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# Velocity Storage in the Vestibulo-Ocular Reflex Arc (VOR)

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**Summary.** Vestibular and optokinetic nystagmus (OKN) of monkeys were induced by platform and visual surround rotation. Vision prolonged per-rotatory nystagmus and cancelled or reduced post-rotatory nystagmus recorded in darkness. Presumably, activity stored during OKN summed with activity arising in the semicircular canals. The limit of summation was about 120°/s, the level of saturation of optokinetic after-nystagmus (OKAN). OKN and vestibular nystagmus, induced in the same or in opposite directions diminished or enhanced post-rotatory nystagmus up to 120°/s. We postulate that a common storage mechanism is used for producing vestibular nystagmus, OKN, and OKAN. Evidence for this is the similar time course of vestibular nystagmus and OKAN and their summation. In addition, stored activity is lost in a similar way by viewing a stationary surround during either OKAN or vestibular nystagmus (fixation suppression).

These responses were modelled using direct pathways and a non-ideal integrator coupled to the visual and peripheral vestibular systems. The direct pathways are responsible for rapid changes in eye velocity while the integrator stores activity and mediates slower changes. The integrator stabilizes eye velocity during whole field rotation and extends the time over which the vestibulo-ocular reflex can compensate for head movement.

**Key words:** Vestibular – Eye movements – Visual – Monkeys – Nystagmus

In the monkey the velocity of nystagmus induced by angular rotation about a vertical axis in darkness adequately opposes head velocity over a wide range of frequencies (Skavenski and Robinson, 1973). If head velocity is held constant in darkness, slow phase velocity decays to zero. In light, however, nystagmus is maintained for as long as rotation persists. Both vestibular nystagmus and optokinetic nystagmus (OKN) are followed by after-reactions, post-rotatory nystagmus and optokinetic after-nystagmus (OKAN). In contrast, there is little or no after-nystagmus following rotation in light (Ter Braak, 1936). This is

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believed to be due to OKAN and post-rotatory nystagmus cancelling each other (Ter Braak, 1936; Mover, 1936; Jung, 1948; Koening et al., 1978).

Recently we identified a storage mechanism that is closely associated with the vestibular system and is a key element in generating OKN and OKAN (Cohen et al., 1977). We designate it a "velocity storage" mechanism because it appears to hold or store activity that produces slow phase eye velocity. In this paper we present data that show the role of the velocity storage mechanism in generating vestibular nystagmus and in mediating visual-vestibular interactions in the monkey. A mathematical model was formulated that shows how the visual and vestibular systems might utilize velocity storage to produce slow phases of nystagmus. It provides a theoretical basis for understanding the interaction of the visual and vestibular system in driving the eyes.

## Methods

Five juvenile rhesus monkeys (Macaca mulatta) were used in this study. Monkeys sat with head fixed in a primate chair. Amphetamine sulfate (0.5 mg/kg) was given 30 min before testing to maintain alertness. Eye movements were recorded by DC electro-oculography (EOG) with implanted silver-silver chloride electrodes (Bond and Ho, 1971). The EOG was differentiated to obtain slow phase eye velocity. The EOG and voltages representing stimulus parameters were recorded on paper and stored on FM magnetic tape. A digital computer was used to analyze the data and generate the graphs and model predictions. Eye movements to the right are represented in the figures by upward deflections in the EOG. The EOG was calibrated using the response to rotation at 60°/s in light. It is assumed that the gain under these conditions is 1 (Skavenski and Robinson, 1973). The data were normalized with respect to this value. Calibrations were done just before each trial and repeated after periods in darkness to control for gain changes in the EOG.

Eye movements were induced by optokinetic stimulation or by angular rotation about a vertical axis. An OKN drum surrounded the animal and filled its field of vision. We have assumed that the system mediating slow phases of vestibular and optokinetic nystagmus is piecewise linear (Stern, 1965); then the step response is important for determining parameters that govern the dynamic response. To deliver steps of surround velocity, the light was switched on and off while the OKN drum was rotating. To give steps of angular velocity, animals were rotated on a rim-driven platform located under the OKN drum. The drum and platform could be rotated at speeds up to 240°/s. Peak accelerations of the platform were about 100–120°/s$^2$. Decelerations were faster, extending to 300°/s$^2$. Both accelerations and decelerations were fast enough to appear as step functions to the vestibular system since its short time constant is in the order of 10-20 ms (Goldberg and Fernández, 1971). Effects of visual fixation on post-rotatory nystagmus and OKAN were determined by illuminating the stationary OKN drum for variable periods. The time constants of the onset and end of light were about 20 ms. Dominant time constants of the declines in slow phase velocity during post-rotatory nystagmus and OKAN were determined using a technique that we have previously described (Cohen et al., 1977). This reference also contains a detailed description of eye movement stimulation and recording techniques.

## Results

### *Vestibular Nystagmus*

Slow phase velocity profiles induced by steps of angular velocity in darkness are shown in Fig. 1A–C. At the onset of stimulation slow phase velocity rises

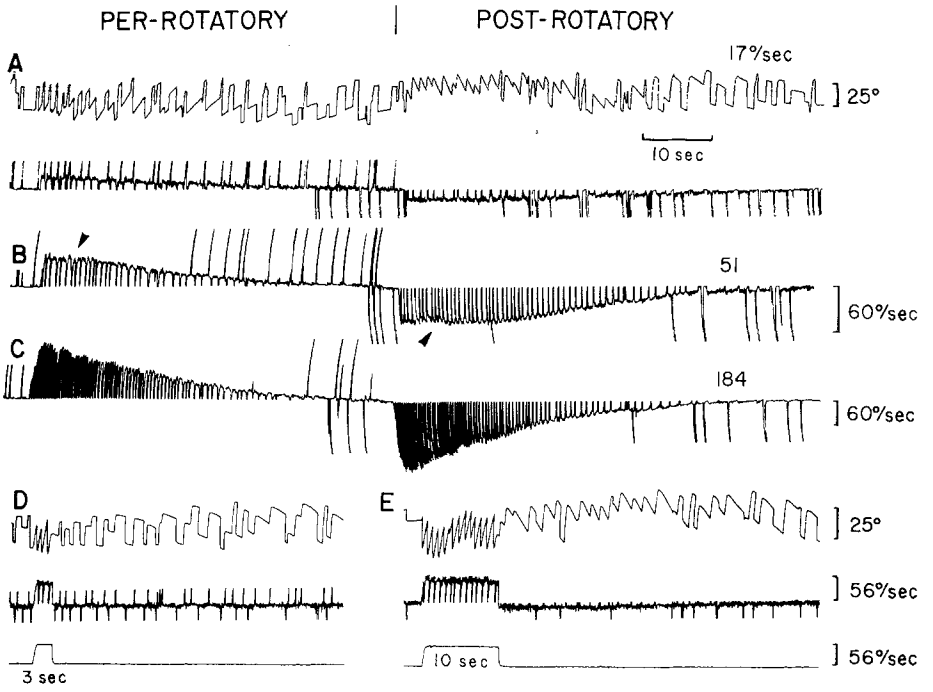


**Fig. 1. A–C** Per and post-rotatory nystagmus of alert monkey in darkness induced by steps in velocity. The stimulus velocity was 17°/s in **A**, 51°/s in **B**, and 184°/s in **C**. The top trace in **A** is the horizontal EOG recorded with DC-coupling. The bottom trace in **A** and traces **B**, **C** are slow phase velocity. Quick phases have been rectified. **D and E** Eye movements (top trace) and slow phase eye velocity (middle trace) in response to short steps of platform velocity at 56°/s (bottom trace). The monkey was in darkness. **D** There was little or no post-rotatory nystagmus after a step of 3 s. **E** The intensity of the post-rotatory nystagmus increased as the step was extended concurrent with the drop in per rotatory slow phase eye velocity

