[33] L. Stark and P. M. Sherman, "A servoanalytic study of consensual pupil reflex to light," *J. Neurophysiol.*, vol. 20, 1957, pp. 17–26.
[34] L. Stark, H. van der Tweel, and J. Redhead, "Pulse response of the pupil," *Acta Physiol. Pharmacol. Neer.*, vol. 11, 1962, pp. 235–239.
[35] J. F. Terdiman, "Neurophysiological mechanisms in the pupillary control system," Ph.D. dissertation, Univ. Illinois, Urbana, 1970.
[36] J. F. Terdiman, J. D. Smith, and L. Stark, "Pupil response to light and electrical stimulation: static and dynamic characteristics," *Brain Res.*, vol. 16, 1969, pp. 288–292.
[37] G. W. H. M. Van Alphen, S. L. Robinette, and F. J. Macri, "The adrenergic receptors of the intraocular muscles of the cat," *Int. J. Neuropharmacol.*, vol. 2, 1964, pp. 259–272.
[38] L. H. van der Tweel and J. J. Denier van der Gon, "The light reflex of the normal pupil of man," *Acta Physiol. Pharmacol. Neer.*, vol. 8, 1959, pp. 52–88.
[39] D. Whitteridge, "The transmission of impulses through the ciliary ganglion," *J. Physiol.*, vol. 89, 1937, pp. 99–111.
[40] B. L. Zuber, J. L. Semmlow, and L. Stark, "Frequency characteristics of the saccadic eye movement," *Biophys. J.*, vol. 8, 1968, pp. 1288–1298.

A Model of Eye Movements Induced by Head Rotation

NOBORU SUGIE AND G. MELVILL JONES

Abstract—**It is well known that head rotation will induce eye movements known as rotational nystagmus, the slow phase of which compensates for head rotation fairly well, and the quick phase of which takes place intermittently in the opposite direction to the preceding slow phase. From both frequency and transient responses, it is confirmed that the slow phase velocity is proportional to the output of the semicircular canal, the main transducer of head rotation. The relationship between the canal output and the quick phase is also discussed. A simple model is proposed in which the quick phase and slow phase are separately generated.**

In cats under controlled ether anesthesia, it is found that both phases of the rotational nystagmus can be decomposed into primary and secondary components, and a new model of the vestibulo-ocular system is proposed which includes the simultaneous influence of these two components. The model is analyzed to find a condition where the summed effect of primary and secondary components of response constituting the slow phase of rotational ocular nystagmus can be made proportional to the canal output. Many simulation results are presented to demonstrate the validity of the model.

Introduction

IT IS WELL KNOWN that head movements (especially head rotation) elicit eye movements [1], that is, the eyes tend to move to compensate for head motion. This would be very useful if one tries to fixate one's eyes on a stationary target. The nonvisual component of this response is known as the vestibulo-ocular reflex.

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Visual target tracking ability with the head remaining still is known to be relatively poor in man [1]. Thus a target motion at more than 2–3 Hz cannot be tracked satisfactorily. However, one's head in daily life is under frequent active and passive rotation, the highest frequency component of which often exceeds 4 Hz [1], and yet under such a situation, one can see stationary targets fairly well. This is most likely due to the automatic compensation mechanism for head rotation, that is, the vestibulo-ocular reflex.

The studies in this field have been undertaken for many years by otolaryngologists. However, there have been few attempts to model the system mathematically [3]. In this paper new experimental data obtained by the present authors as well as data from other authors are investigated to yield a mathematical model. In this model the eye movements induced by head movements consist of two components. The two components seem to have close affinity to the saccadic and smooth components in visual target tracking [4]. Through the model, the contribution of each component is analyzed to show the condition of perfect compensation for head movements. The theoretical analyses are confirmed by analog computer simulation.

Responses to Head Rotation

In order to obtain the eye movements induced purely by head rotation, the subject is asked to sit on a turntable which can be turned around a vertical axis with his eyes shut or covered. The horizontal eye movements can be recorded by so-called dc electro-oculography (EOG) [2]. Photoelectric or contact lens method of recording cannot easily be used in such a situation. The eye movements thus measured are relative to the head.

Under certain conditions, when the subject seems to be well alerted, the so-called rotational nystagmus can be observed as is typically shown in Fig. 1. It can be seen that there are two components in the eye movements. One of them (slow phase) tends to compensate for the head rotation, whereas the other (quick phase) consists of a train of flicks. It is to be noted that each flick takes place in the opposite direction to the preceding slow phase; also that left and right are reversed in Fig. 1.

The direct signal flow in this system is fairly well known [1]. The head rotation detected by the three pairs of semicircular canals is coded into a train of neural pulses and sent to the vestibular nuclei in the brain stem. The vestibular nuclei connect with the three motor nuclei (oculomotor, trochlear, and abducens nuclei), which send the final signals to the extraocular muscles via the corresponding cranial motor nerves; other connections of the vestibular nuclei are not considered here.

The dynamics of the semicircular canal has been studied by various authors [1], [6]–[8]. The transfer function can be represented as

$$\frac{C(s)}{V(s)} = \frac{K_1 s}{(T_1 s + 1)(T_2 s + 1)} \tag{1}$$

where

$C(s)$	Laplace transform of canal output.
$V(s)$	Laplace transform of head angular velocity.
K_1	A constant.
T_1, T_2	Time constants ($T_1 \gg T_2$).
s	Laplace operator.

It is reported that in man and cat

$$T_1 = 10\text{–}20 \text{ s}$$
$$T_2 < 0.1 \text{ s}. \tag{2}$$

Then it can easily be seen that the canal output is approximately proportional to the head angular velocity over the frequency range of $(1/T_1 - 1/T_2)$ rad/s.

The relationship between canal output and slow phase of nystagmus will now be considered. Several authors pointed out that the slow phase velocity seems to be proportional to the canal output [8], [9]. Actually, some authors measured the canal time constants T_1, T_2 from the slow phase of nystagmus. With newly obtained neurophysiological data the aforementioned inference seems to have been confirmed [7], [10]. Therefore, it is possible to calculate the proportionality constant between the canal output and slow phase velocity in the following way.

