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Model for the Translational Vestibuloocular Reflex (VOR)

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Musallam, Wissam S. and R. D. Tomlinson. Model for the translational vestibuloocular reflex (VOR). *J. Neurophysiol.* 82: 2010–2014, 1999. The function of the translational vestibuloocular reflex (tVOR) and the angular vestibuloocular reflex (aVOR) is to stabilize images on the retina during translational and rotational motion, respectively. It has generally been assumed that these two reflexes differ in their central processing because they differ significantly in their primary afferent behavior and characteristics at the motor level. So far, models of the tVOR have focused on the type of processing that the primary afferent signal must undergo before reaching the neural integrator. Here, we propose a model that does not require any prefiltering. It is known that the eye plant requires signals in phase with velocity and position. We propose that the velocity signal is obtained directly from the neural integrator, whereas the position signal is obtained directly from the primary afferents synapsing onto the oculomotor nuclei. This design proved sufficient to simulate eye movements in response to translational motion.

INTRODUCTION

The translational vestibuloocular reflex (tVOR) stabilizes gaze in response to translational movements. This is a complicated task because the peripheral otolith organs respond identically to tilt and to linear translation, whereas the ocular response to these stimuli differ; tilting of the head (sensing gravity) elicits torsional eye movements, whereas translations elicit horizontal eye movements. In addition, to accurately maintain images on the fovea, the brain must consider the target distance and eccentricity of the image with respect to each eye (Paige and Tomko 1991a,b; Telford et al. 1997).

Perhaps the greatest obstacle to understanding the tVOR has been the elucidation of the necessary central processing required to transfer the otolith primary afferent signals into appropriate oculomotor commands. Otolith primary afferents encode a signal that is in phase with, and even leads, linear head acceleration, although these data are only available for frequencies up to 2 Hz (Fernandez and Goldberg 1976; Goldberg et al. 1990). In contrast, the canal afferents, measured to frequencies up to 8 Hz, encode head velocity (Fernandez and Goldberg 1971).

The oculomotor plant is known to require signals in phase with velocity and position (Robinson 1981). Single-cell recordings from position-vestibular pause (PVP) cells and eye-head-velocity (EHV) cells in the vestibular nucleus have shown that these cells carry otolith signals in phase with linear velocity, indicating that the acceleration signal coming from the peripheral organs has already been processed (McConville et al. 1996; McCrea et al. 1996). Several models have been proposed

that attempt to produce a velocity and a position signal from the incoming acceleration signal while attempting to maintain consistency with observed high-pass filtering behavior (Angelaki 1998; Paige and Tomko 1991b; Telford et al. 1997). The most popular hypothesis is that of integrating the primary otolith afferent signal and then sending the integrated signal to the neural integrator and the oculomotor plant (Paige and Tomko 1991b; Telford et al. 1997). Alternatively, it has been proposed that the acceleration signal be differentiated and then integrated (Angelaki et al. 1993). However, we propose a simpler model. Specifically, primary afferent signals are passed through the neural integrator to yield the velocity signal. The position signal is obtained directly from the primary afferents because a signal in phase with acceleration is just a signal in phase with position but with the sign reversed. Therefore a second integrator is not needed, suggesting that the brain stem dynamics of the tVOR reflex are different from those of the angular vestibuloocular reflex (aVOR) only because the respective inputs to the brain stem differ. There is evidence that there exists monosynaptic primary afferent innervation of the oculomotor nuclei of otolithic origin (Uchino et al. 1996). We propose that this signal acts as the position input in response to translational movement. A simple model is presented that has as its input an acceleration signal. Then, given this model and the selected parameters and after computing the difference between the output of the model and the experimental data, we shall deduce the exact input (high-frequency primary afferent behavior) necessary to accurately reproduce the experimental data.

METHODS

The model shown in Fig. 1A was written using Matlab's Control System (Mathworks) package. The neural integrator and the oculomotor plant are expressed as (Fuchs et al. 1988).

$$H_p = \frac{(1 + s\tau_1)}{(1 + s\tau_2)(1 + s\tau_3)(1 + s\tau_4)}$$

$$H_{\text{int}} = \frac{1}{(1 + s\tau_{\text{int}})}$$

where $\tau_1 = 0.14$ s, $\tau_2 = 0.28$ s, $\tau_3 = 0.037$ s, $\tau_4 = 0.003$ s, and $\tau_{\text{int}} = 20$ s.

