 1977) and that each pathway subserves a somewhat different function (Miles and Lisberger, 1981; Robinson, 1981).

The accuracy of the VOR is regulated by a long-term adaptive mechanism that is mobilized whenever the VOR is persistently in error (for reviews see Collewijn, 1979; Ito, 1982; Melvill Jones, 1977; Miles and Lisberger, 1981). To study the adaptive mechanism, we fit monkeys with spectacles that require a VOR different from normal (Miles and Eighmy, 1980). For example, ×2 telescopic spectacles provide retinal images that can be stabilized only if smooth eye movements are opposite in direction and twice the amplitude of head movements. After adaptation to these spectacles, passive oscillation in darkness causes a VOR with an amplitude up to 1.8× normal. Miniaturizing spectacles cause an analogous decrease in the amplitude of the VOR to as little as 0.3× normal. The changes are acquired gradually over several days and are retained if either visual or vestibular input is withheld (Miles and Eighmy, 1980; Paige, 1983; Robinson, 1976).

We have recently shown that the VOR pathways with the shortest latencies are not subject to modification (Lisberger, 1984). The VOR was evoked by a "rapid change in head velocity" that had a sudden onset, a high acceleration, and a short duration. Comparing the eye velocity evoked by this stimulus in normal and adapted monkeys showed that the latency of the VOR was 14 msec and the latency of the modifiable pathways was 19 msec. The present experiments begin to eludicate the neural basis for these latency differences by comparing the firing of vestibular primary afferents during rapid changes in head velocity to the properties of the VOR.

It has been common to think of the vestibular inputs to the oculomotor system in terms of a single vestibular primary afferent having response properties defined by the population averages. However, each semicircular canal gives rise to a large number of afferents with a wide range of anatomical and physiological properties. In general, these properties are correlated. Afferents with faster conduction speeds (thicker axons) have more "irregular" discharge as measured by the variability in their spontaneous interspike intervals (Goldberg and Fernandez, 1977). In addition, discharge regularity is related to the dynamic response properties during sinusoidal vestibular stimulation. Afferents with more irregular discharge show modulation of firing rate that leads head velocity, while those with regular discharge modulate nearly in phase with head velocity (Fernandez and Goldberg, 1971). We have now obtained evidence that afferents with different response properties have different roles in adaptive modification of the VOR. Our data imply that VOR pathways receive inputs from afferents with a wide range of discharge regularities but that the modifiable pathways receive inputs only from the most regular afferents.

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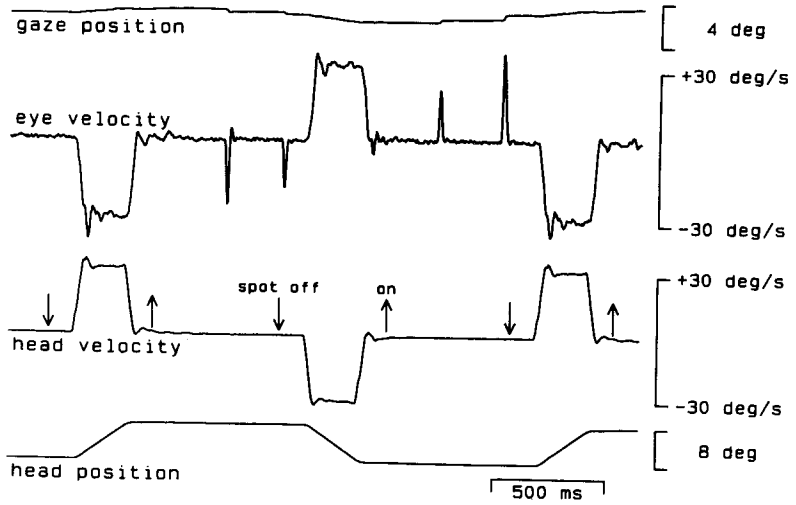


*Figure 1.* Data records showing how rapid changes in head velocity were presented in darkness with the eyes and head initially stationary. *Top to bottom traces,* horizontal gaze position (eye position in space), eye velocity, head velocity, and head position. The monkey fixated a target that was stationary at straight-ahead gaze. The target was extinguished before the head began to move (*downward arrows*) and was reilluminated after the head stopped (*upward arrows*). The small, brief deflections in the eye velocity record are caused by small corrective saccades.

## Materials and Methods

Experiments were conducted on five rhesus monkeys. Under halothane anesthesia, the monkeys were implanted with a scleral search coil to monitor eye movement (Judge et al., 1980). At the same time, four bolts were implanted in the skull to secure a dental acrylic pedestal. The pedestal provided a mount for spectacles and a means to hold the monkey's head during recordings (Miles and Eighmy, 1980). In three monkeys we studied the eye movements evoked by rapid changes in head velocity before and after adaptive changes in the VOR; these monkeys had been trained to fixate a small target. In two other monkeys we recorded the activity of single fibers in the vestibular portion of the VIIIth nerve during rapid changes in head velocity. Under halothane anesthesia, these monkeys had undergone a second surgery so we could trephine a hole in the skull and implant a stainless steel cylinder. The cylinder was tilted 26° back from the coronal plane and was aimed stereotaxically at 11 mm lateral to the midline along the interaural line. It was filled with saline, cleaned daily, and securely capped between recording sessions.

### *Vestibular stimulation*

During experiments, each monkey sat in a specially designed primate chair. The implanted head holder was secured to the chair, and passive horizontal head motion was provided by a servocontrolled turntable (Contraves-Goertz model 813, 20 ft-lb peak torque) that oscillated the monkey, the chair, and a set of 18 in. magnetic field coils together. A tachometer on the turntable provided a direct monitor of angular head velocity, while a precision potentiometer attached to the shaft of the turntable monitored angular head position. Rapid changes in head velocity were driven by the outputs from the digital-to-analog converters of a DEC 11/23 computer. The resulting head acceleration began abruptly, averaged 600°/sec$^2$, and lasted 50 msec, so that head velocity changed by 30°/sec. Sine wave stimuli were provided at frequencies of 0.2, 0.5, 1.0, 2.0, and 4.0 Hz; table excursion was adjusted so that peak-to-peak head velocity was 50°/sec at each frequency (62.5°/sec at 0.5 Hz). Since the monkey's head was held in the stereotaxic plane, horizontal motion activated the horizontal semicircular canals slightly suboptimally and the anterior and posterior canals weakly (Estes et al., 1975).

