Physiology of Peripheral Neurons Innervating Semicircular Canals of the Squirrel Monkey.

II. Response to Sinusoidal Stimulation and Dynamics of Peripheral Vestibular System

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A CENTRAL PROBLEM in vestibular physiology has been the elucidation of the dynamics of the peripheral vestibular apparatus. Following the development of the torsion-pendulum model by Steinhausen (24, 25), many efforts have been directed toward the determination of the time constants of the model. That the model, itself, might not adequately describe the transfer characteristics of the peripheral system has seldom been questioned. Further, the procedures employed to estimate the time constants have had their drawbacks.

The dynamics of the system have sometimes been deduced from hydrodynamic principles (5, 6, 15, 17, 22, 23). Such an analysis permits the inference of only some of the relevant variables. In the torsion-pendulum model, it will be recalled, the angular deflection of the cupula $\xi(t)$ is related to the angular acceleration $a(t)$ by the equation

$$\frac{d^2\xi(t)}{dt^2} + \frac{\Theta}{\Pi} \frac{d\xi(t)}{dt} + \frac{\Delta}{\Pi} = \Theta a(t)$$

$\Theta$ being the moment of inertia of the cupula-endolymph, $\Pi$ the viscous-damping couple, and $\Delta$ the elastic-restoring couple. The response of the system is governed by two time constants, $\tau_1 = \Pi/\Delta$ and $\tau_2 = \Theta/\Pi$. Some estimate can be made by hydrodynamic calculations of the time constant $\tau_2$. However, the more important time constant $\tau_1$ cannot be determined, since the elastic-restoring couple has not been directly measured. Another limitation of the hydrodynamic approach is that it ignores the question as to what extent the dynamics of the peripheral apparatus are determined by the mechanics of the cupula-endolymph system and to what extent by the physiology of the hair cells and of the nerve fibers innervating them.

An alternate approach has been to study the dynamics of the overall response of the system, usually in humans. Response measures have included subjective estimates of the sensation of bodily rotation (5, 10, 13), vestibular-induced nystagmus (10, 13, 19), and the oculogyral illusion (11). Such studies have resulted in estimates of both $\tau_1$ and $\tau_2$. But it is not clear whether these estimates simply reflect peripheral sensory mechanisms, as is usually assumed, or are influenced by the complicated physiology of the central vestibular pathways.

One aim of the present series of experiments was to describe the dynamics governing the response of peripheral vestibular afferents to angular accelerations. Some observations of relevance were presented in the previous paper (9). There the response to steps of constant angular acceleration was considered. The present paper continues this analysis. The response to sinusoidal stimulation will be described. From these observations, a descriptive model of the peripheral vestibular system is devised; the model, even though it is the simplest linear system which will approximate the response dynamics, is more elaborate than
a torsion pendulum. The theoretical response of the model to acceleration steps is then calculated and compared to the actual step response of the neurons.

**METHODS**

Observations were made in the same animals as were used in the previous paper (9), where a full description of techniques may be found. Sine-wave inputs to the velocity servomechanism were produced by a function generator (Wavetek, model 116), whose distortion was less than 0.5%. The frequency was varied over a range extending from 0.006 to 8.0 Hz (see Table 1). Over this range, the velocity of the rotating device faithfully followed the input signal except that, at 8.0 Hz, the device exhibited a phase lag of some 8°. A stimulus sequence consisted of an initial stationary period of 20 sec, during which the resting discharge could be determined. There followed a period of sinusoidal oscillations, succeeded by a stationary or recovery period of 40 sec duration. The number of oscillations was varied from 4 at the low end of the frequency spectrum to 256 at the high end. The response was assumed to reach a steady state in not less than 20 sec after the start of stimulation.

Estimates of the amplitude and phase of the steady-state response were made from its fundamental component, extracted by a Fourier analysis of the average response to several successive sine-wave cycles. Gains were calculated by dividing the response amplitude, obtained from the Fourier analysis, by the magnitude of the peak acceleration (or that of the peak velocity). The analysis was also used to measure the nonlinear distortion and to compute the 2nd through 10th harmonics. Such calculations are meaningful only if the neuron's discharge is not silenced during a portion of the stimulus cycle. When the discharge was silenced, amplitude, phase and distortion measurements were based on a nonlinear regression scheme which fitted the average response to a sine wave clipped at a value of zero.