abruptly. For stimulus velocities up to 90–120°/s, peak values are maintained for several seconds (Fig. 1A and B). This "plateau" in eye velocity is shown by the arrows in Fig. 1B. Slow phase velocity then declines to zero over the next 30–60 s with a dominant time constant of 15–28 s. For higher stimulus velocities, peak velocity is held for a shorter time and the decline in slow phase velocity is more rapid (Fig. 1C).

If head velocity is set to zero during the plateau before per-rotatory nystagmus has begun to decay, there is little or no post-rotatory nystagmus (Fig. 1D). For longer periods of stimulation there is a greater decline in per-rotatory eye velocity with a concurrent increase in the velocity of post-rotatory nystagmus (Fig. 1E). If head velocity is set to zero after per-rotatory nystagmus has entirely disappeared, the characteristics of the per- and post-rotatory nystagmus are approximately equal (Fig. 1A–C).

The relationship between stimulus velocity and peak eye velocity during per and post-rotatory nystagmus is linear up to 240°/s, with a slope close to 1 (Fig.

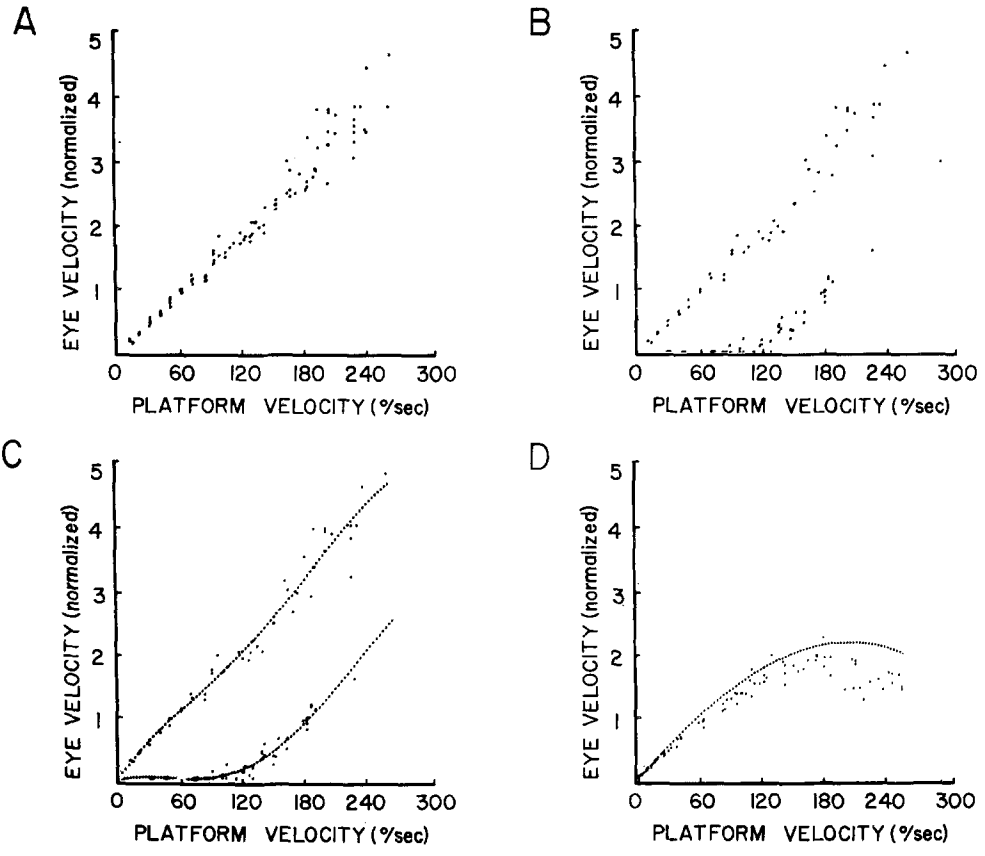


**Fig. 2. A** Peak eye velocity (ordinate) induced by steps of platform velocity in darkness (abscissa). Eye velocity was normalized in relation to values recorded during 60°/s rotation in light. Each dot is the peak velocity of one sample of per or post-rotatory nystagmus. **B** Post-rotatory slow phase eye velocities recorded in darkness after rotation in darkness and in light. The points on the left going through the origin are from rotation in dark. Data from post-rotatory responses to steps of velocity in light are below and on the right. The velocity of the nystagmus following rotation in light was less than 10°/s for stimulus velocities up to 120°/s. Above this eye velocity increased linearly parallel to post-rotatory nystagmus in dark. **C** Data of Fig. 2B approximated by polyomials. **D** Comparison of difference between polynomial curves of Fig. 2C (dotted line) with OKAN data from Fig. 4C of Cohen et al. (1977)

2A). This indicates that the gain of the VOR (eye velocity/head velocity) for steps of head velocity is approximately equal to 1. Consequently the VOR can compensate adequately for angular head movements that do not last longer than the plateau in eye velocity; such head movements will not be followed by after-nystagmus, even in darkness. Compensation becomes progressively poorer and post-rotatory nystagmus stronger for longer periods of head rotation. This is consistent with the idea that the VOR compensates best in the high frequency region during angular rotation about a vertical axis (Skavenski and Robinson, 1973).

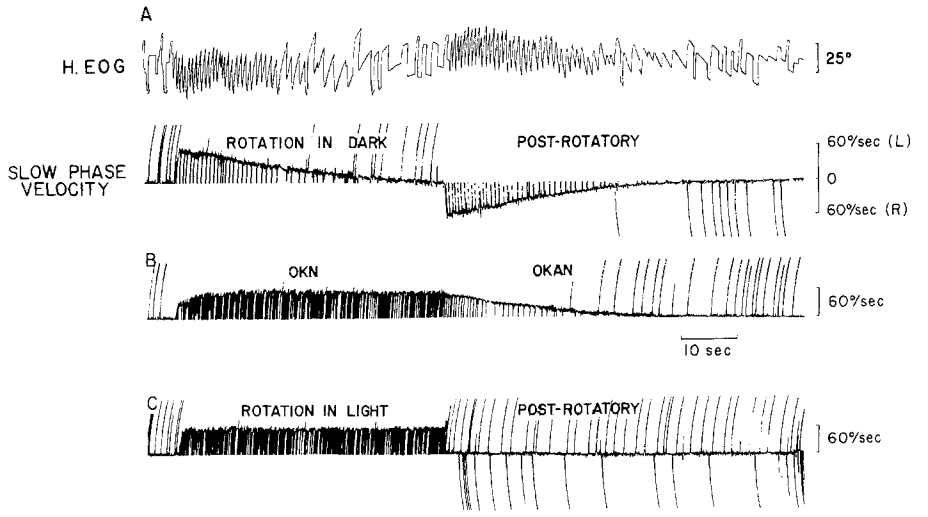


**Fig. 3. A–C** Nystagmus induced by a step of platform rotation in dark (**A**), by a step of surround rotation (**B**), and by a step of platform rotation in light (**C**). The velocity was 60°/s in each instance. Note that post-rotatory nystagmus and OKAN are oppositely-directed for OKN and per-rotatory nystagmus in the same direction (**A** and **B**), and that there was only a slight post-rotatory response to the right after rotation in light (**C**)