### *Modification and measurement of the VOR*

We produced adaptive changes in the VOR by having the monkeys view through various spectacles while undergoing the vestibular stimulation produced by their own active head turns. The first monkey wore the spectacles used by Miles and Eighmy (1980) and underwent adaptation in a generously sized primate chair that allowed him free head, body, and limb movements. Increases in the gain of the VOR were achieved with ×2 telescopic spectacles, and decreases were achieved with goggles containing a visual scene that moved exactly with each head turn (×0 goggles). In the second and third monkeys, we employed a new set of spectacles that the animals wore while free to move within their home cages. The spectacles were made of dental acrylic and were custom fitted to a plaster of paris mold of each monkey's face. Brass rings were mounted on the spectacles in front of the eyes and were used to hold either ×2.2 or ×0.25 telescopes purchased from Designs for Vision in New York. The spectacles were then secured to the monkey's implanted head holder. The monkeys tolerated the spectacles remarkably well and made no efforts to remove or damage them. The new spectacles caused adaptive changes in the VOR comparable in amplitude and time course to those achieved with our previous methods. In addition, the results of the present study were the same, whether VOR adaptation proceeded with the monkey in a primate chair or in his home cage.

To study the performance of the VOR, we used the strategy illustrated in Figure 1. Rapid changes in head velocity were imposed in total darkness, with the spectacles off, and with the eyes near straight-ahead gaze. Between stimuli the head was stationary, and the monkey fixated on a spot directly in front of him in an otherwise dark room. At regular intervals of 1.096 sec, the target was extinguished (downward arrows in Fig. 1). Then, 100 msec later, the head underwent a rapid change in velocity from 0 to 30°/sec, moved for 250 msec, and underwent another rapid change back to 0°/sec. Another 100 msec later, the target came back on (upward arrows in Fig. 1), and the monkey made corrective saccades, which were necessary whenever the gain of the VOR was not 1.0. Alternating the direction of the initial head acceleration produced a trapezoidal head position stimulus.

Data were recorded on-line by the computer, which sampled voltages related to eye velocity, head velocity, and gaze position at 500 Hz/channel. The data were analyzed after the experiment, by aligning 20 stimuli and averaging eye and head velocity around the onset of the rapid change in head velocity. We included in the averages only head accelerations from rest and only responses that were free of saccades. Right- and leftward accelerations were analyzed separately. The steady-state gain of the VOR was estimated as the average eye velocity 150–200 msec after the onset of the stimulus divided by the imposed head velocity (30°/sec).

The VOR was also studied during sinusoidal oscillation in darkness. For analysis, the eye velocity records were edited by using a cursor to remove the rapid deflections caused by saccades. At least 10 consecutive cycles were then averaged, and the averages were subjected to Fourier analysis. The gain of the VOR was estimated as the ratio of the amplitudes of the fundamental components of eye and head velocity. This yielded values that agreed well with those obtained during the steady-state of rapid changes in head velocity.

### *Vestibular nerve recordings*

In monkeys with normal VOR gains, we used glass-insulated platinum-iridium microelectrodes to record extracellular potentials from axons in the vestibular nerve. We approached the nerve through the cerebellar flocculus, which can be recognized by the presence of background activity related to eye movement (Lisberger and Fuchs, 1978). Successful approaches to the nerve were marked by silence as the electrode left the flocculus, followed by the positive-going potentials usually associated

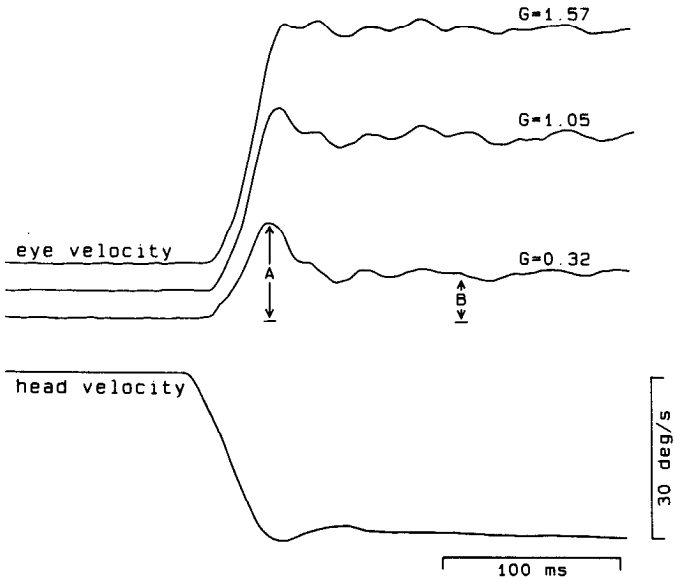


*Figure 2.* Eye velocity caused by rapid changes in head velocity before and after adaptive changes in the VOR. Each eye velocity trace is the average of 20 responses to the same stimulus. Data are all from one monkey and were obtained when his gain was normal ($G = 1.05$) and after 3 d of adaptation to ×2 spectacles ($G = 1.57$) or ×0 goggles ($G = 0.32$). The records have been offset vertically to facilitate viewing of the early part of the responses; eye velocity was always zero at the onset of the change in head velocity. Lines *A* and *B* show the times at which peak and steady-state eye velocity were measured for the computation of "dynamic index."