**RESULTS**

The response to sinusoidal stimulation was studied in 57 units; 28 of the neurons innervated the horizontal canal, 19 the superior canal, and 10 the posterior canal. In all the units, sinusoidal stimulation was begun only after the response to a series of velocity trapezoids had been studied. We first investigated the response at several sine-wave frequencies, the peak acceleration for each being that listed in Table 1. The table also includes the number of units studied at each frequency. Then, if the unit was still isolated, we held the frequency constant and varied the peak acceleration in 6-dB steps from the maximum listed in the table down to 10° (and sometimes 5°)/sec². Intensity series for at least one frequency were completed in 12 units.

**Pattern of response to sinusoidal stimulation**

The typical course of the response to sine waves is illustrated by data obtained from unit 51-8 (Fig. 1). The stimulus frequency was 0.0504 Hz. The discharge, after a transient period lasting roughly .5 cycle, waxes and wanes around the resting level in an approximately sinusoidal manner. When the stimulus ends, the firing rate returns to the resting level with an exponential time course.

A more detailed steady-state analysis of the unit's response is presented in Fig. 2. The stimuli were all 0.0504 Hz and the peak acceleration was varied from 5.0 to 80.6°/sec². Each curve represents the average of the response to 6 successive cycles, the period of analysis beginning 1.5 cycles after the start of stimulation. The resulting functions are roughly sinusoidal in shape, though it will be noted that they are not symmetrically disposed around the resting level. When the different curves are compared, the response is seen to be proportional to acceleration magnitude and the phase lag, amounting to some 58°, is more or less constant. Gain

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**TABLE 1. Stimulus conditions**

<table>
<thead>
<tr>
<th>Frequency, Hz</th>
<th>Maximum Peak Acceleration, deg/sec²</th>
<th>No. of Units</th>
</tr>
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<tbody>
<tr>
<td>.006</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>.0125</td>
<td>20</td>
<td>30</td>
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<td>.025</td>
<td>40</td>
<td>52</td>
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<td>.050</td>
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<td>.25</td>
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<td>54</td>
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<tr>
<td>1.00</td>
<td>160 or 320</td>
<td>51</td>
</tr>
<tr>
<td>2.00</td>
<td>160 or 320</td>
<td>27</td>
</tr>
<tr>
<td>4.00</td>
<td>160 or 320</td>
<td>22</td>
</tr>
<tr>
<td>8.00</td>
<td>160 or 320</td>
<td>8</td>
</tr>
</tbody>
</table>
FIG. 1. Response of unit 51-8 (superior canal) to 8 cycles of a sinusoidal stimulus, 0.0504 Hz, 80.6°/sec². Stimulus ends at 185.72 sec. Each point, average discharge rate for 1/40th of sine-wave cycle (0.496 sec). Vertical marks, instants of peak excitatory acceleration. Lower and upper horizontal lines, respectively, resting discharge before and after stimulation.

FIG. 2. Steady-state responses of unit 51-8 (superior canal) to series of 0.0504 Hz sine waves. See key for accelerations. Zero degrees, peak excitatory acceleration. Inset plots gain and phase as functions of acceleration; included are points for one acceleration (10.1"/sec²) not shown in main graph.
PHASE ANGLE re MAXIMUM EXCITATORY ACCELERATION

FIG. 3. Steady-state response to sinusoidal stimulus. Points, average response to three successive sine-wave cycles; curve, best-fitting sine-wave function centered around resting discharge (horizontal continuous line). A: unit 55-8 (horizontal canal), 0.0243 Hz, 38.9°/sec²; resting discharge, 114.0 spikes/sec; average discharge during sine-wave cycle, 114.5 spikes/sec. B: unit 49-3 (superior canal), 0.0241 Hz, 38.8°/sec²; resting discharge, 150.8 spikes/sec; average discharge (horizontal dashed line), 159.9 spikes/sec.

and phase data are summarized in the inset. The gain did not vary by more than 10% over the 16-fold range of stimulus magnitudes and the phase angle varied by no more than 3°. A similar constancy of gain and phase values was observed in the other 11 units studied in this way.

