Phase-plane analysis of gaze stabilization to high acceleration head thrusts— a continuum across normal subjects and patients with loss of vestibular function

Grace C.Y. Peng1, 6, David S. Zee1, 2, 3, 4, and Lloyd B. Minor2, 3, 5

1Department of Neurology, 2Department of Otolaryngology--Head and Neck Surgery, 3Department of Neuroscience, 4Department of Ophthalmology, 5Department of Biomedical Engineering; The Johns Hopkins University, Baltimore, MD 21287
6Department of Biomedical Engineering; The Catholic University of America, Washington, DC 20064

Running Header: Phase-plane analysis of gaze stabilization

Corresponding Author:
David S. Zee, M.D.
The Johns Hopkins University
Department of Neurology
Pathology 2-210
600 N. Wolfe Street
Baltimore, MD 21287-6921

(410)955-3319 (Phone)
(410)614-1746 (FAX)
Email: dzee@dizzy.med.jhu.edu
Abstract

We investigated the vestibulo-ocular reflex (VOR) during high-acceleration, yaw-axis, head rotations in 12 normals and 15 patients with vestibular loss (7 unilateral (UVD) and 8 bilateral (BVD)). We analyzed gaze stabilization within a 200ms window after head rotation began, using phase planes, which allowed simultaneous analysis of gaze velocity and gaze position. These ‘gaze planes’ revealed critical dynamical information not easily gleaned from traditional gain measurements. We found linear relationships between peak gaze-velocity and peak gaze-position error when normalized to peak head speed and position, respectively. Values fell on a continuum, increasing from normals, to normals tested with very high acceleration (VHA = 10,000 – 20,000°/s²), to UVD patients during rotations toward the intact side, to UVD patients during rotations toward the lesioned side, to BVD patients. We classified compensatory gaze corrections as gaze-position corrections (GPC) or gaze-velocity error corrections (GVC). We defined patients as better-compensated when the value of their end gaze position was low relative to peak gaze position. In the gaze plane this criterion corresponded to relatively stereotyped patterns over many rotations, and appearance of high velocity (100–400°/s) GPCs in the gaze plane ending quadrant (150–200ms after head movement onset). In less compensated patients, and normals at VHA, more GVCs were generated, and GPCs were generated only after gaze-velocity error was minimized. These findings suggest that challenges to compensatory vestibular function can be from vestibular deficiency or novel stimuli not previously experienced. Similar patterns of challenge and compensation were observed in both patients with vestibular loss and normal subjects.
Introduction

Best vision requires that the image of an object of interest be placed and held still on the fovea (Carpenter 1991). This same requirement holds during head movements. To maintain fixation upon an object that is stationary in space, the vestibular system must produce eye movements that perform two functions: 1) match the amplitude and direction of the head movement (more specifically the rotation and the translation of the orbit) to maintain zero gaze velocity, and 2) take into account the need to keep the fovea of both eyes aimed at the point of regard to maintain a constant gaze position. Under normal conditions, this function is fulfilled by the equal and opposite slow-phase eye response to head rotation (the rotational vestibulo-ocular reflex, VOR), with necessary adjustments for translation of the head (orbits), viewing distance, and target eccentricity. The limits within which the vestibular slow-phases are adequate, however, are set by the nominal operating parameters of the vestibular system of each individual. The system is considered ‘challenged’ when the properties of the head stimulus (velocity, acceleration, frequency content) exceed these limits. The system can be challenged with labyrinthine hypofunction (when the operating range and sensitivity are reduced), or with unaccustomed head motion at the limits of normal operation, or with both hypofunction and unusual motion of the head. In the last condition, locomotion would certainly present a natural challenge to the system. Under these conditions during head motion, not only is there unwanted motion of images on the retina (gaze-velocity error) or oscillopsia (the illusory motion of the visual environment), but also the image of the object of interest moves away from the fovea (gaze-position error) leading to a decline in visual acuity. Persons with vestibular loss often complain of problems with vision while walking in the
environment, such as the case of “J.C.” (1952). Accordingly, adaptive mechanisms are triggered to restore the amplitude and direction of the slow-phase response, though there are probably enduring limitations to the degree to which normal vestibular slow phases can be recalibrated (Pulaski et al. 1981; Takahashi et al. 1989). The degree to which pathological vestibular slow phases can be recalibrated is not known.

