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Final Technical Report

NORMAL AND ABNORMAL HUMAN VESTIBULAR OCULAR FUNCTION

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Robert J. Peterka, Ph.D.
and
F. Owen Black, M.D.

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Good Samaritan Hospital and Medical Center
Department of Neuro-otology
1040 N.W. 22nd Ave.
Portland, OR 97210

INTRODUCTION

The major motivation of our research is to understand the role the vestibular system plays in sensorimotor interactions which result in spatial disorientation and motion sickness. We felt that it was necessary to clearly define exactly what normal is in the vestibular system using the most complete and accurate tests currently available. A second goal was to explore the range of abnormality as it is reflected in quantitative measures of vestibular reflex responses. The results of a study of vestibular reflex measurements in normal subjects and preliminary results in abnormal subjects are presented in this report.

METHODS

Vestibular and oculomotor reflexes were tested in 216 normal human subjects aged 7 to 81 years. Tests included 1) horizontal vestibuloocular reflex, 2) optokinetic reflex, 3) caloric irrigations, and 4) moving platform posturography. Various parameters which characterize the dynamic response properties of these reflexes were measured. Statistical methods were used to define the range of normal responses, and determine age related changes in function.

Subjects were required to meet the following criteria:

1. normal age corrected auditory pure tone responses.
2. middle ear reflexes present bilaterally.
3. normal middle ear impedance.
4. negative history of head blows of sufficient magnitude to cause loss of consciousness.
5. negative neurological findings.
6. normal corrected vision.
7. negative history of use of ototoxic drugs.
8. negative history of dizziness or equilibrium problems.

We did not reject subjects based on the results of any of the vestibular or oculomotor tests performed. Details of the testing procedures are presented below.

Rotation tests

Subjects sat in a chair mounted on an 80 ft-lb velocity servo controlled motor (Contraves Goerz Corp, Model 824) which rotated them about an earth vertical axis. The subject was surrounded by a circular cloth cylinder 6 feet in diameter. The cylinder acted as a projection screen for an optokinetic stimulus. A full field optokinetic stimulus was provided by a pin hole type projector mounted on a 5 ft-lb servo motor (Genisco Technology Corp Model 1100) attached to the ceiling directly above the subjects head. The projector produced randomly placed vertical stipes of light against a mostly dark background. The vertical stipes were projected images of a 3 inch long filament of a 40 Watt clear light bulb (General Electric FG 1048-AX2) commonly used to light window displays. The length of the projected images on the cloth screen was about 24 inches.

Subjects performed tests of vestibuloocular reflex (VOR) function with eyes closed in a darkened room. Horizontal and vertical eye movements were recorded by electrooculographic (EOG) techniques using silver/silver chloride pellet electrodes. Horizontal EOG was recorded using bitemporal electrodes, and vertical EOG was recorded by electrodes placed above and below one eye. The EOG amplifier bandwidth was set to DC-80 Hz using a two pole Butterworth filter. Stimulus delivery and data collection were controlled by computer (DEC LSI 11/73). Chair tachometer signals as well as horizontal and vertical EOG were digitized and stored on a disk file for later analysis. Digitizing rates were 200 per second for the horizontal EOG and 50 per second for vertical EOG and stimulus velocity (12 bit A/D converter).

Calibrations of the EOG were performed before and after each rotation test. Three red LED's mounted on the screen at 0 degrees and +/- 10 degrees were successivly illuminated. As the subject looked at the illuminated LED's, the computer recorded the voltage associated with each gaze angle. The testing technicians adjusted the EOG amplifier's gain to provide adequate EOG signal amplitude relative to the digitizing resolution of the A/D converter.

Rotational stimuli for VOR tests included both single frequency sinusiodal stimuli and a pseudorandom stimulus. Single frequency sinusoidal stimuli were at 0.05, 0.2, and 0.8 Hz with peak velocities of 60 deg/sec. The duration of sine tests were 100 seconds (5 cycles) for 0.05 Hz, 45 seconds (9 cycles) for 0.2 Hz, 26.25 seconds (21 cycles) for 0.8 Hz. The first cycle in each data record was considered a transient response and was ignored in the data analysis.

The pseudorandom stimulus consisted of the summation of eight discrete sinusoidal frequencies ranging from 0.0092 to 1.535 Hz. The eight frequencies were 0.0092, 0.021, 0.046, 0.095, 0.180, 0.388, 0.766, 1.535 Hz. The nominal amplitudes of these components were all 15.625 deg/sec except the highest frequency component which was 7.813 deg/sec. The highest instantaneous stimulus velocity was approximately 80 deg/sec. The duration of the stimulus was about 440 seconds. Data from the last 327.68 seconds of the trial were digitized and saved on disk for later analysis. The first 115 seconds of data (about equal to one period length of the lowest frequency component of the stimulus) were considered a transient response and were not analyzed.

The frequency components in the pseudorandom stimulus were selected to minimize corruption of the results of the data analysis due to possible nonlinear responses of the VOR system. A typical result of the testing of a nonlinear system with a sinusoidal stimulus is the presence of response components not only at the stimulus frequency, as expected for a linear system, but also at harmonics of the stimulus frequency. If, in the case of a complex stimulus consisting of the summation of many sinusoidal components, one of the harmonics of a lower stimulus frequency is identical to one of the higher stimulus frequencies, then it is not possible to separate out the two components of the response during subsequent data analysis. The particular stimulus frequencies were chosen to minimize this problem while at the same time providing a wide bandwidth of test frequencies.

The repetition period of the pseudorandom stimulus for the VOR test was 327.68 seconds (fundamental frequency = 0.003052 Hz). However there was no energy at this low frequency. The lowest frequency component which did contain some energy had a period of one third the fundamental period and therefore a frequency of three times the fundamental frequency (lowest frequency = 3 x 0.003052 = 0.009156 Hz). The multiples for all eight frequencies of the pseudorandom stimulus were 3, 7, 15, 31, 59, 127, 251, and 503 times the fundamental frequency. The choice of stimulus frequencies followed the general outline of Victor and Shapley (1980).

The optokinetic reflex (OKR) of each subject was tested by recording horizontal eye movements evoked as a projected visual stimulus rotated around the stationary subject. Under such conditions the subject's eyes tend to follow the moving visual scene. The optokinetic projector moved under the control of a pseudorandom stimulus. The pseudorandom stimulus was similar, but not identical to the one used for VOR testing. Seven sinusoidal signals were added together to form the stimulus. The seven frequencies were 0.018, 0.043, 0.092, 0.189, 0.360, 0.775, and 1.532 Hz. These frequencies are the 3, 7, 15, 31, 59, 127, and 251 multiples of the stimulus fundamental frequency of 0.006184 Hz (163.84 second period). The amplitudes of the components were nominally 7.813 deg/sec except the highest frequency component which was 3.91 deg/sec. The peak instantaneous velocity was approximately 40 deg/sec. Therefore the amplitude of the optokinetic stimulus was lower than that of the VOR stimulus. The lower amplitude avoided the saturation type nonlinearity which is known to exist in the optokinetic system. The total stimulus duration was about 220 seconds. Only the last 163.84 sec of data were recorded so that transient responses were avoided. A few subjects were tested using other optokinetic stimuli including single frequency sinusiods and other pseudorandom stimuli of varying bandwidths and amplitudes. Complete OKR data were not obtained on all subjects since the stimulus induced motion sickness symptoms in some subjects, requiring the early termination of the stimulus profile.

Selected normal and abnormal subjects were given a visual-vestibuloocular reflex test (VVOR) in which the subject was rotated with eyes open while the projected visual stimulus remained stationary. The VVOR test conditions mimic the every day experience where subjects move relative to a stationary, earth fixed visual environment. Both the vestibular and oculomotor systems contribute to the generation of compensatory eye movements during the VVOR test. A pseudorandom rotational stimulus identical to the one used for VOR testing was used for the VVOR test.

Subjects were given verbal tasks by the testing technician throughout the VOR, OKR, and VVOR rotation tests in order to maintain a constant level of alertness. The tasks consisted of alphbetically naming names, places, foods, etc.

Rotation Test Data Analysis

The analysis of horizontal eye movements evoked by rotation tests followed the same basic scheme which was:

1. Scale recorded horizontal EOG signals to calculate eye position with respect to the head. Scaling is done according to the average of the calibrations recorded before and after each test.

2. Smooth the EOG data using a digital low pass filter which does not introduce phase shifts into the analysis.
3. Differentiate the eye position signal to calculate eye velocity. Units of eye velocity are degrees/second.
4. Apply additional low pass digital filtering to the eye velocity signal and reduce the sampling rate from 200 to 50 samples/second.
5. Separate the slow and fast phases of nystagmus and mark the occurrence of the fast phases so that they can be ignored in later stages of analysis.
6. Calculate the average value of the remaining slow phase eye velocity data. This gives a measure called response bias with units of deg/sec.
7. Calculate the Fourier transform of the slow phases of eye velocity.
8. From the Fourier transform coefficients at the stimulus frequency, calculate the response amplitude and phase relative to the stimulus.
9. Calculate response gain which is the ratio of the amplitude of the slow phase eye velocity to the amplitude of the stimulus velocity at the stimulus frequency.

The application of these procedures to data from a 0.05 Hz VOR test is shown in Figure 1. The lower trace shows one period (20 sec) of the rotational chair velocity versus time. The trace immediately above shows the horizontal eye movements evoked by the rotational stimulus. During the first half of the stimulus period, the chair is moving to the left evoking slow phases of nystagmus to the right. Interspersed among the slow phase movements are fast phases which generally are directed opposite to the slow phases. When the chair slows and then reverses its direction of rotation in the second half of the stimulus period, the eye movements slow and then reverse their direction. The solid vertical bars between the stimulus velocity and eye position traces show where the computer algorithm detected the fast phases of the nystagmus.

The upper trace in Figure 1 displays eye velocity of the slow phases of nystagmus versus time. The solid line through the data points show curve fits to the slow phase eye velocity obtained from the Fourier transform calculation of response bias, amplitude, and phase at the stimulus frequency. For sinusiodal stimuli, measures of bias, amplitude, and phase were made on each period of the response. Stimulus periods which contained corrupted data could be rejected before the final averaging of the bias, amplitude, and phase values from the remaining periods.

The curve fits to sinusiodal responses were of the form:

$$r(t) = B_r + A_r \sin(2\pi f + P_r) \qquad (1)$$

where B_r is bias in deg/sec, A_r is response amplitude in deg/sec, P_r is response phase in degrees, and f is the stimulus frequency. The recorded chair velocity data was separately analyzed to calculate stimulus velocity amplitude, A_s, and phase, P_s. The gain of the reflex is defined as the ratio A_r/A_s, and the phase of the reflex as P_r-P_s. Since the VOR and VVOR are compensatory reflexes, the values of P_r-P_s were close to -180 degrees. For the convenience of working with smaller numbers, a value of 180 deg was added to P_r-P_s for both VOR and VVOR tests. This is equivalent to inverting the horizontal eye position data. Since the OKR is a following reflex, the value of P_r-P_s is close to zero degrees for low frequency stimuli. Therefore no offset factor was applied to the OKR phase data.

Positive reflex phases are referred to as phase leads, and negative phases as phase lags. A reflex response with a zero phase value is said to be in phase with the stimulus meaning that at any point in time, the response is directly proportional to the stimulus velocity. A phase lead does not mean that the response occurred earlier in time than the stimulus. That would be impossible. Response parameters are not measured at the beginning of the stimulus when the system is in the transient response period, but rather at a later period when the response is in steady state. A phase lead during the steady state portion of the response simply means that there is a component of the response that is proportional to the time rate of change of the stimulus. Since the stimulus is considered to be rotational velocity, the time rate of change of the stimulus is rotational acceleration. If the phase lead were exactly 90 degrees, then the response would be directly proportional to rotational acceleration, with no component proportional to rotational velocity. A phase lag of 90 degrees would correspond to a response which was proportional to the integral of rotational velocity which is rotational position.

Responses to sinusoidal stimuli were analyzed by an second procedure designed to identify and quantify nonlinear response properties. After the first analysis of the data described above, the horizontal eye velocity data was shifted in time by an amount determined by the calculated phase of each period of the response. The time shift was in a direction which brought the response into phase with the stimulus. Slow phase eye velocity was then plotted against stimulus velocity to yield a scatter of points which generally lie along a negatively sloping line (positively sloping for the OKR). Examples are shown in Figure 2. The negative slope for the VOR reflects the fact that slow phase eye velocity is in the opposite direction to stimulus velocity. The slope of the line is equal to VOR gain.

If the VOR were a linear system, the slope of the data would be the same for eye movements to the right and left, and would be independent of stimulus amplitude. However experience with abnormal subjects has shown that the slopes are not always equal for chair rotations in opposite directions. This type of nonlinearity can be quantified by separately calculating the slopes of the eye velocity versus stimulus velocity data while the chair was rotating in opposite directions. The slopes were calculated by a least squared error fit of a two segment line to the data. One line segment was for positive (to the right) stimulus velocities, and the other for negative (to the left) velocities. The two line segments were constrained to intersect one another at zero stimulus velocity.

Therefore the two-part linear curve fit yields three parameters: the reflex gain for slow phase eye movements to the right, G_r, the gain for slow phase eye movements to the left, G_l, and the eye velocity when the stimulus velocity is zero. The eye velocity at the zero stimulus velocity is referred to as the response offset. Offset has units of deg/sec.

From the two gain measures G_r and G_l, a measure of response asymmetry was calculated according to the formula $100*(G_r-G_l)/(G_r+G_l)$. A zero percent asymmetry is consistent with a linear system response where gain is independent of the direction of the stimulus direction.

Bias, offset, and asymmetry parameters are all measures of response symmetry, although they are not identical measures. Bias is a relatively gross measure of symmetry since different factors can influence its value. For example, both an underlying spontaneous nystagmus as well as a nonlinear response could give equal values of bias. Our definition of response offset and asymmtry were designed to distinguish between average shifts of eye velocity and nonlinear response characteristics, respectively.

The relationship of bias to offset and asymmetry measures can be clarified by several examples. Bias and offset measures give identical values when the response asymmetry is zero. If there is a positive asymmetry (greater gain during eye movements to the right than to the left), then the bias measure will have a value more positive than the offset measure. For negative asymmetry, the bias will be more negative than the offset. There may be a positive bias even when the offset is zero, in which case it is clear that the source of the bias was from a nonlinear response rather than from an underlying spontaneous nystagmus.

The analysis of eye movement data from pseudorandom stimuli follows the same general method used in single sine analysis. However the Fourier analysis of the slow phase eye movement data provides estimates of the response parameters given by the following equation:

$$r(t) = B + \sum_{i=1}^{N} A_i \sin(2\pi f_i + P_i) \qquad (2)$$

were B is bias or average slow phase velocity, with units of deg/sec, N is the number of sinusoidal components in the pseudorandom stimulus, A_i is the response amplitude at the i^{th} frequency f_i, and P_i is the response phase at the i^{th} frequency. A Fourier analysis of the stimulus velocity is performed to calculate the amplitudes and phases of the stimulus waveform. Then the reflex gains and phases at the N stimulus frequencies are computed as the ratio of response amplitude to stimulus amplitude, and the difference between response phase and stimulus phase, respectively. As with single frequency sinusoidal stimuli, a value of 180 degrees is added to the calulated phases for tests of the VOR reflex.

The gain and phase values of the VOR reflex were fitted with a transfer function equation of the following form:

$$H_{vor}(s) = \frac{K_v T_v s}{T_v s + 1} \qquad (3)$$

where T_v is an estimate of the VOR time constant (units of seconds), K_v is the VOR gain constant (unitless ratio of response amplitude to stimulus amplitude), and s is the Laplace transform variable. By substituting $s=j2\pi f$, with j equal to the square root of -1 (the unit imaginary number) and f equal to frequency in Hertz, the transfer function $H_{vor}(j2\pi f)$ can be expressed in terms of real and imaginary parts which are functions of frequency. The real and imaginary parts can be further expressed in terms of gain and phase as a function of frequency.

The form of the transfer function equation in equation 3 was selected since it provided a reasonable approximation to the actual data obtained from subjects. The term reasonable approximation is used here as a very qualified statement since there were systematic discrepancies between the actual data values and the curve fit. These discrepancies were particularly evident at frequencies above 0.1 Hz and one could logically argue that transfer functions with more parameters or of a different form could better approximate the actual data. However the transfer function parameters in equation (3) are consistent with the VOR parameters measured by other researchers and provide a means of comparing our results to theirs.

OKR gain and phase data for all subjects were well fit by a three parameter transfer function of the form:

$$H_{okr}(s) = \frac{K_o}{T_o s+1} \exp(-T_d s) \qquad (4)$$

where T_o is a time constant with units of seconds, K_o is the OKR gain constant relating slow phase eye velocity to stimulus velocity, and T_d is a time delay parameter with units of seconds describing the lag between the stimulus movement and the following eye movement. The $T_o s+1$ factor represents a lowpass filter which accounts for the declining gain with increasing frequency observed in some subjects. Larger values of T_o are consistent with gain declines beginning at lower frequencies. A value of zero for T_o (i.e. the transfer function reduces to $H_{okr}(s) = K_o \exp(-T_d s)$) accounts for subjects whose gain did not decline with increasing frequency.

ENG tests

Standard electronystagmography (ENG) tests were performed on normal subjects. ENG testing involved the recording horizontal and vertical eye movements under various conditions of gaze direction, head postion, and body position. These conditions include:

1. Gaze center
2. Gaze right 20 degrees
3. Gaze left 20 degrees
4. Pendular tracking
5. Supine body position with head straight
6. Supine with head right
7. Supine with head left
8. Left side body position
9. Right side body position
10. Supine with head up 30 degrees

The ENG test also included the Hallpike test where the subject's upper body was lowered rapidly from a sitting to a lying position with the head directed either to the right or left. This maneuver provides a strong stimulus to the vertical semicircular canals and may initiate a pathological nystagmus known a benign paroxysmal positional nystagmus and vertigo (BPPN and BPPV) in some subjects.

Finally the ENG test included four irrigations of the external ear canals using a Brookler-Grams closed loop caloric irrigator. Caloric irrigations artificially stimulate the horizontal semicircular canals and thereby invoke horizontal nystagmus. Due to the uncomfortable nature of the caloric test, it was not performed on subjects under 12 years. Complete data was not obtained on other older subjects since some became nauseated or simply choose not to continue with the caloric irrigations. Each ear was stimulated with a 45 second irrigation a 30 degrees and 44 degrees Celcius. Horizontal and vertical eye movements were recorded with EOG techniques identical to those described for rotation tests. Eye movements were recorded during and after each irrigation for a total of 3 minutes. Horizontal eye movements were analyzed to calculate peak slow component eye velocity. A comparison of peak velocities for the two ears provides an indication of the balance of sensitivity between the two ears. This comparison is quantified by the unilateral weakness measure defined by:

$$UW = \frac{(RW + RC) - (LW + LC)}{RW + RC + LW + LC} \times 100 \qquad (5)$$

where RW, RC, LW, LC are the absolute values of peak slow phase eye velocities recorded during right warm, right cold, left warm, and left cold irrigations respectively.

Subjects were tasked during ENG testing to maintain their alertness. Tasking routines were identical to those used in rotation tests.

Data Quality

The overall quality of each rotation and caloric test for each subject was subjectively given a rating of good, fair, or poor. Only good and fair quality data are included in the data summaries in the results section. Quality judgements were based on the standard deviation of response parameters (such as gain, phase, and bias from rotation tests),on the consistency of the responses throughout the duration of the stimulus, and on the accuracy of the eye movement analysis in the separation of slow and fast phases of nystagmus. The actual values of response parameters were not used in judgement of data quality. The test results from approximately 4 percent of subjects were rated poor for each test. Poor quality data for one subject on one test did not affect the judgment of data quality of the same subject on other tests.

Posture tests

In conjunction with a NIH grant on posturography (NIH grant # NS19222), 214 of the 216 subjects participating in the ENG and rotation tests were also tested on a moving posture platform. This platform test provided a functional evaluation of the ability of a subject to effectively use vestibular, proprioceptive, and visual information in the control of their upright posture (Nashner, 1981; Black et al., 1983). The subject's task during posture testing was to maintain upright stance for 21 seconds with as little postural sway as possible while they were presented with six different sensory environments. Anterior-posterior (AP) sway angle was measured using a potentiometer attached to the hips at approximately the level of the subject's center of gravity. Changes in the sway angle was a measure of the postural stability of the

subject. The first second of the 21 second trials provided a baseline measure of the subjects upright stance position since the altered sensory environments occurred only during the final 20 seconds of the trial.

The simplest two trials required the subjects to stand on a stable surface for 21 seconds with eyes open facing an earth fixed visual field and with eyes closed. These are the standard Romberg tests which are typically performed in a clinical setting in order to qualitatively judge postural stability. The remaining four trials placed the subject in more demanding sensory environments. These envirinoments were created by altering either the visual field orientation or the platform support surface orientation in proportion to the subject's AP sway angle. For example, as the subject swayed forward, the visual field rotated forward about an axis through the ankle joint. Under this condition the subject saw no change in orientation with respect to himself. This is referred to as sway-referenced vision as opposed to the gravity-referenced vision which we normally experience. The same technique was applied to the support surface by rotating the platform about the ankle joint in proportion to AP sway angle. This sway-referenced support condition resulted in no change in ankle joint angle as the subject swayed forward and back and therefore minimized the proprioceptive cues contributing to posture control.

The entire posture test sequence included all combinations of eyes closed, sway-referenced, and gravity-referenced vision and support surface conditions given by the following table:

	TEST CONDITIONS			SENSORY CONDITIONS	
Trial	Sensory Conflict	Visual Reference	Support Surface Reference	Accurate	Inaccurate
1. SnVn	no	gravity	gravity	vis, vest, prop	
2. SnVc	no	eyes closed	gravity	vest, prop	
3. SnVs	yes	sway	gravity	vest, prop	vis
4. SsVn	yes	gravity	sway	vest, vis	prop
5. SsVc	yes	eyes closed	sway	vest	prop
6. SsVs	yes	sway	sway	vest	prop, vis

The abbreviated identifiers for the trials, e.g. SsVn, use capital letters to signify support surface, S, and the visual field,V. The small letters refer to the stimulus condition of the support surface or the visual field with n standing for normal or gravity-referenced condition, s for sway-referenced condition, and c for eyes closed. For example, SsVc stands for sway referenced support surface with eyes closed.

AP sway was quantified by various methods, all of which gave roughly equivalent results when the data from the entire population were compared. The first method calculated the average rectified sway for the final 20 seconds of the 21 second trials. The sway during the final 20 seconds was referenced to the average sway position recorded in the first second of the trial when the visual field and/or the support surface were gravity referenced. Average rectified sway has units of degrees. This measure often did not reflect how close a

given individual was to a fall since, for example, a subject who leaned forward by a few degrees and stayed in that position throughout the remainder of the trial could score the same as a subject who oscillated back and forth during the trial with the peak of the oscillations being close to the threshold of a fall.

Therefore several other measures of stability based on peak sway angles were made. These included peak forward sway, peak backward sway, peak forward minus peak backward sway, and peak forward sway normalized to the approximate theoretical limit of forward sway without falling (7 degrees) minus peak backward sway normalized to the approximate peak backward sway (4.5 degrees). Measures based on peak sway also had disadvantages. For example, a few subjects would thrust their hips forward and backward in an attempt to maintain balance (often referred to as a hip strategy) resulting in large peak values of sway angle measured by the potentiometer attached to the hips. When a subject used the hip strategy, our sway measure did not accurately reflect the movement of the subject's center of gravity with respect to their support base (the length of their feet), and therefore did not reflect the nearness of the subject to their threshold for a fall.

Data Base

All VOR, OKR, Caloric, and posture test results were entered into a Digital Equipment Corporation Datatrieve data base program running on a VAX 11/750 computer. The data base permitted convenient sorting and comparisons of the various response parameters.

RESULTS

Normal subjects showed a wide range of responses on most measures of vestibuloocular, oculomotor, or postural stability function. The only exceptions occurred on tests which most resembled normal environmental situations, such as the VVOR test and the first two posture test trials. The tests which gave low variability were all situations in which accurate orientation information was available from more that one sensory system. It was clear that the restriction of sensory information to a single system, or the presence of conflicting or inaccurate sensory information revealed the variable functional capacity present within a presumed normal population.

Age related changes were identified in almost all response measures. All the changes were in a direction indicating a decline in function. For example, VOR gains declined, OKR time delays increased, and the incidence of falls during posture testing increased with increasing age.

VOR Rotation Tests

Typical VOR test results from 0.05, 0.2, and 0.8 Hz sinusoidal tests are shown in Figure 2. Response gain was generally highest at 0.8 Hz and lowest at 0.05 Hz. The gain was less than unity in most subjects. Response phase was near zero at 0.2 and 0.8 Hz and showed a phase lead at 0.05 Hz.