Distortion analysis

In the preceding paper (9) it was shown that the response of the peripheral vestibular system could, under certain circumstances, be quite nonlinear. Such nonlinearities are also seen in the response to sine waves. Unit 55-8 (Fig. 3A) was unusual in that the data points were quite closely fit by a sinusoidal function symmetrically placed around the resting level. The total nonlinear distortion was 8.8%. Unit 49-3 (Fig. 3B) was more typical. Here there are obvious discrepancies between the best-fitting sine wave and the actual response. The distortion is largely of an asymmetrical type, the peak excitatory response being larger than the peak inhibitory response. One consequence is that the average discharge rate during stimulation is higher than the resting level. In such a situation, it is useful to quote two distortion figures. The first, the unrestricted distortion, reflects the deviation of the response from a sine wave whose mean value is adjusted to the average rate and, hence, involves only the harmonics of the fundamental frequency. The other, the restricted distortion, also includes the distortion introduced by the shift in d-c level. For unit 49-3, the unrestricted distortion was 8.6%, the restricted distortion 13.9%.

Figure 4 summarizes the distortion data for 19 units. In all cases, the stimulus had a frequency of 0.025 Hz and a peak acceleration of 40°/sec². The 19 units were chosen from a larger population of 52 units. To be included, a unit had to meet two criteria. First, the resting discharge, measured after stimulation, had to differ from that measured before stimulation by less than 5% of the amplitude of the response. And second, the response amplitude could not exceed the resting level, i.e., the neuron’s discharge could not be silenced during any portion of the sine-wave cycle. The response to the 0.025-Hz stimulus was reasonably large, the response amplitude in the 19 units averaging 65.0 spikes/sec. The mean unrestricted distor-
tion was 7.3%, the mean restricted distortion 13.6% (Fig. 4, left).

The asymmetrical nature of the distortion is reflected in the amplitude spectrum (Fig. 4, right). The major distortion products are the d-c component and the even harmonics, particularly the 2nd harmonic. That the d-c distortion was not simply due to random variations in the resting level is indicated by the fact that, in 16 of the 19 units, the average discharge was larger than the resting discharge, the probability of this occurring by chance being small ($P < .005$). Further, in calculating the average amplitude of the d-c component, the sign of the component was taken into consideration. The average d-c distortion was 11.6 ± 1.6%, which was significantly different from zero ($t$ test, $P < .001$). Finally, the variation between the resting levels measured before and after stimulation averaged, in this selected sample of units, only 2.9% of the response magnitude and could have had only a negligible influence on the d-c distortion figure of 11.6%.

A source of spurious distortion needs to be considered. This is the random distortion introduced by moment-to-moment variations in discharge rate. A study of such variations occurring during the resting activity provided an estimate of 3.2% for the average amplitude of this distortion, the value for any particular neuron being closely related to the regularity of spacing of its action potentials. When the contribution of the random component was subtracted, the average unrestricted distortion was reduced to 6.6%, the average restricted distortion to 13.2%. Further, on the not unreasonable assumption that the power contained in the random distortion was uniformly distributed throughout the spectrum, the amplitudes of the individual harmonics could be corrected. The corrected values are indicated in Fig. 4 by dashed lines. As might be expected, the random distortion had an appreciable effect only on the amplitudes of the upper harmonics.

**Gain and phase as functions of sine-wave frequency**

Gains and phase lags were relatively unaffected when stimulus magnitude was varied over wide limits (see Fig. 2). This justifies presenting the data in the normalized form of Bode plots. Such plots for two units—64-11 and 51-8—are included in Fig. 5. The points at the left are the gains re acceleration, those at the right the phase lags re acceleration.

Consider first unit 64-11 (Fig. 5, solid circles). The behavior of the unit deviates from that of a torsion pendulum (indicated by the solid line) at both low and high frequencies. At high frequencies, there is a gain enhancement; also the phase lag, rather than continuously increasing with frequency, reaches a maximum at 0.25 Hz and then begins to diminish. The discrepancy at low frequencies is mainly reflected in the phase lag being less than expected. The simplest linear transfer function which will approximate the data is of the form

$$H(s) = \frac{\tau_A s}{(1 + \tau_A s)(1 + \tau_B s)(1 + \tau_C s)}$$

The factor $H_{TP} = 1/[(1 + \tau_A s)(1 + \tau_B s)]$ is the transfer function of the torsion pendulum with $\tau_1 = \Pi / \Delta$ and $\tau_2 = \Theta / \Pi$. The choice of the values $\tau_1 = 5.7$ sec and $\tau_2 = .003$ sec will be gone into in the next section. The term $H_A = \tau_A s / (1 + \tau_A s)$ results in a phase lead at low frequencies and is the frequency-domain representation of the Young-Oman adaptation operator (9, 27). The constant $\tau_A$ is the adaptation time constant. The operator $H_A$ should also lead to a gain attenuation at very low frequencies, well below 0.0125 Hz. The term $H_L = (1 + \tau_L s)$ is a lead component and reproduces the high-frequency deviations from the torsion-pendulum model, including the gain enhancement and the progressive phase lead. If one assumes that the transfer function $H_{TP}$ provides an adequate depiction of the relation between angular acceleration and cupular displacement, then the meaning of the operator $H_L$ can be deduced. The presence of the high-frequency lead component $H_L$ would then imply that the system is sensitive both to cupular displacement and to the velocity of the displacement, the time constant $\tau_L$ reflecting the relative sensitivities to these two aspects of cupular motion.
In unit 51-8 (Fig. 5, open circles), there is less discrepancy from the torsion-pendulum model. This is reflected in the corresponding transfer function. The adaptation operator is omitted and the value of $\tau_1$ is only 0.015 sec.