From the transient and frequency responses of the slow phase, the time constant T_1 can be measured first. The time constant T_2 is so small that it is neglected in the following discussions since the frequency range of interest is relatively low. Then, assuming that $K_1 = T_1$ in (1), the canal output $c(t)$ in response to an arbitrary head rotation can be calculated. For example, the unit step change in head angular velocity will yield

$$c(t) = \exp(-(t/T_1)). \tag{3}$$

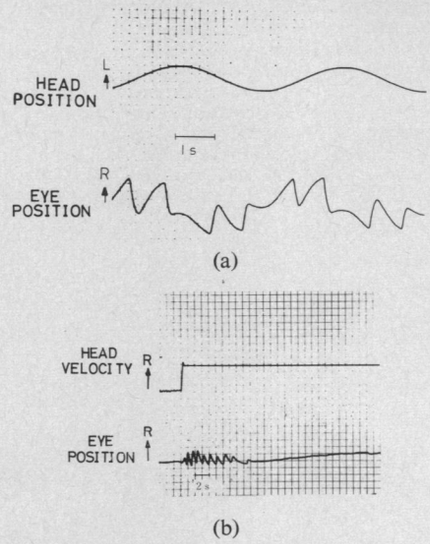

Fig. 1. Typical rotational nystagmus in cat. (a) Frequency response. Top trace gives head position relative to space, amplitude of which is about 30° (peak to peak). Bottom trace gives eye position relative to head. It may be said that slow phase approximately compensates for head movement. (b) Transient response. Top trace shows jump of head angular velocity from 0 to 60°/s. Bottom trace gives eye position relative to head. Slow phase velocity of eye around onset of nystagmus is likely to be approximately equal to head angular velocity.

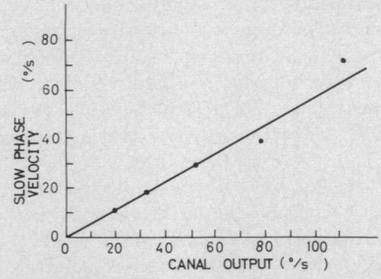

Fig. 2. Relationship between calculated canal output and slow phase velocity. Abcissa represents canal output at end of 9 s of constant angular acceleration at various values. Ordinate represents slow phase velocity corresponding to canal output.

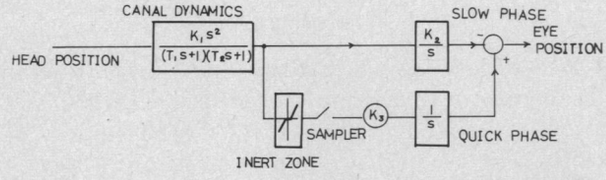

Fig. 3. Quick phase–slow phase model. Sample period is constant.

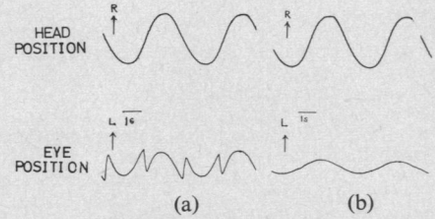

Fig. 4. Frequency responses [5]. (a) With quick phase. (b) Without quick phase.

Thus one can correlate the canal output with the slow phase velocity. In Fig. 2 an example of such calculation is shown for which it is assumed that slow phase eye velocity is equated to the canal output. The raw experimental data are from Brown and Crampton [11]. The time constant T_1 in this case turns out to be 14.0 s. From Fig. 2, the proportionality constant is approximately 0.57. Similarly from frequency responses [8], the constant turns out to be 0.64. Therefore, even if the canal output is equal to the head angular velocity in the angular frequency range of $(1/T_1 - 1/T_2)$ rad/s, the slow phase of rotational nystagmus apparently compensates for the head rotation by only 60 percent or so.

The mode of quick phase generation is next considered. Since the quick phase takes place in opposite direction to the preceding slow phase, the direction of quick phase is directly related to the canal output. That is, the sign of the canal output uniquely determines the direction of quick phase. Dependency of the frequency and amplitude of quick phase upon the canal output is not clear. But by and large, one can say that amplitude and frequency increase with canal output. It is interesting to note that the quick phase (saccadic) interval is seldom shorter than about 200 ms, as is the case in visual tracking. In the following discussions it is assumed for simplification of analysis that the frequency of quick phase is constant and that the amplitude is proportional to the canal output.

From the preceding discussions, a mathematical model as shown in Fig. 3 can be conceived in which the slow phase and quick phase are generated separately. But in the next section, new experimental results are shown suggesting that the model in Fig. 3 is inadequate.

Decomposition of Responses

The experimental results described in this section are mainly from cats under controlled ether anesthesia. By appropriately controlling ether anesthesia, one can selectively suppress the quick phase of nystagmus. The control of functional depth of anesthesia is not easy and the suppression of quick phase could be observed only transiently, which of course imposes limitations on the accuracy of results.

In Fig. 4 eye movements from a cat with and without quick phase are shown. It can be seen that the peak eye positions without quick phase (Fig. 4(b)) are leading those of the corresponding slow phase when quick phase is present (Fig. 4(a)). The eye movement with absent quick phase will be termed the primary component.

Primary Component

The frequency characteristics of the primary component relative to head position (i.e., including canal dynamics) are shown in Fig. 5. The amplitude of the response is, of course, not stable since as already indicated, it is dependent on the anesthetic level. But the phase characteristics are relatively stable. Therefore, only the phase characteristics are shown in Fig. 5. Since the primary component is

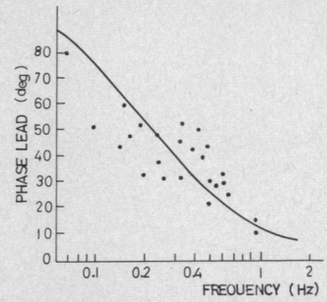

Fig. 5. Phase characteristics of eye movements relative to head position with suppressed quick phase. Phase angle is considered to be zero when eyes are in opposite phase to head. Experimental data $(\cdot)$ and $[K_1K_3 s^2/(T_1s + 1)(T_3s + 1)]$ $(-)$ are shown, where $T_1 = 10$ s and $T_3 = 0.69$ s.