The open loop transfer function of the model in Fig. 1A is simply

$$\frac{\dot{E}}{a} = (K_1 + K_2 H_{\text{int}}) H_p e^{0.01s} \quad (1)$$

where K_1 provides the system with position information, $K_2 H_{\text{int}}$ with velocity information, $e^{0.01s}$ represents a 10-ms delay, and a is the acceleration. The output from Eq. 1 (Eye velocity) was compared with Angelaki's data (Angelaki 1998) and the values K_1 and K_2 optimized to minimize both phase and gain errors. Because the Angelaki data are

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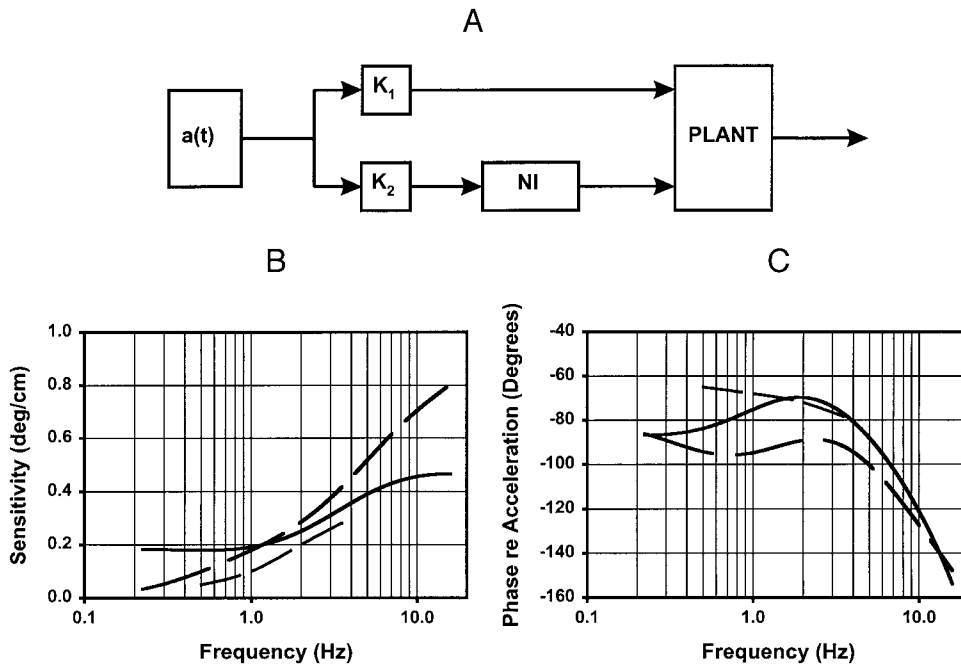


FIG. 1. A: model presented in this paper. $a(t)$, acceleration; NI, neural integrator; EP, eye plant; K_1 and K_2 are as discussed in METHODS. B: comparison of sensitivity (deg/s/cm/s = deg/cm) and phase (C) values for the output produced by the model (solid line) shown in A, with $K_1 = 1.0$ and $K_2 = 100$ for a pure acceleration signal, and data from Angelaki (1998) (long dashed line) and Telford (1997) (short dashed line up to 4 Hz). The phase from the model leads by up to 20° at 2 Hz while the sensitivity has a smaller slope and a greater intercept. Phase for this and all subsequent plots is relative head acceleration.

available to frequencies beyond 15 Hz, we have chosen to model this data instead of the Telford data, which is included for completeness. Nevertheless, our results show that the phase of the model's output differs from the Telford data by $\sim 10^\circ$ between 1 and 4 Hz (Fig. 3). For a pure acceleration input, the appropriate values for K_1 and K_2 were deduced by minimizing the least-squared difference between the experimentally obtained complex number ge^{ip} (where g is the gain and p is the phase) and the one produced by the model. Other minimization methods were also used without any significant change to the values of K_1 and K_2 . Then, the difference between the output of the model using the derived K_1 and K_2 and the experimental data were computed. This difference corresponds to the required filtering of the acceleration signal to adequately simulate the experimental data. The difference between the two outputs was fitted to an equation according to the Goldberg et al. (1990) classification of afferents (see RESULTS).

RESULTS

Figure 1, B and C, depicts the output of the model (solid line) for $K_1 = 1.0$ and $K_2 = 100$ as compared with experimental data [Angelaki 1998 (long dashed line up to 15 Hz) and Telford et al. 1997 (short dashed line up to 4 Hz)] for a pure acceleration input. There is a fairly good phase agreement between the two plots with the largest phase difference occurring at 2 Hz where the output of the model lags the experimental results of Angelaki by $< 20^\circ$. However, the value of the model's sensitivity curve is greater than those found experimentally below 1 Hz. As the frequency increases, the slope of the model's sensitivity curve is smaller than the experimental one, and as the frequency increases further, the sensitivity curve levels off.