The distributions of gain, phase, bias, offset, and asymmetry are shown for our normal population at all test frequencies in Figures 3-7. Table 1 summarizes the statistics of these distributions. The small differences in N's are due to

data eliminated from consideration because of its poor overall quality. These distributions are reasonably symmetrical about their means. Gain increased with increasing frequency and had lower variance at 0.8 Hz as compared to 0.2 and 0.05 Hz. The phase variance also decreased with increasing frequency. The higher variance of the 0.05 Hz phase probably reflects the variability of the VOR time constant in the normal population. Since phases at higher frequencies are not affected by the VOR time constant their distributions had lower variances. The variance of the offset distributions are all somewhat less than the variances of the bias distributions. This is consistent with fact that both nonlinear responses (asymmetric gains) and nonzero average slow eye velocity contribute to the bias measure. That is, a portion of the bias measure is accounted for by the presence of asymmetrical gains. The remaining portion, which is the response offset, is therefore smaller than the bias.

A typical response to a pseudorandom VOR stimulus is shown in Figure 8. The data analysis separates slow and fast phases of the nystagmus. The vertical bars between the horizontal EOG and slow eye velocity traces show where the program found fast phase eye movements. A spectral analysis of slow eye velocity and the recorded stimulus velocity provide measures of response gain and phase as a function of stimulus frequency. An example of gain and phase data are shown in Figure 9. Typically the gain is lower at the lowest test frequency and increases with increasing frequency. In some subjects the gain monotonically increases over the frequency range tested and in others it appears to reach an asymptote. The dotted lines through the data points represent a minimum least squared error curve fit of a two parameter transfer function (equation 3) to the data.

The distribution of the VOR gain constants and time constants are shown in Figure 10 and 11 and summarized in Table 2. The gain constant distribution was symmetrical with an average value of 0.72. The VOR time constant distribution was skewed toward longer time constants. Mean value was 24.4 sec and median value was 23.0 seconds. Only two subjects had time constants below 10 seconds, and one of these subjects (time constant = 8.2 sec) had a unilateral loss of vestibular function as judged by caloric testing. A short time constant is consistent with a unilateral loss of vestibular function as reported by others (Paige, 1983) and also verified by test results in our lab. The other subject (time constant = 7.0 sec) had normal caloric test results. Therefore the abnormally short time constant for this subject remains an unexplained anomaly.

Pseudorandom test results are displayed in Figure 12 as percentile plots of VOR gain and phase data as a function of stimulus frequency. As with single frequency sinusoidal results, the variance of gain data were fairly constant across all frequencies. The variance of phases data was larger at low frequencies than at high probably reflecting the variance of the VOR time constant amoung individuals. The variance of the phase at 1.535 Hz was larger than the phase data at adjacent lower frequencies. This probably resulted from the fact that the stimulus amplitude of the highest frequency component was half that of the lower frequencies resulting in a lower signal-to-noise ratio at the 1.535 Hz test frequency.

Comparison of Single Frequency and Pseudorandom VOR Results

If the VOR were a linear system, then gain and phase data obtained from single frequency sinusoidal and pseudorandom stimulation should be identical within the random variability introduced by measurement errors. Statistical comparisons were made between the single frequency and pseudorandom gains and phases and are shown in Table 3. Single sine and pseudorandom results were not significantly different at the lowest frequency (0.05 Hz). Small but consistent differences were evident at higher frequencies. In particular, the single frequency gain was higher at 0.8 Hz than the pseudorandom derived gain and the pseudorandom phase values at 0.2 and 0.8 Hz were phase advanced by about 3 degrees compared to single frequency sine results.

Phases values from single sine tests at 0.2 and 0.8 Hz were nearer to zero than the phases from pseudorandom results. One could argue that the improved phase response from sine tests represented a small predictive effect. However this effect did not apparently carry over to the 0.05 Hz data. Usually one expects a lower test frequency to be the most likely to be influenced by any predictive mechanisms.

The gain value from the 0.8 Hz test was higher than the pseudorandom test result. This might also be due to a predictive effect. However, in this case it seems likely that the data analysis methods could have contributed to this higher value. During the analysis of the single frequency sine tests, the experimenter had the ability to reject data from stimulus cycles which were obviously corrupted. These corrupted data cycles could easily be identified based on gain, phase, and/or bias values which deviated greatly from the values for other cycles. There were several causes for poor data cycles including transient increased EMG interference as a subject squinted, excessive blinking, looking up or down, inattentiveness to tasking by the testing technician, and failure of the fast phase eye movement detection algorithm. With experience, it becomes a simple task to detect and correct these problems by rejecting the affected cycles. The net effect usually increases the average gain measure. Clearly there exist during pseudorandom testing the identical problems which transiently corrupt the eye movement data. However there does not exist in our current analysis methods a means of correcting or eliminating these problems and they are therefore averaged into the final result. A likely consequence is that there will be systematic differences between pseudorandom and single sine parameter measurements resulting from the differences in analysis. The magnitude of these differences will depend on how often transiently corrupted eye movements occur within a data record.

OKR Rotation Test

Typical OKR test results from pseudorandom stimulation are shown in Figure 13. Gain and phase data for two subjects are shown in Figure 14. The main features are the following. Response gain was less than unity. The gains of most subjects were approximately flat across the bandwidth of frequencies tested (0.02 to 1.5 Hz) as in Figure 14A. Phase was near zero degrees at the lowest frequencies and showed monotonic increasing phase lags as frequency increased. Since perfect tracking of the visual stimulus is represented by unity gain and zero phase at all frequencies, subjects demonstrated very imperfect tracking in terms of both amplitude (gain) and timing (phase). OKR response bias was near zero deg/sec for all subjects.

The major variation on the typical OKR result was the presence of declining gain with increasing frequency in some subjects. Figure 14B shows the OKR transfer function data from one such subject.

The means, standard deviations, and ranges of OKR gain constant, time constant, and time delay are given in Table 4. The distribution of these parameters are shown in the histograms in Figures 15-17. Both the gain constant and time delay have approximately symmetric distributions. In contrast, the OKR time constant has a highly skewed distribution with about 40 percent of the values near zero. OKR time constants near zero reflect the fact that OKR gains for these subjects were approximately constant over the frequency range tested.

OKR gain and phase measures were dependent on both the amplitude and bandwidth of the pseudorandom stimulus. The results for one subject tested at constant bandwidth but varying amplitude are shown in Figure 18. At the lowest stimulus amplitude the gain was approximately flat. As the stimulus amplitude increased the gain decreased at every frequency. The gain decrease was proportionally larger at the higher frequencies resulting in a change in shape of the gain function from an approximately flat one to a function which declined with increasing frequency. Phase values showed the least amount of lag for the smallest amplitude stimulus and generally showed increasing phase lags with increasing stimulus amplitude. The exception to this were the two lowest test frequencies, 0.02 and 0.04 Hz, where phase values apparently were not affected by the increasing stimulus amplitude.

Pseudorandom stimulus bandwidth affects on OKR responses in one subject are shown in Figure 19. This subject showed the typical constant gain result when tested by the lower bandwidth signal. The higher bandwidth result showed both lower gains and a change in shape of the gain function so that gain was now decreasing with increasing frequency. This decrease in gain with increasing frequency was clearly apparent at frequencies which overlapped with those of the lower bandwidth stimulus. The phase data from the higher bandwidth stimulus showed increased lags compared with the lower bandwidth stimulus except at the lowest overlapping test frequencies where the phase difference was small. The effects on gain and phase are apparently similar when pseudorandom stimulus bandwidth and amplitude are increased.

The standard OKR pseudorandom stimulus proved to be quite provocative in the initiation of motion sickness symptoms. Twenty subjects requested the termination of testing as a result of the onset of motion sickness symptoms. Approximately an equal number experienced motion sickness symptoms but were able to complete the 220 second duration OKR stimulus. It was not possible to calculate OKR gains and phases from incomplete trials using our current analysis methods. Therefore it was not possible to test the hypothesis that abnormal OKR responses were related to motion sickness sensitivity in these highly susceptable subjects. However, OKR gains and phases from subjects who reported the onset of motion sickness symptoms but were able to complete the test did not show any obvious differences compared with subjects who did not report symptoms. Also comparisons of VOR rotation test results of OKR motion sickness susceptable subjects with nonsusceptable subjects did not reveal any differences between these populations.

VVOR Rotation Test

A limited number of normal and abnormal subjects were given pseudorandom stimulus VVOR tests during which the subject rotates within a stationary visual field. Since the VOR and OKR reflexes both contribute to the generation of compensatory eye movements, it was expected that VVOR eye movements would be better (more nearly perfect compensation) than those provided by either the VOR or OKR alone. The results in Figure 20 reveal that this was indeed the case. Within experimental error, gains were unity and phases were zero across the full bandwidth of test frequencies.

Particular attention was paid to the VVOR responses in subjects whose VOR results were phase advanced from zero degrees at higher frequencies. These subjects were of interest because they provided a population in which it was possible to test the hypothesis that the OKR makes a constructive contribution to the generation of compensatory eye movements at higher frequencies.

The classical description of VOR and OKR interactions suggest a frequency separation of function (Robinson, 1977). That is, the VOR provides the drive for compensatory eye movements at high frequencies (unity gain and zero phase), and the OKR provides the drive at low frequencies (once again, unity gain and zero phase). The actual VOR results in many subjects were not in agreement with the classical description of the VOR which predicts zero degree response phases for frequencies above about 0.2 Hz.

Caloric Test

Figure 21A shows a histogram of the absolute value of the unilateral weakness measure. These results are generally in agreement with those of other researchers (see Barber and Stockwell, 1980) who report normal ranges of unilateral weakness of about 15 to 25%. Our results are consistent with a 25% definition of normal since 95% of our subjects had unilateral weakness measures below this value.

The histogram of the average peak value of slow phase eye velocity evoked from caloric irrigations is shown in Figure 21B. The distribution is skewed to the right and had an average value of 17.0 deg/sec.

Age Related Changes in VOR, OKR, and Caloric Test Results

Most VOR and OKR response parameters showed aged related changes (Figures 22-25), however caloric response parameters did not (Figure 26). In particular, all single sine gains and the pseudorandom gain constant decreased with increasing age. Single sine response phases increased with increasing age at all frequencies tested, although the effect was more pronounced at 0.2 and 0.8 Hz than at 0.05 Hz. VOR time constant derived from pseudorandom results decreased with increasing age. OKR gain constant decreased with increasing age and OKR time delay parameter increased with increasing age. The OKR time delay showed the clearest age related trend (correlation coefficient = 0.53 and slope = 1.2 msec/year) of any of the VOR and OKR parameters studied. Table 5 summarizes the observed age related changes with age.

The age trends of VOR and OKR parameters mentioned above appeared to be linear with age. The only parameter studied which showed an age related effect which was clearly not linear was the OKR time constant. Data in Figure 25B show that

a large number of subjects between about 20 and 60 years had OKR time constants close to zero indicating that their OKR gain was constant across frequency. In contrast, there were very few subjects under 20 years and proportionally fewer of the subjects over 60 which had zero OKR time constants, indicating that on average their OKR gain declined with increasing frequency. A robust locally weighted regression curve fit (Cleveland, 1985) is plotted in Figure 25B. This curve fit indicates that age related trends were minimal for subjects between 20 and 60 years. Subjects under 20 years showed an age related decline in their OKR time constant with increasing age. Subjects over 60 years showed an age related increase in their OKR time constant with increasing age.

Age related effects on caloric test results were ambiguous. A linear regression curve fit to the data in Figure 26A showed an average decrease in slow phase eye velocity with increasing age. The linear regression had an associated correlation coefficient of -0.15 which is significantly different form zero ($P<0.05$). However the robust locally weighted regression fit shown in Figure 26A indicated that a linear regression was probably not an appropriate description of the data. Decreases in slow phase velocity were most pronounced for subjects under about 35 years, and showed small increases for older subjects.

Figure 26B plots the absolute value of the unilateral weakness measure against age. The robust locally weighted regression shows essentially no change over the first 6 age decades, and a slight increase in older subjects. Due to the large variance in the data, a much larger sample would be required to determine if the small increase in older subjects was significant.

Abnormal VOR, OKR, and VVOR Examples

As an example of the nonlinearities revealed and quantified in the VOR analysis, Figure 27 shows the results of a VOR test from a subject following a left side labyrinthectomy. There is a large shift of the slow phase eye velocity versus time data to the left resulting in a large negative bias measure. However the nonlinear analysis of the eye velocity versus stimulus velocity plot reveals that the VOR gains for eye movements to the right and left are not equal. This gives a large negative asymmetry measure. Since part of the response bias is accounted for by the asymmetrical response gains, the measured offset is smaller than the bias by more than a factor of two.

An example of the VOR pseudorandom test gain and phase data obtained from a subject with a large (approximately 3 cm) acoustic neuroma in the left ear is shown in Figure 28. The VOR gain constant is normal but the VOR time constant is very small, T_v = 6.33 sec. Response bias is equal to -4 deg/sec. The direction of the bias is consistent with a loss of vestibular nerve activity on the left side. This pattern of results is consistent with a fairly well compensated complete loss of function on the affected side.

Figure 29 shows the VVOR test results from a subject who experienced a nearly total bilateral loss of vestibular function due to streptomycin ototoxicity. Since this subject's VOR is nearly absent, eye movements evoked by rotation within a stationary visual field are nearly identical to their eye movements obtained from the OKR. That is, if you have no vestibular function, it is not possible to distinguish between the world rotating around yourself and your rotating in a stationary visual environment.

Posture Test Results

As visual and proprioceptive sensory information is removed or made inaccurate, subjects are forced to rely on their vestibular systems for posture control. Under these circumstances subjects become less stable and falls become more likely. Figure 30 shows typical results from one subject who was attempting to stand as still as possible under different sensory conditions. On average subjects were very stable, as judged by their peak forward minus peak backward sway angle, with eyes open on a stable surface, and with eyes closed on a stable surface (trials SnVn and SnVc). No subjects fell under SnVn and SnVc conditions. Postural stability was decreased on the SnVs trial when the visual surround was sway referenced to the subject's sway. Under this SnVs condition, proprioceptive and vestibular inputs are normal, but visual references are inaccurate. The median of the SnVs sway distribution in Figure 31 is only about one degree higher than the SnVc trial with eyes closed indicating that most subjects had only slightly more difficulty controlling their posture under the SnVs condition than under the eyes closed condition. However, the SnVs distribution is highly skewed toward larger sway amplitudes than the SnVc distribution indicating that a significant fraction of the normal population had a great deal of difficulty maintaining their upright posture when visual orientation information was present but inaccurate. In fact 23 of 214 subjects (10.7%) fell on the SnVs trial. This indicates that these subjects, and others near the tail of the distribution, were highly dependent on visual orientation cues. These subjects chose visual orientation cues in preference to accurate available vestibular and proprioceptive cues. Since visual cues during the SnVs trial were in fact inaccurate, the postural sway increased to the point where they fell.

The SsVn trial provided accurate visual cues but inaccurate sway referenced proprioceptive cues as the platform upon which the subject stood rotated in proportion to their sway. On average subjects swayed more under these conditions than during the first two trials. This distribution was skewed toward larger sway angles in a similar manner to the SnVs distribution. However subjects on average were more stable then in the sway referenced vision trial since only one subject out of 214 (0.5%) fell on the SsVn trial.

On the SsVc trial visual cues were absent (eyes closed) and proprioceptive cues were inaccurate since the platform was sway referenced. Under this condition only vestibular orientation cues could be used for posture control. Average sway angle was larger than on any of the previous trials and was also skewed toward larger sway angles. Twenty two of 214 subjects (10.3%) fell on this trial.

The SsVs trial was by far the most difficult of the six conditions for subjects to maintain their balance. Under this condition both the visual surround and the platform were sway referenced and therefore were providing inaccurate proprioceptive and visual orientation cues. Only the vestibular system was providing accurate information for posture control. In order to maintain their balance, subjects had to select the accurate vestibular cues and ignore the inaccurate sensory cues. Apparently this was a difficult task as the average sway for subjects who completed the trial was larger than on any other trials, and 57 of 214 subjects (26.6%) fell.

Fall Patterns in Posture Tests

Falls during sensory trials were not random occurrences, but rather were associated with the inability of some subjects to obtain and/or coordinate the sensory information available for the control of posture. Table 6 summarizes the data on subjects who fell during one or more of the six trials. Seventy three of 214 subjects (34.1%) fell on one or more trials. Of these, 45 (45/214 or 21.0%) fell on only one trial, 25 of 214 (11.7%) fell on two trials, and 3 of 214 (1.4%) fell on three trials. Of the 45 subjects who fell on only one trial, 32 of 45 (71.1%) fell on the SsVs trial, 7 (15.6%) on the SsVc trial, 5 (11.1%) on the SnVs trial, and 1 (2.2%) on the SsVn trial. Of the 25 subjects who fell on two trials, 13 of 25 (52%) fell on SnVs and SsVs trials, 10 (40%) fell on SsVc and SsVs trials, and 2 (8%) fell on SnVs and SnVc trials. Of the 3 subjects who fell on three trials, all 3 fell on SnVs, SsVc, and SsVs trials.

Consider subjects who fell on two of the four trials which presented them with sensory conflict situations. There are six combinations of paired falls within a grouping of four trials. If paired falls occurred randomly they would be evenly distributed across the six possible combinations. This was clearly not the case in our population since three of the six combinations of paired falls were not observed. That is, no subjects fell on SnVs-SsVn, SsVn-SsVs, and SsVn-SsVc paired trials. Only two subjects fell on the SnVs-SsVc trials. Paired falls were primarily limited to only two of the six possible paired combinations. Ten subjects fell on the SsVc-SsVs trials and 13 fell on the SnVs-SsVs trials.

The three subjects who fell on three trials were also not randomly distributed among the 4 possible combinations of the 4 sensory conflict trials taken 3 at a time. Rather all three subjects fell on the same set of three trials which was the SnVs-SsVc-SsVs combination. This combination is interesting since it combines the features of the two most common paired trial falls, SnVs-SsVs and SsVc-SsVs. That is, these subjects show both an increased visual orientation reference dependency and a vestibular deficit. Patients which show this pattern of falls have previously been identified by Black and Nashner (1984a). These subjects, who constituted only 1.4% of our subjects, could be considered to be quite seriously impared relative to the remainder of the population. All of these subjects were in the older age decades; their ages being 60, 69, and 70 years.

There was evidence of vestibular reflex abnormalities on other vestibular tests for subjects who fell on two or more posture trials. A comparison was made of VOR rotation test response parameters in the 28 subjects who fell on two or more posture trials to the VOR parameters in 8 sets of 28 subjects which were randomly selected from the entire population of 214 subjects. This comparison involved a count of the number of subjects in each group who had VOR parameters at the fringes of the respective parameter distributions (above and below the upper and lower 2.5 percentile points, respectively). The VOR parameters included gain, phase, bias, offset, and asymmetry at 0.05, 0.2, and 0.8 Hz as well as gain constant, time constant, and bias obtained from pseudorandom test results. 54% of subjects with multiple falls had single sine test VOR parameters on the fringes of the distributions compared to an average of 37% (8.4% s.d.) for the 8 control groups. The largest control group percentage was 46%. When pseudornadom VOR results were included with single sine results, 61% of the multiple fall subjects had parameters on the fringes compared to an

average of 44.4% (8.6% s.d., largest 54%) for the control groups. Using a criteria which required a subject to have two or more VOR parameters at the fringes of their distributions, then 25% of the multiple fall group met this stricter condition compared to 10.8% (4.7% s.d., largest 17.9%) for the control groups.

Clearly there is evidence that subjects who fell on multiple posture tests were more likely to have abnormal or borderline normal VOR test results. This quantifies the expected result that a peripheral vestibular pathology is likely to effect both horizontal canal function and vertical canal/otolith function. However the fact that about half of the multiple fall subjects did not have unusual horizontal plane VOR results indicates that either there was a separation of peripheral vestibular pathology or that central mechanisms associated with the vestibulospinal system were faulty while VOR pathways were normal.

Age Related Changes in Posture Test Results

The general trend was for falls to increase with increasing age. These results are summarized in Table 7. The incidence of falls was generally lowest for subjects 20 to 40 years of age. Subjects ages 10 to 20 years had a high incidence of single trial falls (30%) but low multiple trial falls (3%). The occurrence of single trial falls increased rapidly for subjects 40 years and older, although the incidence of multiple trial falls remained quite stable through the 50's age decade before showing a jump in the 60 to 70 age group. A possible anomolous result was obtained in the over 70 age group for multiple falls. Their multiple fall rate was much less than the fall rate for 60 to 70 year old subjects and approximately the same as for subjects younger than 60 years. This might be a result of the small sample size of the over 70 age group. Alternatively the anomoly might be in an exceptionally high fall rate for subjects in the 60-70 age group.

DISCUSSION

The major accomplishments of this study are the following: 1) a determination of the ranges of normal function for a variety of vestibular tests, 2) a detection and quantification of age related changes in vestibuloocular, oculomotor, and vestibulospinal reflex function and, 3) a documentation of the incidence of vestibular abnormalities within an otherwise normal population. These points are discussed in more detail below.

Almost all vestibular and oculomotor reflexes tested showed a wide range of what must be considered normal function. The execeptions to this rule involve reflex responses which function under "normal" operating conditions including the VVOR rotation test and the first two posture test trials where the subject stands on a earth fixed platform with eyes open and closed. Without exception, our normal population performed these tests with very low variance.

Both the VVOR and the two posture tests are characterized by the presense of multiple sensory system inputs which converge and cooperate in the generation of appropriate and accurate motor responses. Apparently subjects are very well adapted for dealing with their environment under normal operating conditions. However it is clear that the "sum of the parts" which make up the "whole" are

subject to a great deal of variability. In VOR and OKR test results this variabiltiy is manifest in the wide range of response parameters observed. In posture testing, the variability is demonstrated by the wide range of postural sway trajectories recorded under varying conditions of accurate, inaccurate, and absent visual and proprioceptive cues to orientation. In some individuals during posture testing, there is a complete failure to accomodate (resulting in a fall) to some of the sensory conditions imposed while in others these same unusual sensory conditions hardly increased sway compared to the first two "normal" trials.

Although there exists a wide range of normal function in VOR and OKR responses, this range is not so wide as to exclude the possibility of detecting abnormal function based on these tests. Clearly there is room for subjects with abnormally low gains on VOR and OKR tests. Clinical experience with VOR tests have shown that low gains and large phase leads are associated with bilateral losses of vestibular function (Honrubia et al., 1985; Peterka and Black, 1985). If bilateral loss is sufficient, then VVOR test results (Figure 29) begin to show abnormal phase lags at higher frequencies. Functionally these phase lags result in blurred vision during rapid head movments and often lead to reports of oscillopsia where the subject feels that the world jumps when they move their heads. Low gains in OKR tests have been associated with cerebellar degeneration which probably resulted in the loss of cerebellar centers related to the control and/or generation of the fast component of the OKR response. Subjects with absent OKR responses will also show abnormal VVOR phase results at low frequencies. That is, without the aid of vision in the control of eye movements, the low frequency phase leads which are characteristic of normal VOR function remain in the VVOR results. Clinical experience has also led to the identification of subjects with abnormal VOR response phases. Abnormally large low frequency VOR phase leads, and equivalently a small VOR time constant, but with normal VOR gains, bias, offsets, and asymmetries are characteristic of subjects with a well compensated unilateral loss of vestibular function (Figure 28). One of our "normal" subjects had this type of VOR data, and their caloric test results were consistent with a diagnosis of unilateral loss. One other "normal" subject had the same pattern of VOR data, but their caloric results were normal. This result remains unexplained.

High VOR gains have been clinically associated with cerebellar degeneration. However, the high end of the normal range of VOR gains identified in this study are greater than unity and therefore partially overlap with the high gains associated with cerebellar disease. Therefore care must be exercised in interpreting high VOR gains as an indicator of central pathology.

In contrast to the VOR and OKR tests, the range of results obtained from posture tests from our presumed normal subjects does include much of the range from patients with vestibular pathology. One could speculate that the posture test sequence was simply too difficult and therefore was overly sensitive to minor deficencies in sensory system function or in neuromuscular control. This later conclusion is not supported due to several observations. First is the number of subjects who were able to perform all posture trials with little or no difficulty. Second is the way in which many subjects fell on posture trials. Some subjects showed no postural correction strategy indicating that the posture control mechanism was totally nonfunctional under the given sensory conditions. Third is the shape of the sway distributions in Figure 31. It is clear from the distributions in which a significant number of subjects fell,

that there was a separation of falls from the average performance of subjects who did not fall. Fourth is the fact that falls on the posture trials were not random occurrences but rather occurred in distinct patterns which were the same patterns as those shown in patients suffering from clearly defined abnormalities.