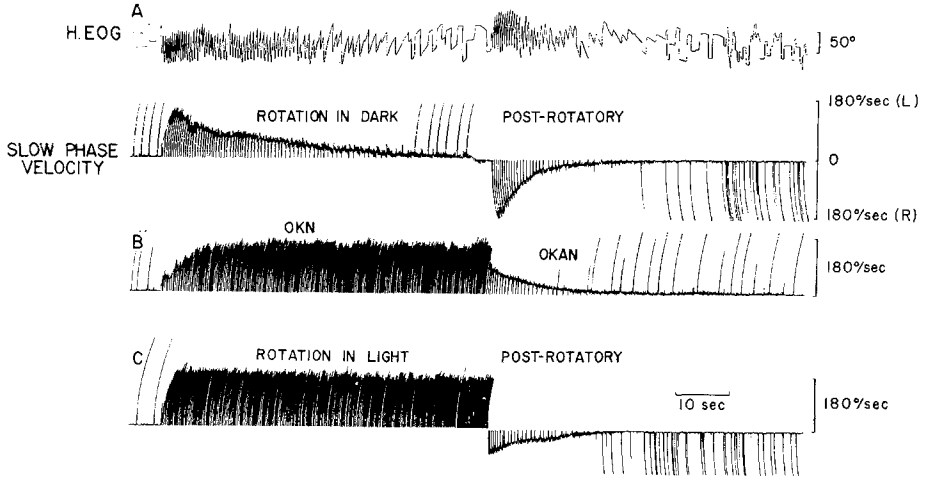


**Fig. 4. A–C** Nystagmus induced by steps of platform (**A**) and surround (**B**) rotation at 180°/s. Scheme as in Fig. 3. The buildup of steady state OKN in response to stimulation at 180°/s was slower than for 60°/s (Fig. 3B). At the end of OKN, OKAN slow phase velocity dropped to about 120°/s, the saturation level for OKAN. **C** During a step of rotation in light peak velocity of OKN was reached immediately and declined slightly as rotation continued. The oppositely-directed post-rotatory response (**C**) was weaker than the post-rotatory nystagmus after rotation in dark (**A**). Nystagmus eye velocity after rotation in light (**C**) is approximately equal to the difference between postrotatory nystagmus (**A**) and OKAN (**B**)

*OKN and OKAN*

OKN and OKAN have been characterized in our previous study (Cohen et al., 1977) and will be briefly summarized. For steps in visual surround velocity, there is an initial rapid rise in eye velocity during OKN, and steady state values are reached over a time course of 5–10 s (Figs. 3B, 4B). The peak values are maintained for as long as the stimulus persists. At the end of stimulation OKAN is brief if animals are in light. If they are in darkness, OKAN is prominent (Figs. 3B, 4B). Up to the saturation levels of OKAN (90–120°/s), there is a small rapid fall in slow phase velocity at the onset of OKAN followed by a slower decline (Fig. 3B). For OKN of higher velocity the rapid fall at the onset of OKAN is larger (Fig. 4B). The time constant of the slow decline varies with starting velocity (Cohen et al., 1977) and duration of stimulation (Buettner et al., 1977), and generally is in the range of 12–20 s.

*Rotation in Light*

Rotation in a stationary lighted surround causes both vestibular and optokinetic stimulation, and the resultant nystagmus has characteristics of the responses of both. Peak eye velocity is reached immediately (Figs. 3c, 4C, 5A) as during vestibular nystagmus. For stimulus velocities of less than 90–120°/s, eye velocity is maintained for the duration of stimulation (Figs. 3C, 5A) as during OKN. For stimulus velocities above 120°/s, eye velocity declines slightly as rotation continues, so that the steady state value is slightly less than the initial value (Fig. 4C).

If per-rotatory nystagmus and OKN are induced in the same direction, their after-responses are oppositely directed and have a similar time course (compare post-rotatory nystagmus and OKAN in Figs. 3A and B, and 4A and B). Consequently, if they are induced together their after responses should summate and eye velocity should be reduced. At the end of rotation in light if the animals are put in darkness, there is weak after-nystagmus in the post-rotatory direction (Figs. 3C, 5A). At higher stimulus velocities the oppositely-directed after-nystagmus becomes more prominent (Fig. 4C), but it is never as vigorous as the after-nystagmus that follows rotation in darkness (compare Fig. 4A and C).

Peak velocity of after-nystagmus recorded in darkness after rotation in light and in darkness is compared in Fig. 2B. The values of post-rotatory nystagmus after rotation in dark go through the origin. Following rotation in light there was only about 10°/s of after-nystagmus for stimulus velocities up to 90–120°/s. Above this after-nystagmus became progressively stronger, and slow phase velocity rose linearly (Fig. 2B). The data of Fig. 2B were approximated by polynomials (Fig. 2C). The slope of the rise in slow phase velocity following rotation in light was the same as the slope of the rise for rotation in darkness (Fig. 2C). If the two polynomials curves were subtracted from each other, the difference (Fig. 2D, dotted line) approximated the peak values of OKAN induced by the same speeds of stimulation (Fig. 2D, data from Cohen et al.,