From Fig. 1, it can be seen that to accurately simulate the experimental data, the model still needs a slowly rising high pass filter and an almost flat phase response, exhibiting a 20° phase lag as the frequency increases. Up to 2 Hz, this is the behavior of utricular regular afferent neurons recorded by Goldberg et al. (1990). The two curves shown in Fig. 1 extending up to 15 Hz (dashed line is Angelaki data, solid line is model output) were divided into each other and the result

labeled H_{aff} . H_{aff} represents the required filtering of the acceleration signal (primary afferent behavior) so that the output of the model agrees with the Angelaki data. H_{aff} was then fitted according to Goldberg et al. (1990) classification of primary afferents. Specifically, the overall transfer function describing otolith primary afferent behavior is

$$H(s) = H_M(s) \cdot H_V(s) \cdot H_A(s)$$

where

$$H_M(s) = \left[\frac{1}{(1 + s\tau_{M1})(1 + s\tau_{M2})} \right]^{K_M}$$

$$H_V(s) = [(1 + s\tau_{V1})(1 + s\tau_{V2})]^{K_V}$$

$$H_A(s) = [(1 + s\tau_A)]^{K_A}$$

Representative values of the parameters are $\tau_{M1} \approx 3$ s, $\tau_{M2} \approx 0.10$ s, $K_{M1} \approx 0.15$, $\tau_A \approx 15$ s, $K_A \approx 0.13$, $\tau_{V1} \approx 200$ s, and $\tau_{V2} \approx 1$ s (Goldberg et al. 1990). For H_{aff} , all values were as above except for $\tau_{V2} = 0.25$, $K_V = 0.15$, and $H_V = [(1 + s\tau_{V1})(1 - s\tau_{V2})]^{K_V}$. This results in a signal that has a phase response consistent with a very regular primary afferent but with a gain described by a dimorphic afferent (see Fig. 2). Indeed, this could be the behavior of some afferents above 2 Hz, because for primary afferents recorded by Goldberg et al. (1990) the phase began to lag acceleration as the frequency increased.

Figure 2 depicts the output of the model in response to an input of a regular primary afferent and a dimorphic primary afferent. The dimorphic afferent ($K_V = 0.16$; Fig. 2, A and B) converges onto the experimentally deduced sensitivities but with as much as a 50° phase lead at 1 Hz. For the dimorphic afferents, least-square optimization resulted in $K_1 = 3.5$ and $K_2 = 80$. In contrast, the regular afferent ($K_V = 0.01$, $K_1 = 0.8$, and $K_2 = 1$) approximates the experimental phase curve almost perfectly but with a large loss in sensitivity (Fig. 2, C and D). Figure 3 depicts the output of the model in response to the