The two paired fall combinations which produced the majority of the paired falls can be logically associated with specific types of sensory problems. The 13 subjects who fell on the SnVs-SsVs trials were highly dependent on vision for their orientation reference. Black and Nashner (1984a and b) have shown that this type of visual dependency is common amoung individuals who have a syndrome known as benign paroxysmal positional nystagmus and vertigo (BPPN or BPPV). The reason for this increased visual dependency is unknown. One hypothesis would be that the visual dependency is the result of a central nervous system compensation in response to a vestibular deficit associated with the posterior semicircular canal and/or otolith organs. The posterior canal is implicated since histological studies on subjects who suffered from BPPV revealed dense deposits on the posterior canal cupula (Schuknecht, 1969). This would cause the canal to be responsive to linear accelerations of the head rather than the normal situation where canals are only responsive to rotational movements. Since it is likely that the central nervous system requires both rotational information from the vertical canals and linear motion signals from otoliths for the synthesis of an internal orientation reference with respect to gravity, the distorted information arrising from the affected canal could initiate a central reorganization of orientation control which relied on an alternative orientation cue (vision) rather than the faulty vestibular signals.

The subjects who fell on SnVs-SsVs trials present an interesting paradoxical behavior. The fact that these subjects did not fall on the SsVc trial indicates that they had sufficient vestibular function to properly maintain a stable stance posture. However they chose to ignore the vestibular input when visual information was present, even though the visual information conflicted with the vestibular. On the SnVs trial, both proprioceptive and vestibular inputs were in disagreement with visual cues. However these subjects still chose vision as their orientation reference.

The second most common paired fall situation (SsVc-SsVs) forces a subject to rely on their vestibular systems for posture control since proprioceptive and/or visual cues are either absent or inaccurate on both trials. Subjects who fell on these trials could reasonably be called vestibular deficient. There are several possible sources of a particular subject's vestibular deficiency. A subject with total bilateral peripheral loss of function would clearly qualify as one extreme form of vestibular deficiency, and indeed clinic patients with bilateral loss (as judged from absent caloric and rotation responses) will invariably fall on both the SsVc and SsVs trials. Since we did not perform independent tests of vertical canal and otolith function (e.g. vertical plane rotations or ocular counterrolling) in subjects we cannot rule out the possibility that these paired fall subjects had absent vertical canal and otolith function. However horizontal plane rotation tests and caloric tests did not reveal the typical flat responses seen in subjects with total bilateral loss of vestibular function.

Another source of vestibular deficit could be a bilateral reduced, but not absent vertical canal and/or otolith function. Again, independent tests of vertical canal and otolith function would be required to test this hypothesis.

A vestibular deficit posture response could also arise from central mechanisms. The central mechanisms are performing a quite complex task which includes both the selection of the appropriate sensory orientation reference in the face of conflicting cues from several sensory systems and the generation of the correct motor commands to the muscles. It is possible that peripheral vestibular signals may be normal, but central mechanisms which make use of this information are faulty. The fault may have more than one source. For example, the processing of the sensory information may simply be too slow, in which case the appropriate motor commands arrive at the muscles but are too late to prevent a fall. Alternatively, the central processing which must deal with conflicting sensory information may produce inappropriate responses based on the available sensory signals. These inappropriate responses could drive the system into instability with a resulting fall.

Finally, subjects with weak muscles relative to their body masses would be more susceptible to a fall than stronger subjects since the lower forces generated by weaker subjects would diminish their ability to correct for postural purturbations. This effect would be particularly important near the limits of stability where maximal forces are necessary to move the subject away from the brink of a fall. Since the SsVc and SsVs trials are the most difficult in the sense that they bring subjects closer to the threshold of a fall than do the other four trials, the relative strength of subjects would be more likely to play a role in the last two trials.

We feel that the large failure rate on the posture trials probably reflects a combination of peripheral vestibular deficits and inappropriate central nervous system coordination of sensory information. In support of the hypothesis that posture results reflected underlying vestibular deficits is the fact that subjects who fell on two or more trials also had significantly greater number of VOR response parameters which were on the tails of the population distributions of those parameters. Since it is reasonable to assume that distance from the mean reflects vestibular abnormality, there was at least a partial correlation between subjects with abnormal VOR results and abnormal posture control results. The correlation was not perfect however. We can think of three possiblities which would degrade the correlation. First is the fact that our VOR tests were indicative of horizontal canal function whereas head movements during posture testing primarily stimulate vertical canals and otoliths. To the extent that a vestibular abnormality would only affect one or a limited number of the vestibular receptors in each ear, VOR and posture results would be different. For example, the BPPV syndrome is believed to be related to abnormal posterior canal function and therefore would affect posture test results but not tests of VOR function in the horizontal plane of motion. A second reason for differences between our VOR and posture test results would relate to central nervous system problems in the organization of sensory system interactions. That is, it is reasonable to imagine that the vestibular signals from the ears are normal in some subjects, but that their posture test results are abnormal due to their inability to effectively deal with conflicting information from vestibular, proprioceptive, and visual systems. This implies a central problem rather than a peripheral problem. A third possibility is that both peripheral vestibular and central interactions mechanisms are normal but that sensory information from either visual and or proprioceptive systems are abnormal. For example, in light of the intersubject variabilty observed in the OKR, a similar variability in the visual motion detection system involved in posture control could reasonably influence the posture control patterns of

subjects in unusual environments. In particular, the wide range of time delays associated with visual system motion processing would likely have important affects on the closed loop posture control system.

We were able to clearly identify age effects on vestibular and oculomotor reflexes. The direction of change of some reflex parameters were expected, such as declining VOR gains and increased time delays in visual processing in the OKR with increasing age. The rate at which the OKR time delay increased with age was quite large. If this increased time delay is representative of general changes in the speed of visual motion information processing associated with all orientation control systems, then this would have great implications for the stability of feedback control mechanisms involving vision since longer feedback loop lags contribute to decreased stability.

The increases in VOR phase leads at higher frequencies with increasing age were not anticipated. On the surface they would seem to represent a degradation of function since the increased phases take the system response away from the goal of perfect compensatory eye movements (unity gain and zero phase). Perhaps the phase advances are an artifact of an adaptation which improves overall VOR function. For example, studies of peripheral semicircular canal function in the squirrel monkey have shown that higher gain peripheral nerve fibers have dynamic properties which include phase advances at higher frequencies (Fernandez and Goldberg, 1971). Phase advances indicate a sensitivity to the velocity of cupula deflection in addition to the cuplula position (which is assumed in the classical description of canal dynamics). In contrast, lower gain canal fibers show cupula position sensitivity and therefore, due to the integrating accelerometer characteristics of canal biophysics, the nerve responses are in phase with head velocity at higher frequencies of rotational movements.

We can postulate that in young people, a mixture of low gain and high gain canal fibers contribute to the overall VOR. As the subject ages and there is a gradual loss of peripheral canal input due to cell death, adaptive mechanisms in the central nervous system may be able to selectively increase the relative contribution of high gain canal nerve input as compared to the low gain fibers. The sum effect would be to maintain the gain of the VOR at a reasonable level allowing for the generation of adequate compensatory eye movements. However this mechanism of gain enhancement would be accompanied by the possibly undesireable phase leads associated with the dynamics of the high gain canal fibers. One could envision that a tradeoff is occurring between maintaining the desireable feature of high VOR gain and the undesireable feature of phase advances in a direction away from perfect eye movement compensation. Simply stated, this is an response amplitude versus timing tradeoff.

The demonstration of age related changes in vestibular function has implications for the assessment of normal function. Part of the wide spread of VOR parameters is caused by this age effect. The square of the correlation coefficient gives an estimate of the proportion of variance related to changes with age. The VOR gain versus age measures had correlation coefficients between 0.3 and 0.4. Therefore approximately 10 to 15% of the variance of gain data is accounted for by the aging affect. The largest correlation coefficient was 0.53 for the OKR time time delay indicating that 28% of the variance was due to the effect of aging. It is clear that the development of normal scales of vestibular function must account for age effects.

Since the majority of the observed response variability is independent of age, it is clear that the functional characteristics vary widely within any given age group in a normal population. To the extent that the aging affects are deleterious and that our reflex measures accurately characterize the general decline in function, it can be stated that a significant proportion of subjects within any age group look "older" than their chronological ages and are therefore less functional with regards to their orientation control abilities. One could postulate that these subjects would be more susceptable to the development of balance and orientation control problems as they age since we could expect their function to further decline with age. Perhaps there is some threshold beyond which the brain's adaptive mechanisms are not able to compensate for the declining function. After this point is reached, subjects may develop dizziness and equilibrium control complaints, or perhaps subjects will restrict their activities so as to avoid situations which stress their remaining capabilities.

We feel that it would be reasonable to conclude that approxiamtely 10 to 15% of the normal population studied had significant equilibrium control deficits. A few are clearly traceable to vestibular deficits, e.g. the subject with unilateral loss of vestibular function. Others are stongly suggestive of peripheral vestibular problems based on results of rotation and posture testing. However since the role of the central nervous system in the coordination of sensory signals from multiple systems is poorly understood, it is not possible to rule out the contribution of central interaction problems as opposed to peripheral sensory problems. But in any event, the overall effect is that equilibrium control deficits exists in a normal population, and that these deficits can only be detected using the sophisticated test batteries which only recently have become available to researchers.

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Table 1 - VOR Response Parameters for Single Sine Stimuli

VOR phase is in degrees, bias in deg/sec, offset in deg/sec, and asymmetry in percent. Gain is the ratio of slow phase eye velocity to stimulus velocity and therefore has no units. Seven percentile values on the distributions of the parameters are given.

Frequency = 0.05 Hz, N = 208

	Gain	Phase	Bias	Offset	Asymmetry
Mean	0.68	10.5	-0.44	-0.35	-0.9
S.D.	0.15	4.85	3.56	2.90	7.5
2.5%tile	0.39	0.94	-8.21	-6.11	-18.8
5%	0.44	2.47	-6.34	-4.84	-15.1
25%	0.58	7.54	-2.55	-2.03	-5.7
50%	0.67	10.27	-0.36	-0.32	-0.5
75%	0.78	13.41	1.89	1.46	4.0
95%	0.96	18.10	5.00	3.84	10.8
97.5%	1.02	19.19	6.73	6.46	13.8

Frequency = 0.2 Hz, N = 207

Mean	0.75	1.65	-0.62	-0.36	-1.4
S.D.	0.16	3.15	2.92	2.32	6.6
2.5%	0.40	-4.27	-6.59	-5.01	-14.5
5%	0.51	-3.44	-5.70	-4.00	-11.7
25%	0.64	-0.35	-2.46	-1.80	-5.5
50%	0.75	1.97	-0.64	-0.37	-1.5
75%	0.85	3.97	1.34	1.11	3.18
95%	0.99	5.97	4.33	3.48	9.72
97.5%	1.02	6.40	4.88	4.51	10.4

Frequency = 0.8 Hz, N = 203

Mean	0.84	0.80	-0.29	0.03	-1.4
S.D.	0.13	2.59	2.77	3.13	6.0
2.5%	0.59	-3.95	-6.28	-6.78	-14.6
5%	0.62	-3.08	-5.30	-5.21	-11.9
25%	0.75	-0.57	-2.01	-1.64	-4.0
50%	0.84	0.77	-0.21	-0.10	-1.1
75%	0.93	2.07	1.53	1.86	1.6
95%	1.05	5.37	3.91	5.40	8.5
97.5%	1.07	6.99	5.14	6.51	10.3

Table 2 - VOR Response Parameters for Pseudorandom Stimulus

VOR time constant is in seconds and bias is in deg/sec. Gain constant is ratio of slow phase eye velocity to stimulus velocity and therefore has no units. Seven percentile values on the distrubutions of the parameters are listed. N = 206 subjects.

	Gain Constant	Time Constant	Bias
Mean	0.720	24.50	-0.08
S.D.	0.156	8.58	2.14
2.5%	0.423	13.15	-4.96
5%	0.482	14.20	-3.78
25%	0.611	18.40	-1.34
50%	0.728	23.02	-0.01
75%	0.814	28.16	1.34
95%	0.974	43.60	3.18
97.5%	1.015	47.37	3.70

Table 3 - Comparison of Single Sine and Pseudorandom Gain and Phase Results

Positive differences indicate that average single sine parameter value was larger than average pseudorandom parameter value. A * indicates that the difference was significant at $P<0.05$ using a paired variable comparison Student t test. The average differences listed are corrected for the difference in test frequencies between the single sine and pseudorandom stimuli. Gain and phase corrections were based on the VOR transfer function in equation (3) with average time constant of 24.5 sec and gain constant of 0.72. N's are smaller than those in Tables 1 and 2 since comparisons were not made if either test had poor quality data.

Parameter	Single Sine	Pseudorandom	Average Difference	N	Significance
Gain	0.05 Hz	0.046 Hz	-0.0106	199	
	0.2 Hz	0.180 Hz	0.0185	199	*
	0.8 Hz	0.766 Hz	0.0951	195	*
Phase	0.05 Hz	0.046 Hz	-0.34	199	
	0.2 Hz	0.180 Hz	-3.20	199	*
	0.8 Hz	0.766 Hz	-3.44	195	*

Table 4 - OKR Response Parameters for Pseudorandom Stimulus

OKR time constant is in seconds, time delay is in seconds, and bias is in deg/sec. Gain constant is a ratio of slow phase eye velocity to stimulus velocity and therefore has no units. Seven percentile values on the distributions of the various parameters are listed. N = 179 subjects.

	Gain Constant	Time Constant	Time Delay	Bias
Mean	0.651	0.080	0.180	-0.12
S.D.	0.116	0.080	0.043	0.84
2.5%	0.403	0.002	0.099	-1.67
5%	0.469	0.003	0.114	-1.52
25%	0.586	0.008	0.147	-0.68
50%	0.664	0.063	0.187	-0.10
75%	0.721	0.115	0.216	0.32
95%	0.807	0.226	0.248	1.13
97.5%	0.867	0.252	0.253	1.36

Table 5 - Age Effects on VOR and OKR Responses Parameters

All parameter values which showed significant or nearly significant linear trends with age are listed. Correlation coefficients which were significantly different from zero ($P<0.05$) are marked with a * in the last column of the table. OKR time constant showed apparent age related trends which were not linear with age.

Parameter	Slope (change/year)	Intercept at 0 years	Correlation Coefficient	N	Significance
Gain - 0.05 Hz	-0.00295	0.795	-0.39	208	*
- 0.2 Hz	-0.00263	0.851	-0.34	207	*
- 0.8 Hz	-0.00216	0.927	-0.33	203	*
Phase - 0.05 Hz	0.0289 deg	9.32 deg	0.12	208	
- 0.2 Hz	0.0404 deg	0.04 deg	0.26	207	*
- 0.8 Hz	0.0493 deg	-1.19 deg	0.38	203	*
VOR Gain Constant	-0.00189	0.794	-0.24	206	*
VOR Time Constant	-0.0625 sec	27.0 sec	-0.15	206	*
OKR Gain Constant	-0.00153	0.712	-0.26	179	*
OKR Time Delay	1.15 msec	134 msec	0.53	179	*

Table 6 - Normal Subject Falls on Posture Test

Single Falls

Trials	# Subjects	% of Total N = 214
SsVs	32	14.9%
SsVc	7	3.3%
SnVs	5	2.3%
SsVn	1	0.5%
	45	21.0%

2 or 3 Falls

Trials	# Subjects	% of Total N = 214
SnVs,SsVs	13	6.1%
SsVc,SsVs	10	4.7%
SnVs,SsVc	2	0.9%
SnVs,SsVc,SsVs	3	1.4%
	28	13.1%

1, 2, or 3 Falls	73	34.1%

Table 7 - Posture Test Falls Sorted by Subject Age

Age Group	#Subjects	Single Falls N	Single Falls %	Multiple Falls N	Multiple Falls %	Total Falls N	Total Falls %
7 - 12	21	1	4.8%	0	0.0%	1	4.8%
13 - 19	27	9	33.3%	1	3.7%	10	37.0%
20 - 29	28	4	14.3%	2	7.1%	6	21.4%
30 - 39	32	1	3.1%	2	6.3%	3	9.4%
40 - 49	32	8	25.0%	4	12.9%	12	37.5%
50 - 59	26	8	30.8%	4	15.4%	12	46.2%
60 - 69	35	9	25.7%	13	37.1%	22	62.9%
70 and over	13	5	38.5%	2	15.2%	7	53.9%
Total	214	45	21.0%	28	13.2%	73	34.1%

FIGURE LEGENDS

Figure 1. Example of data from sinusoidal stimulus VOR rotation test. Upper trace shows slow phase eye velocity response to a 0.05 Hz, 60 deg/sec sinusoidal rotational stimulus. Solid curve through the data is the curve fit to each cycle. Response gain, phase, and bias are obtained from these curve fits. Middle trace shows eye movements evoked by one period of the rotational stimulus shown in the lower trace. Solid vertical bars under the middle trace show the location of fast phase portions of the nystagmus identified during the analysis.

Figure 2. VOR responses to sinusiodal rotation at three different test frequencies. Data were from a single subject. Stimulus amplitude was 60 deg/sec in each case. Slow phase eye velocity is plotted versus time in the three traces on the left. Solid lines through the data points are curve fits to the data. The three traces on the right show slow phase eye velocity plotted against stimulus velocity after correction for the phase of the response. Straight line curve fits through the data are used in the calculation of right and left VOR gain, asymmetry, and offset.

Figure 3. Normal population distributions of VOR gain at 0.05, 0.2, and 0.8 Hz.

Figure 4. Normal population distributions of VOR phase at 0.05, 0.2, and 0.8 Hz.

Figure 5. Normal population distributions of VOR bias at 0.05, 0.2, and 0.8 Hz.

Figure 6. Normal population distributions of VOR offset at 0.05, 0.2, and 0.8 Hz.

Figure 7. Normal population distributions of VOR asymmetry at 0.05, 0.2, and 0.8 Hz.

Figure 8. Example of data from pseudorandom stimulus VOR rotation test. Top trace shows 30 sec portion of the pseudorandom stimulus velocity. Second trace shows slow phase eye velocity. Third trace shows horizontal eye movements. Fourth trace shows vertical eye movements. Solid vertical bars between second and third traces show locations of fast phase portions of nystagmus identified during the analysis.

Figure 9. Example of VOR gain and phase data from a single subject derived from their response to a pseudorandom rotation test. Solid line through the data show the transfer function curve fit. VOR response parameter values estimated by the curve fit were VOR gain constant = 0.91 and VOR time constant = 17.4 seconds.

Figure 10. Normal population distributions of VOR gain constant derived from responses to pseudorandom stimulation.

Figure 11. Normal population distributions of VOR time constant derived form responses to pseudorandom stimulation.

Figure 12. Box graphs of normal population VOR gain and phase values versus stimulus frequency derived from responses to pseudorandom stimulation. Upper set of eight box graphs show VOR gain, lower set of eight show VOR phase at the eight component frequencies of the pseudorandom stimulus. Within each box graph, the central line marks the median, or 50^{th} percentile point of the distribution. The upper and lower edges of the boxes surrounding the median value mark the 75^{th} and 25^{th} percentile points on the distribution. The upper and lower error bars above and below the boxes mark the 97.5 and 2.5 percentile points. Individual data points whose values were outside the 2.5 to 97.5 percentile marks are plotted separately. Open circles are for gain values and open diamonds are for phase values.

Figure 13. Example of data from pseudorandom stimulus OKR rotation test. Top trace shows stimulus velocity. Second trace shows slow phase eye velocity. Third trace shows horizontal eye movements. Fourth trace shows vertical eye movements. Dark vertical bars between the second and third traces show locations of fast phases of nystagmus detected by the analysis algorithm. Data is a 30 second sample of the 163.84 second stimulus period.

Figure 14. Examples of OKR gain and phase data from two individuals. Gain and phase values were derived from responses to pseudorandom optokinetic stimulation. Solid lines show transfer function curve fits to data. Equations of the curve fits are inset.

Figure 15. Normal population distribution of OKR gain constant derived from transfer function curve fit.

Figure 16. Normal population distribution of OKR time constant derived from transfer function curve fit.

Figure 17. Normal population distribution of OKR time delay derived from transfer function curve fit.

Figure 18. OKR gain and phase data obtained from one subject at four different pseudorandom stimulus amplitudes. Stimulus amplitude values inset by gain data indicate the amplitudes of each of the seven sinusoidal components of the pseudorandom stimulus. Phase data points obtained at a given stimulus amplitude are shown by open symbols with shapes identical to the corresponding gain data points. Data points are connected by straight lines for clarity.

Figure 19. OKR gain and phase data obtained from one subject at two different pseudorandom stimulus amplitude bandwidths. Open symbols correspond to the lower bandwidth and filled symbols to the higher bandwidth. Data points are connected by straight lines for clarity.

Figure 20. VVOR gain and phase data obtained from ten subjects. Error bars are at plus and minus two standard deviations from the mean.

Figure 21. Normal population distributions of caloric test unilateral weakness and average slow phase eye velocity.

Figure 22. Age effects on VOR gain obtained from sinusoidal rotational stimulation at three different test frequencies. Curve fits are from a linear regression analysis.

Figure 23. Age effects on VOR phase obtained from sinusoidal rotational stimulation at three different test frequencies. Curve fits are from a linear regression analysis.

Figure 24. Age effects on VOR gain constant and time constant parameters. Parameter values were estimated from transfer function curve fits to gain and phase data obtained from pseudorandom rotation test results. Curve fits are from a linear regression analysis.

Figure 25. Age effect on OKR gain constant, time constant, and time delay parameters. Parameter values were estimated from transfer function curve fits to OKR gain and phase data obtained from pseudorandom rotation test results. Curve fits in A and C are from a linear regression analysis. Curve fit in B is from a robust locally weighted regression analysis (Cleveland, 1985).

Figure 26. Age effect on caloric test unilateral weakness and average slow phase eye velocity. Curve fits are from a robust locally weighted regression analysis of the data (Cleveland, 1985).

Figure 27. VOR responses at 0.2 Hz from a subject following a left labyrinthectomy. Response phase is advanced relative to the normal range. Slow phase eye velocity has a large negative, or left, bias. Slow phase eye velocity versus stimulus velocity plot in lower left shows that the large bias results from both an offset of 4.66 deg/sec to the left and a response asymmetry where the gain of slow phase eye movements to the left, 0.72, is larger than the gain of slow phase eye movements to the right, 0.39.

Figure 28. VOR gain and phase data obtained from a subject with an acoustic neuroma on their left side. Dotted lines through data points show transfer function curve fit to data. VOR gain constant is 0.75, time constant is 6.33 seconds, and bias is 4.0 deg/sec to the left.

Figure 29. VVOR gain and phase data obtained from a subject with nearly complete bilateral loss of vestibular function. Gains are lower than normal and phases show increasing lags with increasing frequency. Data points are connected by straight lines for clarity.

Figure 30. Typical posture test results showing anterior-posterior sway measures versus time obtained from a normal subject. The subject was exposed to different sensory conditions during the six trials shown. See the methods section for the definition of the sensory conditions.

Figure 31. Normal population distributions of subject anterior-posterior sway under the six different posture test sensory conditions. Subject sway was quantified by calculating peak forward minus peak backward sway angle during the 20 second trials. Gray bar to the right of each graph indicates the number of subjects who fell on a given trial.