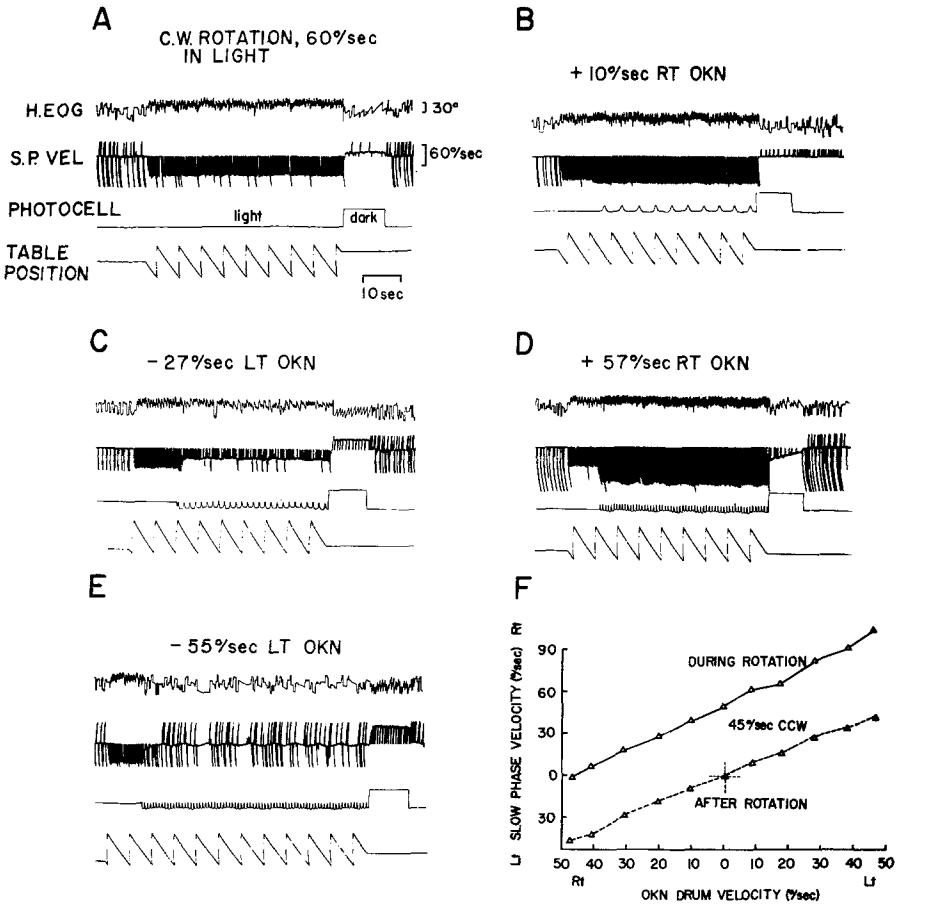


**Fig. 5. A–F** Effects of adding OKN (**B** and **D**) or subtracting it (**C** and **E**) from eye velocity during rotation in a lighted stationary surround. The control response is shown in **A**. After-nystagmus was recorded in darkness for only 10 s to minimize EOG gain changes. **A** There was a weak post-rotatory nystagmus of about 10°/s after rotation in light, **B** If 10°/s of OKN was added by rotating the drum in the direction opposite to the platform, post-rotatory nystagmus was abolished. **D** Further addition of OKN caused a shift of the after-nystagmus in the direction of OKAN. **C** Subtraction of OKN velocity caused a shift of the after-nystagmus in the direction of post-rotatory nystagmus. **E** If per-rotatory nystagmus is completely abolished, the intensity of the after-nystagmus is equal to that after rotation in darkness. **F** Effects of adding or subtracting OKN on post-rotatory nystagmus recorded in darkness for only 10 s to minimize EOG gain changes. **A** There was a weak post-rotatory nystagmus of about 10°/s after rotation in light, **B** If 10°/s of OKN was added by rotating the drum changes in post-rotatory eye velocity were linear for increments or decrements in per-rotatory velocity

1977). This suggests that activity responsible for OKAN had summated with that for post-rotatory nystagmus to reduce the after-response to rotation in light.

Summation of OKAN and post-rotatory nystagmus is also suggested in experiments in which the OKN induced by constant velocity platform rotation in

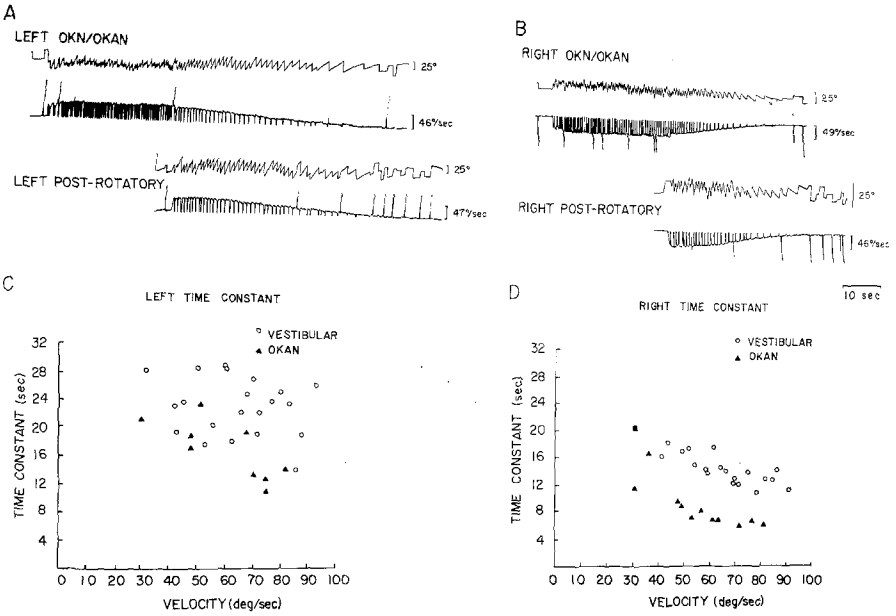


**Fig. 6. A** and **B** OKN, OKAN and post-rotatory nystagmus in a monkey with directional asymmetry. Both vestibular nystagmus and OKAN were longer to the left (**A**) than to the right (**B**). **C** and **D** Time constants of vestibular nystagmus (dots) and OKAN (triangles) declined with increasing stimulus velocity. Time constants of nystagmus to the left (**C**) were greater than to the right (**D**), and were larger for vestibular nystagmus than OKAN

light is increased or reduced. If OKN is added by rotating the drum in the direction opposite to that of platform rotation, the after-nystagmus is shifted in the direction of OKAN (Fig. 5D). If OKN is subtracted by rotating the drum and platform in the same direction, the after-nystagmus is shifted in the direction of post-rotatory nystagmus in darkness (Fig. 5C). If the drum and table move at the same rate, there is no nystagmus during rotation and the intensity of the after-nystagmus is equal to that of after-nystagmus recorded after rotation in darkness (Fig. 5E). A drum velocity of about 10°/s was necessary to completely abolish any after-nystagmus (Fig. 5B). Presumably more OKN than rotation velocity was necessary for the two to cancel because OKAN can only be charged to about 85% of the preceding OKN velocity (Cohen et al., 1977). The shift in eye velocity caused by adding or subtracting OKN was linear over a wide range of velocities (Fig. 5F). The limit of summation was about 90–120°/s, the level of OKAN saturation.

Summation of OKAN and post-rotatory nystagmus was not limited to initial or peak values, but extended for all time (Figs. 3C, 4C). Several animals developed directional asymmetry during the course of testing. Peak velocities of vestibular nystagmus were symmetrical but the nystagmus lasted longer and its time constant of decline was greater in one direction than the other. Despite the directional asymmetry, OKAN and post-rotatory nystagmus summated (Fig.

5A). If OKAN and vestibular nystagmus had been induced by separate processes, in order for one to cancel the other, the asymmetry should be opposite. However, when vestibular nystagmus was longer to one side (Fig. 6A), OKAN was always longer to the same (Fig. 6A), not to the opposite side (Fig. 6B). This suggests that a common mechanism is utilized for producing both OKAN and vestibular nystagmus.