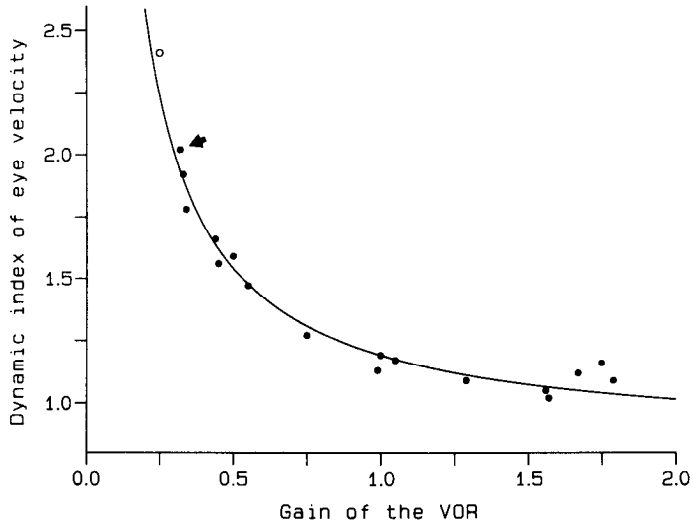


*Figure 3.* Effect of changes in VOR gain on the degree of transient overshoot during rapid changes in head velocity. Each *point* shows data from 1 d during adaptation and plots dynamic index (see text) as a function of the steady-state gain of the VOR (eye velocity divided by head velocity) 150–200 msec after the onset of the rapid change in head velocity. *Filled circles* show measurements made following adaptation with active head turns. The *arrow* indicates the values obtained from one monkey after 3 d of adaptation to ×0 goggles. The *open circle* represents measurements taken after the same monkey was subjected to a further 2 hr of adaptation to the ×0 goggles with passive vestibular stimulation provided by rapid changes in head velocity. The *smooth curve* was fitted to the filled symbols by performing linear regression of dynamic index on the reciprocal of VOR gain.

with axon recordings (Fuchs and Luschei, 1971). At the bottom of the penetration we could recognize (and avoid) the base of the skull by the sudden appearance of large-amplitude 60 Hz noise. Within the vestibular nerve, we encountered only fibers with the discharge patterns previously identified as typical of vestibular primary afferents (Fernandez and Goldberg, 1971; Miles and Braitman, 1980). Spike potentials were amplified conventionally (bandpass 100 Hz to 10 kHz) and converted to trigger pulses by a window discriminator.

Whenever possible, afferent firing was recorded during at least two sequences of 80 or more rapid changes in head velocity in each direction and during sinusoidal rotation at frequencies ranging from 0.2 to 4.0 Hz. In the first monkey, rapid changes in head velocity were imposed by alternating head velocity between 15°/sec rightward and leftward, as shown in Figure 4. In the second monkey, we obtained a smoother baseline firing rate by imposing rapid changes in head velocity from rest, as in Figure 1. Twenty-four afferents studied with both methods showed no systematic differences in any of the response properties we measured. During recordings, one monkey was rewarded for fixating a stationary spot and the other sat quietly in the light. As expected from previous studies (Büttner and Waespe, 1981; Keller, 1976), we saw no evidence that the different behavioral circumstances affected the responses of vestibular primary afferents.

### *Acquisition and analysis of neural data*

Data were digitized on-line by the computer. Analog traces were sampled as in the eye movement experiments, and interspike intervals were recorded to the nearest 10 μsec through Schmitt trigger inputs. In some of the early experiments, interspike intervals were recorded to the closest 100 μsec. Comparison of the two sampling rates in 16 fibers revealed no systematic differences in any of the response properties we measured.

Rapid changes in head velocity evoked a series of relatively subtle changes in the rate of discharge in a continuous spike train. We obtained an analog representation of the change in spike frequency by aligning 50–100 stimuli at the onset of the acceleration. Head velocity and firing rate were then averaged at 1 msec intervals for 200 msec before and 300 msec after the stimulus. To avoid obtaining a response that preceded the stimulus, we computed the firing rate at time $t$, $fr(t)$, according to the following algorithm:

$$fr(t) = \frac{1}{T_i - T_{i-1}} \quad \text{if} \quad t - T_i < T_i - T_{i-1}$$

$$= \frac{1}{T_{i+1} - T_i} \quad \text{if} \quad t - T_i \geq T_i - T_{i-1}$$

where $T_i$ represents the absolute time of occurrence of the $i$th spike in the train and time $t$ falls between the $i$th and $(1 + i)$th spike. The acquisition and analysis procedures were calibrated with a spike train generated by using a square wave stimulus to modulate the frequency of a pulse generator.

Averages of the reciprocal interspike interval are sensitive to artifacts created when unit spikes are missed or extra triggers are generated through the window discriminator. We have therefore analyzed only those recordings in which we achieved excellent isolation of the spikes under study. Some trigger artifacts (about 1/sec) were nonetheless unavoidable. They had a clear effect on the raw reciprocal interval records and could be spotted unambiguously during analysis in all except the afferents with the most irregular discharge. If only one or two artifacts were present in a stimulus, we manually instructed the computer to repair the spike train; otherwise the stimulus was not added to the average. Repairs were made only to insure a smooth baseline firing rate before and after a stimulus and were never attempted near the time of a rapid change in head velocity.

The spike trains evoked by sinusoidal stimuli were analyzed by dividing each cycle into 512 bins and averaging firing rate and head velocity for 10–40 cycles. Firing rate was computed as the reciprocal of the interspike interval that contained the center of each bin. The averages were subjected to Fourier analysis, and the amplitude and phase of the fundamental components were used to compute the sensitivity to head velocity (in spikes/sec per deg/sec) and phase shift with respect to head velocity for each afferent at each stimulus frequency. Harmonic distortion of firing rate was almost always less than 10%.

We analyzed the regularity of spontaneous discharge in each afferent from 10 sec of data taken with the head stationary. The coefficient of variation was computed as the SD divided by the mean of the interspike intervals. The method of Goldberg et al. (1984) was then used to obtain CV*, the coefficient of variation normalized to a mean interspike interval of 15 msec.