A comparison of units 64-11 and 51-8 suggests that neurons exhibiting a relatively high degree of adaptation are also characterized by a pronounced high-frequency lead component. This was generally true. The phase lag at 1.0 Hz may be taken as a measure of the magnitude of the lead component, that at 0.0125 Hz as a measure of the degree of adaptation. Figure 6 plots these two phase lags against one another for 29 units. A strong positive relation is indicated. The product-moment correlation is 0.59 (t test, $P < .001$). The figure also indicates a great variability in the observed phase angles for the 0.0125- and 1.0 Hz stimuli. The spread of phase angles at low frequencies reflects, in large part, differences among units in their adaptive properties; that at high frequencies differences in the magnitude of the high-frequency lead component. Figure 7 (right) shows the mean and standard deviation of phase angles at different frequencies, as determined from the entire population of units. The standard deviation is least at 0.25 Hz—a frequency where the phase angle should be only imperceptibly affected by either adaptation or the high-frequency lead component—and systematically increases as one departs from this frequency in either direction.

**Determination of time constants**

The transfer function presented in equation 2 requires the specification of four time constants. We begin with a considera-

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**Fig. 5.** Bode plots (re acceleration) for two units. Left: gain plots. Right: phase plots. Points, experimental values; curves, values calculated from transfer functions. In Figs. 5, 7, and 12, log-gain at 0.25 Hz arbitrarily set to value expected from torsion-pendulum model. Acceleration magnitudes as in Table 1. Unit 64-11, posterior canal; unit 51-8, horizontal canal.

**Fig. 6.** Comparison for 29 units of phase lags (re acceleration) at 0.0025 and 1.0 Hz. Acceleration magnitudes as in Table 1. N, number of units; $r$, product-moment correlation; $P$, significance level of correlation (t test).
FIG. 7. Average Bode plot (re acceleration). Points, mean values; bars, standard deviation of values; curves, values calculated from transfer functions. See Table 1 for sample size at each frequency and for acceleration magnitudes.

estimation of the time constants of the torsion pendulum. The constant $\tau_1 = 11/\Delta$ can be estimated from the phase lag $\phi$ at a sufficiently low frequency $f$ by the formula $\tau_1 = (1/2\pi f)\tan \phi$. The equation is valid provided that adaptation has a negligible effect. Otherwise the value of $\phi$ and, hence, that of $\tau_1$ will be reduced. A convenient frequency is 0.025 Hz. The only neurons used in the estimation were those—the LA units (see ref 9)—whose response to velocity trapezoids was characterized by a relatively small degree of adaptation. Twelve such units were available. Their average phase lag was $42.0 \pm 1.1^\circ$, equivalent to a $\tau_1$ of $5.73 \pm 0.23$ sec. This value corresponds reasonably well with that determined from previous time-domain analyses (9). The estimated time constant, based on the response during constant accelerations, was $5.41 \pm 0.38$ sec. That based on the recovery from such accelerations was $5.99 \pm 0.16$ sec.

Our observations, since they extended only to 8.0 Hz, are insufficient to provide a direct experimental measure of $\tau_2 = \Theta/\Pi$. Some estimate of this constant can, however, be made from hydrodynamic considerations. What seems to be required is a solution of the Navier-Stokes equation (ref 18, p. 642) for the complicated geometry represented by the canal, the associated ampulla, and the utriculus. No one to our knowledge has accomplished this, though Steer (23) has solved the equation for a straight tube. The approximate value of the time constant, so derived, is

$$\tau_2 = \left(\rho\beta_1^2/\eta\right)^{1/2}$$

(3)

where $\beta_1$ is the first zero of the zeroth-order Bessel function of the first kind; $\rho$ and $\eta$ are, respectively, the density and viscosity of the endolymph; and $r$ is the internal radius of the tube, in this case the radius of the membranous canal. This radius has been measured by Igarashi (14) and leads to a value of $\tau_2$ equal to 0.005 sec in man and 0.003 sec in the squirrel monkey.