### *Fixation-Suppression*

Exposure to a stationary surround during OKAN causes a rapid decline in slow phase velocity (Krieger and Bender, 1956; Cohen et al., 1977), similar to the suppressive effects of fixation on caloric nystagmus (Takemori and Cohen, 1974). If fixation is maintained for longer than several seconds, OKAN is lost completely and does not reappear if animals are once again put in darkness (Cohen et al., 1977). For shorter periods of fixation, the activity responsible for OKAN is partially lost, so that the recovery velocity of OKAN is less than it would have been without fixation. This is demonstrated in the response to a short (1.8 s) period of fixation shown in Fig. 7B.

Effects were similar for vestibular nystagmus. Post-rotatory nystagmus was truncated by continuous exposure to a stationary lighted surround (Fig. 7A, arrows). The peak velocity reached during post-rotatory nystagmus in light was 40% or less of that in darkness, and declined with a time constant of about 3–5 s. Once the nystagmus had disappeared, as in Fig. 7A, it did not reappear if animals were put back in dark. As with OKAN, post-rotatory nystagmus could be partially lost after shorter periods of fixation (Fig. 7D). These data support the idea that both types of nystagmus share a common storage mechanism.

Although similar, the effects of fixation-suppression on OKN and vestibular nystagmus were not identical. For the same periods of fixation the loss of slow phase velocity was greater for OKAN than vestibular nystagmus (Fig. 7B and D). This is reflected in different time constants of the recovery velocity curves. During OKAN slow phase velocity fell with a time constant of 1.7 s (Fig. 7C), while during vestibular nystagmus the time constant of decline was 2.6 s (Fig. 7E). There are other differences between vestibular nystagmus and OKAN. The plateau in velocity just after the onset of vestibular nystagmus is not present in OKAN (Fig. 6A and B). Moreover, vestibular nystagmus lasts longer than OKAN. Reflecting this, time constants of decline of vestibular nystagmus are larger than those of OKAN over a wide range of stimulus velocities. This was found both in normal animals and in animals with directional asymmetry (Fig. 6C and D).

### *Model Formulation*

The persistence of nystagmus in darkness after OKN indicates that the nervous system stores activity related to slow phase eye velocity during full-field rotation that is subsequently used to drive the eyes during OKAN. OKN has been shown

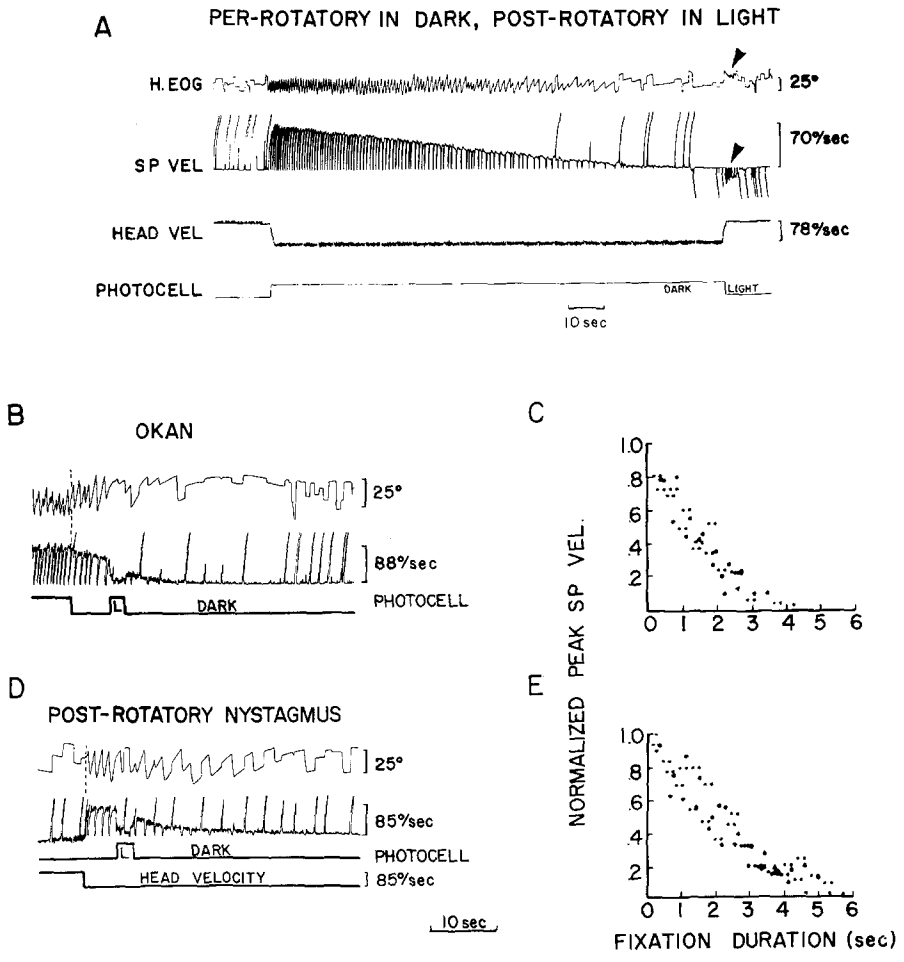


**Fig. 7. A** Per-rotatory nystagmus in darkness and post rotatory nystagmus in light (arrows). Maximum slow phase velocity in light was about 40% of that in darkness and declined with a time constant of about 3–5 s. **B** and **D** Effects of 1.8 s period of fixation on OKAN (**B**) and post-rotatory nystagmus (**D**). Recovery velocity was greater for post-rotatory nystagmus than OKAN. **C** and **E** Effects of fixation suppression on OKAN (**C**) and post-rotatory nystagmus (**E**). The time constant of decline of vestibular nystagmus (**E**) was greater than that of OKAN (**C**)

to be separable into two components, one that rises rapidly and another that rises more slowly (Cohen et al., 1977; Figs. 3B and 4B). Activation of direct pathways from the visual to the oculomotor system is believed to be responsible for the rapid rise in slow phase velocity at the onset of OKN. The more slowly rising component of OKN is presumably due to activation of a storage mechanism that is responsible for OKAN. Activity from the storage mechanism is postulated to sum with activity in direct pathways that extend around it to form the velocity signal that drives the eyes during OKN (Cohen et al., 1977).

In contrast to OKN, slow phase velocity of vestibular nystagmus rises immediately to a peak velocity and then decays to zero (Fig. 1). This has classically been explained as being due to the mechanical storage properties of the cupula-endolymph system (Steinhausen, 1933). However, neural activity in the vestibular nerve induced by a step in velocity decays with a time constant of about 3–5 s (Goldberg and Fernández, 1971), not 15–30 s, the dominant time constant of nystagmus induced by a similar step of velocity (Fig. 1). This suggests that in addition to peripheral storage of energy by the cupula, there is also central storage of the signal emanating from the cupula. Phase and gain characteristics of the VOR in the cat are also inconsistent with a time constant of 3–5 s (Robinson, 1976) and could best be explained by central storage in the VOR (Raphan et al., 1977).