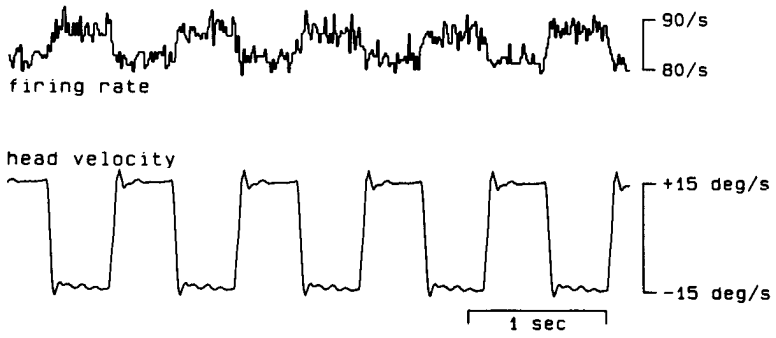


*Figure 4.* Firing of a typical primary afferent during a sequence of rapid changes in head velocity. The record labeled *firing rate* was obtained by taking the reciprocal of each interspike interval. This afferent had a normalized coefficient of variation (CV*) of 0.04.

## Results

We report two experiments in which we used the same vestibular stimulus, but monitored different outputs in different experimental conditions. In the first experiment, we measured the eye movements evoked by rapid changes in head velocity before and after the VOR had been adapted with spectacles. In the second experiment, we studied the responses of vestibular primary afferents during rapid changes in head velocity in monkeys with normal VOR gains. We report the two experiments together to facilitate their interpretation in relation to the properties of the pathways subserving plasticity of the VOR.

### *Eye movements evoked by rapid changes in head velocity*

Adaptive changes in the gain of the VOR caused a change in the shape of the eye velocity produced by rapid changes in head velocity. Figure 2 compares average eye velocity responses in one monkey before and after several days of adaptation to magnifying or miniaturizing spectacles. When the VOR was normal ($G = 1.05$), the change in head velocity caused eye velocity to exceed transiently its steady-state value by more than the overshoot in the stimulus. When the gain of the VOR was low ($G = 0.32$), the steady-state response was small, but the phasic response was emphasized so that peak eye velocity during the head acceleration (A in Fig. 2) was almost twice eye velocity in the steady-state, 150 msec later (B in Fig. 2). When the gain of the VOR was high ($G = 1.57$), eye velocity underwent a large and rapid change, but peak velocity exceeded the steady-state value only slightly.

To quantify the overshoot in the eye velocity responses, we calculated the "dynamic index," defined as the maximum change in eye velocity divided by the steady-state change in eye velocity (A divided by B in Fig. 2). In two of the three monkeys, we then measured the gain of the VOR and the dynamic index daily during adaptation; in one monkey we obtained data only at high, low, and normal VOR gains. All the measurements appear in Figure 3, which plots the dynamic index as a function of the steady-state gain of the VOR. Low VOR gains were consistently associated with large values of dynamic index and the data were fitted by the equation

$$\text{dynamic index} = (0.35/\text{gain}) + 0.84$$

The inverse relationship between dynamic index and VOR gain could represent selective adaptation of VOR pathways transmitting low-frequency vestibular inputs, and minimal adaptation of pathways transmitting higher frequencies. Our earlier work on frequency-selective adaptation (Lisberger et al., 1983) suggested that the failure to adapt high-frequency "channels" might result simply from the lack of adequate high-frequency vestibular stimulation during adaptation with the monkey's active head turns. To assess this possibility, we adapted one monkey during passive high-frequency vestibular stimulation. Initially, the monkey had worn $\times 0$ goggles for several days during active head turns and had achieved an asymptotically low VOR gain of 0.33 with a dynamic index of 2.0 (point indicated by the arrow in Fig. 3). We then subjected it to passive vestibular stimulation with rapid changes in head velocity while it viewed through $\times 0$ goggles. After two hr, the gain of the VOR had decreased and the dynamic index had increased to 2.4. The datum for adaptation with passive vestibular stimulation (open symbol in Fig. 3) fell on the curve fitted to data obtained during adaptation with active head turns (filled symbols).

### *Responses of vestibular primary afferents during rapid changes in head velocity*

We analyzed in detail the firing of 73 fibers that showed increased firing in relation to head velocity toward the side of the recordings and therefore innervated the horizontal semicircular canal. Figure 4 shows the reciprocal interspike interval record for one such afferent during rapid changes in head velocity. The

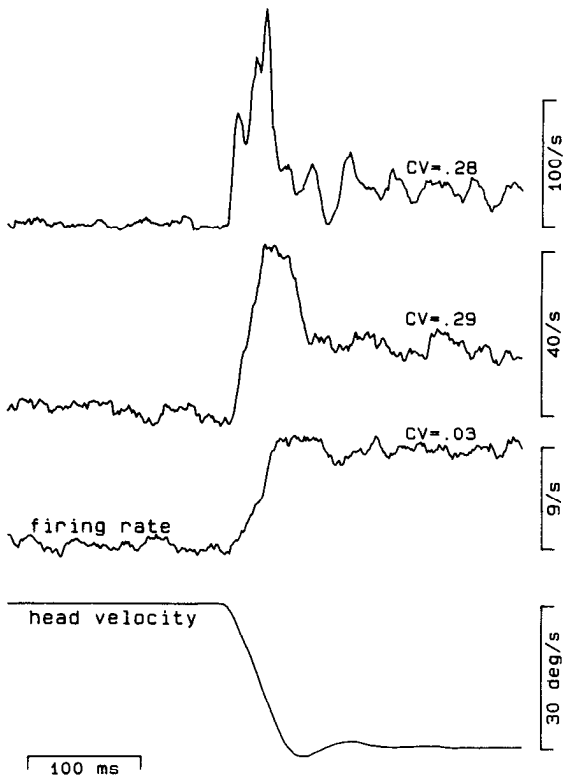


*Figure 5.* Representative responses to rapid changes in head velocity for three primary afferents with different degrees of transient overshoot. Each firing rate trace was obtained by averaging the responses to 50–100 sequentially applied stimuli. *Top to bottom,* the dynamic indexes of the three afferents were 5.99, 2.53, and 1.19. *CV,* The normalized coefficient of variation of each afferent.