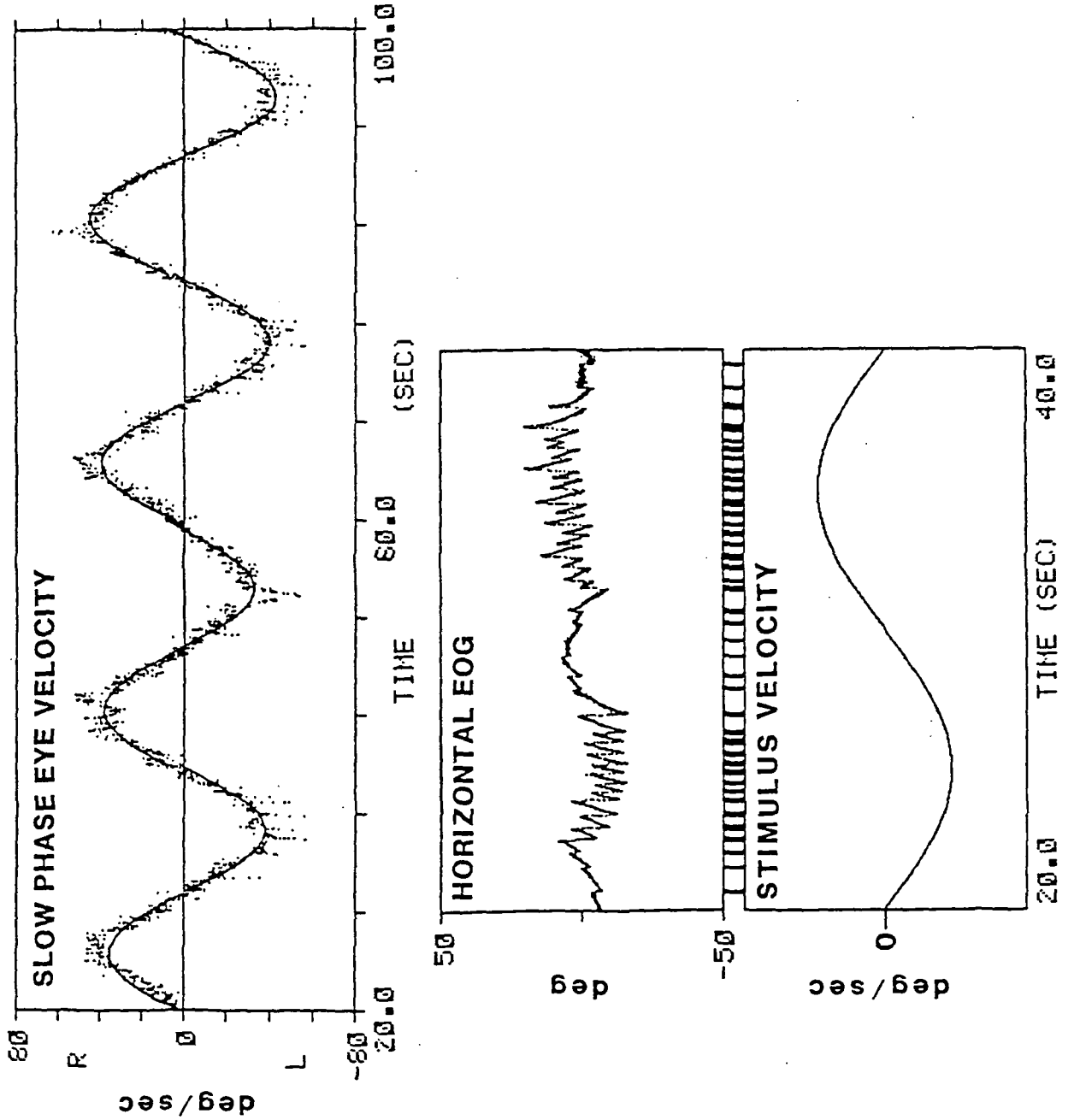
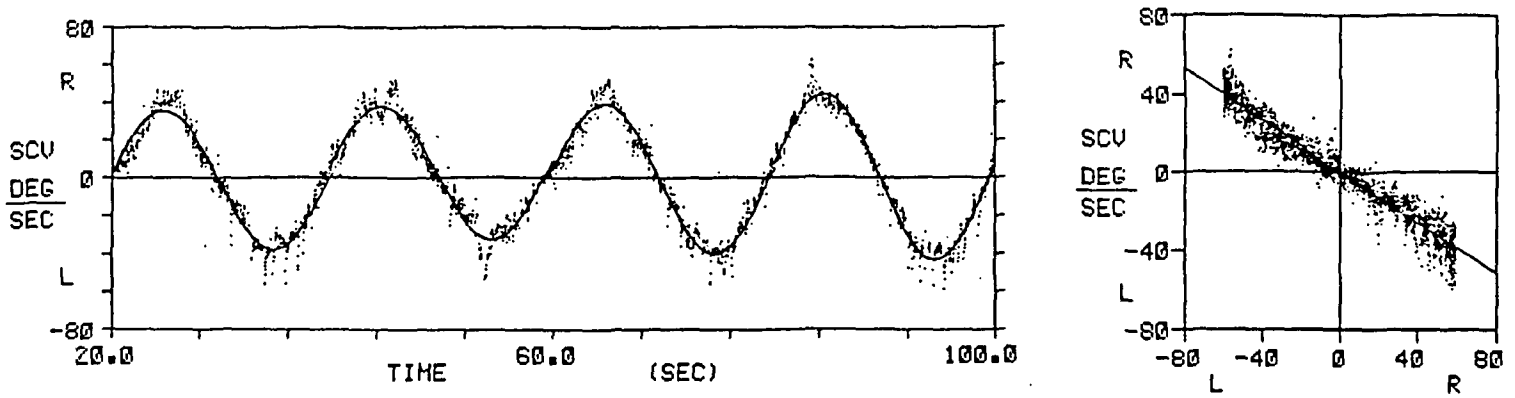
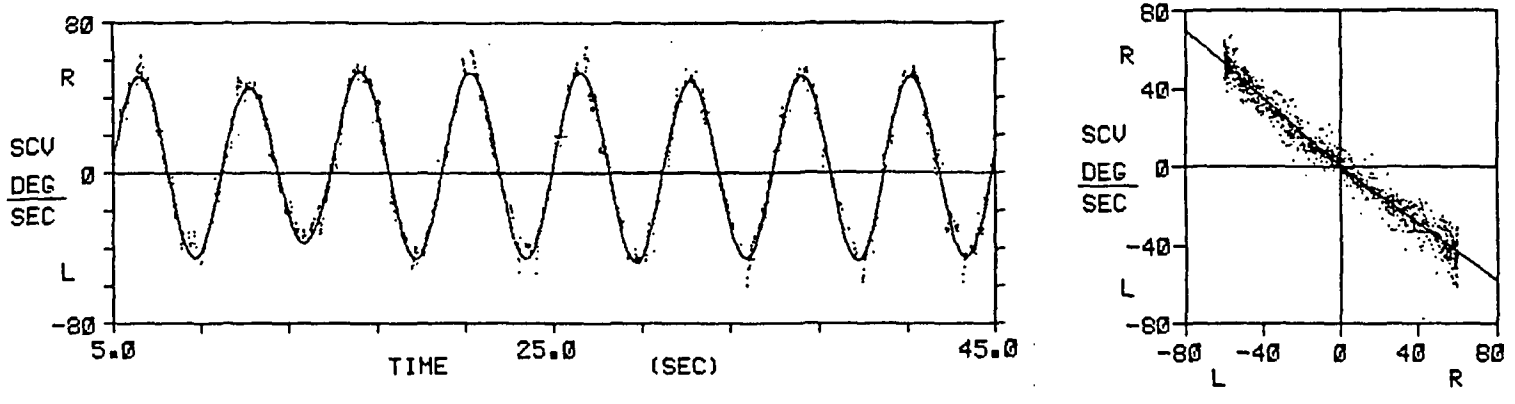

SLOW PHASE EYE VELOCITY
80
R
0
L
-80
deg/sec
-20.0
60.0
100.0
TIME (SEC)
HORIZONTAL EOG
50
deg
-50
STIMULUS VELOCITY
0
deg/sec
20.0
40.0
TIME (SEC)

Figure 1

0.05 Hz



0.2 Hz



0.8 Hz

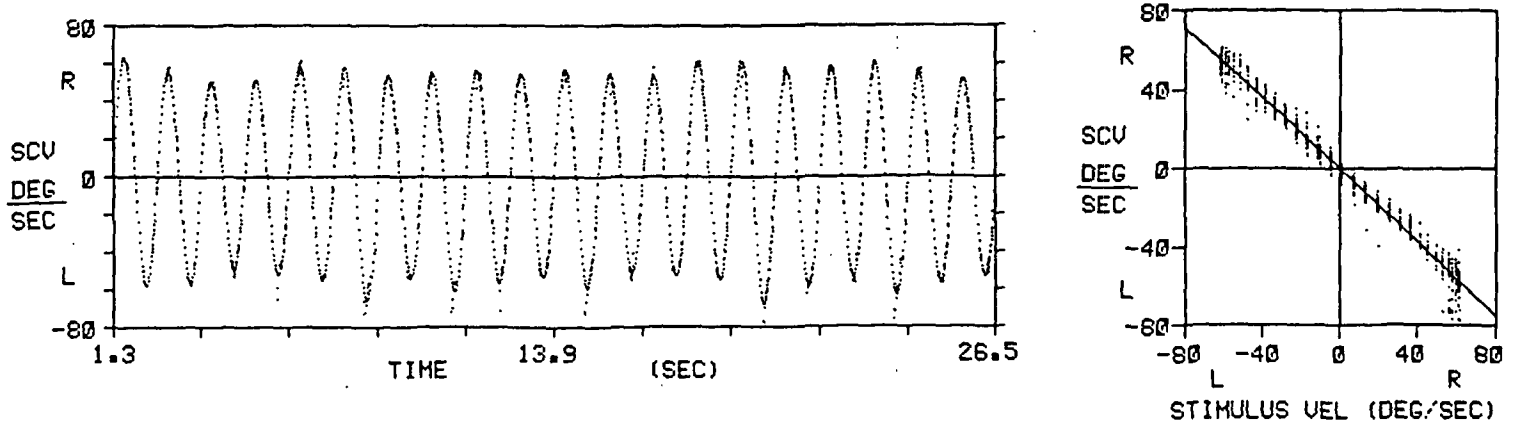
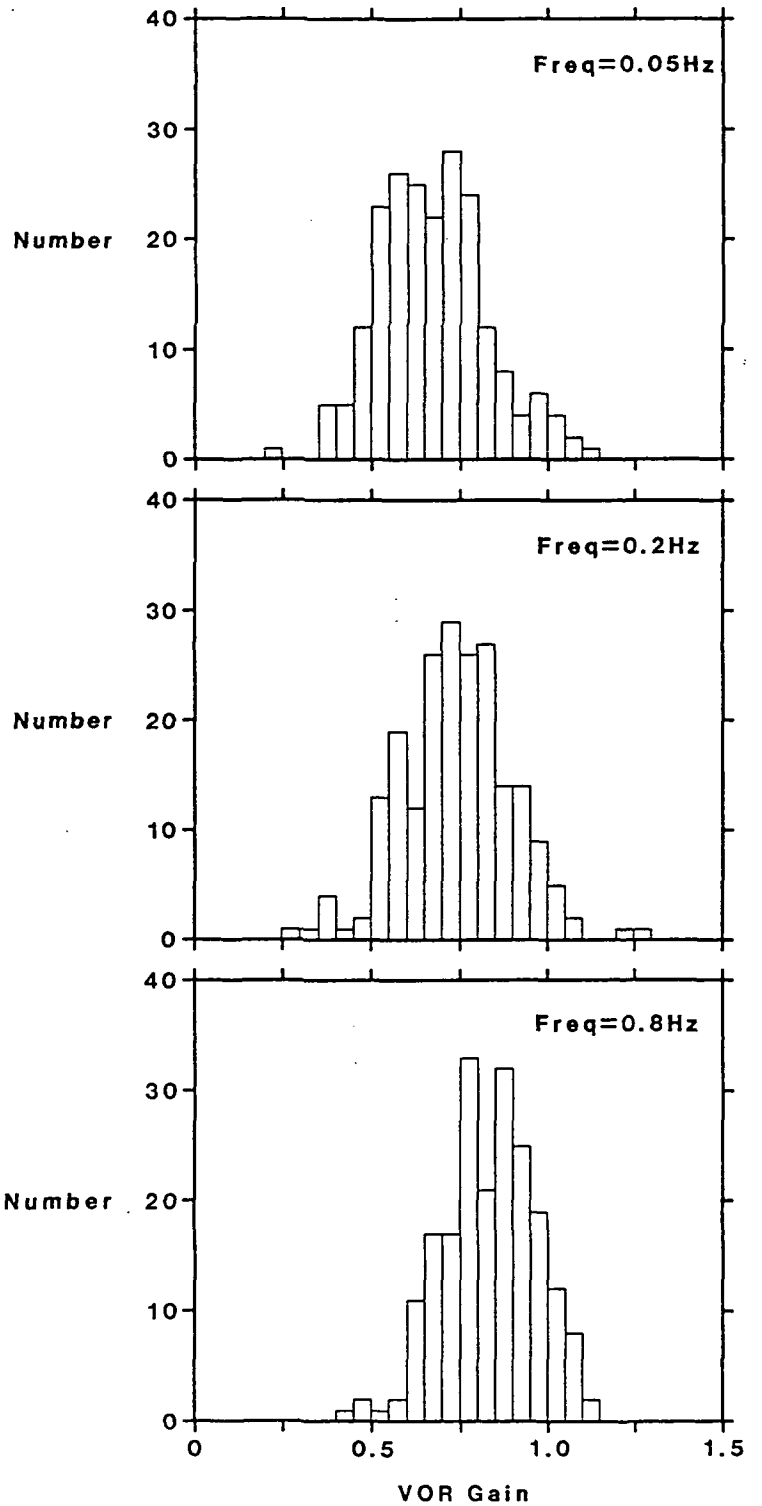


Figure 2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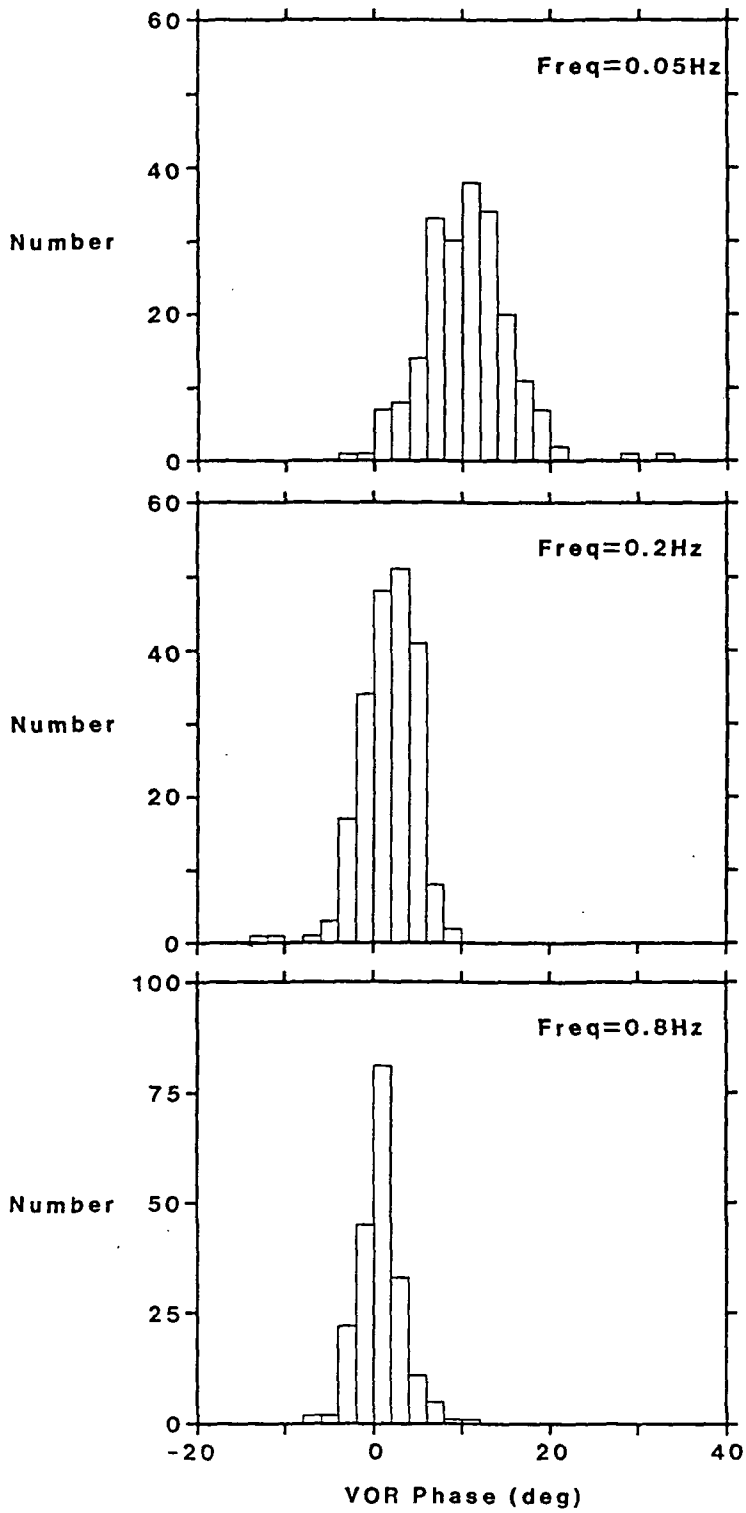
Freq=0.2Hz
Freq=0.8Hz
Number
40
30
20
10
0
0
0.5
1.0
1.5
VOR Gain

Figure 3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Freq=0.2Hz
Freq=0.8Hz
Number
Number
Number
60
40
20
0
60
40
20
0
100
75
50
25
0
-20
0
20
40
VOR Phase (deg)

Figure 4

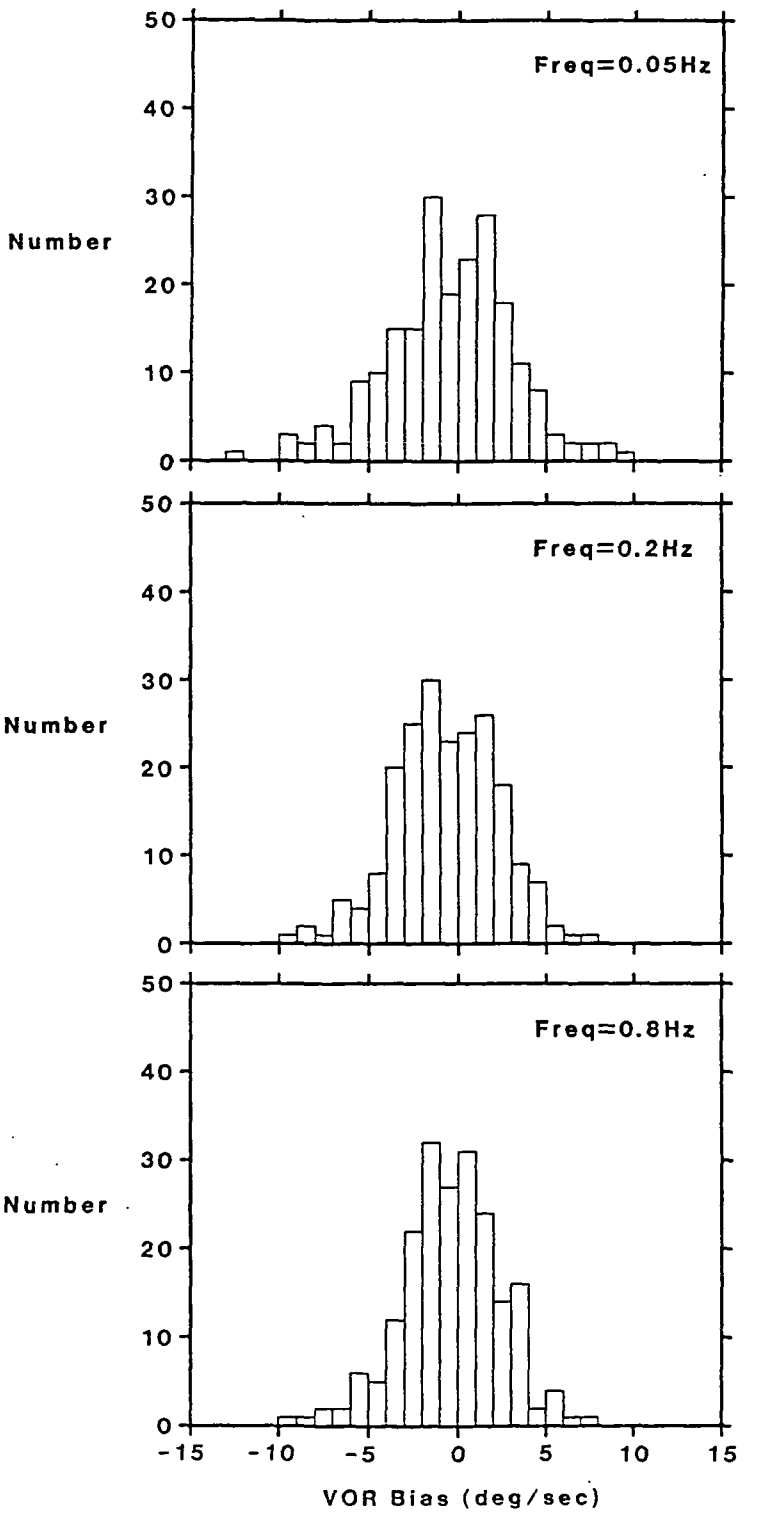
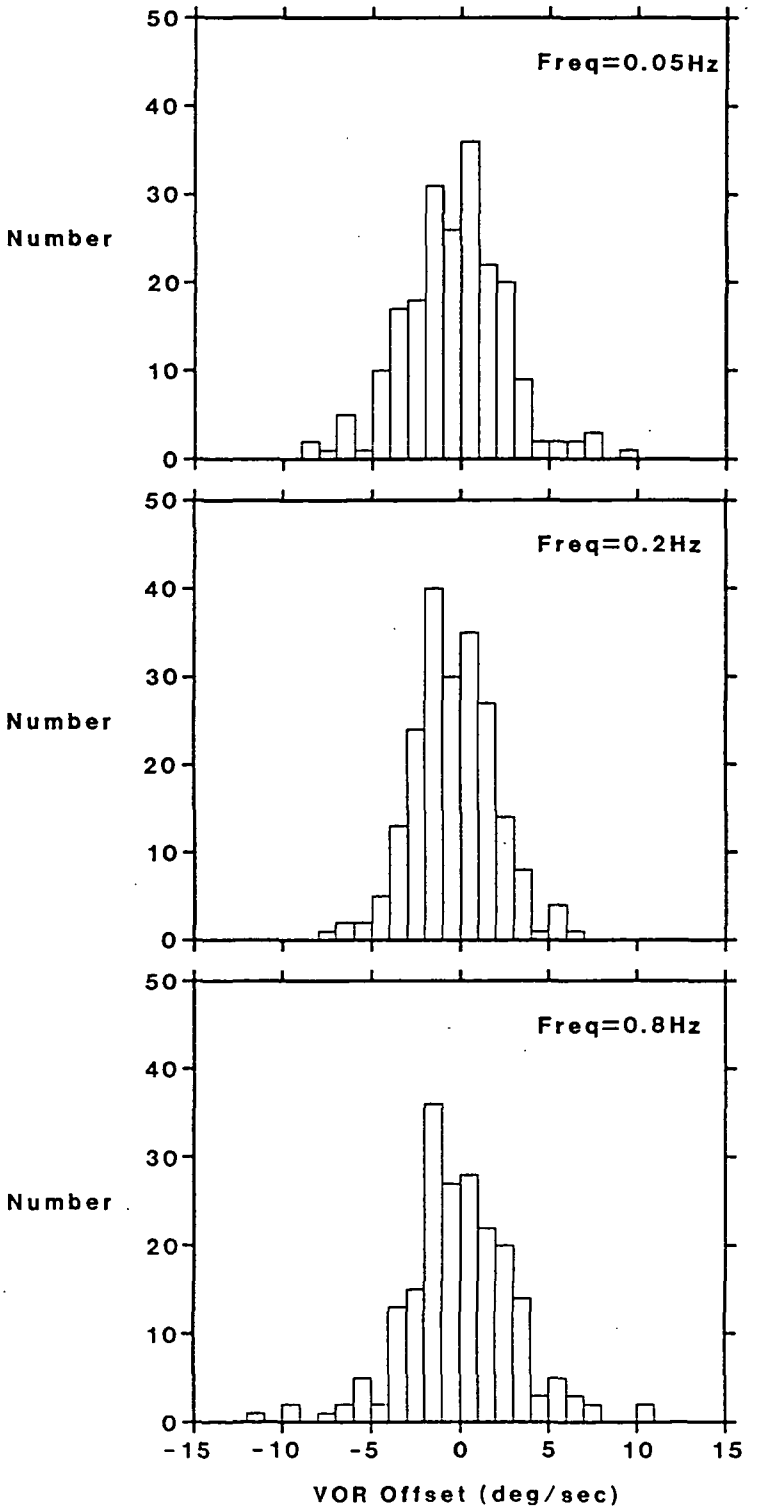

Freq=0.05Hz
Freq=0.2Hz
Freq=0.8Hz
Number
Number
Number
50
40
30
20
10
0
-15
-10
-5
0
5
10
15
VOR Bias (deg/sec)

Figure 5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Freq=0.2Hz
Freq=0.8Hz
Number
Number
Number
50
40
30
20
10
0
-15
-10
-5
0
5
10
15
VOR Offset (deg/sec)