We have assumed in the model that a non-ideal integrator that is part of the VOR is utilized in producing vestibular nystagmus, OKN and OKAN. Evidence for this comes from the finding that the velocity storage mechanism that is prominent during OKN and responsible for OKAN is impaired by bilateral labyrinthectomy (Uemura and Cohen, 1973). This is also suggested by the close approximation of firing rates of vestibular nuclei neurons to slow phase eye velocity during vestibular nystagmus and OKAN (Waespe and Henn, 1977a, 1977b).

It has been shown that OKAN and vestibular nystagmus are similar but not identical. OKAN is assumed to represent activity associated with discharge of the integrator. Vestibular nystagmus, on the other hand, is assumed to be composed of two parts, one referable to the contribution made by the cupula dynamics and the second to the contribution made by the integrator.

### *Mathematical Modeling of Nystagmus and the Visual-Vestibular Interaction*

Phenomenological aspects of vestibular nystagmus, OKN and OKAN were formalized in the model shown in Fig. 8. Vestibular nystagmus and OKN are produced by combined activation of direct and indirect pathways. The indirect pathways include a velocity storage mechanism, represented by an integrator, whose output is the VOR-OKAN state. Activity in the direct and indirect pathways summate to form a velocity signal that drives the velocity to position integrator identified by Skavenski and Robinson (1973). This final integrator and direct pathways around it drive the oculomotor plant (Robinson, 1964, 1965). For simplicity, the final stage of processing is not shown. The model has different parameter values for nystagmus to the right and to the left. Only the left side will be considered for simplicity.

The inputs to the model are vestibular and visual signals representing cupula deflection $r_v$ and surround velocity $r_o$. Presumably, surround velocity $r_o$ is obtained by combining retinal error with an efference feedback of eye velocity from the oculomotor system and a signal related to cupula deflection from the vestibular system (Yasui and Young, 1975; Lanman et al., 1978). In darkness no information related to surround velocity is available. This is represented by the switch L which opens in dark. Consequently, in dark the system is driven

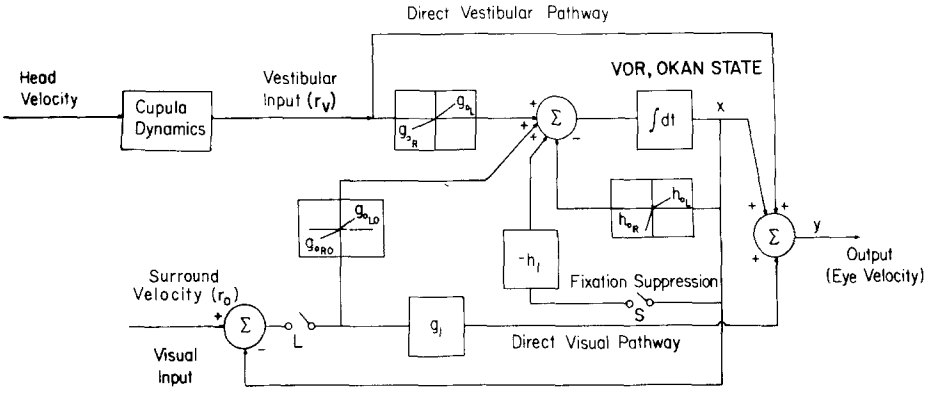


**Fig. 8.** Model of OKN, OKAN and vestibular nystagmus. Parameter values used in the model are $g_{OLO} = 0.25$, $g_I = 0.6$, $h_{OL} = 0.085$, $h_I = 0.7$, $g_{OL} = 0.25$, $g_{OR} = 0.222$, $g_{ORO} = 0.222$, $h_{OR} = 0.111$. The cupula dynamics have been represented as having a dominant time constant $T_c = 4$ s

only by $r_v$. This signal is transmitted over the direct vestibular pathway and is also coupled to the integrator, $\int dt$, via the forward gain element, $g_{OL}$. The rate of charge of the integrator for a step in activity is determined by its characteristic time constant, $T_o = 1/h_{OL}$ and the strength of the coupling coefficient, $g_{OL}$. The equivalent closed-loop charging time constant is $T_r = 1/h_{OL} + g_{OL}$). The time constant of the integrator, $T_o$ is also the dominant time constant of OKAN (Cohen et al., 1977).

The dominant time constant of the vestibular response, $T_v$, is given by $T_v = \frac{T_c}{T_r}$. $T_o$ (see Appendix for deviation). If it is assumed that the integrator has a characteristic time constant $T_o = 12$ s, that $T_r = 3.3$ s and that the cupula has a time constant $T_c = 4$ s, vestibular nystagmus would have a time constant $T_v = \frac{4.0}{3.3}(12) \cong 15$ s. Thus, $T_v$, the dominant time constant of vestibular nystagmus, would be about 25% greater than $T_o$, the time constant of the integrator and of OKAN. This agrees with the data of Fig. 6C and D.

Figure 9A shows the eye velocity predicted by the model in response to a step of vestibular excitation in dark. The overall characteristics are similar to those of vestibular nystagmus shown in Fig. 1. Figure 9B shows the predicted activity in the eighth nerve and integrator induced by a step of head velocity, and how this activity is combined to give slow phase eye velocity. The inflection point in the response of Fig. 9A simulates the plateau in eye velocity (Fig. 1). The plateau occurs because of the difference between the cupula time constant $T_c$ and the equivalent, closed-loop, charging time constant of the integrator $T_r$.

For a step in surround velocity during optokinetic stimulation the cupula signal $r_v = 0$, and the model behaves as described in Cohen et al. (1977). The directional asymmetry of OKAN has been incorporated into the g and h parameters that govern the charge and discharge characteristics of the integrator for nystagmus to either side. A typical response to a step in optokinetic

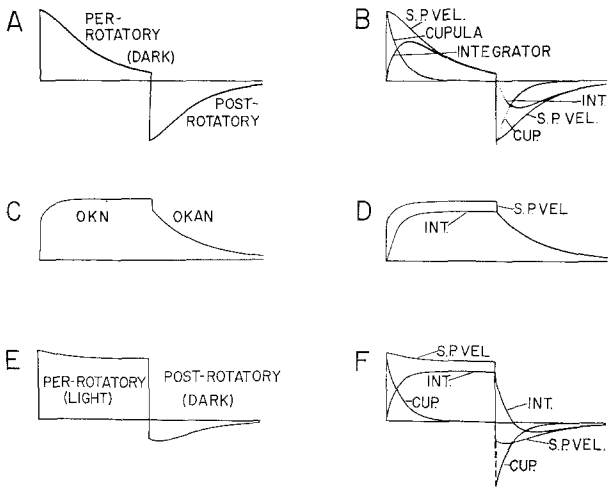


**Fig. 9. A, C, E** Model predictions of slow phase eye velocity for a step of angular velocity in darkness (**A**), for a step of surround velocity (**C**) and for a step of angular velocity in light (**E**). **B, D, F** Comparative changes in slow phase velocity, cupula deflection, and output of the integrator for the responses shown in **A, C** and **E** respectively

stimulation and the ensuing OKAN is shown in Fig. 9C. Note the rapid and slow rise in velocity during stimulation, and the slight fall in velocity just at the onset of OKAN. Figure 9D shows a comparison of the integrator response and the overall response. The difference between them is due to the contribution made by the direct pathway from the visual system.