Once $\tau_1$ and $\tau_2$ have been calculated, some estimate can be made of the other two time constants. Figure 7 presents Bode plots based on the entire population of units. The gain plot is to the left, the phase plot to the right; mean values and standard deviations are included. In determining time constants, phase information is more revealing than is gain information. Note that at all frequencies the phase lag falls short of that predicted by the torsion-
pendulum model. The discrepancy will be denoted by $\Delta \phi$ and, by convention, will be taken as positive. At a very low frequency $f$, $\tau_A$ should be given by the expression $(1/2\pi f)\tan(\pi/2 \Delta \phi)$. Thirty units were studied at a frequency of 0.0125 Hz. The average phase angle was 15.1°, giving a $\Delta \phi$ of 9.1° and a $\tau_A$ of 80 sec. The standard deviation of $\Delta \phi$ at this frequency was appreciable, in large part presumably reflecting variations in $\tau_A$. From the standard deviation, it may be surmised that $\tau_A$ ranges among the population from 30 sec to values which are so large as to be indistinguishable from infinity.

The time constant $\tau_L$ may be estimated from the formula $(1/2\pi f)\tan(\Delta \phi)$, where $\Delta \phi$ is the phase discrepancy at a relatively high frequency $f$. The average value of $\tau_L$, based on all data available between 1.0 and 8.0 Hz, was 0.049 sec. The standard deviations of $\Delta \phi$ at these frequencies indicates that $\tau_L$ may vary within the population from 0.013 to 0.094 sec.

The average transfer function thus arrived at is

$$H(s) = \frac{80s}{(1 + 0.049s)} \frac{(1 + 0.003s)}{(1 + 5.7s)(1 + 0.008s)}$$

The agreement between the transfer function and the data is, as indicated by Fig. 7, reasonably good in the low- and high-frequency ranges. There is, however, a discrepancy of some 5° between the observed and predicted phase angles in the range of 0.1–0.5 Hz. The discrepancy cannot be significantly reduced by adjusting any of the four time constants in $H(s)$, without at the same time upsetting the fit in other parts of the frequency spectrum. One could of course add an appropriate lead-lag compensation factor to the transfer function. A factor—$(1 + 0.64s)/(1 + 0.52s)$—would eliminate the discrepancy. But such an ad hoc procedure has little to recommend it since the compensation factor does not have a ready physiological interpretation.

Comparison between time- and frequency-domain analyses

The transfer function of equation 2 can be used to obtain a theoretical response to velocity trapezoids. A comparison between a theoretical and an actual response is presented in Fig. 8. The empirical points represent the average of the excitatory and inhibitory responses to a 40-sec velocity trapezoid. The parameters for the calculation were, with the exception of the sensitivity factor, determined from sine-wave data. Thus, $\tau_A$ was deduced from the phase lags at 0.006 and 0.0125 Hz, $\tau_L$ from the phase lags at 1.0 and 2.0 Hz. The value of the sensitivity factor used in the calculation, 3.44 spikes-sec⁻¹/deg-sec⁻², is in reasonable agreement with the value of 3.00 derived from the response to a sine wave of 0.25 Hz.

As is exemplified in Fig. 8, there should be a close relation between the responses to sine waves and to velocity trapezoids. Each kind of stimulus may be used to measure the sensitivity of the neuron. The degree of adaptation, observed in the response to velocity trapezoids, should be reflected in the response to low-frequency sine waves. And the high-frequency lead component, best seen when sine waves are presented, should also affect the initial
FIG. 9. Sensitivity of individual units determined by response to constant excitatory acceleration (ordinate) and by response to sinusoidal stimulus (abscissa) in spikes/sec×deg/sec−1. Constant acceleration: 10 sec duration, 30°/sec2. Sine-wave stimulus: 0.25 Hz, 80°/sec2 (peak acceleration). Solid curve, regression line; dashed curve, theoretical relation based on equation 4. N, number of units; r, product-moment correlation; P, significance level of correlation (t test).

FIG. 10. Adaptation of individual units as determined by recovery from constant accelerations (ordinate) and by phase lag (acceleration) of response to 0.0125-Hz, 20°/sec2 sinusoidal stimulus (abscissa). Curve, theoretical relation based on equation 2 with \( \tau_1, \tau_2, \) and \( \tau_L \) as in equation 4. N, number of units; r, product-moment correlation; P, significance level of correlation (t test).