Brief transient head rotations have long been used (e.g., Gauthier and Robinson 1975) to quantify the human VOR. A position ‘step’, which results from these head rotations, is a complex signal composed ideally of an infinite number of frequencies. The amplitude and duration of the step determine the range of dominant frequencies, velocities and accelerations. By varying these step parameters one can examine the response of a control system across a large range of stimulus properties, easily placing the vestibular system in a ‘challenged’ state when the velocities and accelerations are high enough. The ‘step’ displacement of the head also closely resembles natural head movements (Zangemeister et al. 1981). Also, since the function of the VOR varies with mental set (effort of spatial localization), the brevity of the step stimulus (< 500ms) allows a constant mental set to be assumed while the head is moving. Recently, many studies have applied high acceleration steps to replicate the clinical “head-impulse test” (Halmagyi et al. 1988, 1990) or “head thrust.” Experimental accelerations of the head range from 700-2,800°/s² (electromechanical rotational devices of Tabak et al. 1997a; Tian et al. 2000) to 1,500-4,000°/s² (manual perturbations of Halmagyi et al. 1990; Aw et al. 1996a) to 1,900-7,100°/s² (head taps of Maas et al. 1989) to ~10,000°/s² (manual perturbations of Schmid-Priscoveanu et al. 1999), with stimulus durations of 100-200ms.
The ranges of accelerations in these head thrusts are comparable to those accelerations imposed on the system during natural locomotion (Grossman et al. 1988).

Studies using transient head rotations have assessed the vestibular slow-phase response in normal and challenged vestibular systems. Studies have shown that when slow-phase responses are inadequate (in a challenged state), other mechanisms are triggered to help stabilize images on the fovea after the head begins to move. They include the substitution and enhancement of other sensory reflexes, such as the cervico-ocular reflex; the substitution of mechanisms to satisfy a function, such as pre-programming of slow-phases in anticipation of head motion; and the substitution of other behaviors, such as the use of saccades to generate rapid eye movements in the direction of the deficient slow-phase response (e.g., Kasai and Zee 1978). Quantitatively, the vestibular (position) step response has been used to estimate the gain (eye motion/ head motion) of inadequate slow phases (Halmagyi et al. 1990; Aw et al. 1996b; Allison et al. 1997; Foster et al. 1997; Tabak et al. 1997b; Cremer et al. 1998; Schmid-Priscoveanu et al. 1999) and to estimate the contributions of compensatory saccades to gaze stabilization (Segal and Katsarkas 1988a,b; Bloomberg et al. 1991; Tian et al. 2000). The latter studies showed that when compensatory saccades are observed in the step response, they interact synergistically with the slow-phase components to augment gaze stabilization, such that smaller slow-phase components during the step were matched with larger saccadic components and vice versa. In these studies, compensatory saccades were observed in unilateral vestibular deficient (UVD) subjects during ipsilesional rotations and in bilateral vestibular deficit (BVD) subjects. With the exception of one study (Tian
et al. 2000), compensatory saccades were also observed in normal subjects and in UVD subjects during contralesional rotations.

Quantitative measures of gain provide an assessment of vestibular function limited by the specific conditions in each study. For example, in the studies using step-like inputs, the stimulus characteristics and the method of gain calculation have varied widely. The method for calculating slow-phase gain is usually determined at a set interval of time after the onset of the transient head motion, averaged across 35-45 ms for slow-phase gain (Tian et al 2000), 40-50ms acceleration gain (Collewijn and Smeets 2000), 30 and 100ms velocity gain (Aw et al. 1996a, b). Carey and colleagues (2002) used the largest velocity gain value within the 30ms interval prior to the onset of peak head velocity during each head thrust. The properties of each stimulus, therefore, tested a different portion of the operating range, and the resulting characteristics of the responses reflected these differences in terms of gain. In order to achieve an overall assessment of gain, studies used their own averaging, threshold, and fitting methods on selected time intervals or point(s) in time. In this manner, contributions from vestibular slow-phase components and saccadic components were reduced to two numbers. It is also understood that the nominal operating parameters for each system will vary, especially among the patients. Factors such as degree of deficit, age, duration of symptoms and rate at which deficits were acquired may be critical in setting the nominal operating range for each subject. Therefore, it is not surprising that the gains differed among studies. The gains in effect provide ‘static snapshots’ of the approximate vestibular and saccadic components for a given set of calculation constraints. They do not relate the dynamic response characteristics over the time period of the stimulus. Gain ratios have meaning
when there is a causal relationship between the input and the output. In a challenged vestibular system a single input (head motion) may trigger multiple outputs (e.g., from the vestibular and saccadic systems) and these outputs in turn may be more causally related to other inputs (e.g., neck afferents). The slow-phase gain, therefore, describes an overall performance level composed of slow-phase responses from multiple sources, rather than just being of vestibular origin. Finally, since the function of the vestibular system is to optimize gaze velocity and gaze position simultaneously, it is unclear from previous studies how gaze velocity is minimized in relation to gaze position. Dell’Osso and colleagues (1992a-c) studied the effects of congenital nystagmus - another condition that disturbs steady foveal fixation, grappling with similar issues of analysis. Among their many methods, they used phase planes, which provided a means to analyze the intricate dynamics of ‘foveation’ and allow them to follow the interactions between ‘foveating- and braking-saccades’ and the slow phases of congenital nystagmus.