The response of the model to a step in head velocity in light is shown in Fig. 9E. The direct vestibular pathway is activated and immediately drives the eyes to the velocity of the stimulus. The initial surround velocity signal is approximately zero since the cupula signals that the head is turning and the environment is stationary. As the cupula signal $r_v$ decays, the visual system signal $r_o$ begins to activate the direct pathway as well as the integrator. When the cupula signal has decayed to zero, head velocity becomes the inertial frame of reference to which surround motion is referenced, and the steady state response is due entirely to optokinetic excitation. During the steady state in Fig. 9E there is a slight decline in velocity. This is due to a shift in dependence of the response from the vestibular nystagmus with an initial gain of approximately 1 to OKN with a steady-state gain of about 0.92 (Cohen et al., 1977).

Figure 9F shows the contributions of the integrator, cupula and direct pathways to the total response. Because the eye velocity following rotation in light is low (Fig. 9E), the plateau in eye velocity of the post-rotatory nystagmus lasts longer than after rotation in darkness (Fig. 9A). The model predicts that after-nystagmus that follows rotation in light in a stationary surround would always be in the post-rotatory direction. This is because the response is dependent on a summation of activity in the direct vestibular pathway with that in the integrator. Since the direct pathway has a gain of 1 while the integrator

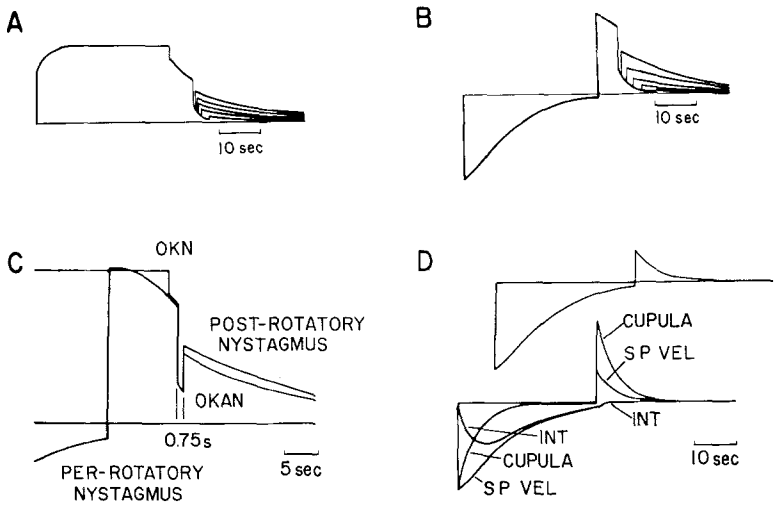


**Fig. 10. A** and **B** Model predictions of effects of varying periods of fixation-suppression on slow phase velocity during OKAN (**A**) and post rotatory nystagmus (**B**). Effects were similar but not identical in that OKAN lost activity more rapidly than vestibular nystagmus. This is shown in **C**. Superimposed OKAN and post-rotatory curves were subjected to a 0.75 period of fixation. The recovery velocity was greater for vestibular nystagmus than OKAN. **D** Model predictions for step of per-rotatory nystagmus in darkness and post-rotatory nystagmus in light. SP Vel is small in light despite the large cupula deflection because of activity in the direct visual pathway which cancels it. The time constant of the SP Vel decline in light is 4 s, the time constant of the cupula

can only be charged to about 0.85 of the total response (Cohen et al., 1977), an additional 15–20% of OKN would be necessary to abolish the post-rotatory response. This is consistant with the findings of Fig. 5B. Adding or subtracting OKN to rotation in light would give proportional increments in the velocity of the after-nystagmus, similar to findings in Fig. 5.

Fixation in the model is accounted for by the switches L and S. They are open in darkness. Switch L closes in light. When a stationary visual environment is observed after rotation or during OKAN, surround velocity is zero, but the integrator is still charged. This difference activates the fixation-suppression mechanism by closing switch S and discharges the integrator rapidly. The model predicts that vestibular nystagmus and OKAN would be discharged in a similar but not identical fashion (Fig. 10A and B). For an equal starting velocity vestibular nystagmus has a greater recovery velocity than OKAN and takes longer to decline (Fig. 10C). The same happens in the monkey (Fig. 7B and D).

The difference in recovery velocity of vestibular nystagmus and OKAN after fixation suppression occurs because the integrator but not the direct vestibular pathway, is susceptible to having its activity diminished by fixation suppression. During OKAN only the integrator is driving the eyes; consequently, its activity is discharged rapidly by fixation-suppression. During vestibular nystagmus, however, the cupula deflection continues to activate the integrator, making it less susceptible to suppression. The response predicted by the model for

per-rotatory nystagmus in darkness and post-rotatory nystagmus in light is shown in Fig. 10D. Slow phase velocity during after-nystagmus in light reaches a peak of about 40% of the response in dark and decays with a time constant of 4 s. This is essentially the response of the cupula. The integrator is not charged because the fixation-suppression mechanism is activated when the animal is in a lighted stationary surround. The model prediction closely approximates the experimental results shown in Fig. 7A.

## Discussion

The data demonstrate the importance of the interaction of the visual and vestibular systems in producing compensatory eye movements. Underlying this interaction is storage of activity related to eye velocity. It plays a key role in generating OKN, OKAN and vestibular nystagmus. The model predicts each of these responses as well as the visual-vestibular interaction. Specifically it accounts for the plateau in eye velocity during vestibular-nystagmus at low velocities, the response to combined vestibular and optokinetic stimulation during rotation in light, the sudden drop in slow phase velocity at the onset of OKAN, the larger time constant of vestibular nystagmus than OKAN, the post-rotatory response in light, and the greater effect of fixation suppression in reducing slow phase velocity of vestibular nystagmus.

The model predictions do not fit the data perfectly. At low eye velocities there is more overshoot in the model response (Fig. 9E) than in the monkey (Fig. 5A). The predicted time constant of OKAN and vestibular nystagmus does not decrease at higher velocities of stimulation in the model as in the monkey but this can be simulated if the parameter $h_o$ is modified by the velocity of stimulation. Because the model is piecewise linear, i.e., it is incrementally linear for specific ranges of input and state of the integrator (Stern, 1965), all waveforms will be exponential. Some of the data appear to change non-exponentially (see Figs. 6 and 8 of Cohen et al., 1977). This suggests that there are nonlinearities in the VOR that we have not taken into account. The usefulness of a simple approach is that single numbers (time constants) can be compared easily to experimental data to verify the theoretical predictions.

The storage element that we have identified is probably realized in a complex way by the nervous system, but can be represented by a single non-ideal or "leaky" integrator. In response to a step function the output of an ideal integrator rises linearly with a slope equal to the size of the step. When the input falls to zero, the output is maintained indefinitely. A non-ideal or "leaky" integrator has a continuous loss of activity that is proportional to its state or output; the greater the output, the greater the loss. For a step input its output rises exponentially to a steady level until the rate of loss equals the input. It will then hold this value for as long as the input continues, subserving a storage function. When the input ceases, its output falls to zero with a characteristic time constant.