Perhaps the most unexpected finding of the present paper, given our observations on velocity trapezoids, was the demonstration of a high-frequency lead component. Can the effects of this factor be identified in the response to constant-acceleration stimuli? Were no lead component present, the initial response to such a stimulus should increase linearly with time. Consider the transfer function (equation 4) with the lead component \( H_L \) removed. The ratio of the average responses during the periods 0.5–1.0 and 0.0–0.5 sec after the start of the constant acceleration should be slightly greater than 3.0. Reasonably wide variations in time constants, including large, but reasonable, decreases in \( \tau_1 \) and \( \tau_L \), would only reduce the constant-acceleration ratio to on the order of 2.9. In contrast, the lead component can have a dramatic effect. If, for example, \( \tau_L \) is set to 0.05 sec, the resulting ratio will be 2.66. Increasing the time constant to 0.1 sec will decrease the ratio to 2.41. Measures of the ratio were obtained in 50 neurons. The accelerations were 150°/sec2 and only excitatory responses were used. The average value of the ratio was 2.47 ± 0.06, which would correspond to an average \( \tau_L \) of 0.086...
FIG. 11. Magnitude of high-frequency lead component as measured by early part of excitatory response to constant-acceleration stimulus (ordinate) and by phase lag (re acceleration) of response to 1.0-Hz sinusoidal stimulus (abscissa). Constant acceleration, 150°/sec². Peak acceleration of sine wave as in Table I. Dashed curved line, theoretical relation based on equation 2 with \( \tau_p, \tau_b \), and \( \tau_A \) as in equation 4. N, number of units; \( r \), product-moment correlation; \( P \), significance level of correlation (t test).

\[ \pm 0.013 \text{ sec.} \] Note that this estimate of \( \tau_p \) is roughly double that obtained from the sine-wave analysis.

Despite the discrepancy, the results may be taken as a verification in the time-domain of the existence of a high-frequency lead component. A further comparison is possible. It would be expected that the phase lag in the response to a 1-Hz sinusoidal stimulus would be positively related to the value of the constant-acceleration ratio. These two variables are plotted against one another in Fig. 11. There is a wide spread in the data, which is largely attributable to the fact that the acceleration ratios were calculated, with few exceptions, from the response to a single velocity trapezoid. Nevertheless, a statistically significant trend is observable. Again, the discrepancy is apparent. In 38 of the 50 units, the ratio is less than that predicted from the sine-wave response (\( P < .05 \)).

DISCUSSION

Most treatments of the dynamics of the peripheral vestibular apparatus (5, 6, 10, 11, 13, 15, 17, 19, 22–25) have assumed that the system behaves like a heavily damped torsion pendulum. It is probable that the torsion-pendulum model adequately represents the motion of the cupula and endolymph (22, 23). But the model cannot account for the response dynamics of the first-order afferents innervating the semicircular canals. Here two additional factors come into play. They affect the response to both velocity trapezoids and sinusoidal stimulation. One factor is adaptation. The second is a high-frequency lead component, which suggests that the neurons, in addition to being sensitive to cupular displacement, are also sensitive to the velocity of the displacement. The adaptation need not be considered further, since it was dealt with in a previous paper (9). The high-frequency lead component does merit some comment.

One of the main theoretical deductions made on the basis of the torsion-pendulum model was that the peripheral vestibular system acts as a quite faithful velocity transducer for the brief angular accelerations encountered in everyday life. The conclusion must be modified. Figure 12 replots the data of Fig. 5 in terms of angular velocity, rather than angular acceleration. Consider first the behavior expected of the torsion pendulum (solid lines). The gain re velocity is flat in the physiological range between 0.25 and 8.0 Hz and the phase lag is less than \( \pm 10^\circ \). Introduction of the lead component, as is illustrated by the curves for unit 64–11, results in a high-frequency gain enhancement and a progressive phase lead. Both effects become substantial only for frequencies above 1 Hz. At 0.5 Hz, for example, the phase lead is only some 15°, an observation in reasonable accord with the phase measurements made by McIvill Jones and Milsum (16) in second-order neurons; further, there is no appreciable gain enhancement. In contrast, the gain enhancement at 8.0 Hz amounts to 10 dB and the phase lead to some 60°.
That the presence of the lead component may profoundly affect the response to a physiological stimulus, such as a rapid rotation of the head, is depicted in Fig. 13A. Shown are a series of calculated responses to a rotation whose velocity profile is 0.5 cycle of a 4-Hz sine wave. The value of $\tau_L$, the time constant of the lead component, is varied over the range encountered experimentally. As might be expected from the steady-state analysis, an increase in $\tau_L$ produces an increase in gain and a phase lead re velocity. The phase lead, in turn, results in the response becoming progressively biphasic. These tendencies become more pronounced, the higher the frequency (Fig. 13B).