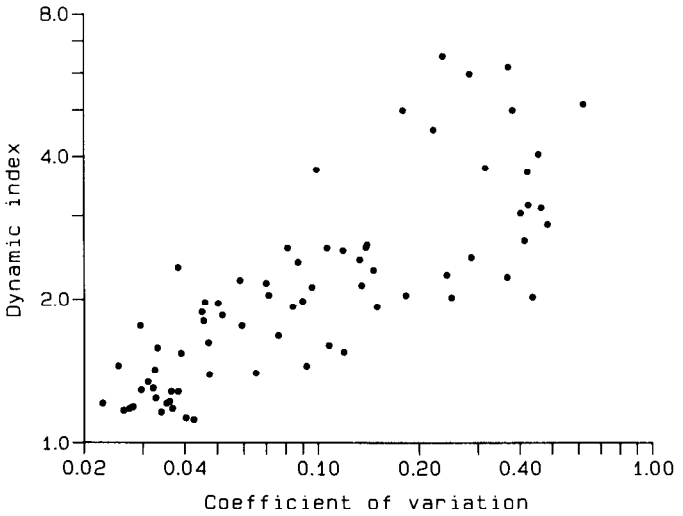


*Figure 6.* Relationship between the transient response during rapid changes in head velocity and discharge regularity. Each *point* represents the response of one primary afferent and plots dynamic index as a function of the normalized *CV*. Dynamic index was measured from rapid changes in head velocity that caused an increase in afferent firing.

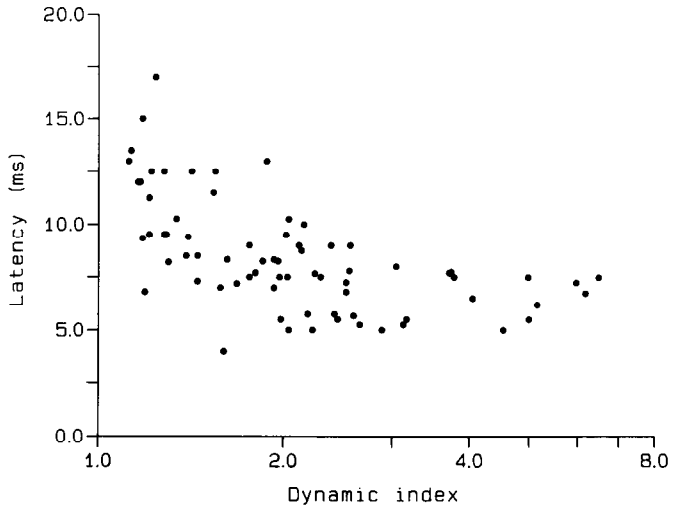


*Figure 7.* Latency of primary afferent responses to rapid changes in head velocity. Each *point* plots the latency for an individual primary afferent as a function of its dynamic index. Latency was measured separately for rapid changes in head velocity that caused increases or decreases in afferent firing and averaged. Fifty-five of the afferents were studied during at least two and sometimes three sets of rapid changes in head velocity. For these, we have averaged four or six estimates of latency to obtain the data shown here.

firing rate alternated between 80 and 90 spikes/sec during rightward and leftward head motion at 15°/sec, respectively. Averaging methods were used to reveal the details of the transition between lower and higher firing (see Methods).

The shape of the change in firing rate during rapid changes in head velocity varied substantially among afferents but was quite repeatable for each individual afferent. Figure 5 contains three averages that represent the full spectrum of responses. The afferent with regular interspike intervals (CV* = 0.03) showed a change in firing that reflected the change in head velocity with high fidelity. One of the two afferents with more irregular interspike intervals (CV* = 0.28) showed a large phasic response during the rapid change in head velocity and a relatively weak response in the steady-state. The other afferent with less regular discharge (CV* = 0.29) showed a transient response of intermediate amplitude.

We have represented the response of each afferent to rapid changes in head velocity by computing the dynamic index, defined as the peak change in firing rate divided by the steady-state change in firing rate. Figure 6 plots dynamic index as a function of normalized discharge regularity (CV*) and shows that afferents with more irregular discharge had larger transient responses. Only those afferents with the most regular discharge (CV* < 0.045) had values of dynamic index close to unity. However, the overall correlation between dynamic index and CV* was relatively weak ($r = 0.68$). The values of dynamic index in Figure 6 represent the responses to rapid changes in head velocity that caused an increase in firing rate. We obtained similar values for rapid changes in head velocity that caused decreases in firing, except in afferents with a high sensitivity to head velocity. These were driven toward a firing rate of zero and therefore had lower values of dynamic index for inhibitory than for excitatory stimuli. Dynamic index is a useful measure of afferent response properties, but it is not an absolute property of vestibular afferents. The presence and magnitude of the transient overshoot in firing rate should depend on the magnitude of head acceleration during the change in head velocity. Thus, data would have to be normalized to compare results obtained in different laboratories. In our lab, mean head acceleration was 600°/sec² and peak head acceleration was 850°/sec²; the head velocity stimulus itself had a dynamic index of 1.05.

Figure 6 also shows that we sampled afferents over the full range of discharge regularities reported by other investigators. The range of CV* in our sample (0.222–0.63) is similar to the range reported by Goldberg et al. (1984) in the squirrel monkey (0.025–0.7). In addition, the distribution of discharge regularities in our sample agrees well with theirs.

### *Latency of primary afferent responses*

Afferents with larger values of dynamic index responded at shorter latencies to rapid changes in head velocity. For example, Figure 7 shows that afferents with dynamic index less than 1.5 responded after latencies of 7–17.5 msec, while those with dynamic index greater than 2.0 had latencies of 5–10.5 msec. Because of the uncertainty of selecting the moment at which a small signal begins to deviate from a noisy baseline, measurements of latency are vulnerable to error. To make reliable estimates, we have studied many of the afferents during two or three sequential presentations of 80 or more rapid changes in head velocity. Latency was measured separately for both the excitatory and inhibitory responses, yielding two to six separate estimates for each fiber. These estimates seldom varied by more than 3 msec and have been averaged to obtain the values reported in Figure 7.