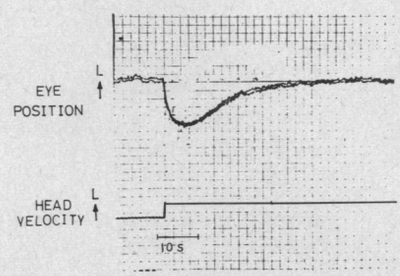

Fig. 6. Primary component in response to step change in head angular velocity. Top trace gives eye position relative to space. Note that there are no saccades at all. Compare response to that in Fig. 1(b). Bottom trace gives head angular velocity. Step change is about 30°/s [5].

considered to be generated by the canal output, the dynamics between the canal output and the primary component can be easily obtained. The dynamics can be approximately represented as a first-order lag system:

$$\frac{K_3}{T_3s + 1}. \tag{4}$$

The transfer function in (4) can be considered as a composite of a muscle-eyeball system and the neural network between the canal output and the muscle-eyeball system.

Fig. 5 shows the phase characteristics of the overall system:

$$\frac{K_1K_3s^2}{(T_1s + 1)(T_2s + 1)(T_3s + 1)} \tag{5}$$

where $T_1 = 10$ s, $T_2 = 0$ s, and $T_3 = 0.69$ s. In a cat the time constant T_1 is approximately 5–10 s [7], [10]. Because of the difficulties in experimentation, the data in Fig. 5 exhibit considerable randomness, but the general tendency is clear and the model in (5) can be said to fit the data reasonably well.

The time constant T_3 can also be measured from transient responses. A typical response of the primary component to a step change in head angular velocity is shown in Fig. 6. In this case the primary component $E_1(t)$ should be represented as

$$E_1(t) = [\exp(-t/T_1) - \exp(-t/T_2)]. \tag{6}$$

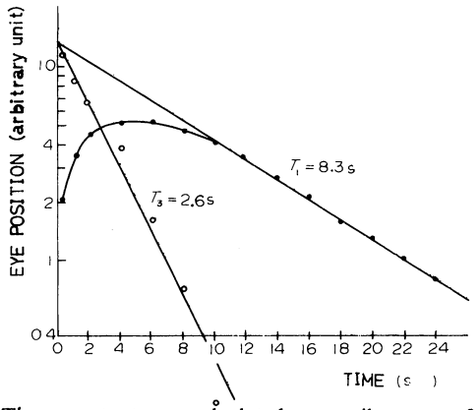

Fig. 7. Time constants are calculated on semilog paper from record in Fig. 6.

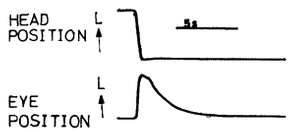

Fig. 8. Primary component in response to step change in head angular position.

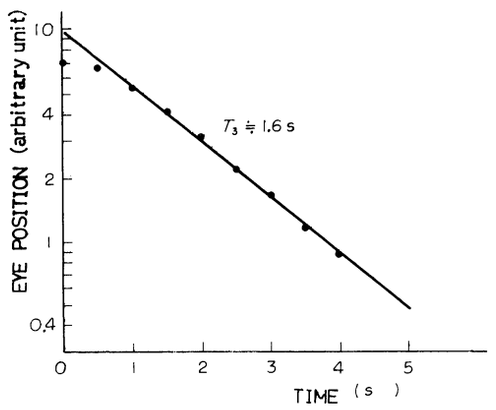

Fig. 9. Time constant T_3 is obtained from data in Fig. 8.

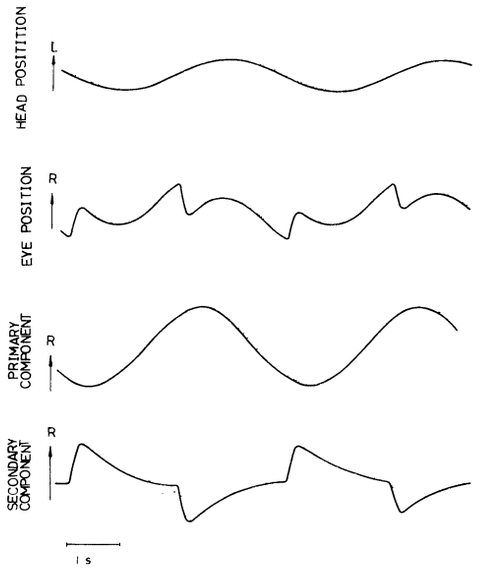

Fig. 10. Decomposition of eye movements into primary and secondary components.

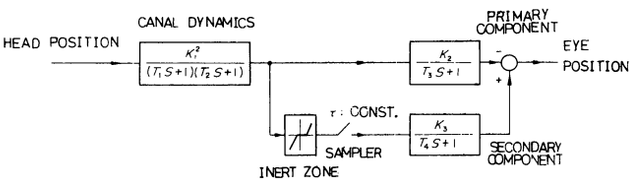

Fig. 11. Improved mathematical model.

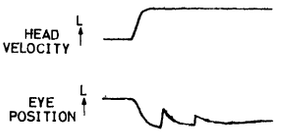

Fig. 12. Eye movements with only two quick flicks.

Therefore, by plotting the data in Fig. 6 on a sheet of semilog paper, one can determine the time constant T_3 as well as T_1 as is shown in Fig. 7. The value of T_3 is rather large compared to that in Fig. 5. In another cat the value of T_3 obtained as in Fig. 7 was 1.3 s. The difference between response dynamics of the primary component alone and that of the slow phase of nystagmus in which quick phase movements are included is remarkable and can be seen by comparing Fig. 6 with Fig. 1(a). Experiments such as that in Fig. 6 were difficult to perform since it proved difficult to apply a sufficiently large stimulus without inducing nystagmus. Consequently, only a few successful results were obtained from this method.