Figure 6

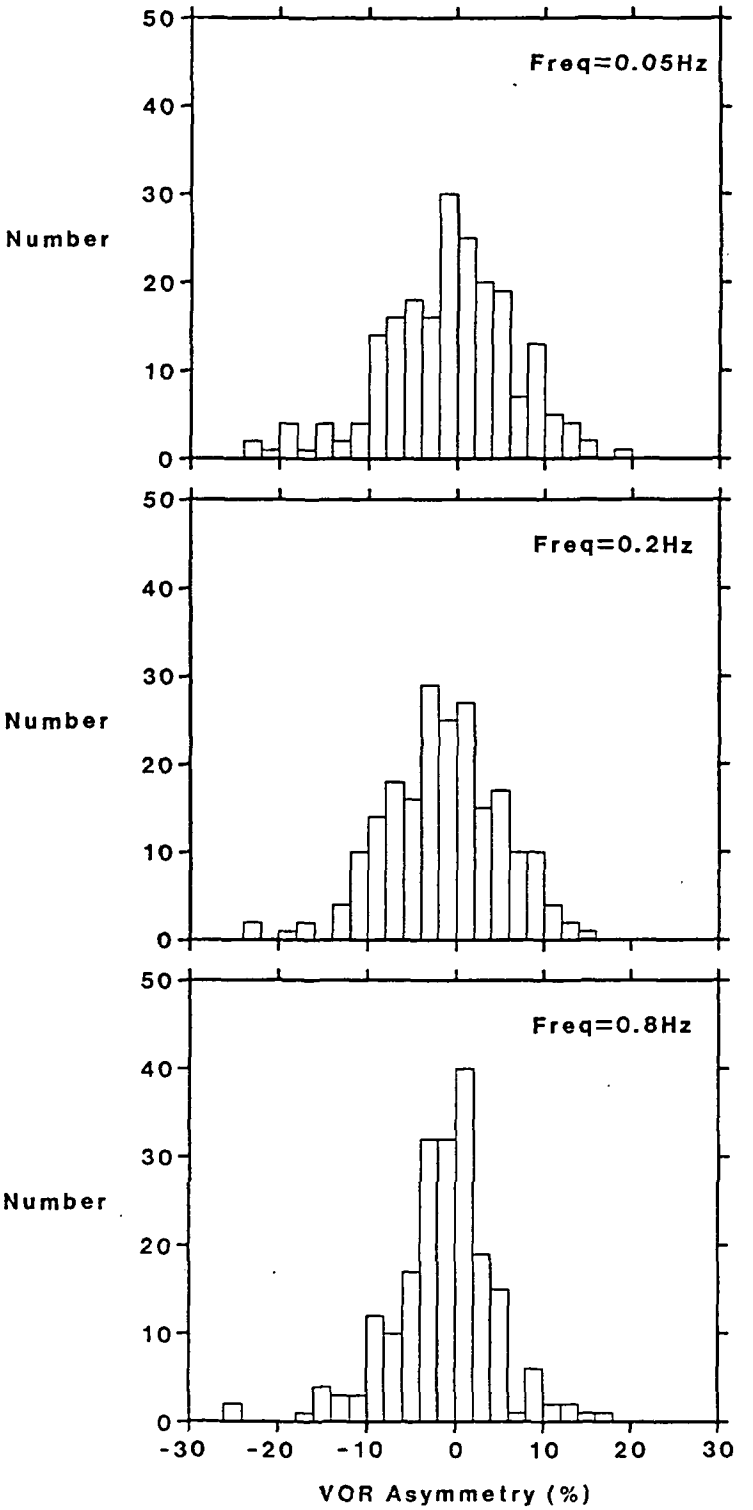

Freq=0.05Hz
Freq=0.2Hz
Freq=0.8Hz
Number
Number
Number
50
40
30
20
10
0
-30
-20
-10
0
10
20
30
VOR Asymmetry (%)

Figure 7

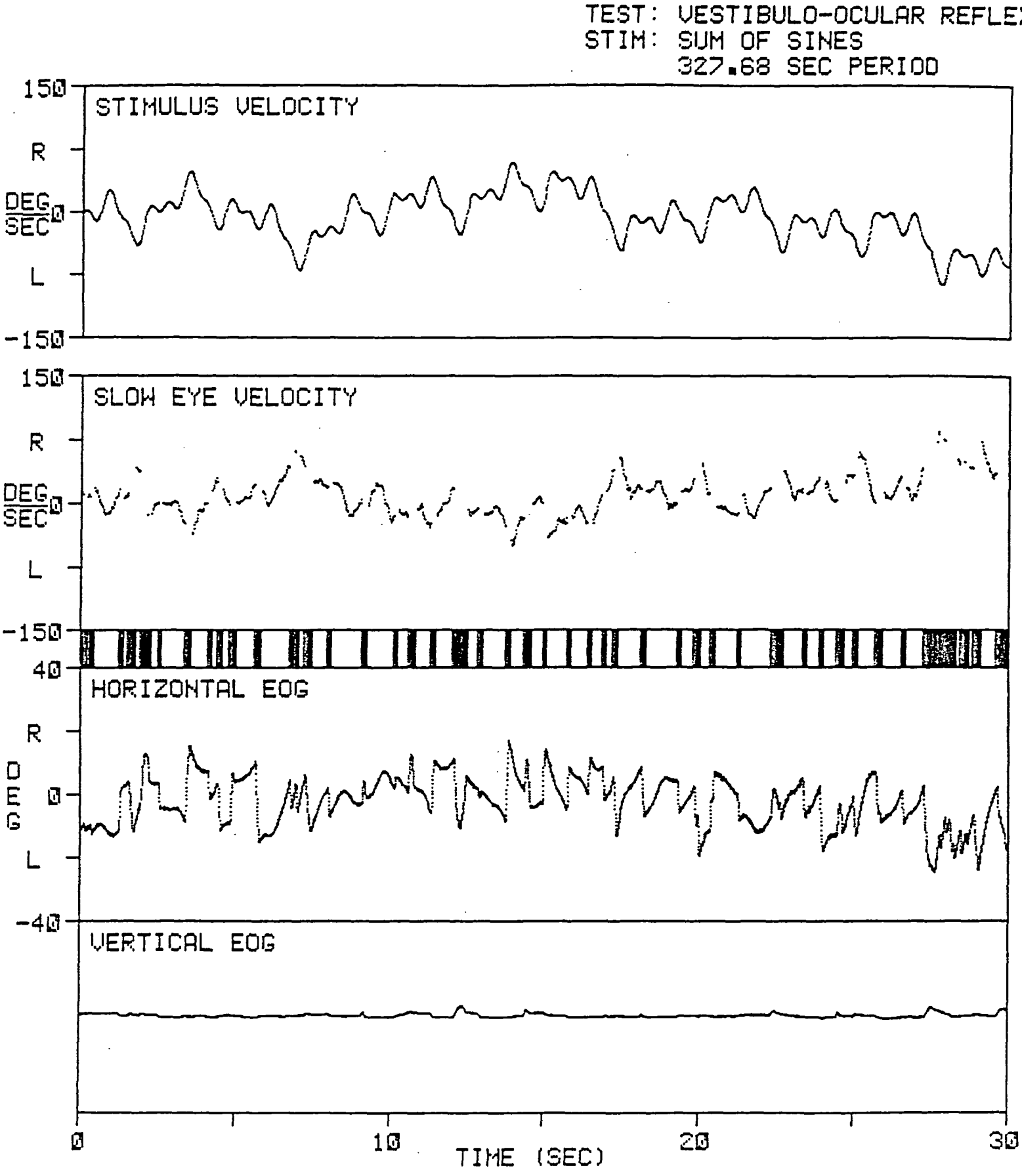

TEST: VESTIBULO-OCULAR REFLEX
STIM: SUM OF SINES
327.68 SEC PERIOD
150
STIMULUS VELOCITY
R
DEG/SEC
0
L
-150
150
SLOW EYE VELOCITY
R
DEG/SEC
0
L
-150
40
HORIZONTAL EOG
R
DEG
0
L
-40
VERTICAL EOG
0
10
20
30
TIME (SEC)

Figure 8

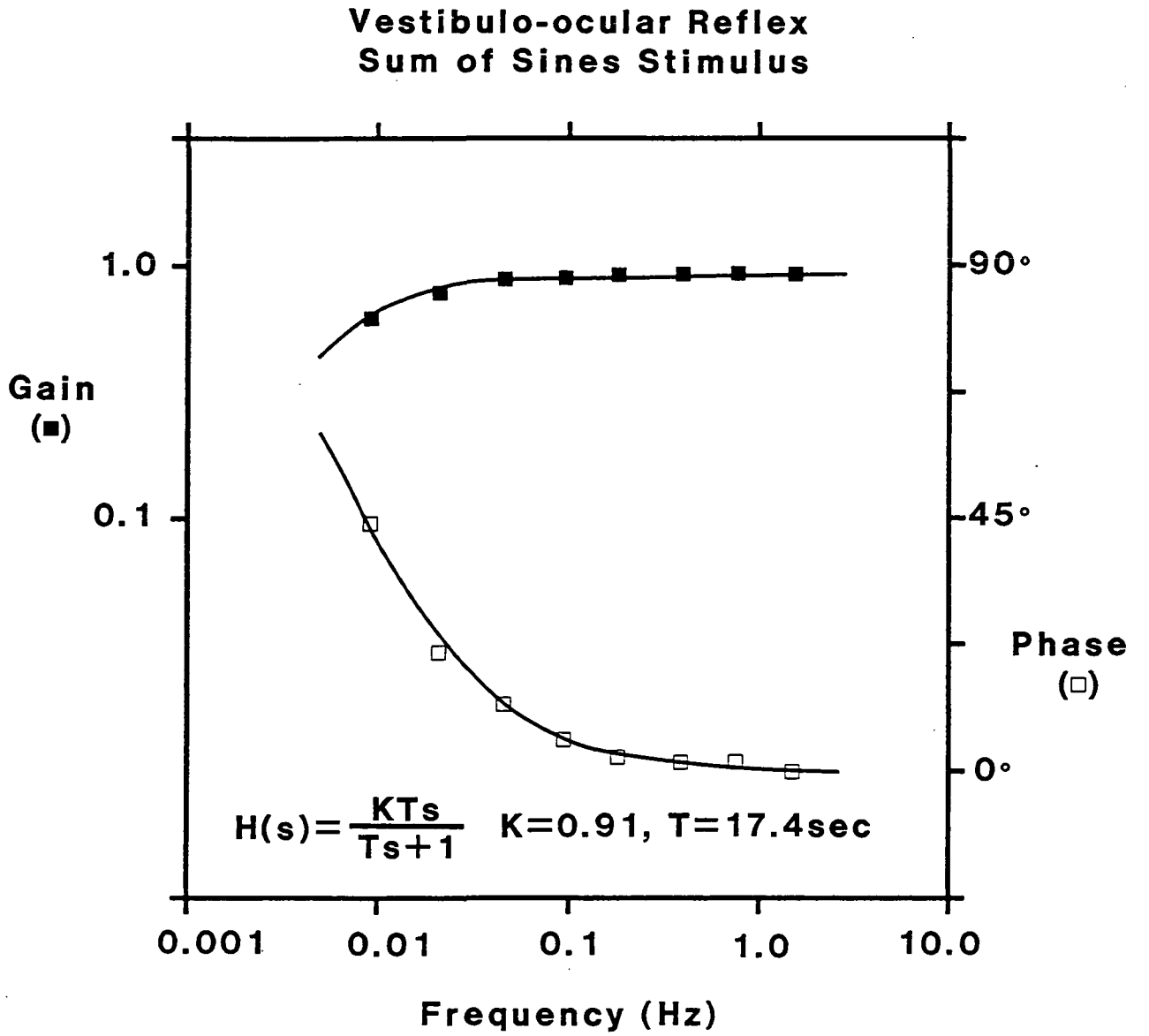

Vestibulo-ocular Reflex
Sum of Sines Stimulus
1.0
0.1
Gain
(■)
90°
45°
0°
Phase
(□)
H(s)=KTs/(Ts+1) K=0.91, T=17.4sec
0.001
0.01
0.1
1.0
10.0
Frequency (Hz)

Figure 9

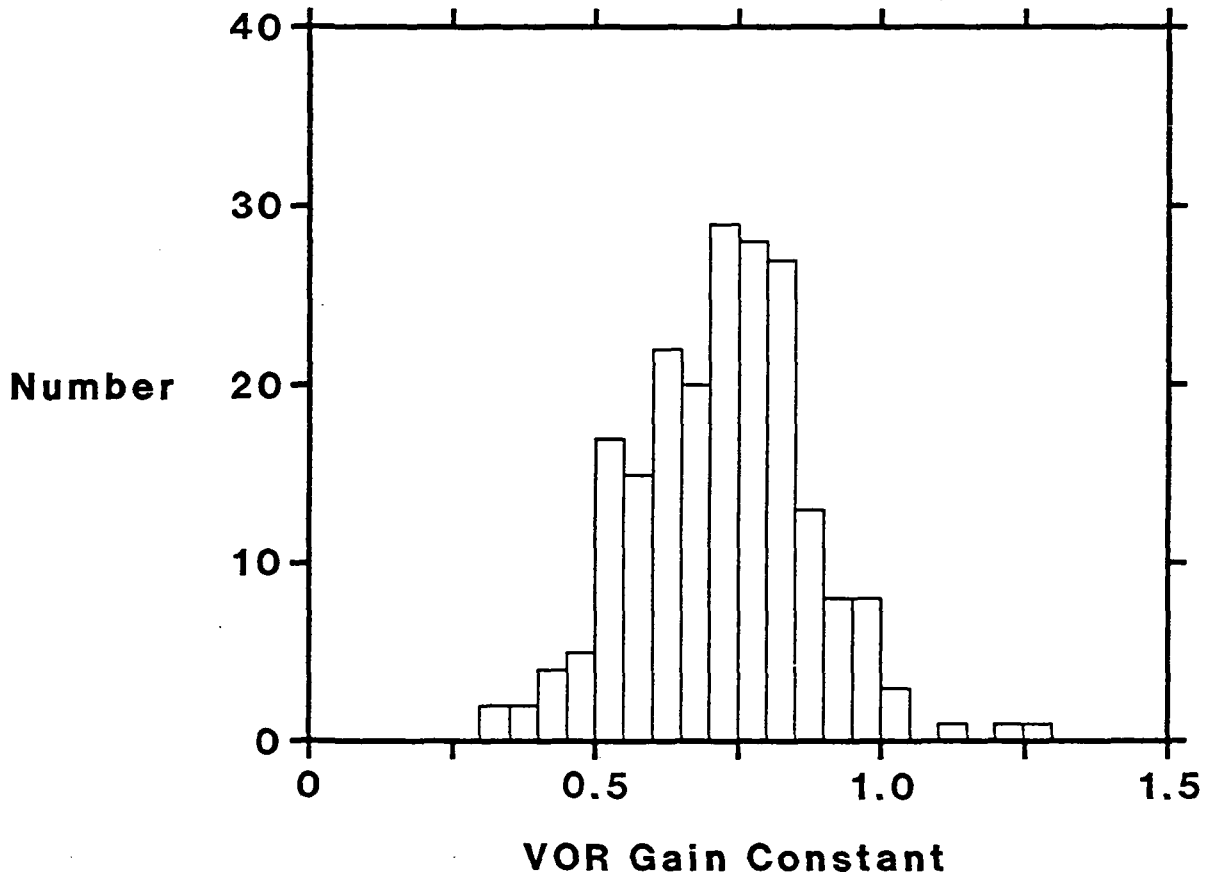


Figure 10

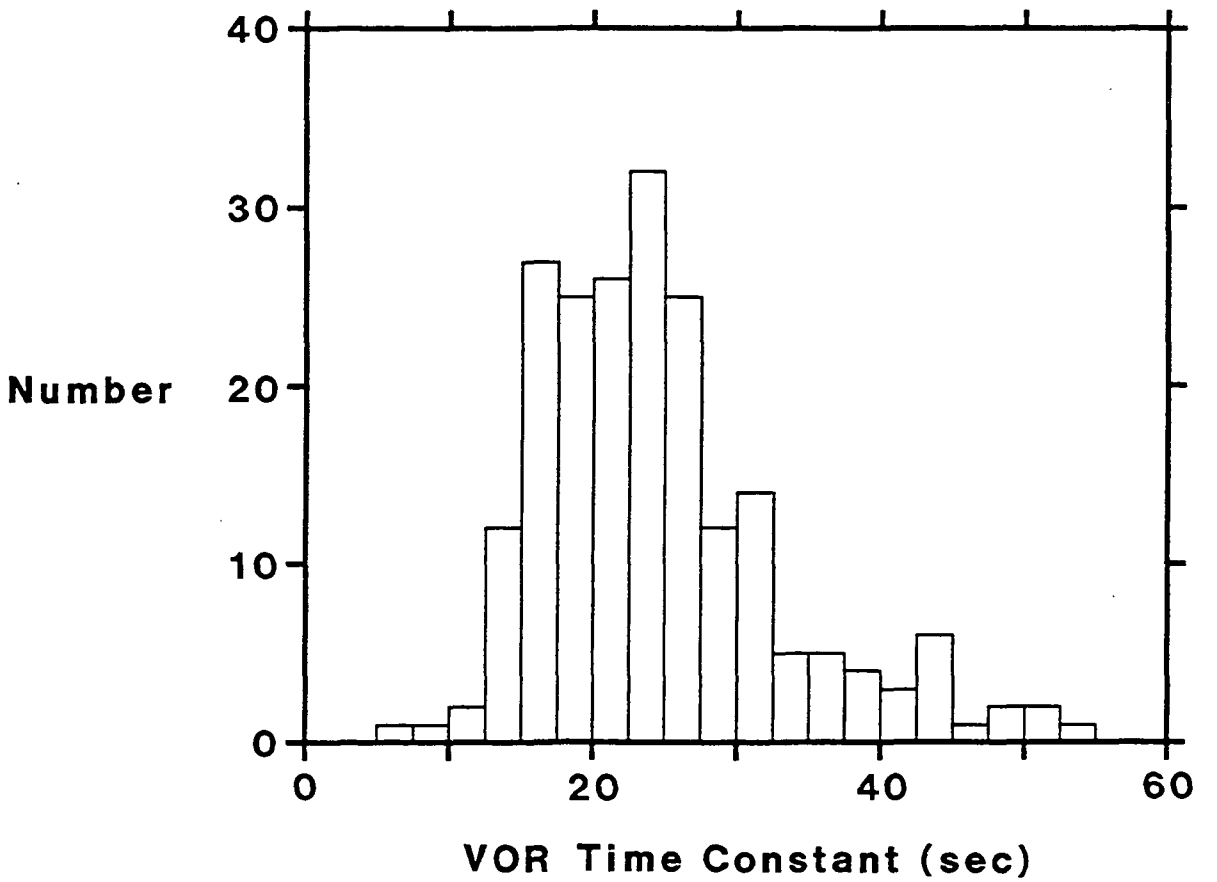


Figure 11

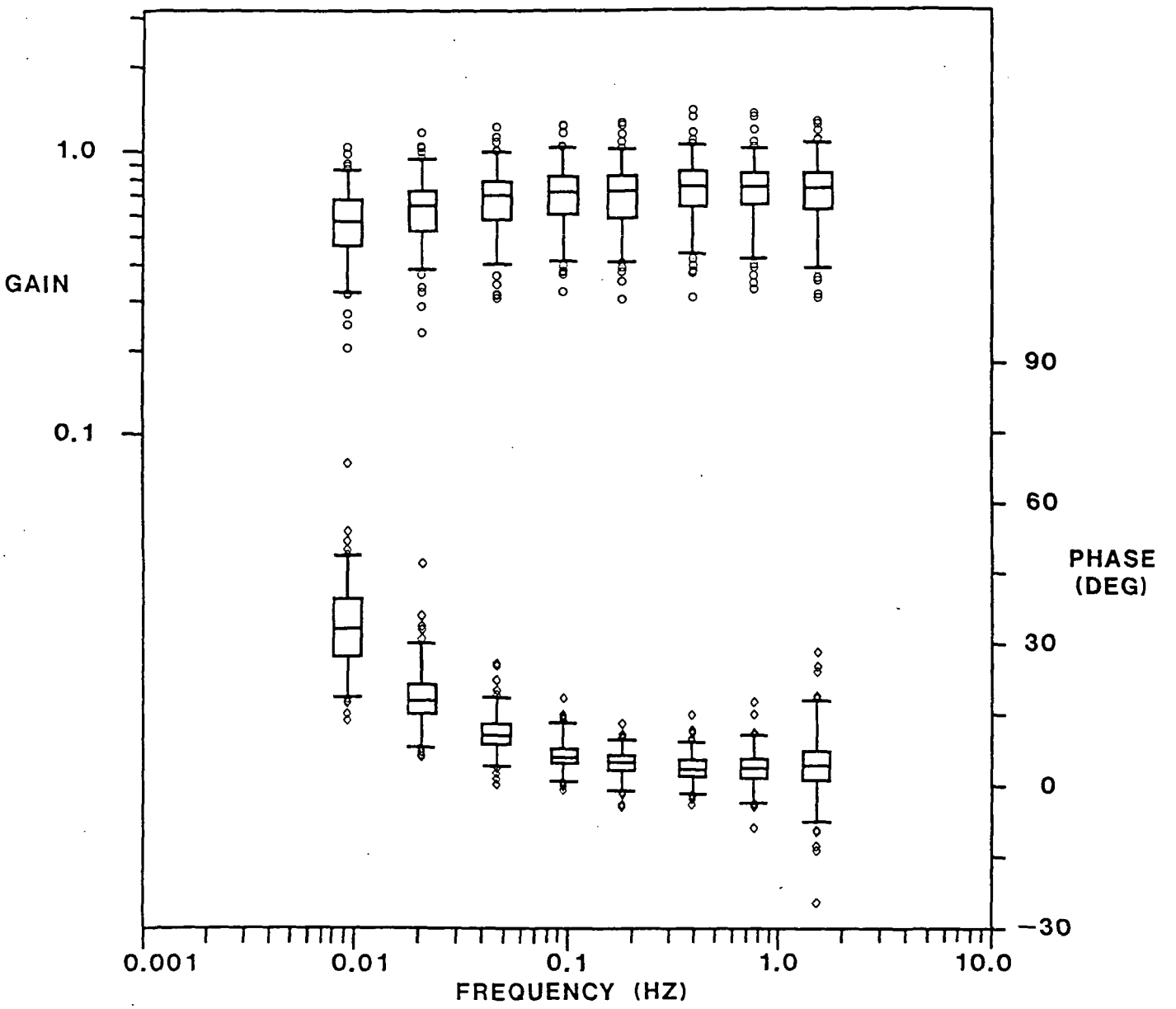

1.0
GAIN
0.1
90
60
PHASE
(DEG)
30
0
−30
0.001
0.01
0.1
1.0
10.0
FREQUENCY (HZ)

Figure 12

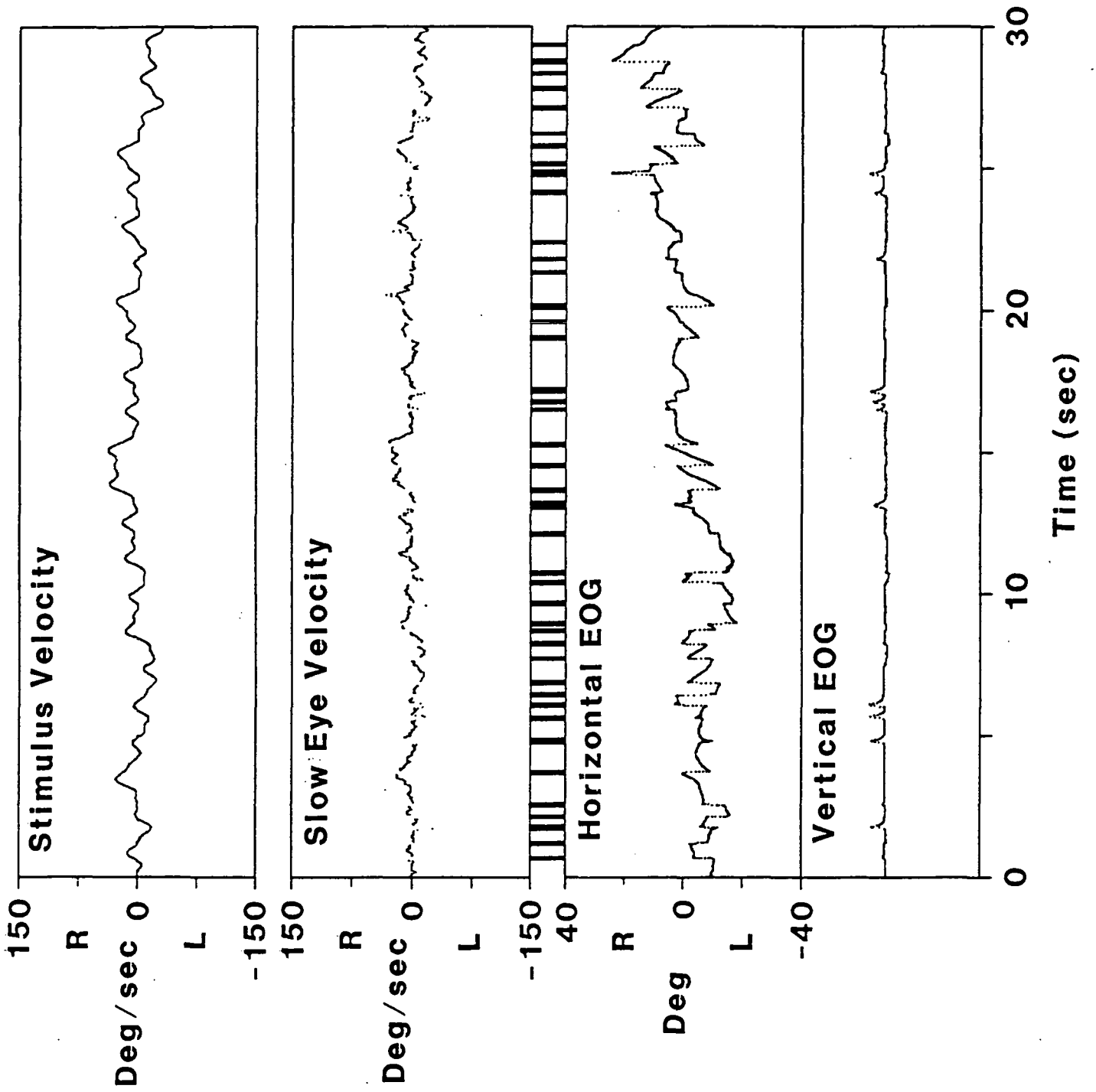

Stimulus Velocity
150
R
Deg/sec 0
L
-150
Slow Eye Velocity
150
R
Deg/sec 0
L
-150
Horizontal EOG
40
R
Deg 0
L
-40
Vertical EOG
0
10
20
30
Time (sec)