What might be the functional significance of the lead component? One way to view its function is in terms of dynamic load compensation. Certainly the lead component compensates, within the frequency range of interest, for the inertia of the cupula and endolymph. But, if this were all that was involved, the value of $\tau_L$ need not be larger than 0.003–0.005 sec. Probably of greater consequence are the loads represented by the various vestibular reflex pathways. Such pathways may introduce delays, phase lags, and high-frequency gain attenuations. The lead component could compensate for these effects. Optimum compensation would be accomplished were each vestibular afferent mainly routed into those pathways whose dynamics most closely matched the magnitude of the neuron’s lead component.

Unfortunately, it is not possible to specify how the lead component works in most vestibular reflexes. The simplest way to gain some insight into its role would be to compare the response dynamics when natural stimuli are employed with the dynamics characterizing the response to direct electrical stimulation of the vestibular nerve. There have been numerous studies of the reflex responses to vestibular nerve stimulation (1–4, 7, 26), but few of these have concentrated on dynamic properties. An exception is a paper by Partridge and Kim (20). Individual ampullary nerve bundles were stimulated with sinusoidally modulated pulse trains and the resulting variations in the isometric tension of the triceps surae muscle recorded. The dynamics resembled those of a first-order lag element with a time constant of 0.18 sec, in series with a 15-msec conduction delay. Obviously the gain attenuations and phase shifts, observed by Partridge and Kim in
FIG. 13. Theoretical responses to series of head rotations, each consisting of 5 cycle of a sine wave. Calculations based on equation 2 with $\tau_1$, $\tau_2$, and $\tau_4$ as in equation 4. Peak velocity assumed same for all rotations. Ordinate in units of response expected from corresponding torsion-pendulum model. A: effect of variation of $\tau_4$ on response to 4.0-Hz stimulus. B: effect of variation of sine-wave frequency, $\tau_L = .045$.

The frequency range above 1 Hz could be most effectively compensated were the afferents involved in the reflex characterized by a lead component with a time constant $\tau_L$ near the time constant of the aforementioned lag element. The lead components observed in our experiments were never this prominent, the highest value of $\tau_L$ being on the order of 0.1 sec. But even such a lead component could serve to extend the bandwidth of the reflex and would bring the variations in isometric tension more closely into phase with the angular velocity of head movement.

An interesting question which may be raised is the extent to which the dynamics of vestibular reflexes are determined by peripheral mechanisms, rather than by the central pathways. The reflex dynamics observed by Partridge and Kim (20) were similar to the dynamics relating motor nerve stimulation to muscle contraction. Similarly, if natural stimulation had been used, it may be assumed that the overall dynamics of the reflex would also reflect the dynamics of the peripheral vestibular apparatus. Although the central nervous system may affect the dynamics in any of a number of ways, its only certain contribution is to the 15-msec conduction delay. That the central nervous system might play only a minor role in determining reflex dynamics is, at first glance, not unreasonable. On both the afferent and efferent sides, the reflex components consist of mechanical systems characterized by time constants which are much larger than those thought to govern the discharge of central neurons and these relatively slow mechanical systems may dominate the dynamics of the overall pathway.

Can it be argued that the central nervous system plays a minor role in the dynamic properties of all vestibular reflexes? One need only consider the vestibuloculomotor system to be disabused of this notion. The fast phase of nystagmus is unquestionably of central origin. Even the dynamics of the slow phase cannot be explained on the basis solely of peripheral mechanisms. This is most clearly indicated by the fact that the discharge of abducens motoneurons, during both phases of nystagmus, parallels the movement of the eye and not the time course of afferent nerve discharge (12, 21). What is suggested then is that, in the study of any particular vestibular function, consideration must be given both to central and peripheral mechanisms. Unfortunately, there has been a tendency in much of vestibular research to explain all observations, including the results of human psychophysical experiments, on the basis of the motion of the cupula and endolymph. This may lead to erroneous conclusions concerning the physics of the semicircular canals. One example may be cited.