### *Comparison of afferent responses during sine wave and transient vestibular stimuli*

The responses of vestibular primary afferents during sine wave stimuli agreed with data in the literature and will not be presented graphically here. Instead, we will show the correlations between the dynamic index of afferent firing during rapid changes in head velocity and the phase shift and sensitivity to head velocity during sine wave stimulation.

The phase shift of afferent firing during sine wave stimuli was tightly correlated with the dynamic index during rapid changes in head velocity. Figure 8 plots phase shift as a function of dynamic index for 41 afferents studied during oscillation at 4.0 Hz (filled symbols) and for 62 studied at 0.5 Hz (open symbols). On semilog plots, the relationships appeared linear and were fitted by the functions: $\text{phase}_{4.0\ \text{Hz}} = 93.5 \times \log(\text{dynamic index}) + 4.1$ and $\text{phase}_{0.5\ \text{Hz}} = 42.9 \times \log(\text{dynamic index}) + 3.4$. The higher slope at 4.0 Hz indicates that afferents with larger

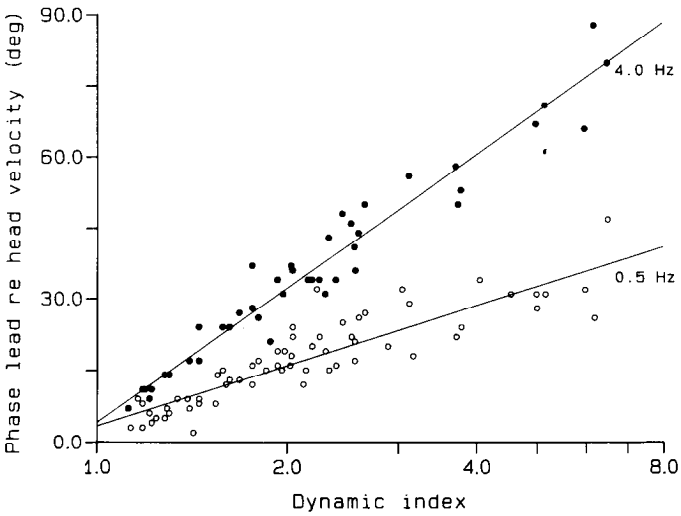


*Figure 8.* Comparison of phase shift during a sinusoidal vestibular stimuli with responses during rapid changes in head velocity. Each *point* represents one afferent and plots phase lead with respect to head velocity as a function of dynamic index. *Open symbols* show phase shift for sinusoidal stimuli at 0.5 Hz, *filled symbols* for 4.0 Hz. Note that the graph is plotted in semilog coordinates. The *lines* were obtained by performing linear regression of phase shift on the logarithm of the dynamic index.

values of dynamic index also showed a steeper increase in phase lead as the sinusoidal stimulus frequency was increased.

The absolute sensitivity to head velocity was not tightly correlated with dynamic index, although the ratio of sensitivity at high and low frequencies was. Figure 9*A* plots absolute sensitivity to head velocity during sine wave stimulation at 0.5 Hz (open symbols) and 4.0 Hz (filled symbols) as a function of dynamic index. There was only a weak tendency for afferents with larger values of dynamic index to have greater sensitivity to head velocity; the correlation coefficients were 0.01 at 0.5 Hz and 0.29 at 4.0 Hz. Figure 9*B* contains data from 39 afferents that were studied at both frequencies, and plots the ratio, sensitivity at 4.0 Hz divided by that at 0.5 Hz, as a function of dynamic index. This ratio measures the enhancement of sensitivity at high-stimulus frequencies and correlates tightly with the dynamic index ($r = 0.92$). The slope of the linear regression line was 0.36.

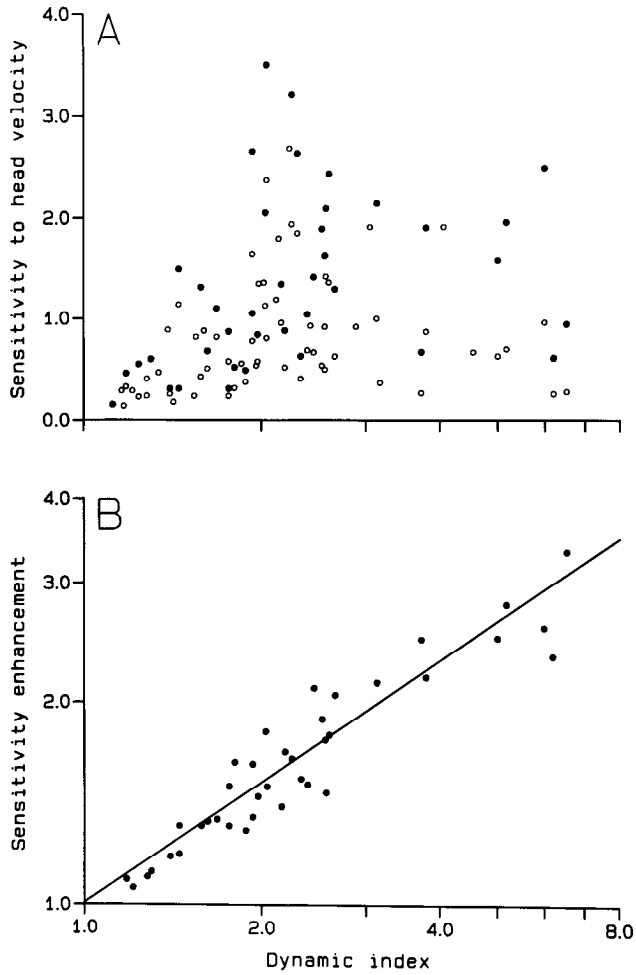


*Figure 9.* Comparison of sensitivity to head velocity during sinusoidal vestibular stimuli with responses during rapid changes in head velocity. *A,* Each *point* represents one afferent and plots sensitivity to head velocity (in spikes/sec per deg/sec) as a function of dynamic index. *Open symbols* show sensitivity for sinusoidal stimuli at 0.5 Hz, *filled symbols* for 4.0 Hz. *B,* Each *point* shows data for one afferent and plots the ratio of sensitivity to head velocity at 4.0 Hz divided by the sensitivity at 0.5 Hz (sensitivity enhancement) as a function of dynamic index. The *line through the points* was obtained by linear regression.