Figure 13

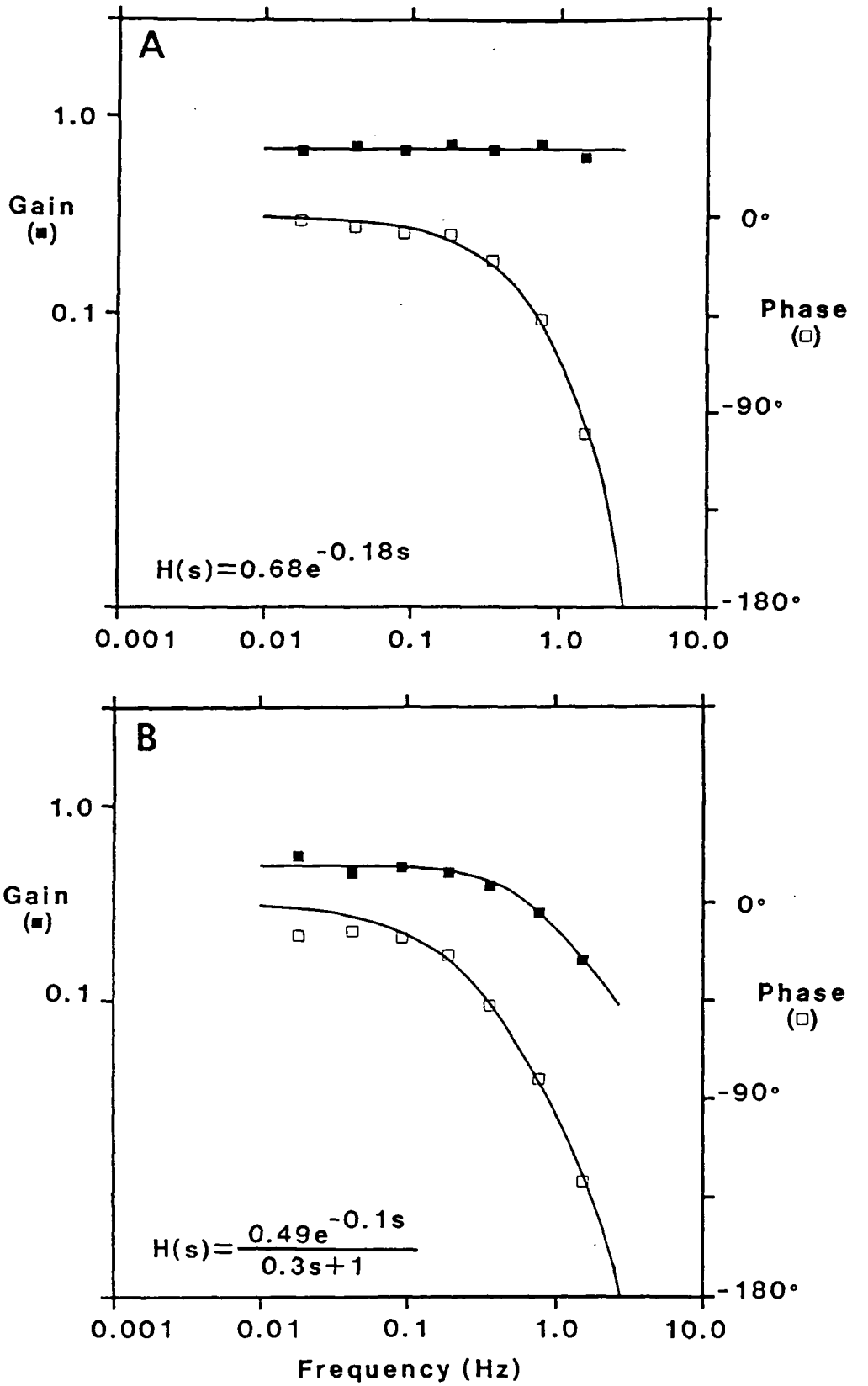

A
1.0
Gain
(■)
0.1
0°
Phase
(□)
-90°
-180°
H(s)=0.68e^-0.18s
0.001
0.01
0.1
1.0
10.0
B
1.0
Gain
(■)
0.1
0°
Phase
(□)
-90°
-180°
H(s)=0.49e^-0.1s / 0.3s+1
0.001
0.01
0.1
1.0
10.0
Frequency (Hz)

Figure 14

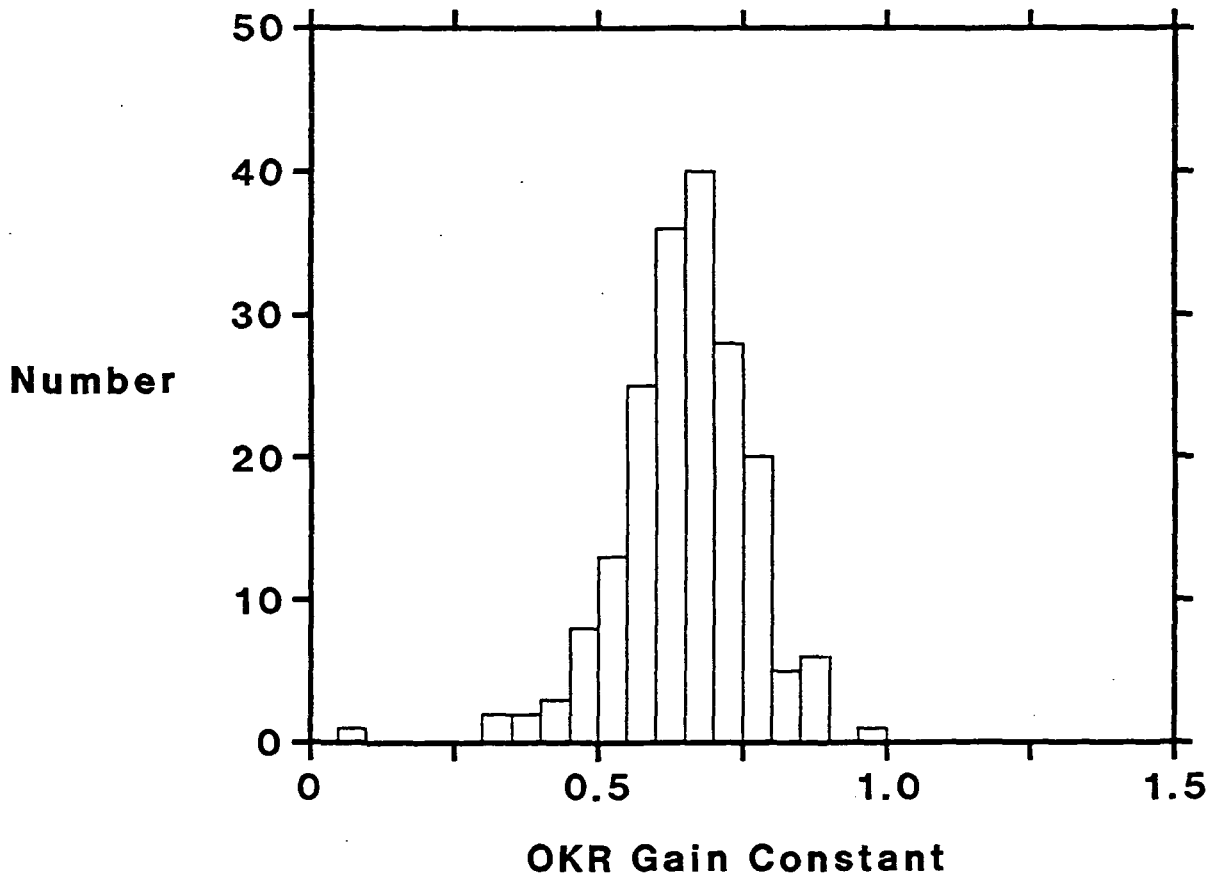


Figure 15

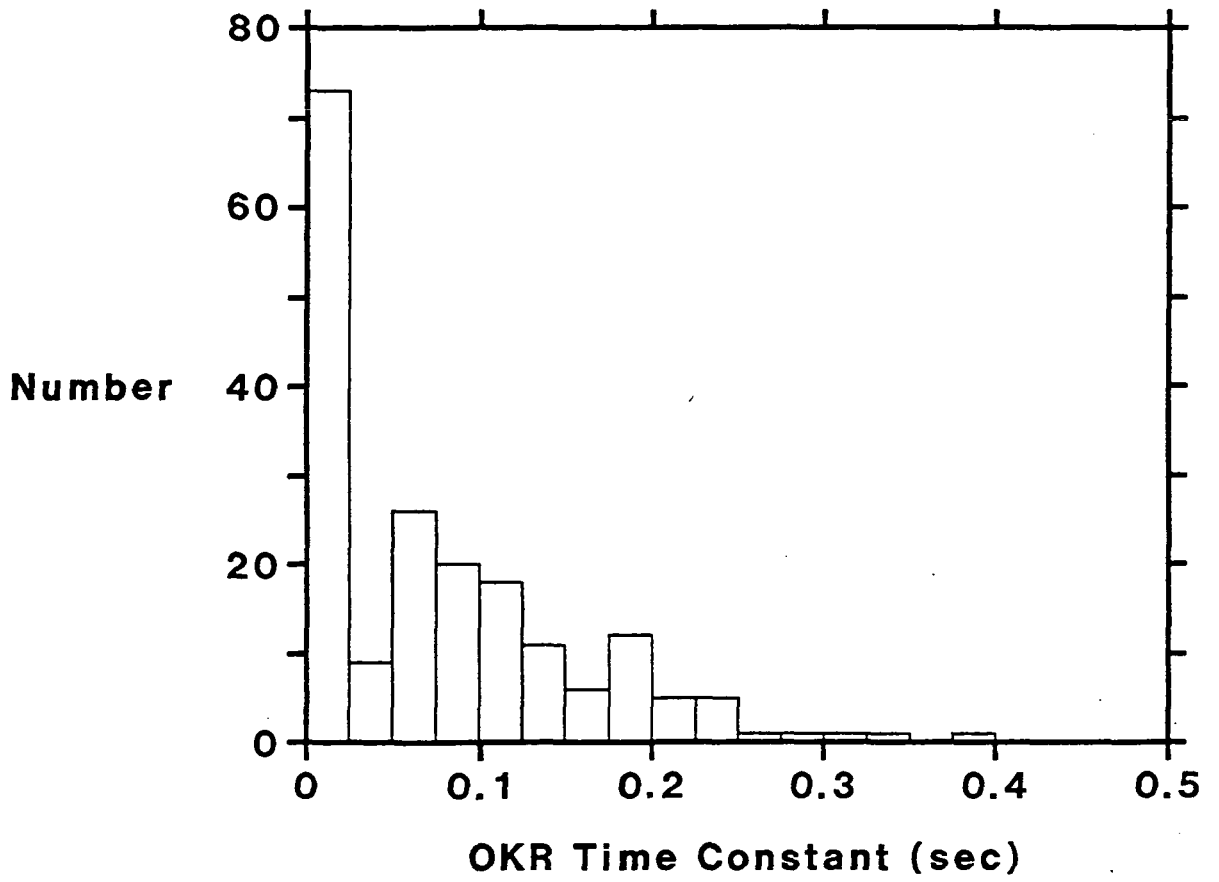


Figure 16

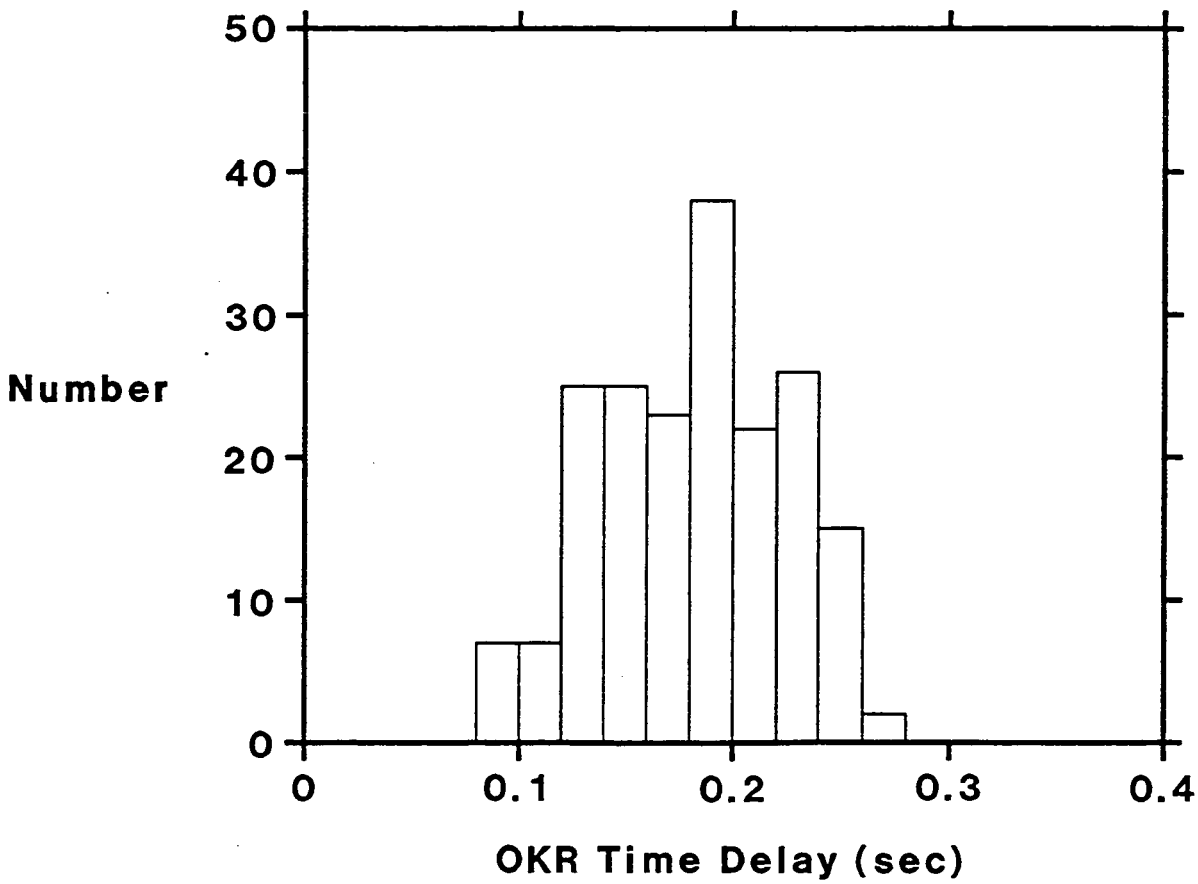


Figure 17

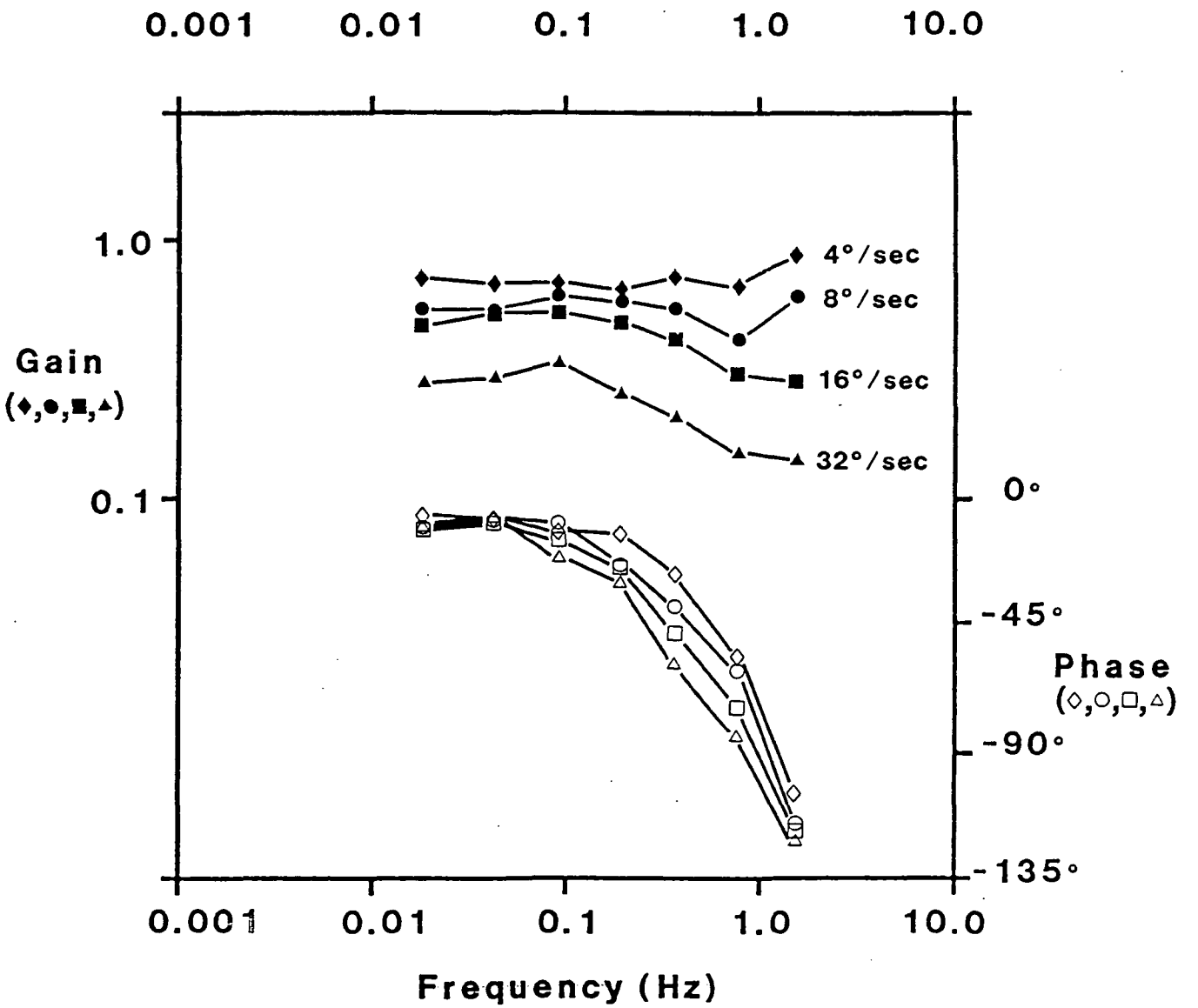

0.001
0.01
0.1
1.0
10.0
1.0
0.1
Gain
(♦,●,■,▲)
4°/sec
8°/sec
16°/sec
32°/sec
0°
-45°
-90°
-135°
Phase
(◇,○,□,△)
0.001
0.01
0.1
1.0
10.0
Frequency (Hz)