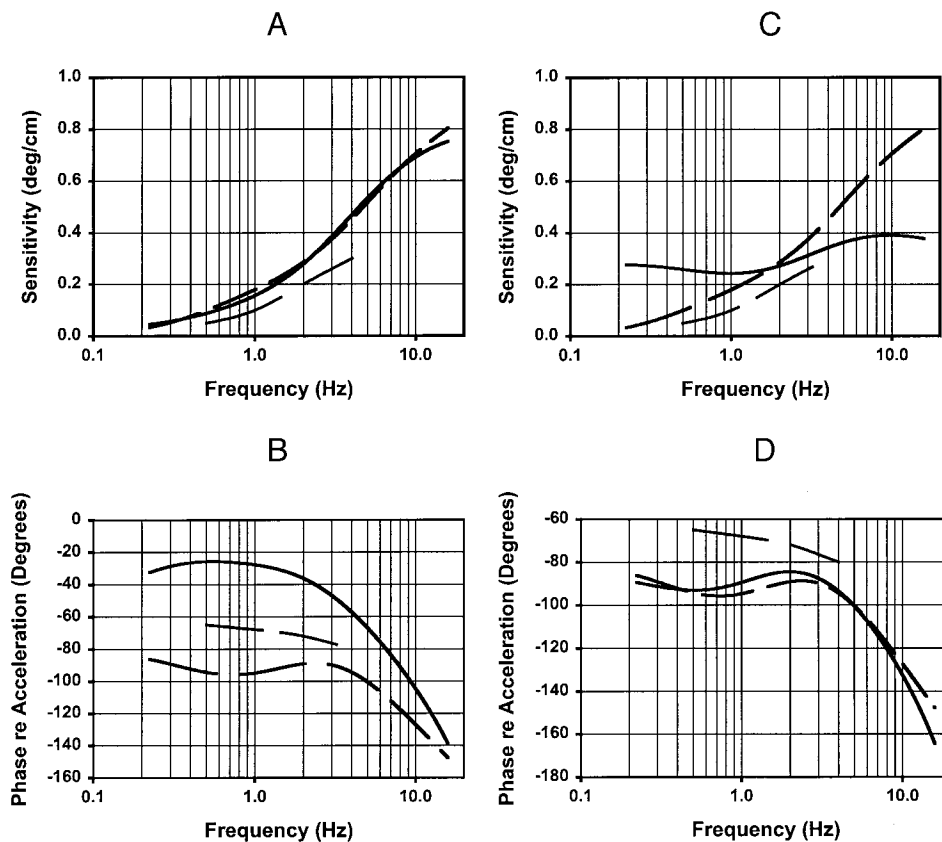


FIG. 2. Comparison of the sensitivity (A) and phase (B) of experimental translational vestibuloocular reflex (tVOR) data (---) and the model (—) in Fig. 1 for a primary afferent input with $K_V = 0.16$ (dimorphic), $K_1 = 3.5$, and $K_2 = 80$. The sensitivity curve is almost identical with that of Angelaki (1998) (long dashed line extending to 15 Hz), and with a slight adjustment in gain, can also be made to accurately reproduce Telford (1997) data (short dashed line extending to 4 Hz). However, up to a 60° phase lead is introduced. Also shown is a comparison of the sensitivity (C) and phase (D) of experimental tVOR data (---) and the model (—) in Fig. 1 for a primary afferent input with $K_V = 0.01$ (highly regular), $K_1 = 0.8$, and $K_2 = 1$. In contrast with A and B, the phase curve is almost identical with that of Angelaki (1998). However, the sensitivity is flat and exhibits a high intercept.

input composed of the combined behavior derived from the regular and dimorphic afferents shown in Fig. 2. Least-square optimization resulted in values of $K_1 = 1.2$, $K_2 = 150$, $\tau_{V2} = 0.25$, and $K_V = 0.15$. The combined behavior of the afferent is represented by H_{aff} derived above. The model's sensitivity curve (solid line) closely resembles that of Angelaki (Angelaki 1998), whereas the maximum phase difference is a lag of 10° and occurs at ~ 1 Hz.

DISCUSSION

As shown in Fig. 1A, the tVOR pathway in our model is identical to the Robinson model for the aVOR. However, because the canal primary afferents encode angular head velocity and the utricular primary afferents encode linear head acceleration, the input to the integrator from the two systems is different. The question is, how are the utricular primary afferent signals processed to provide the eye plant with the necessary velocity and position signals.

The model presented here is simple in that it takes advantage of known pathways in the brain stem. For horizontal conjugate eye movements, the nuclear prepositus hypoglossi (NPH) is an important site for neural integration. There is a large projection of inputs from the lateral vestibular nucleus (LVN) onto the NPH in the squirrel monkey (Belknap and McCrea 1988) and a corresponding large projection of utricular afferents onto the LVN (McCrea et al. 1987). No second integration of the otolith signal is required. The position signal is obtained directly from the primary afferents with a modification in gain. Utricular afferents synapsing directly onto oculomotor nuclei have been

reported by Uchino et al. (1996). Because an acceleration signal is in phase with position, this makes the signal that the otolith primary afferents carry adequate to code position.

In deriving H_{aff} , it became clear that the behavior of regular afferents is more suited to drive the system. Unfortunately, no afferent data are available for frequencies > 2 Hz. However, H_{aff} gives an idea of the type of afferent behavior needed to realize the model presented here. It is consistent with extrapolated regular afferent (bordering on dimorphic) behavior; a slow rising high-pass filter with a flat phase response increasing in lag as the frequency increases. We are not suggesting the existence of a new class of afferents. The behavior of primary afferents for frequencies > 2 Hz is not known. Therefore H_{aff} may represent the response of several primary afferents converging onto central neurons or even the central processing of primary afferent signals. Either way, given H_{aff} , a signal that deviates slightly from observed low-frequency afferent signals, the model shows how existing pathways may be used to reproduce tVOR behavior.

The high-frequency phase lag exhibited by the response of the model is partly due to the 10-ms delay that was used while fitting the afferent transfer function. This short latency is consistent with Angelaki's result (Angelaki 1998).