## Discussion

We have used a transient vestibular stimulus in two sets of experiments that, when interpreted together, provide information about the vestibular signals transmitted by modifiable VOR pathways. The first experiment showed that adaptive changes in the gain of the VOR affect the dynamic response properties of the VOR. The eye velocity during rapid changes in head velocity had a transient overshoot that became more prominent as VOR gain decreased. The second experiment showed that vestibular primary afferents have a wide range of dynamic response properties during rapid changes in head velocity. The afferents with the most regular spontaneous firing showed little or no overshoot in firing rate. Those with more irregular firing showed transient overshoot in which the peak response could be up to 6× as large as the steady-state response. Our data provide no reason to divide afferents into distinct groups based on dynamic response properties, and we view the population as a continuum. For ease of discussion, however, we will refer to the afferents as "regular" or "more irregular," according to whether they have low or high values of dynamic index.

The use of a transient vestibular stimulus affords several advantages over the sine wave stimuli usually employed to study vestibular responses, although in principle the two kinds of stimuli would provide the same information about the VOR, if it is a linear system. First, transient stimuli streamline data collection. A wide range of sinusoidal frequencies would have been needed to obtain the dynamic response properties as revealed by a single sequence of rapid changes in head velocity. Second, transient stimuli reveal the contributions of separate, parallel pathways and provide direct estimates of latency. These properties of the VOR are obscured during sine wave stimuli and must be inferred by making assumptions about the structure of the system and then fitting transfer functions to the data. Finally, transient stimuli provide a useful tool for studying the discharge of central neurons. In particular, the waveform of firing rate during rapid changes in head velocity provides a means to characterize central neurons according to the classes of vestibular afferents that provide their inputs.

### *Dynamic response properties of vestibular inputs to the modifiable pathways*

The relationship between the dynamic index of eye velocity and the gain of the VOR can be accounted for by the block diagram in Figure 10. In this model, there are two parallel VOR pathways

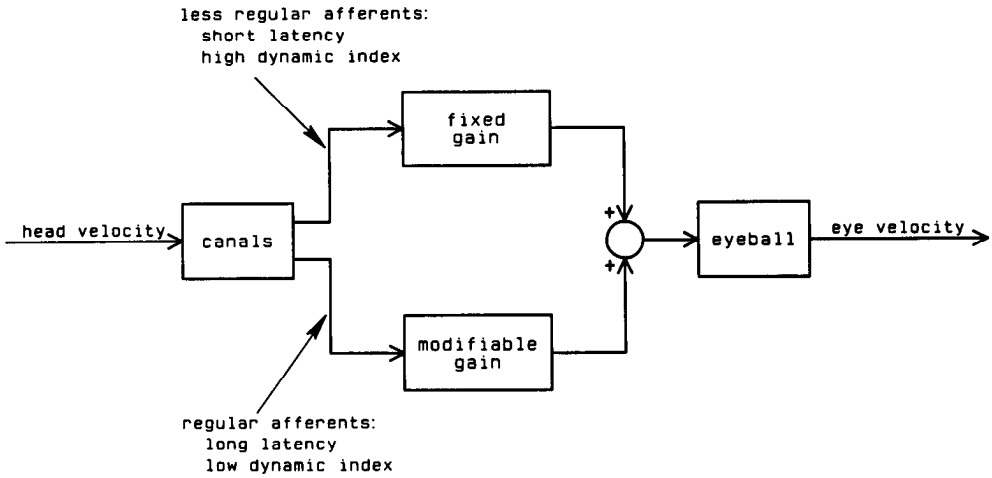


*Figure 10.* A simple model of the VOR showing two pathways from the semicircular canals (*canals*) to the motor output (*eyeball*). The lower pathway has a modifiable gain and transmits vestibular signals from afferents with regular discharge, long latencies, and low values of dynamic index. The upper pathway has a fixed gain and transmits vestibular signals from afferents with less regular discharge, short latencies, and high values of dynamic index. The *circle* represents a junction where the inputs from the modifiable and fixed-gain pathways are summed.

that receive inputs from separate groups of afferents. One pathway has a fixed gain and receives inputs from afferents with moderate to large values of dynamic index (i.e., more irregular discharge). The gain in the unmodifiable pathway defines the minimum VOR gain that can be achieved with adaptation to $\times 0$ goggles; in our monkeys this value was usually about 0.3. The other pathway has a modifiable gain and receives inputs from only those afferents with dynamic indexes close to unity (i.e., the most regular discharge).

Adjustments in transmission through the modifiable pathways would affect both the gain of the VOR and the relative weighting of vestibular inputs with high and low values of dynamic index. If the modifiable gain were zero, vestibular signals reaching the extraocular motoneurons would come only from afferents with large values of dynamic index. The eye velocity during rapid changes in head velocity would have a high dynamic index, and a small steady-state eye velocity, as it does when the gain of the VOR is low. If the modifiable gain were large, the vestibular signals reaching the extraocular motoneurons would be dominated by inputs from afferents with low values of dynamic index. The eye velocity during a rapid change in head velocity would be large and would have a dynamic index close to 1.0, since the phasic effect of the unmodifiable pathways would be swamped by the large, tonic effect of the modifiable pathways. We assume that the value of the modifiable gain is normally about 0.7. Thus, both groups of afferents contribute, the gain of the VOR is normally about 1.0, and the dynamic index of eye velocity is somewhat larger than unity. The proposal in Figure 10 is the simplest of several that could account for our data. For example, a change in the dynamics of central pathways could account for changes in the dynamics of the VOR, but would be more complicated than the mechanism suggested in Figure 10.