Figure 18

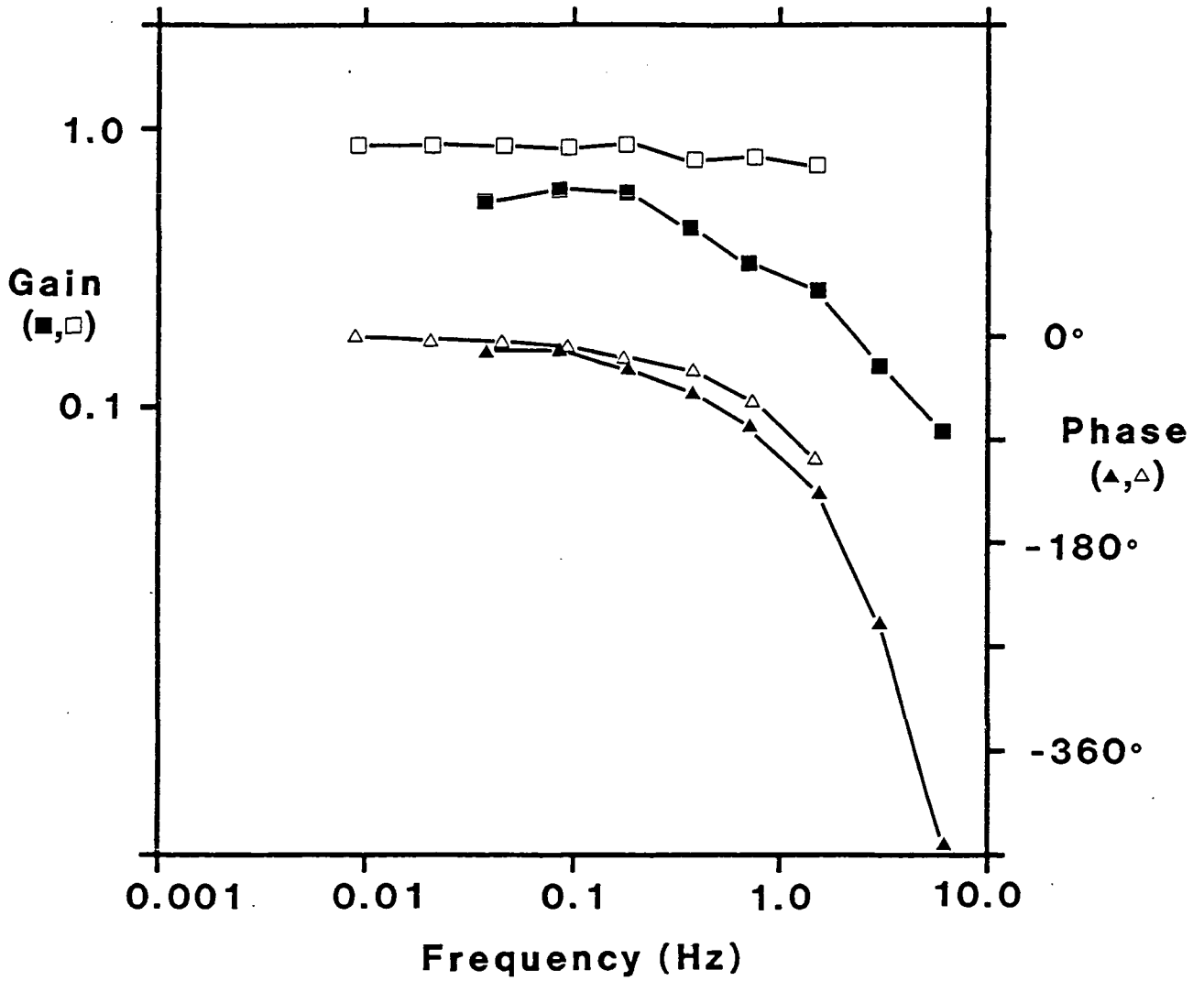


Figure 19

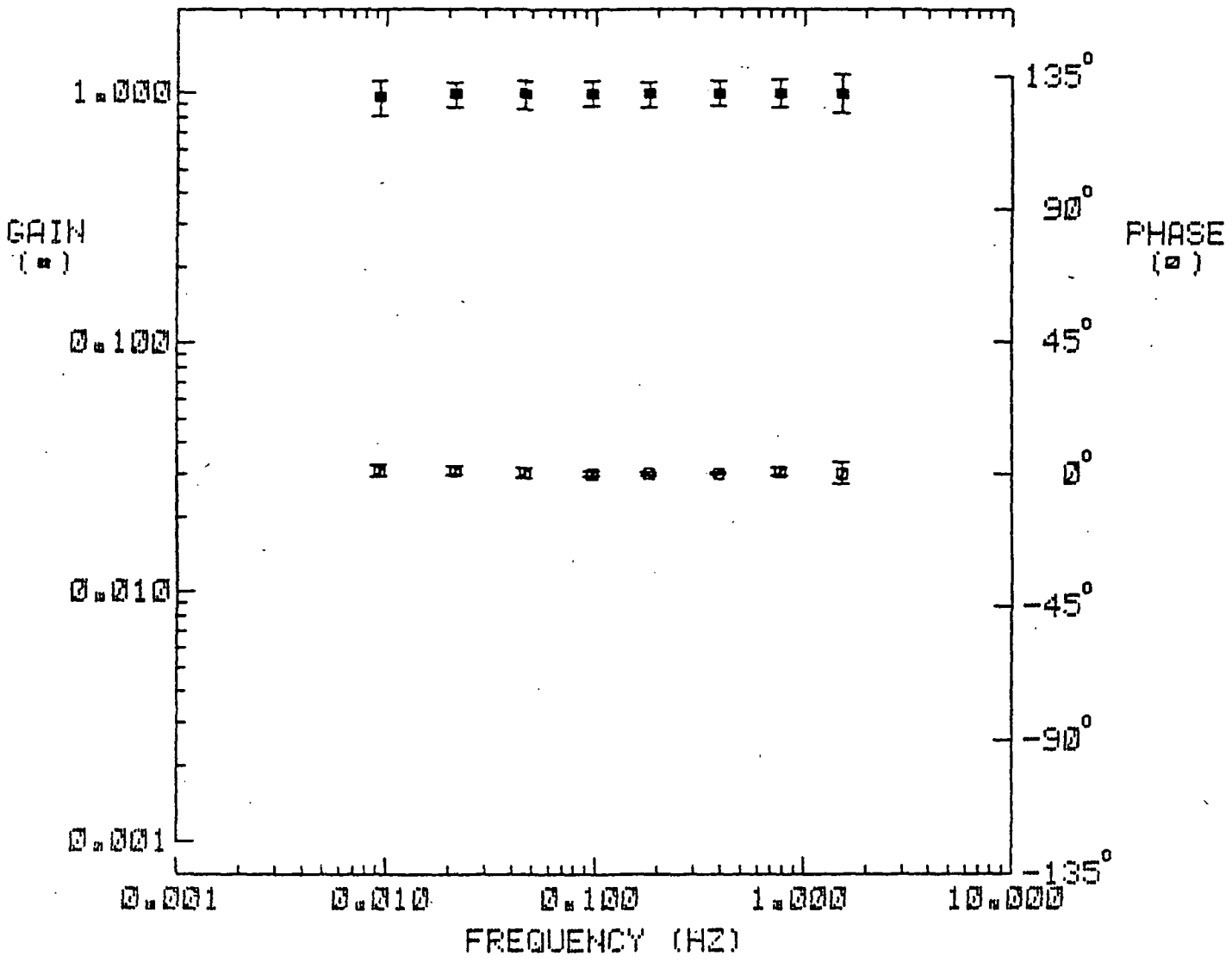
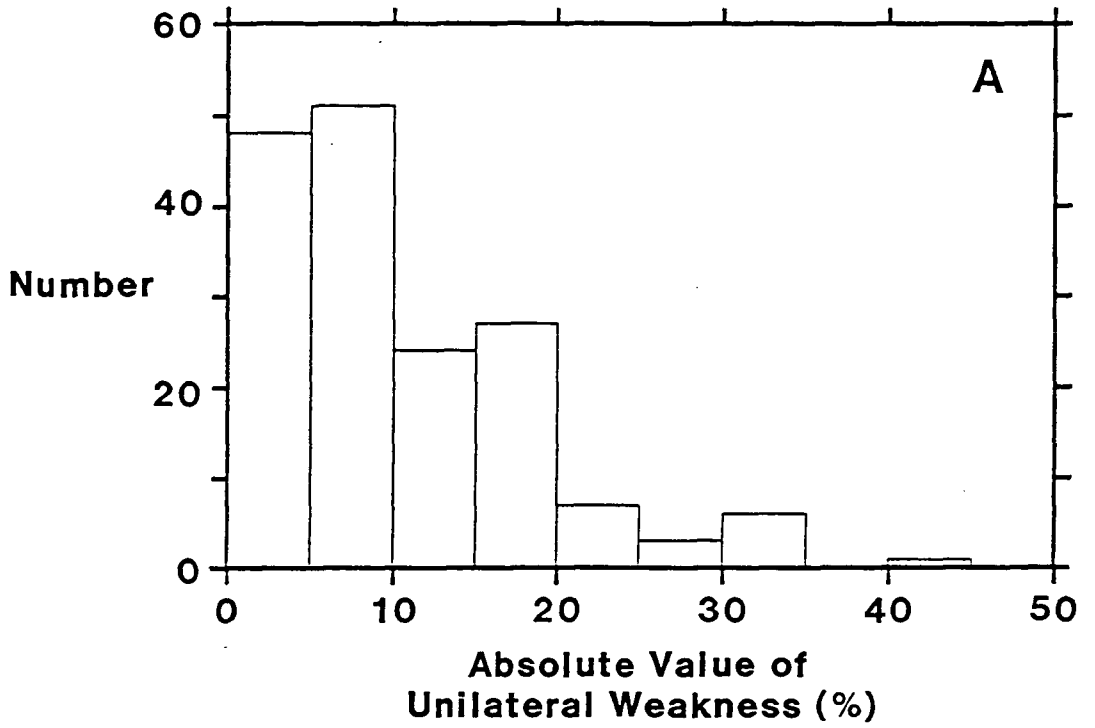

1.000
0.100
0.010
0.001
GAIN
(■)
135°
90°
45°
0°
-45°
-90°
-135°
PHASE
(□)
0.001
0.010
0.100
1.000
10.000
FREQUENCY (HZ)

Figure 20

Caloric Test



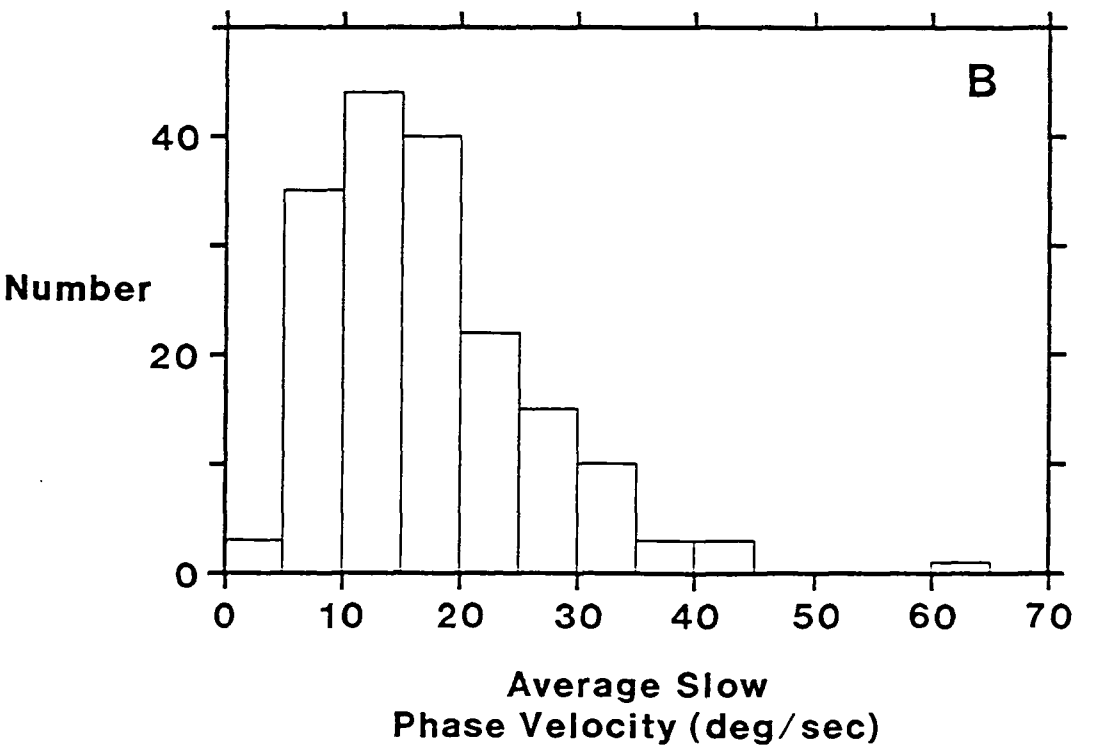


Figure 21

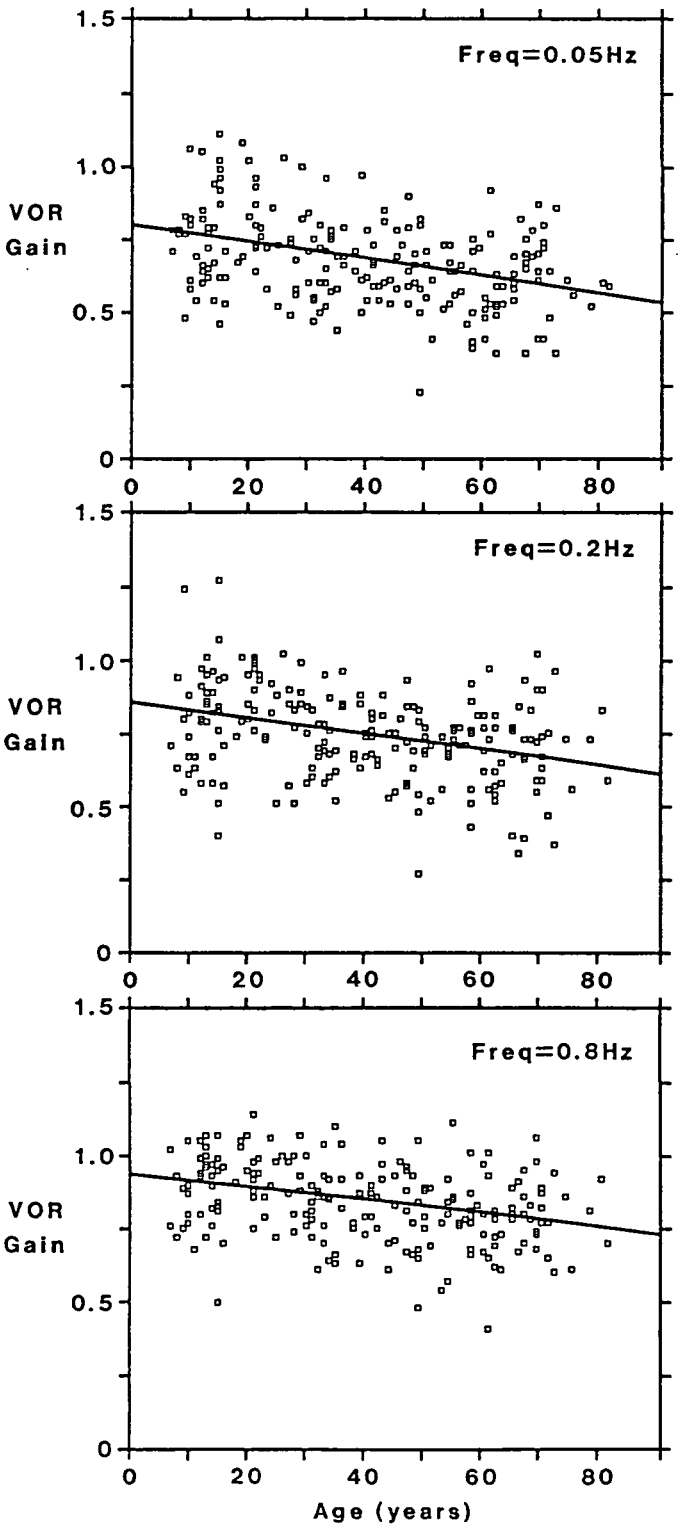

Freq=0.05Hz
Freq=0.2Hz
Freq=0.8Hz
VOR Gain
VOR Gain
VOR Gain
1.5
1.0
0.5
0
0
20
40
60
80
Age (years)

Figure 22

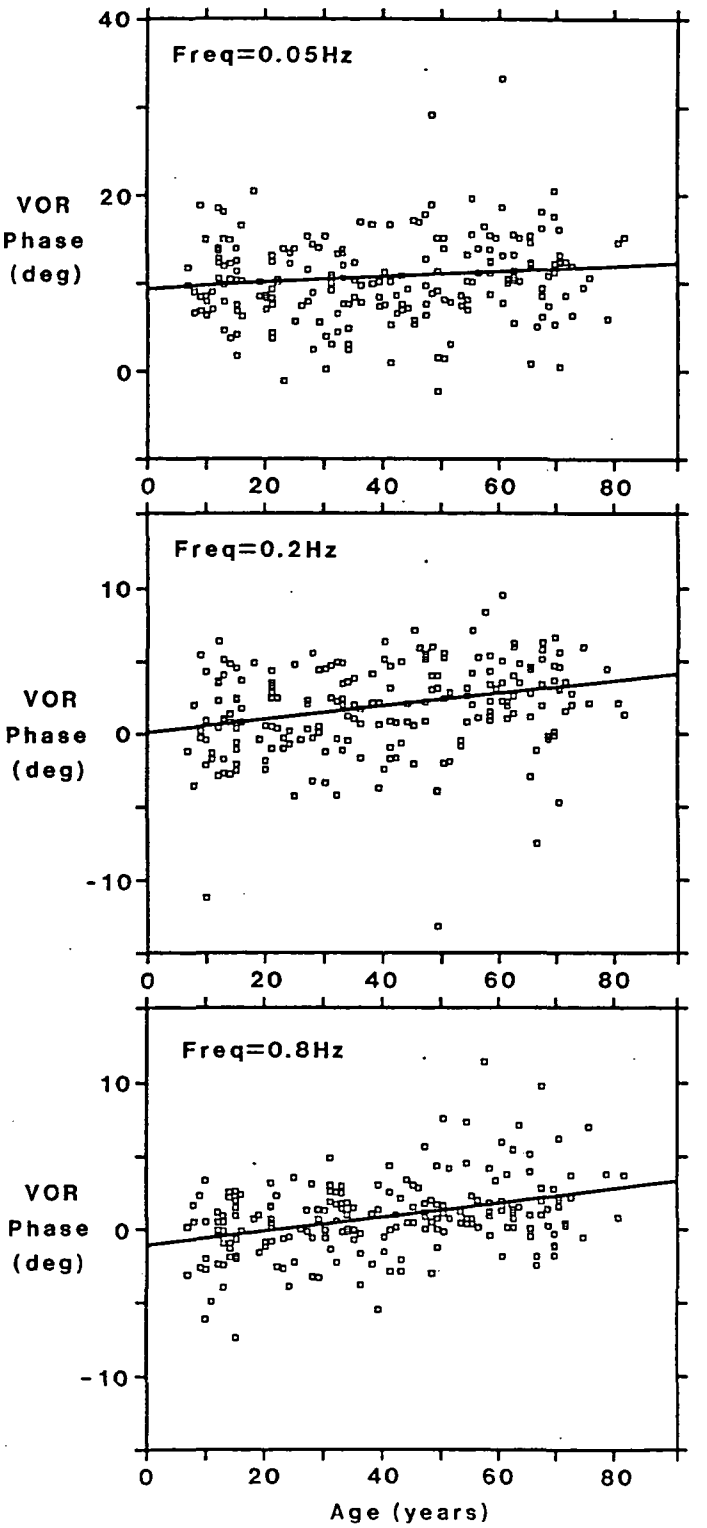

Freq=0.05Hz
VOR Phase (deg)
40
20
0
0
20
40
60
80
Freq=0.2Hz
VOR Phase (deg)
10
0
-10
0
20
40
60
80
Freq=0.8Hz
VOR Phase (deg)
10
0
-10
0
20
40
60
80
Age (years)

Figure 23

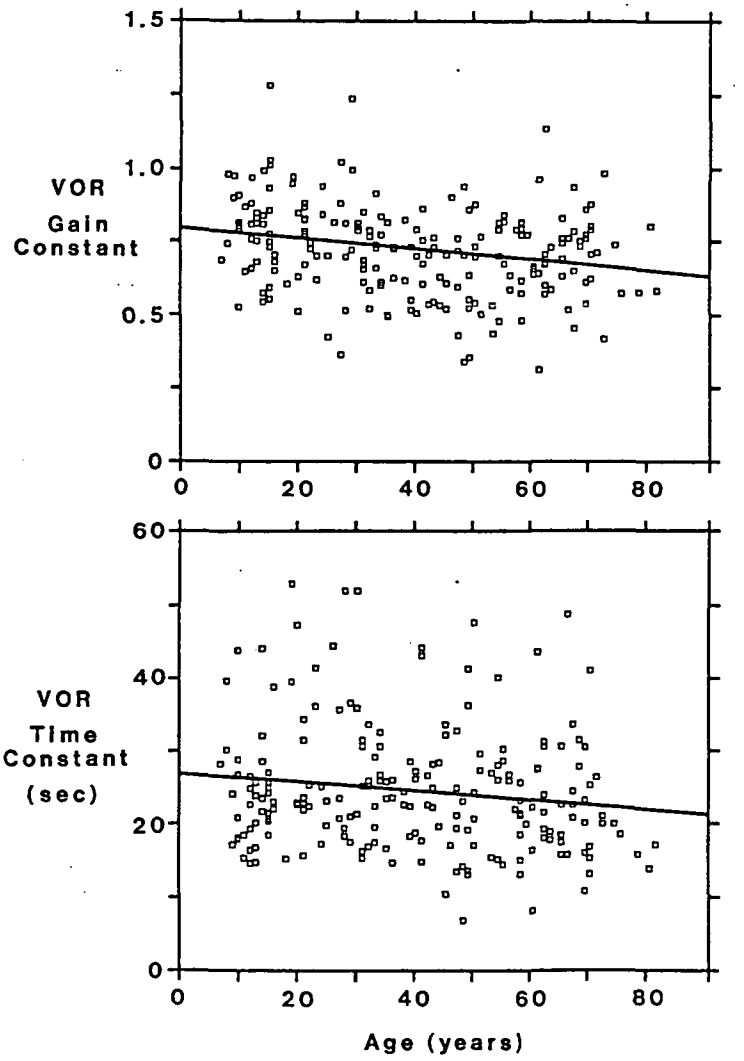

1.5
1.0
0.5
0
VOR
Gain
Constant
0
20
40
60
80
60
40
20
0
VOR
Time
Constant
(sec)
0
20
40
60
80
Age (years)

Figure 24

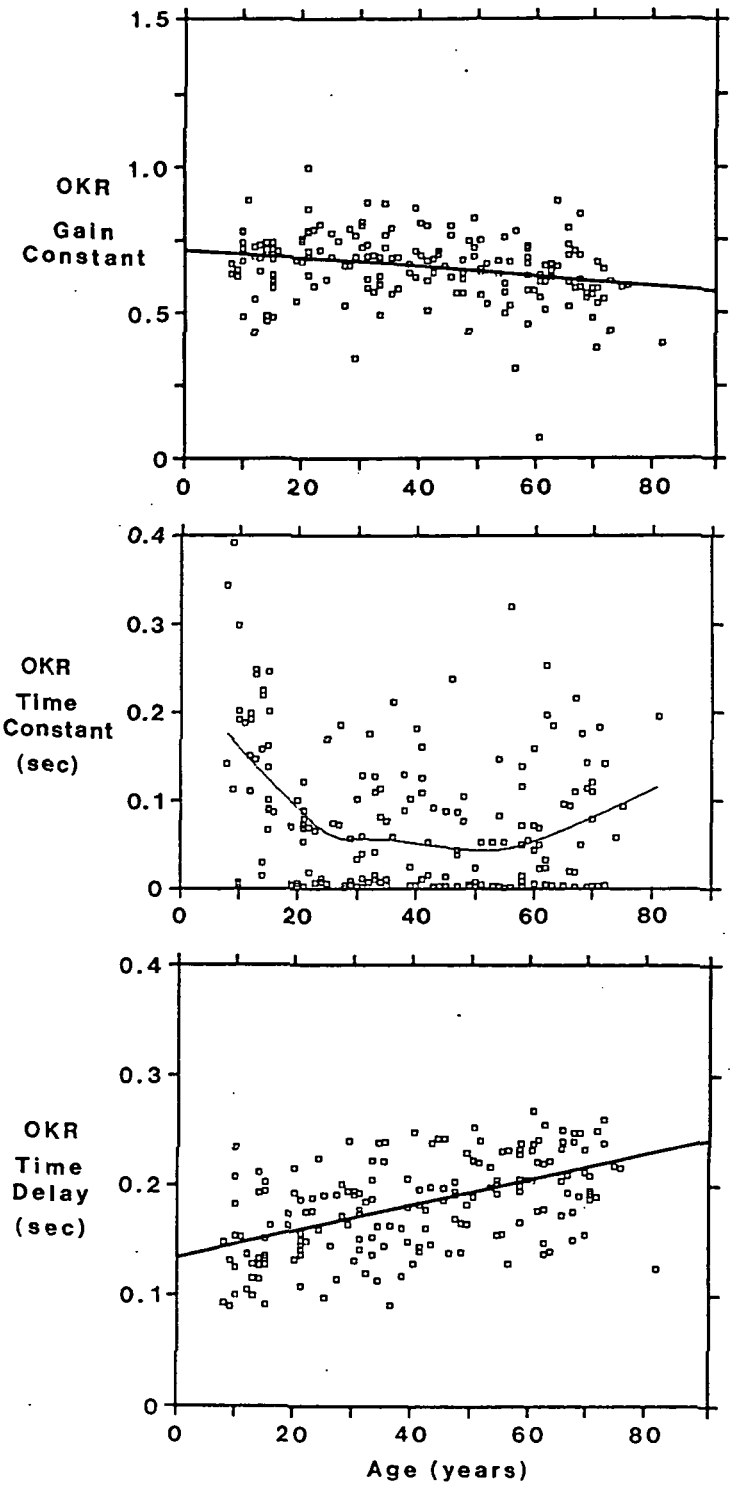

OKR
Gain
Constant
1.5
1.0
0.5
0
0
20
40
60
80
OKR
Time
Constant
(sec)
0.4
0.3
0.2
0.1
0
0
20
40
60
80
OKR
Time
Delay
(sec)
0.4
0.3
0.2
0.1
0
0
20
40
60
80
Age (years)