In the model the integrator performs two major functions. It mediates activity coming from the visual system to stabilize ocular following when the

whole visual field is rotating (Cohen et al., 1977). OKAN is a consequence of having charged the integrator during OKN, and reflects its discharge in the absence of stimulation. The integrator also stores activity related to cupula deflection. This increases the time over which the VOR compensates for head rotation and effectively lengthens the dominant time constant of the ocular response. By storing or integrating the signal coming from the cupula, the storage mechanism gives a more faithful representation of head velocity during slow head movement than the cupula signal itself. Consequently, monitoring of activity from the integrator as well as the peripheral vestibular apparatus and the visual system could be important for perception of self and environmental motion.

A basic assumption in the model is that there is a central representation of environmental velocity from the visual system that excites both the direct pathways and the integrator. This signal is presumably obtained by combining retinal slip with an "efference copy" of eye velocity and a signal representing cupula deflection. The evidence for the existence of efference copy is sparse. It is based largely on a need for an internal feedback signal representing eye velocity to sustain ocular following in the absence of retinal slip. Efference copy has been useful in explaining the behavior of smooth pursuit movements (Yasui and Young, 1975; Lanman et al., 1978) and OKN (Koerner and Schiller, 1972; Cohen et al., 1977; Behrens and Grüsser, 1978). In the model efference feedback summates with retinal slip to supply a reference signal, surround velocity, to which the state of the integrator can be compared. A comparison of environmental velocity and the state of the integrator is a relatively simple way to decide whether to charge the integrator or to invoke the fixation-suppression mechanism that discharges it rapidly in the presence of a stationary surround (Cohen et al., 1977).

According to the model both the vestibular system and the visual system initially activate direct pathways. The word "direct" does not imply that the pathways are monosynaptic, only that they can change their activity levels rapidly and affect eye velocity at short latencies. The indirect pathway associated with the integrator changes its activity level more slowly, over seconds. Thus, in Fig. 9F at the end of the step in light, the cupula deflects instantaneously causing a rapid change in slow phase eye velocity, but the integrator discharges more slowly.

Both the direct visual and vestibular pathways have been represented by simple gain elements in the model, but they have different dynamics. The peripheral vestibular system responds rapidly to rotational stimulation, and the pathways from the eighth nerve to eye muscle motoneurons are direct and of short latency. A step in head velocity can be transmitted to the eye muscle in 5–25 ms, probably utilizing disynaptic and trisynaptic vestibulo-ocular reflex arcs. (See Precht, 1974; Wilson, 1972; Cohen, 1974, for review; also Baker and Highstein, 1975; Highstein, 1977). The direct visual pathways are slower. The response to a step in surround velocity takes about 150–300 ms for the initial rise in OKN slow phase velocity, although visually mediated ocular and postural responses can occur in 50–100 ms (Cohen et al., 1967; Nashner and Berthoz, 1978; Lanman et al., 1978). Direct visual-oculomotor pathways are not clear,

but may utilize the accessory optic system (Maekawa and Simpson, 1973) and cortico-ponto-cerebellar pathways (Baker et al., 1976).

Based on the loss of OKAN after lesions, the velocity storage mechanism appears to be associated with the vestibular nuclei, the medullary reticular formation, and the prepositus nucleus (Uemura and Cohen, 1973, 1975). In the monkey, within limits, frequencies of unit activity in the vestibular nuclei follow the state of the integrator shown in Fig. 9D during OKN and OKAN (Waespe and Henn, 1977b). Unit activity during rotation in dark (Waespe and Henn, 1977a) agrees more closely with slow phase velocity than with activity predicted for the integrator (Fig. 9B). This suggests that the signal in the vestibular nuclei is related to a summation of activity in the direct vestibular pathway and the integrator. It implies that the state of the integrator may be located outside the vestibular nuclei. One possible external site is the prepositus nucleus. Firing rates of prepositus units (Blanks et al., 1977) have a similar time course to predicted activity in the integrator (Fig. 9B). Local circuit neurons within the vestibular nuclei could also participate inthis function.

There are several types of integrators in the VOR that have different functions. The cupula-endolymph system of the peripheral labyrinth performs a mechanical integration on vestibular activity by transforming head acceleration into a signal representing head velocity over a wide range of frequencies (Steinhausen, 1933; Goldberg and Fernández, 1971). Another integrator performs a velocity to position transformation at the end stage of the VOR to establish the activity for the motoneurons to drive the ocular plant (Skavenski and Robinson, 1973). It is involved in holding the eyes steady during periods of fixation (Henn and Cohen, 1974; Keller, 1974). Its state is probably in the periabducens region (Keller, 1974; Raphan and Cohen, 1978). The integrator that we have described in the VOR has a different function, it stores activity related to eye velocity. Thus it is separate from the velocity to position integrator.

Although the two central integrators perform different functions, they could be realized by similar neuronal circuits with different time constants. However, there should be differences in the way they are activated and discharged during nystagmus induced by a constant velocity input. The velocity to position integrator should be discharged by each quick phase and start to recharge at the beginning of each slow phase. Consequently, it should never achieve a steady level of activity. Activity in the velocity-storage integrator on the other hand should be maintained at a steady level during a constant input and not be influenced by the presence or absence of quick phases or the position of the eyes.

As yet neither the way that storage nor integration is achieved by neural circuitry is known. It is clear, however, that the integrator is a basic building block of oculomotor organization. An understanding of how it is realized and used by the central nervous system is fundamental for understanding oculomotor control.

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## Appendix

*Derivation of the relationship between the dominant time constant of vestibular nystagmus and the dominant time constant of OKAN.*

In darkness, all visual inputs to the oculomotor system are inactivated. Consequently $r_o = 0$, $L = 0$, and $S = 0$. For rotation to the left, the state equation may be given as:

$$\begin{aligned} \dot{x} &= -h_{0L}\, x + g_{0L}\, r_v \\ y &= x + r_v \end{aligned} \tag{1}$$

where $r_v = V_1 \exp(-t/T_c)$. $V_1$ is the magnitude of the step in head velocity and $T_c$ is the dominant time constant of the cupula return.

The zero state response to this differential equation is

$$y(t) = \frac{g_{0L}}{h_{0L} - 1/T_c} V_1[\exp(-t/T_c) - \exp(-h_{0L}t)] + V_1 \exp(-t/T_c) \tag{2}$$

The dominant time constant associated with this response may be found by integrating y(t) over $(0,\infty)$ and then dividing by $V_1$. (See Cohen et al. (1977) for definition and details). Therefore,

$$\int_o^\infty y(t)dt = V_1 T_c\, (h_{0L} + g_{0L})/h_{0L} \tag{3}$$

But, $1/h_{0L} = T_o$, the characteristic time constant of the integrator and $1/(h_{0L} + g_{0L}) = T_r$, the equivalent closed loop charging time constant of the integrator. Thus if $T_v$ is defined as the dominant time constant of the vestibular response, dividing Eq. (3) by $V_1$ gives.

$$T_v = (T_c / T_r)\, T_o \tag{4}$$

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