The simplicity of the model presented here would also generate horizontal eye movements during sustained head tilts. The model has as its purpose only the reproduction of horizontal eye movements. Clearly additional circuitry and processing is required to inhibit horizontal eye movements during tilts. The mechanism involved in differentiating between translations and tilts is currently not known and has

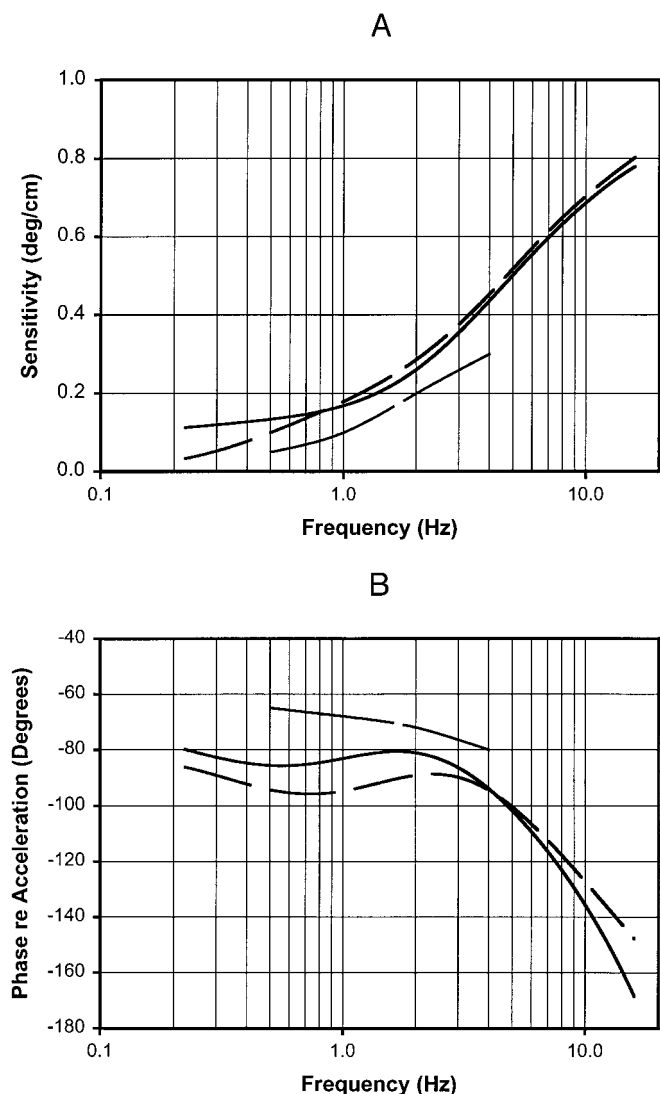


FIG. 3. Output of the model from an input composed of a combination of the behavior of the primary afferents from Fig. 2. The model's sensitivity curve (—) is quite close to experimental values, whereas the phase exhibits up to a 10° lag below 4 Hz. $K_1 = 1.2$ and $K_2 = 150$, $K_V = 0.16$. $\tau_{V2} = 0.25$, $K_V = 0.15$, and $H_V = [(1 + s\tau_{V1})(1 - s\tau_{V2})]^{K_V}$.

not been included in the current model. Several hypotheses have been proposed to account for the tilt/translation duality. The most popular of these hypothesis is frequency filtering (Telford et al. 1997). According to this hypothesis, torsional eye movements are produced by a low-frequency pathway while horizontal eye movements by a high-frequency pathway. Because primary afferent neurons synapse directly onto the motoneurons (Uchino et al. 1996), this hypothesis fails to fully explain the observed behavior. Nevertheless, this mechanism would simply require additional circuitry to differentiate between the two stimuli. Once the brain has decided that the stimulus is translational in nature, then the output of this hypothesized tilt/translational filtering will just feed the model presented here.

For angular rotations, an integrator lesion leads to an inability to keep gaze steady (no position signal) (Cannon and Robinson 1987). Perhaps the most striking consequence

of our model is that on integrator lesions, a partial loss of eye movements in response to translational motion will occur, although some eye movement may still occur due to the monosynaptic primary afferent connection to the plant. Another consequence of this model is that irregular primary afferents have little or no effect on the behavior of the tVOR. Because the behavior of primary afferent neurons for frequencies >2 Hz is not known, this prediction is based solely on theory. Galvanic current studies for the aVOR have shown that irregular afferents do not contribute to the aVOR (Minor and Goldberg 1991). Also, regular and irregular inputs remain segregated at the level of the vestibular nuclei (Goldberg et al. 1987), although this segregation is incomplete. However, this could result in parallel pathways for the primary afferents that have distinct functions. We have shown here that it is possible for the function of the regular afferent to be to provide the integrator with input to obtain the velocity command. The function of the irregular afferent remains a question. The VOR is not the only reflex that these afferents drive. Therefore the irregular afferents could be used for the vestibulocollic reflex (Goldberg et al. 1987) or even to adjust the gain of the tVOR for vergence sensitivity.

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