A response of the primary component to a step change in head angular position is shown in Fig. 8 where again the quick phase has been eliminated as before. The response of the primary component should be

$$E_1(t) = (1/T_3)\exp(-t/T_3) - (1/T_1)\exp(-t/T_1) \fallingdotseq (1/T_3)\exp(-t/T_3). \quad (7)$$

The time constant T_3 is then obtained from Fig. 9 as about 1.6 s. The mean value obtained in this way from six cats is 1.75 s. Thus from both frequency and transient responses, the validity of (5) as representing the primary component is confirmed, although the value T_3 varies somewhat from experiment to experiment.

Secondary Component

The present results suggest that a secondary component of oculomotor response is introduced when quick saccades are interspersed in the response to produce nystagmus. It is assumed then that the overall rotational nystagmus is an algebraic sum of the primary and secondary components. In Fig. 10 an example of decomposition of a frequency response into the two components is shown.

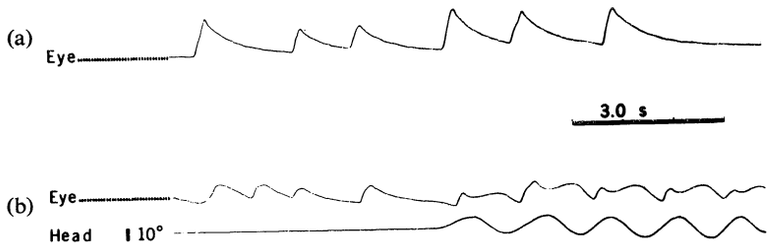

Fig. 13. (a) Spontaneous nystagmus. (b) Spontaneous and induced movements.

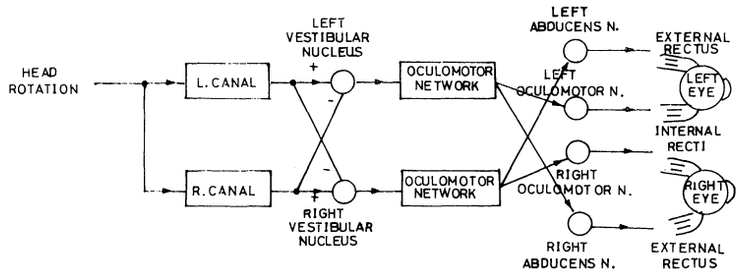

Fig. 14. Horizontal eye movements induced by head rotation.

These records were obtained from an experiment in which the lightly anesthetized cat was rotated sinusoidally at 0.25 Hz and an amplitude of about 30 deg (peak to peak). This frequency lies within the presumed angular velocity range of transduction, and hence the head angular velocity is presumed to give the canal output. Thus the head angular velocity may be considered the effective input to the remaining part of the system, i.e., the output of "canal dynamics" in Fig. 11.

The next trace gives eye position relative to the head and shows a typical nystagmoid pattern of compensatory oculomotor response obtained without vision; it does not differ materially from the response to be expected in the unanesthetized animal [12]. The third trace (primary component) gives the sine wave of response obtained by passing the canal output through the box marked $K_2/(T_3 s + 1)$ in Fig. 11. Note that this trace thus becomes phase-advanced relative to the stimulus and to the nystagmus trace of real eye movement. The bottom trace is the result of algebraic subtraction of the primary response from the real eye movement. Alternatively, it may be said that the recorded trace of real eye movement is quantitatively the sum of the two bottom traces, i.e., the sum of primary and secondary components. It can be seen that the secondary component consists of a series of exponentially decaying functions taking place in synchrony with each quick phase. The quick phase in the secondary component is about equal to the original quick phase. Each slow phase in the secondary component turns out to be an exponential decay. The time constant T_4 of the exponential function in the secondary component in this case is about 0.67 s.

Overall Response

As discussed in the preceding section for analytical treatment, the quick phase is here assumed to be directly related to the canal output in the sense that its frequency of occurrence is constant and its amplitude is proportional to the canal output at every instant.

Then the overall system for the rotational nystagmus can be represented as in Fig. 11. In this model the primary and secondary components are generated separately. Two first-order systems are included in place of the two integrators in Fig. 3. In Fig. 3 the output of each integrator tends to increase infinitely if the canal output is a nonzero constant. Since the infinite output is physically impossible, the model in Fig. 3 is not realistic.

The validity of the model in Fig. 11 can be confirmed from other evidence. In Fig. 12 a transient response where the quick phase takes place only twice through controlled ether anesthesia is shown. This response lies midway between those in Fig. 6 and Fig. 1(b). Such a response can easily be simulated by the model in Fig. 11. The time constant T_4 can be determined from such a response. From Fig. 12, the time constant T_4 turns out to be about 1.3 s.

At times, so-called spontaneous nystagmus can be observed as in Fig. 13(a) and the first part of Fig. 13(b) in spite of no head movement. Such a fairly regular repetitive pattern can easily be simulated if the canal output is a nonzero constant in the model. The primary component becomes constant in the steady state in this case, so that the ac component of the eye movement must be solely due to the secondary component which is of a regular sawtooth-like waveform. The time constant T_4 measured from spontaneous nystagmus is about 1.0 s. It is conceivable that because of anesthesia, some sort of unbalance takes place in the neural network equivalent to the nonzero constant canal output and that this provides a rational explanation of spontaneous nystagmus.

In Fig. 14 the horizontal eye movement system induced by head rotation is shown. In this system a pair of canal outputs is connected differentially at the level of the brain stem nuclei.

TABLE I
MEAN VALUES OF TIME CONSTANTS MEASURED UNDER VARIOUS CONDITIONS

Responses \ Time constants*		T_1	T_3	T_4
Frequency responses	with quick phase	(10.5)	-	0.9
	without quick phase	-	0.7	-
Transient responses (step change in angular velocity)	with quick phase	(14.0) 6.6	-	1.3
	without quick phase	9.5	1.9	-
Transient responses (step change in angular position)	with quick phase	-	-	-
	without quick phase	-	1.8	-
Spontaneous nystagmus		-	-	1.0

* Data in parentheses obtained from humans; other data from cats. Data given in seconds.