Figure 25

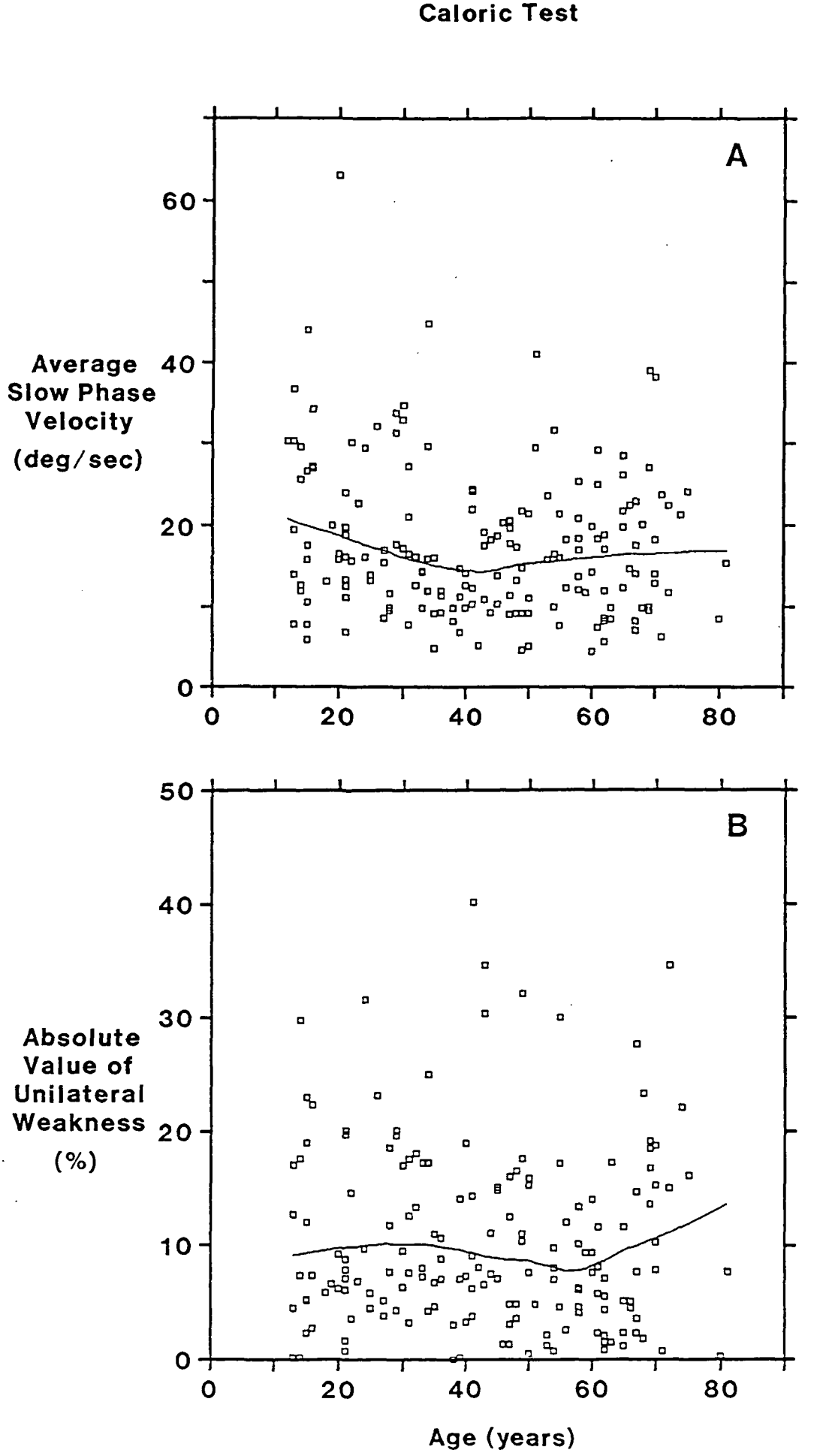
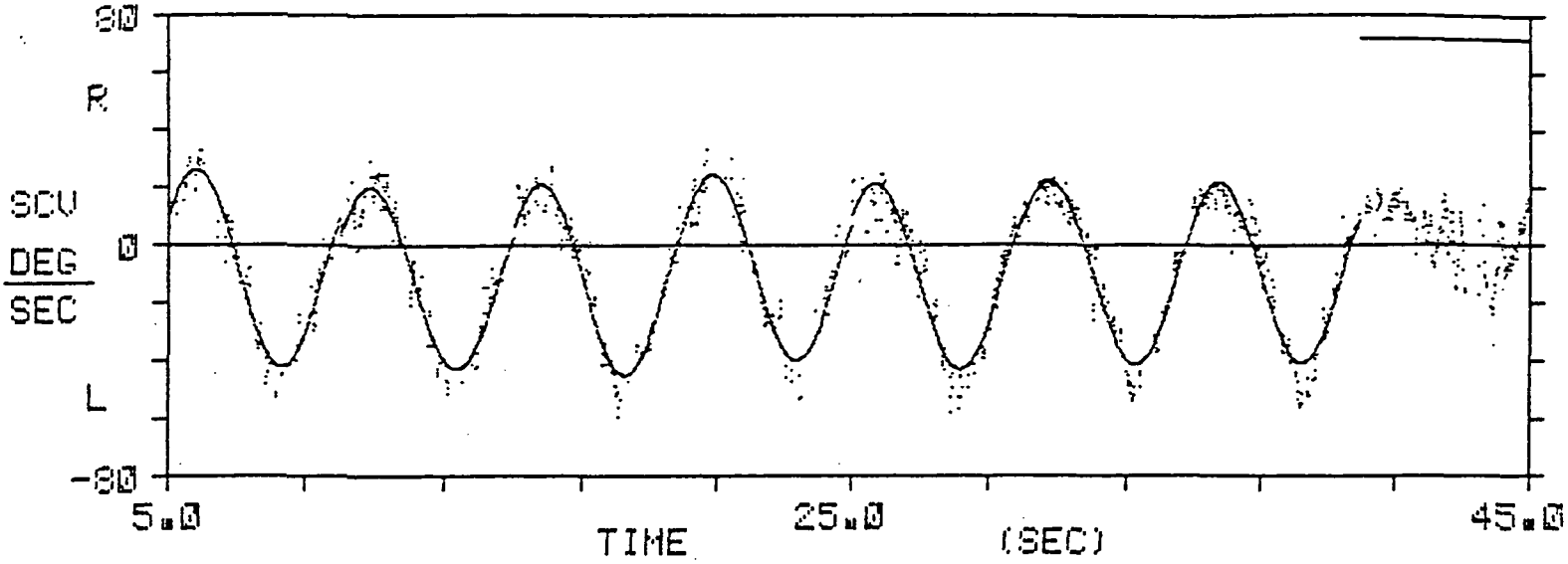
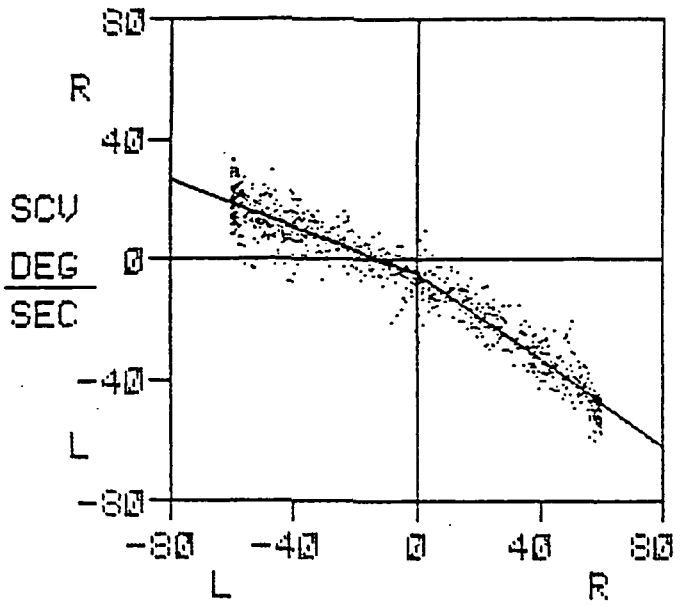

Caloric Test
A
B
Average Slow Phase Velocity (deg/sec)
Absolute Value of Unilateral Weakness (%)
0
20
40
60
80
10
30
50
Age (years)

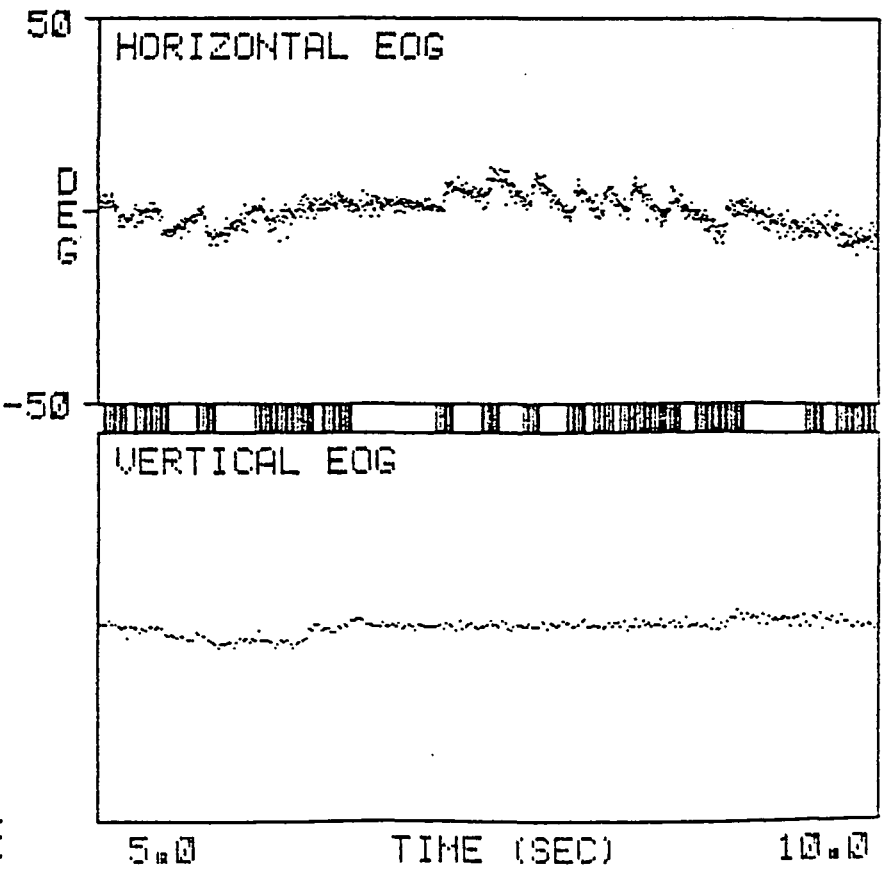
Figure 26

TEST: VESTIBULOOCULAR REFLEX
STIM: 0.2000 HZ SINE
7 PERIODS 60.00 DEG/SEC
PRE-TEST CAL : 41.1 COUNTS/DEG
POST-TEST CAL: 41.8 COUNTS/DEG

	MEAN	STD DEV
STIM AMP :	59.9	
RESP AMP :	32.7	0.98
GAIN :	0.55	0.02
PHASE :	34.48	4.56
BIAS :	-10.22	1.61



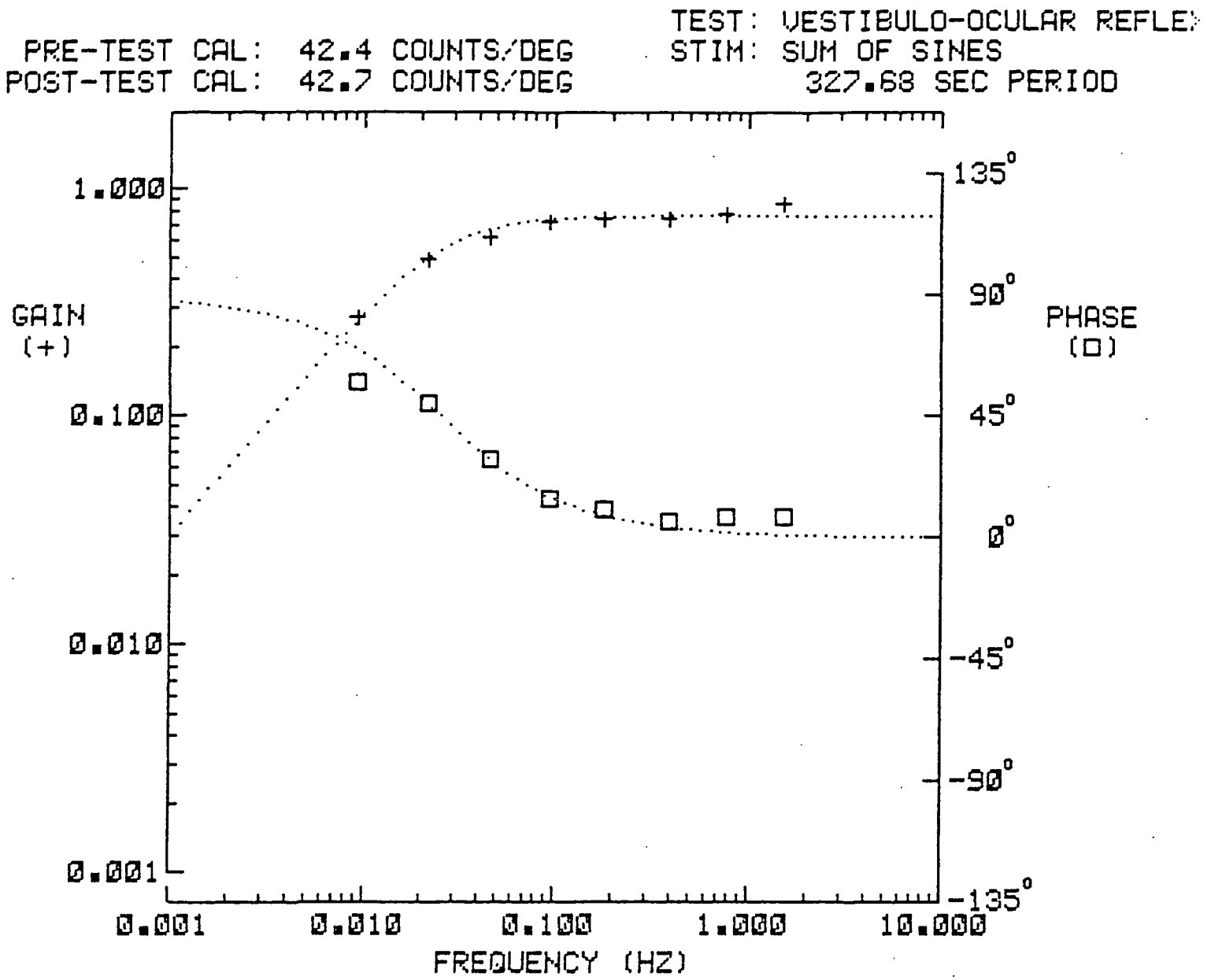




EYE VELOCITY

RHT GAIN	:	0.39
LFT GAIN	:	0.72
OFFSET	:	-4.66 DEG/SEC
ASYMMETRY (R-L)/(R+L)	:	-29.2 PERCENT

Figure 27



CURVE FIT: $\frac{KS^N(S+A1)(S+A2)E^{-ST}}{(S+B1)(S+B2)(S+B3)}$

	FIT1	FIT2	FIT3	FIT4
K	0.7500	0.9068	7288.9097	0.7183
N	1.0000	1.0000	1.0000	1.0579
T				
1/A1		0.1648		
1/A2				
1/B1	6.3340	6.8577	6.3359	8.6104
1/B2		0.1301	0.0001	
1/B3				
ERROR	0.50E-01	0.14E-01	0.50E-01	0.18E-01

BIAS = -4.0 DEG/SEC

FREQ	GAIN	PHASE
0.009	0.27	57.8
0.021	0.49	49.7
0.046	0.61	28.8
0.095	0.70	14.4
0.180	0.73	10.7
0.388	0.73	6.1
0.766	0.77	7.5
1.535	0.85	7.3

Figure 28

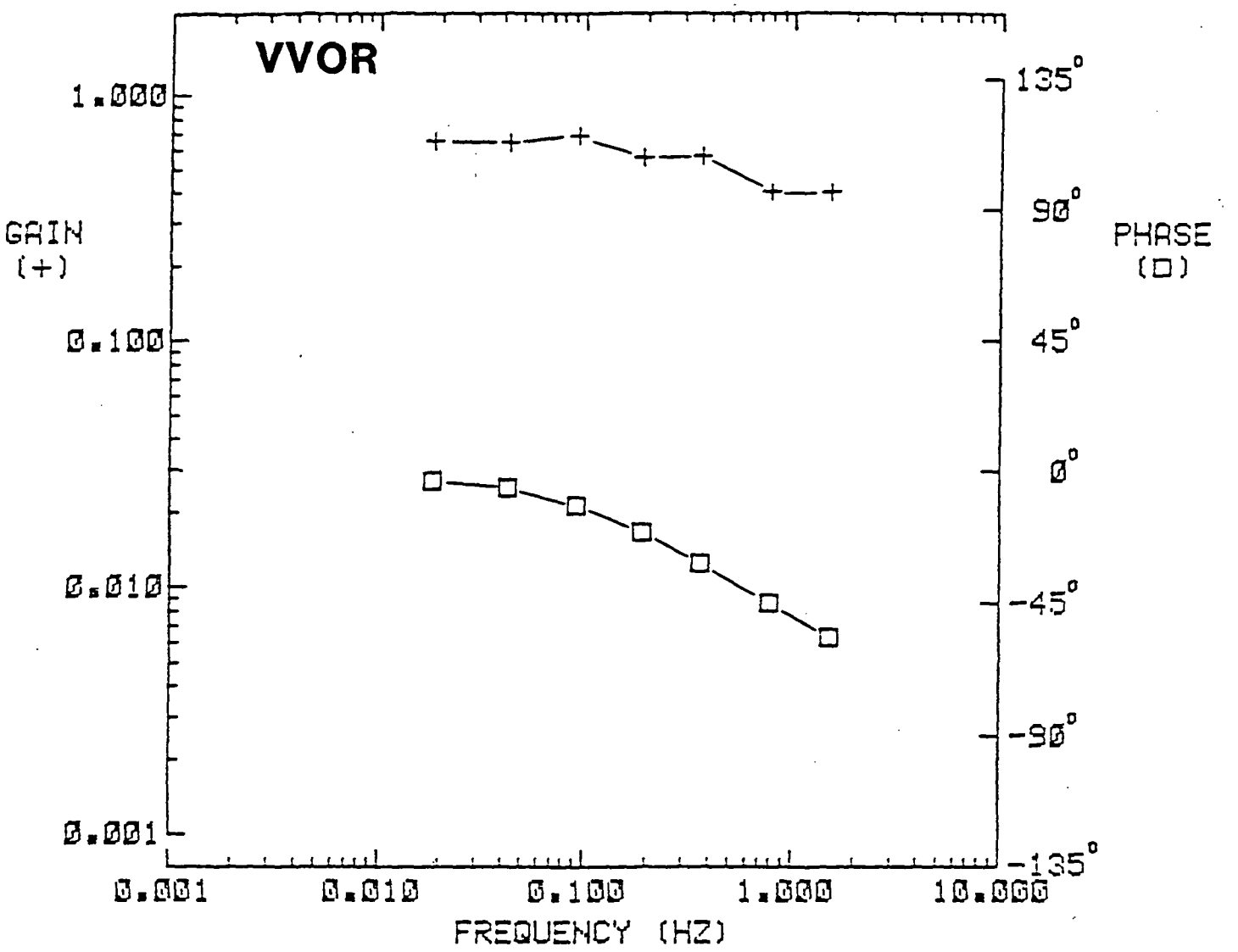

VVOR
1.000
0.100
0.010
0.001
GAIN
(+)
135°
90°
45°
0°
-45°
-90°
-135°
PHASE
(□)
0.001
0.010
0.100
1.000
10.000
FREQUENCY (HZ)

Figure 29

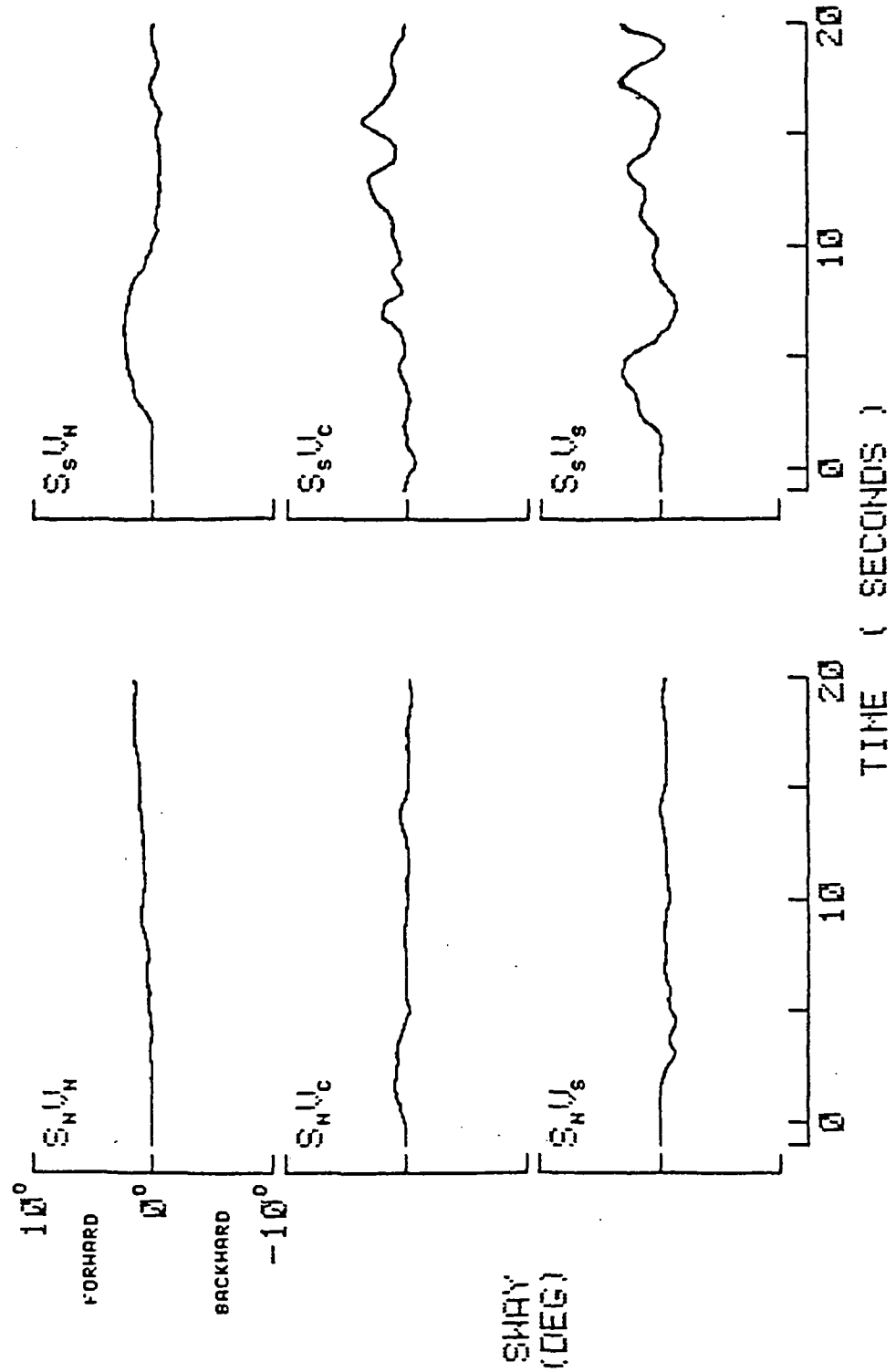

$S_N U_N$
$S_N U_c$
$S_N U_s$
$S_s U_N$
$S_s U_c$
$S_s U_s$
10°
0°
-10°
FORWARD
BACKWARD
SWAY (DEG)
0
10
20
TIME (SECONDS)

Figure 30

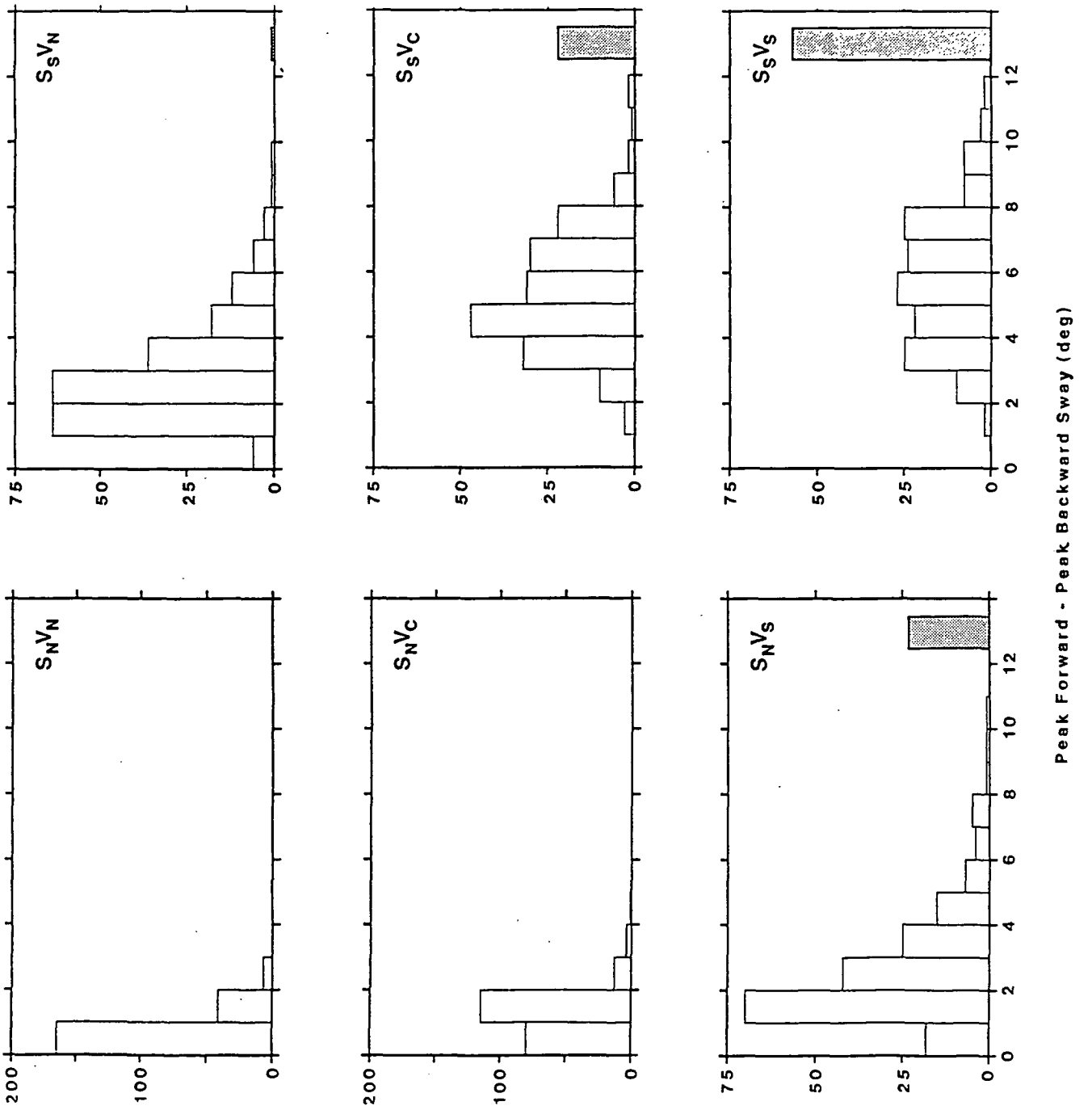

S_SV_N
S_SV_C
S_SV_S
S_NV_N
S_NV_C
S_NV_S
75
50
25
0
200
100
0
2
4
6
8
10
12
Peak Forward - Peak Backward Sway (deg)

Figure 31