In their pioneering studies, van Egmond, Groen, and Jonkees (5) attempted to determine the time constants of the torsion-pendulum model from human judgments of the sensation of turning. The first-order time constant $\tau_1 = \Omega/\Delta$ was estimated from cupulometric studies to be about 10 sec. As was reviewed in a previous paper (9) this value is probably low, in part due to the intrusion of sensory adaptation. In any case, nystagmographic studies consistently provide somewhat larger values of $\tau_1$, usually on the order of 15 sec (10, 13). A more serious discrepancy was encountered in the estimation of $\tau_2 = \Theta/\Omega$. Here van Egmond and his colleagues placed subjects on a torsion swing and determined the resonant frequency $\omega_0$, i.e., the frequency for which the subjective sensation
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would just be in phase with the velocity of the swing. The resonant frequency was 1 rad/sec, which corresponds to a value of \( \tau_2 = .05 \) sec. This differs from hydrodynamic calculations by one order of magnitude. Further, any relation between subjective estimates of \( \omega_0 \) and peripheral mechanisms is, at best, indirect. None of the neurons examined in the present study had a resonant point. This was largely a consequence of the high-frequency lead component. At all frequencies, the discharge led the velocity of the stimulus, though this phase lead reached a minimum at 0.25–0.50 Hz. In some units, the minimum phase lead amounted to no more than 5°. Perhaps coincidentally, the frequency of 0.25–0.50 Hz is quite close to the resonant point defined in the human experiments.

There is no reason to believe that the dynamics governing the discharge of human vestibular afferents differs in any fundamental way from those described in the squirrel monkey. One quantitative difference, though, is striking. The average value of \( \tau_1 \) in the squirrel monkey is 5.7 sec, roughly 2–3 times smaller than the values estimated from human studies. The difference can be partly related to the physical dimensions of the canals in the two species. Let \( R \) be the radius of curvature of the canal and \( r \) the internal radius of the membranous canal. According to the theory elaborated by van Egmond et al. (5) and by Melvill Jones and Spells (17), the time constant \( \tau_1 \) should be proportional to \( R^2 \) and inversely proportional to \( r^2 \). From the physical measurements of Igarashi (14), the expected time constant in man should be 1.68 times larger than that in the monkey. Steer's (22, 23) somewhat more detailed analysis of the motion of the cupula-endolymph system leads to a not dissimilar conclusion. Note that the calculated ratio of 1.68 is less than that obtained experimentally. The discrepancy probably reflects the fact that we still do not possess a complete physical theory of cupular motion. What is particularly lacking is a knowledge as to how the elastic-restoring forces of the cupula are generated.

SUMMARY

A study was made as to how peripheral vestibular afferents innervating the semicircular canals of the squirrel monkey (Saimiri sciureus) respond to sinusoidal stimulation. Frequency was varied from 0.006 to 8.0 Hz. In many ways the responses resembled those expected of a linear system. The nonlinear distortion, which mainly reflected asymmetries between excitatory and inhibitory responses, was reasonably low, averaging some 13%. For a given sine-wave frequency, gains and phase lags were relatively independent of stimulus magnitude. This justified presenting the data in the normalized form of Bode plots. From such plots, linear transfer functions were constructed.

The response to sinusoidal stimulation deviates in two respects from that predicted by the torsion-pendulum model. At low frequencies, the phase lag re acceleration is less than expected. This is a consequence of sensory adaptation. At high frequencies, there is a gain enhancement; also the phase lag re acceleration, rather than continuously increasing with frequency, reaches a maximum and then begins to diminish. One interpretation of this high-frequency lead component is that the afferents are sensitive both to cupular displacement and to the velocity of that displacement. A possible function of the presumed velocity sensitivity is that it may permit the peripheral vestibular system to compensate for the dynamic loads represented by the various vestibular reflex pathways.

The prominence of both sensory adaptation and the high-frequency lead component varied from neuron to neuron and were related to one another. The greater the adaptation exhibited by a unit, the more pronounced tended to be the lead component.

There is, as expected, a close relation between the responses to sine waves and to velocity trapezoids. The transfer function, deduced from the response of a neuron to sinusoidal stimulation, may be used to reconstruct the major features of the response to velocity trapezoids. The two kinds of response provide equivalent measures of the unit's sensitivity. The de-
gree of adaptation observed in the response to velocity trapezoids is also reflected in the response to low-frequency sine waves. Finally, the velocity sensitivity, best seen when high-frequency sine waves are presented, also affects the initial portion of the response to constant accelerations.

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