Interdependence of Primary and Secondary Components

The mean values for T_1, T_3, and T_4 calculated from various kinds of experiments are shown in Table I. It can be seen that T_3 and T_4 are nearly equal to each other. It would be appropriate to consider the interdependence of primary and secondary components. As has been pointed out, the slow phase velocity is proportional to the canal output. The canal output is in turn nearly proportional to the head angular velocity over the frequency range encountered in daily life. Thus in most cases the slow phase can compensate for the head rotation to some extent at least. But without the secondary component, the eyes cannot compensate for the head rotation satisfactorily. For example, in frequency responses the primary component is leading too much as shown in Fig. 5, and in transient responses as in Fig. 6, the primary component would first move in the same direction as the head.

Therefore, the slow phase can be realized only through proper combination of the primary and secondary components. Thus it is concluded that in many situations the slow phase can be made proportional to the canal output only when the secondary component is generated properly. This matter will be discussed in the next section.

ANALYSIS OF MODEL

The model in Fig. 11 is investigated in this section. For simplicity it is examined here only under the condition that the slow phase can be made proportional to the canal output. Therefore, an appropriate value of K in Fig. 15 will be chosen so as to make the slow phase velocity of $r(t)$ proportional to $c(t)$. Let the sample period be constant and τ. The sampling is assumed to be taking place at the instants $t = 0, \tau, 2\tau, \cdots$, and $\dot{r}(+0), \dot{r}(\tau + 0), \dot{r}(2\tau + 0), \cdots$ are compared with $c(+0), c(\tau + 0), c(2\tau + 0), \cdots$.

First, the case of a unit step input will be considered:

$$c(t) = \begin{cases} 1, & t \geq 0 \\ 0, & t < 0. \end{cases}$$

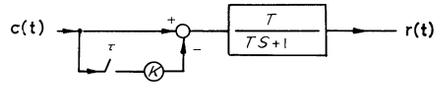

Fig. 15. Model to be analyzed.

It is required that

$$\dot{r}(i\tau + 0) = \dot{r}((i + 1)\tau + 0), \qquad i = 0,1,2,\cdots.$$

Then it can be shown that

$$K = T[\exp(\tau/T) - 1] \fallingdotseq \tau, \qquad \tau \ll T \tag{8}$$

and

$$\dot{r}(i\tau + 0) = \exp(\tau/T) \fallingdotseq 1 = c(t). \tag{9}$$

Namely, if $K \fallingdotseq \tau$, then the slow phase velocity of $r(t)$ is nearly equal to $c(t)$.

As for the case of a ramp input,

$$c(t) = \begin{cases} t, & t \geq 0 \\ 0, & t < 0. \end{cases}$$

The requirement to K is that $\dot{r}(i\tau + 0)$ increase at a constant rate with i. Then it can readily be shown that

$$K = \frac{T^2[1 - \exp(\tau/T)]^2}{\tau \exp(\tau/T)} \fallingdotseq \tau, \qquad \tau \ll T \tag{10}$$

and

$$\dot{r}(i\tau + 0) = iT[\exp(\tau/T) - 1] \fallingdotseq i\tau = c(i\tau + 0). \tag{11}$$

Therefore, if $K \fallingdotseq \tau$, then the slow phase velocity of $r(t)$ is nearly equal to $c(t)$.

Finally, let

$$c(t) = \begin{cases} \sin \omega t, & t \geq 0 \\ 0, & t < 0 \end{cases}$$

and

$$\omega n\tau = 2\pi, \qquad n = 1,2,\cdots.$$

Namely, one period of $c(t)$ is assumed to be integral times of τ. This assumption will be permitted when the period

$2\pi/\omega \gg \tau$. Then it can be shown that

$$K = \frac{T^2}{1 + \omega^2 T^2} \frac{[1 - \exp(n\tau/T)]}{\sum_{i=0}^{n} \sin(\omega i\tau)^{i\tau/T}} \quad (12)$$

and

$$\dot{r}[(mn + l)\tau + 0] = \sin(\omega l\tau) = c[(mn + l)\tau + 0]. \quad (13)$$

Similarly, in the case of

$$c(t) = \begin{cases} \cos \omega t, & t \geq 0 \\ 0, & t < 0 \end{cases}$$

it can be shown that

$$\dot{r}[(mn + l)\tau + 0] = \cos(\omega l\tau) = c[(mn + l)\tau + 0] \quad (14)$$

by proper choice of K. Therefore, from (13) and (14), a similar conclusion can be applied to a sinusoidal input $c(t)$ with arbitrary phase.

The preceding discussion can easily be solved in the following way. The $s(t)$ in Fig. 16 represents the slow phase or $r(t)$. Assume that

$$0 < \omega \leq (1/2)2\pi(1/\tau)$$

where $s = j\omega$. Then from the sampling theorem,

$$S(s) = (T/Ts + 1)C(s) - K(T/Ts + 1)(1/\tau)C(s) + (K/s)(1/\tau)C(s) = (1/s)C(s) \quad (15)$$

if $K = \tau$. From this it may be said that if the input frequency is lower than $1/2\tau(\fallingdotseq 2$ Hz, for $\tau \fallingdotseq 0.25$ s), then the slow phase of $r(t)$ is equal to the integral of $c(t)$.

The overall system in Fig. 11 may now be considered again. When the input frequency is lower than 2 Hz, the slow phase velocity of rotational nystagmus is proportional to the canal output. When the frequency increases above 2 Hz, this relationship does not hold. But the first-order lag system with $T_3 = T_4 = T \fallingdotseq 1$ s acts as an approximate integrator in this frequency range. Therefore, the slow phase velocity will still be proportional to the canal output.