It seems unlikely that the efferent control of the vestibular organ participates in VOR modification by altering the dynamic index of primary afferent firing. In an earlier study using sine wave stimuli, Miles and Braitman (1980) found that the phase of afferent firing did not change in adapted monkeys. We have now found that the dynamic index of each afferent was tightly correlated with its phase shift during sine wave oscillation. The strong correlation suggests that both the dynamic index and the phase shift are measures of the dynamics of an afferent's response and that they should change together. Thus, the findings of Miles and Braitman argue that the dynamic index of primary afferent firing does not change in adapted monkeys.

Our block diagram predicts that adaptive changes in the gain of the VOR should also cause changes in the phase shift of the VOR during sinusoidal stimuli. In Figure 10, decreases in the gain of the VOR are effected by decreasing the transmission of signals from afferents with low values of dynamic index. Because of the relationship between dynamic index and phase shift, this operation selectively decreases the weighting of vestibular signals that are in phase with head velocity. Conversely, it increases the relative contribution of afferents with firing rates that lead head velocity and show increased phase lead at high frequencies. Therefore, sinusoidal oscillation in a low-gain monkey should reveal a small VOR with phase lead that increases as a function of the frequency of oscillation. Figure 1 of Lisberger et al. (1983) verifies this prediction. However, we will need to know the exact dynamic indexes of afferents that project into the modifiable and unmodifiable pathways before we can determine whether the phase shifts in our earlier report are in quantitative agreement with our new data.

*Latency of vestibular inputs to modifiable pathways*

The organization suggested in Figure 10 can account for our earlier report that the modifiable VOR pathways have a longer latency than do the unmodifiable pathways (Lisberger, 1984). Afferents with higher values of dynamic index responded to rapid changes in head velocity with shorter latencies. We are proposing that these short-latency, high-dynamic-index afferents provide inputs only to the unmodifiable pathways. As a result, the start of the eye velocity response to rapid changes in head velocity should be unmodifiable. However, the latencies of afferent responses probably cannot account for the entire 5 msec difference in latency between the modifiable and unmodifiable pathways. The modifiable pathways may also include more synapses than do the unmodifiable pathways.

Our data indicate that at least 9 msec of the 14 msec delay in the VOR is due to the semicircular canals and the motoneuron–muscle–eyeball system. Motoneuron firing precedes eye movement by 4–10 msec (Robinson, 1970), and the transduction of head motion into afferent spike trains takes at least 5 msec. We were surprised to find that vestibular primary afferents had such long response latencies. However, theoretical and empirical evaluation of our procedures verified that the long latencies were not artifactual. In addition, we measured the latency as the earliest possible time that could be construed as a response; more objective procedures would have biased our results toward longer latencies. Miles et al. (1980) reported data that appear to disagree with ours: All 21 afferents they studied had latencies shorter than 5 msec. Unfortunately, it is difficult to evaluate this discrepancy, since Miles et al. did not provide details of their data analysis except to say that their latency measures had an uncertainty of 5 msec. However, they may have biased their sample toward the high-sensitivity, short-latency afferents, which would have been most amenable to study with the relatively low head accelerations available to them.

*Are there different central pathways for different classes of afferents?*

We are proposing that vestibular primary afferents with different dynamic response properties project into different central VOR pathways and that this separation is maintained at least until the signals have passed through the site of adaptive modification. Earlier work has set a precedent for our suggestion. Studies by Goldberg et al. (1981), Shimazu and Precht (1965), and Lisberger and Miles (1980) implied that the projections of primary afferents onto second-order cells are segregated according to the dynamic response properties or discharge regularities of the afferents. Bilotto et al. (1982) have suggested that the segregation is retained throughout the vestibulocollic pathways, which appear to receive inputs predominantly from afferents with high values of dynamic index. We do not intend to imply, with Figure 10, that the primary afferents divide into two anatomically distinct channels. The present study, as well as those mentioned above, suggest that there is really a continuum between the two extremes we have shown.

It is worth comparing two aspects of our present model (Fig. 10) with a model we suggested earlier (Lisberger et al., 1983; Miles et al., 1985), which employed parallel "temporal-frequency" channels to account for the observation that the VOR is subject to frequency-selective adaptation. First, our earlier model suggested that vestibular inputs were channeled, with irregular afferents projecting into *modifiable* pathways that mediated the VOR at high frequencies. However, Figure 10 assumes that the pathways from irregular afferents are *unmodifiable,* since we were not able to lower the dynamic index of eye velocity when we adapted the monkey to $\times 0$ goggles with vestibular inputs provided by rapid changes in head velocity. Thus, our current work suggests that vestibular inputs to VOR pathways may not be channeled in the way envisaged before and that frequency-selective adaptation may represent properties of the central VOR pathways alone. Second, the model in Figure 10 does not include the dynamics of the central VOR pathways, while our earlier model assigned specific dynamics to each channel. Those dynamics are still necessary to reproduce the phase shifts between eye and head velocity during sinusoidal vestibular stimulation. They have not been included in Figure 10 because they are not needed to account for the dependence of the dynamic index of eye velocity on the gain of the VOR.

The model in Figure 10 suggests that afferents with different dynamic response properties play different but complementary roles in the VOR. Inputs from afferents with high dynamic index values and less regular discharge provide an initial response that is in the correct direction and at the shortest possible latency. Inputs from afferents with low dynamic index values and regular discharge provide a high-fidelity representation of angular head velocity that arrives at longer latencies but is appropriate for generating accurate compensatory eye velocities. Changes in the gain of the VOR result from changes in the transmission of the vestibular signals that most accurately represent head velocity. The adaptive mechanism is therefore ideally suited to fine-tuning the *accuracy* of the VOR.

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