For $\omega \leq 1/T_1 \fallingdotseq 0.1$ rad/s, the canal output is leading in phase, and the gain is small in relation to the head angular velocity. Therefore, the slow phase velocity of nystagmus cannot compensate for the head angular velocity except when $1/T_1 < \omega < 1/T_2$ rad/s. For $\omega \leq 1/T_1$, one can track stationary targets with ease, so that it would not cause any trouble in daily life.

Simulation

In this section typical simulated results are shown. In Fig. 11, it is assumed that $T_1 = 10$ s, $T_2 = 0.1$ s, $T_3 = T_4 = 1$ s, and $K_2 = K_3$.

Fig. 17 corresponds to the responses to a step change of head angular velocity. Fig. 17(a) is very similar to Fig. 1(b), noting that in Fig. 17 stimulus is recorded as head position and in Fig. 1 as head velocity. Again, Fig. 17(b) is similar to Fig. 12. In Fig. 17(b) the secondary component is reduced by raising the threshold of the inert zone and by lowering the frequency of quick phase. It may be noted that in the bottom trace of Fig. 17(a) the sum of "head" and "eye" traces gives eye movement relative to space, and it can be seen how the outcome is effectively intermittent eye stabilization.

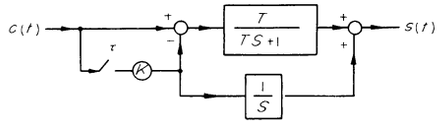

Fig. 16. Modified model $s(t)$ is equal to slow phase of $r(t)$.

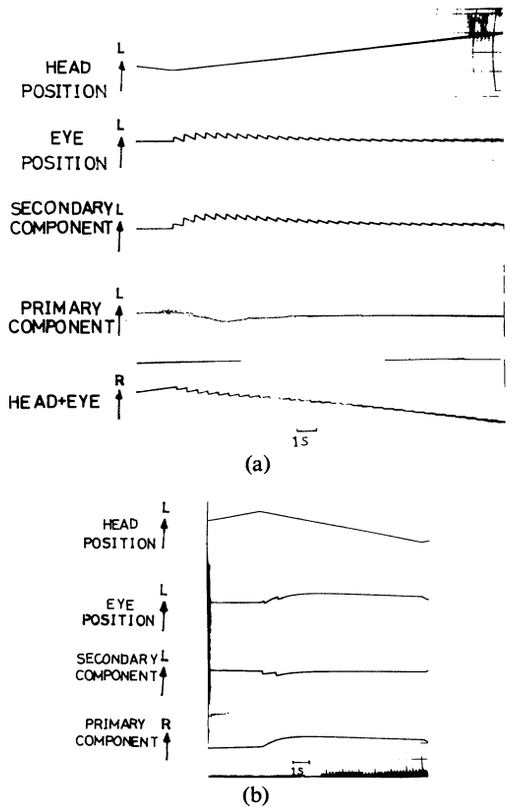

Fig. 17. Simulated transient responses where head-plus-eye is equal to eye relative to space. (a) Balance between primary and secondary components is proper, cf. Fig. 1(b). (b) Balance is not proper. Secondary component is insufficient, cf. Fig. 12.

In Fig. 18 frequency responses are shown. Keeping the primary component intact, the secondary component is adjusted by changing the frequency of quick phase and the threshold of inert zone. In Fig. 18(a) the primary component is too large and the slow phase is overcompensating for the head rotation. On the contrary, the compensation is insufficient in Fig. 18(c). In Figs. 18(b) and (d) the compensation is just enough as can be seen from the staircase-like head-plus-eye movements, i.e., eye movement relative to space. These results correspond rather nicely to Fig. 1(a).

At low frequencies, the frequency response becomes that shown in Fig. 19. Because of the canal dynamics, the slow phase is leading too much to compensate for the head

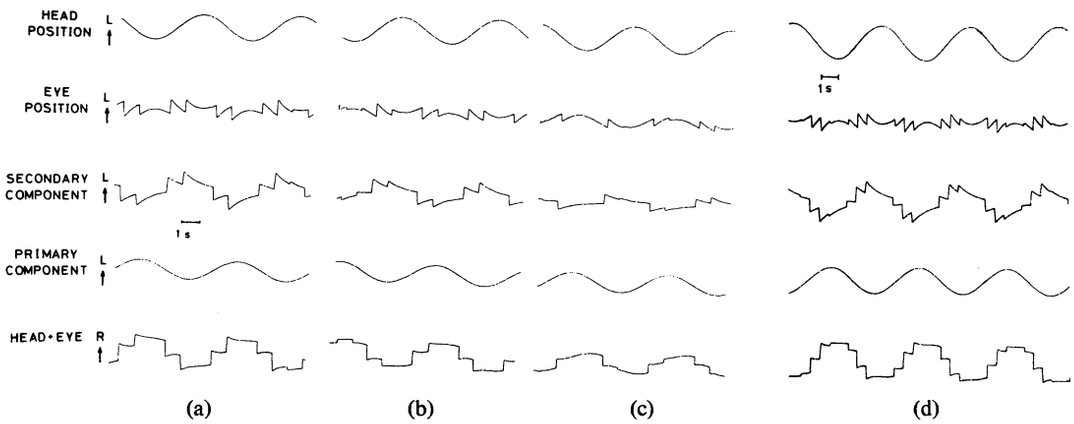

Fig. 18. Simulated frequency responses, cf. Fig. 1(a). (a)–(c) Frequency of quick phase is same, but threshold of inert zone is twice higher in (b) and four times higher in (c) than that in (a). In (a) compensation for head rotation is excessive, in (b) it is just enough, and it is insufficient in (c). (d) Threshold is five times higher than that in (a), but frequency of occurrence of quick phase is higher than in (a); therefore, compensation is just enough.

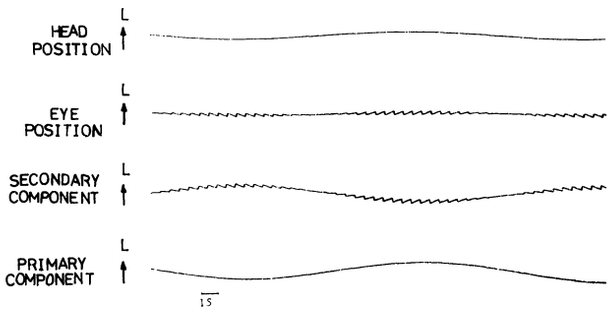

Fig. 19. Simulated frequency response at low frequency. Slow phase is leading too much to compensate for head rotation.

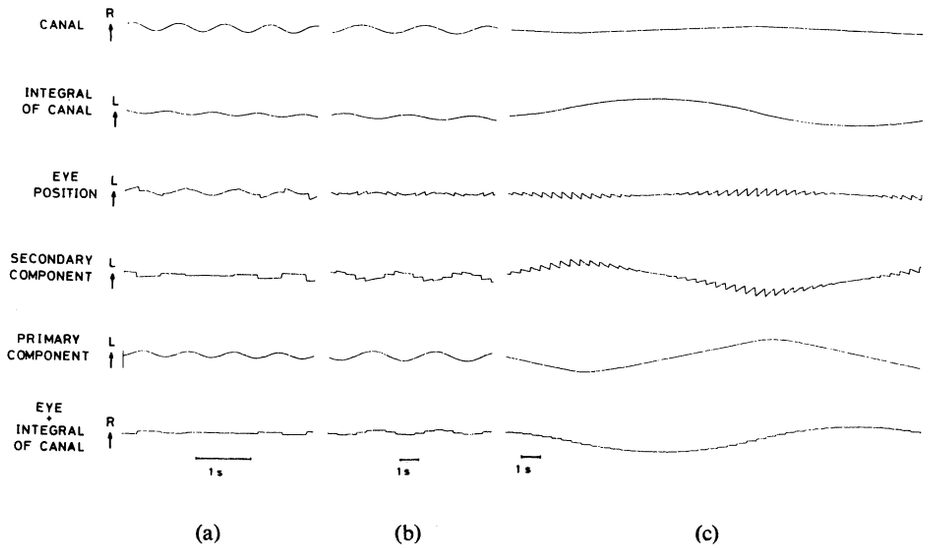

Fig. 20. Simulation is performed in same condition. It can be seen that slow phase is compensating for integral of canal output as suggested in model analysis.

rotation, as has been repeatedly demonstrated to be the case in man [8], [14], [15].

Fig. 20 shows that over a wide frequency range, the slow phase compensates for the integral of the canal output. Similarly, the spontaneous nystagmus can be simulated quite easily. Thus the proposed model seems to satisfy a wide range of requirements imposed by both theory and experiments.

Discussion

It is well known that the vestibulo-ocular reflex arc is susceptible to anesthetic agents and mental states such as sleep. In the attempt to decompose vestibulo-ocular responses into two components under controlled ether anesthesia, it is quite important to confirm that the findings are still meaningful despite this susceptibility. There are several points to be brought out here.

First, in the lightest state of ether anesthesia, the vigorous nystagmus induced by sinusoidal head rotation was indistinguishable from similar nystagmus induced in normal unanesthetized cats in the dark [12].

Second, the first observation of the effect of the time constant T_3 was in animals in which this condition of light anesthesia generating good brisk nystagmus was slightly changed to one in which 50 percent of the cycles contained a fast clear-cut saccade placed in a clear-cut sequence of smooth pursuit movements. In Fig. 21 it can be seen that the peak positions of the slow phase soon after the onset of a quick flick are phase-leading those just before the onset of a quick flick. At this depth of anesthesia, there was no indication of the breakdown of the saccade itself, which comes with deeper anesthesia.

Third, the fact that the phases with and without a preceding saccade were determined over the same stretch of record necessarily implies that both conditions obtained at the same level of anesthesia, i.e., the observed phase difference with and without the preceding saccade, could not be accounted for by a changing level of anesthesia.

A fourth point might be that the smooth response to a step change in head angular velocity produced the expected 10-s canal time constant for T_1 as well as a T_3 value similar to that determined from the sinusoidal data referred to in the preceding (see Figs. 6 and 12).

Young and Stark [4] developed a model of visual tracking. Smooth pursuit movements in visual tracking might correspond to the slow phase in vestibulo-ocular nystagmus. In the model by Young and Stark, the pursuit system acts as a velocity servo to rotate the eyes at the same angular velocity as the target. During the slow phase in the vestibulo-ocular nystagmus, the eyes are rotated at the velocity approximately equal to canal output. The semicircular canal can be regarded as a head angular velocity transducer over a certain frequency range. It should be noted that the slow phase is dependent upon both primary and secondary components. This is due to the existence of T_3. The essence of the model proposed in this paper is T_3, but it does not appear at all in the model of visual tracking.

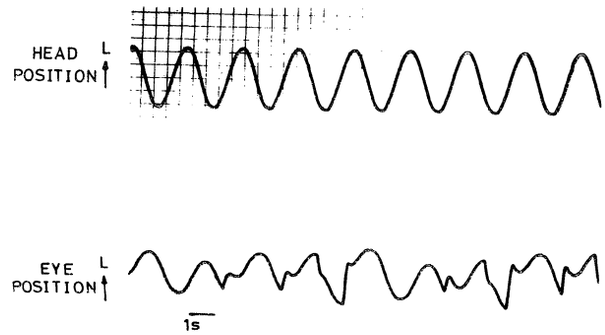

Fig. 21. Frequency response with only one flick per cycle.

In the model of visual tracking the saccadic system acts as a position servo to direct the eyes at the target. Purely saccadic eye movements look like staircases. The secondary component in the vestibulo-ocular system can be compared with saccadic eye movements. The secondary component consists of a series of exponentially decaying functions instead of steps.

A major outcome of the experiments in the cat under controlled ether anesthesia is that the presence of a quick saccade has been demonstrated to quite substantially modify the immediately subsequent slow phase movement. For example, compare Fig. 1(b) with Fig. 6. A little while after the sudden change in head angular velocity, the slow eye movement in Fig. 6 changes its direction, and the eyes move in the same direction as the head. But in Fig. 1(b), the slow phase is always in the opposite direction from that of the head. Thus the slow phase in Fig. 1(b) is due for the most part to the secondary component. The saccades are shown not only to effect the velocity gain of the whole system, but also to have a sizeable effect on the phase of compensatory response. An important systems implication is that the presence of saccades has the effect of markedly extending the frequency band of compensatory response in the oculomotor system which would be substantially curtailed by the filtering effect of the time constant T_3 in the absence of saccades.

The usefulness of the model proposed in this paper is to point quantitatively to the presence of a biological process in the oculomotor system which has not previously been demonstrated and indeed could not have been demonstrated without the quantitative systems approach. In doing so the model pinpoints a particular feature in such a way that it becomes available for further experimental investigation. Obvious questions raised are where does T_3 act (i.e., in the peripheral mechanical plant, or the central generation of the oculomotor neural message) and what physiological mechanisms are responsible (e.g., muscle mechanics, neural rebound, etc.). Relevant studies are attempted [10].

Additional questions in a more applied sphere are the following. Does the same effect manifest in humans? What implications does the presence of T_3 in the oculomotor system have in regard to the use of oculomotor response in clinical assessment of vestibular function? Other questions raised by the model relate to the suggested neural wiring diagram, the mechanism of triggering in the saccadic

pathway, and the degree of optimization achieved in the placement and size of saccades actually incurred. The vestibulo-ocular responses in man during sleep is under study by the present authors in relation to the model. Visual target tracking with active head rotation is also related to the model [16].

Conclusions

1) The slow phase of rotational nystagmus compensates for the canal output which is proportional to the head angular velocity over a certain frequency range.

2) The quick phase is directly related to the canal output, that is, the direction of the quick phase is uniquely determined by the polarity of the canal output.

3) The frequency of occurrence and amplitude of the quick phase are related to the canal output in some complicated way. But in the present model analysis, it is assumed that the frequency is constant and the amplitude is proportional to the canal output at the corresponding instant.

4) In a cat under controlled ether anesthesia, it is found that the rotational eye nystagmus can be decomposed into two components: the primary and secondary components. Thus a mathematical model as in Fig. 11 is proposed.

5) The primary component can be generated when the canal output is fed into a first-order lag system with a time constant of about 1 s.

6) The secondary component can be obtained if the primary component is subtracted from the rotational nystagmus. This component can be modeled by supplying a train of pulses to a first-order lag system with a time constant of about 1 s. The pulses take place in synchrony with the quick phase. The amplitude and polarity of the pulse are nearly equal to those of the quick phase.

7) Analysis of the model shows that by appropriate combination of the primary and secondary components, the slow phase velocity can be made proportional to the canal output.

8) The results of simulation data uniformly support the preceding conclusions.

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References

[1] G. M. Jones and J. H. Milsum, "Spatial and dynamic aspects of visual fixation," *IEEE Trans. Bio.-Med. Eng.*, vol. BME-12, Apr. 1965, pp. 54–62.
[2] E. Marg, "Development of electrooculography. Standing potential of the eye in registration of eye movement," *Arch. Ophthalmol.*, vol. 145, 1951, pp. 169–174.
[3] W. C. Hixson and J. I. Niven, "Application of the system transfer function concept to a mathematical description of the labyrinth," Bureau of Medicine and Surgery, Project MR 005.13.6001, Subtask 1, Rep. 57, 1961.
[4] L. R. Young and L. Stark, "Variable feedback experiments testing a sampled data model for eye tracking movements," *IEEE Trans. Hum. Factors Electron.*, vol. HFE-4, Sept. 1963, pp. 38–51.
[5] N. Sugie and G. M. Jones, "A mathematical model of the vestibulo-ocular system," in *Dig. 6th Int. Conf. Medical Electronics and Biological Engineering*, 1965, pp. 422–423.
[6] A. A. J. Van Egmond, J. J. Groen, and L. B. W. Jongkees, "The mechanics of the semicircular canal," *J. Physiol.*, vol. 110, 1949, pp. 1–17.
[7] H. Shimazu and W. Precht, "Tonic and kinetic responses of cat's vestibular neurons to horizontal angular acceleration," *J. Neurophysiol.*, vol. 28, 1965, pp. 991–1013.
[8] W. C. Hixson and J. I. Niven, "Frequency responses of the human semicircular canals, pt. II: nystagmus phase as a measure of nonlinearities," U.S. Naval School of Aviation Medicine, Rep. 73, 1962.
[9] G. Aschan, "The mechanism of the cupula ampullaris in man," *Acta Soc. Med. Upsal.*, vol. 60, 1955, pp. 77–98.
[10] N. Sugie, N. Mano, and T. Kasai, "System analysis of eye movement system at neuronal level," in *Proc. 1968 IFAC.*
[11] J. H. Brown and G. H. Crampton, "Quantification of the human nystagmic response to angular acceleration. Prediction formulae and nomograph," *Acta Oto-Laryngol.*, vol. 58, 1962, pp. 555–564.
[12] H. Zuckerman and G. M. Jones, "Adaptation to unilateral semicircular canal inactivation," in *Proc. Aerospace Medical Ass.*, 1968, pp. 3–4.
[13] H. Shimazu and W. Precht, "Inhibition of central vestibular neurons from the contralateral labyrinth and its mediating pathway," *J. Neurophysiol.*, vol. 29, 1966, pp. 467–492.
[14] J. L. Meiry, "The vestibular system and human dynamic space orientation," NASA, Rep. CR-628, 1966.
[15] J. Outerbridge, "Experimental and theoretical study of reflex vestibular control of head and eye movement," Ph.D. dissertation, McGill Univ., Montreal, P. Q., Canada, 1969.
[16] N. Sugie and M. Wakakuwa, "Visual target tracking with active head rotation," *IEEE Trans. Syst. Sci. Cybern.*, vol. SSC-6, Apr. 1970, pp. 103–109.