We undertook this study to characterize the dynamic function of the vestibular system over the duration of a step-like stimulus. Many studies over the past 30 years have analyzed the VOR in subjects with normal vestibular function or with unilateral or bilateral vestibular hypofunction. These studies, however, have not examined how the vestibular system acts to simultaneously minimize gaze position and gaze velocity – the two fundamental functions of the system. Here, we devised a phase plane method for systematically presenting the gaze dynamics of the head thrust response. In this study, we describe a method that offers three fundamental advantages in the analysis of these responses in comparison to techniques that have been used previously. First, the method provides a robust depiction of responses over different periods of time during the
stimulus. Second, variability in responses between subjects and between stimulus repetitions in a single subject can be readily visualized and interpreted. Third, the method allows eye-movement responses to be analyzed in terms of the contributions to minimization of gaze-velocity and gaze-position error. We tested the system across a large range of accelerations: 1,400-20,000°/s², applying the stimulus in unpredictable directions and amplitudes, with the following questions and hypotheses in mind: How do the gaze-position and gaze-velocity errors that are produced in vestibular systems challenged by pathology compare with that in normal vestibular systems challenged by unnatural stimuli? We hypothesized that gaze errors are produced along a continuum depending on the challenge. How do patients and normal subjects compensate for gaze-position and gaze-velocity errors? We hypothesized that phase-plane analysis will more easily show whether strategies from recovery of vestibular challenge will preferentially reduce gaze-position or gaze-velocity errors. We hypothesized that patients with more residual function would exhibit better compensation for gaze-position errors than those with less residual function. We hypothesized that patients with bilateral loss rely more on gaze-position correction mechanisms, such as saccades. Preliminary analyses of this data were presented elsewhere (Peng et al. 1997, 1998, 2000).

2 Methods

2.1 Experimental Setup

We present data from eight patients with unilateral vestibular deficits (UVD), ages 30 to 67 years, who had undergone surgical resection of the superior and inferior
vestibular nerves, for removal of an acoustic neuroma (6) or for Ménière's disease (2). The UVD patients were tested 10 days to 37 months after their surgeries. We also present data from eight patients with bilateral vestibular deficits (BVD), ages 20 to 85 years. The etiology of the loss of function in the BVD patients was gentamicin toxicity (3), presumed bilateral vestibular neuritis (1), and of unknown origin (4). One BVD patient (LO) was tested twice, 9 and 21 months after developing symptoms. Eleven normal subjects, ages 23 to 54 years were also tested. These normal subjects had no clinical symptoms of a vestibular disorder and had a normal neuro-otological examination. All experimental protocols were approved by the Joint Committee on Clinical Investigation of the Johns Hopkins University School of Medicine. Each subject gave informed consent for participation in this study.

Subjects were seated in a chair, with their heads free to move inside a triaxial magnetic field search coil system. Subjects wore search coils that allowed for measurement of eye motion around all three axes of rotation. The coils were embedded in an annulus that adhered to the sclera of the eye (Skalar, Delft, Netherlands). All patients wore one eye coil, except for BVD patients MI, LO2, RO and GR who wore coils on both eyes. Two of ten normal subjects wore coils on both eyes. Each eye coil was placed on the eye after applying a topical anesthetic (Ferman et al. 1987). Each subject used a dental bite plate to hold a triaxial, hand-made search coil, which transduced the motion of the head. In each search coil, a current was induced by the oscillating magnetic fields, composed of a superposition of the three frequencies of each magnetic field. Movement of each search coil thereby modulated the amplitude of the three frequencies (on the coil) with respect to the three-dimensional orientation of the coil.
in space. Synchronous detection of each frequency component produced voltages proportional to the amplitude of coil rotation relative to each field (linear across all the fields). These voltages were low-pass filtered with a single pole, Butterworth, anti-aliasing filter at 90Hz, and sampled at 500Hz through a 12 bit A/D converter. The resolution of the eye coil was 0.1° for eye rotations in the horizontal and vertical planes, and 0.2° in the torsional plane. More details about the coil system are described in Bergamin et al. 2001.

Before each head thrust, the experimenter centered the head (within 1.5°) in the yaw, pitch and roll planes using a video feedback screen. The experimenter stood behind the subject and applied head thrusts: the experimenter held the subject’s head and imposed an abrupt, ‘position-step’ stimulus, rotating the subject’s head at a high acceleration to bring the head 10-25° from center. Head thrusts were produced in the horizontal plane (yaw head thrusts), over a range of accelerations in random rightward and leftward directions, with 10 to 20 head thrusts towards each side. The experimenter observed the video screen to ensure that components of pitch and roll head movements were close to zero during each yaw head thrust. The subject was asked to maintain fixation on an LED at a distance of 124 cm from the eyes, in an otherwise dark room.

Peak head velocities of head thrusts ranged from 70°/s to 410°/s. The head reached peak velocity between 30 to 140 ms after it began rotating. The peak head accelerations ranged from 1,400°/s² to 10,600°/s². In normal subjects, the kinematics of head motion during the high acceleration (HA) head thrusts were in the same ranges of those of the patients. For the very high acceleration (VHA) head thrusts (only for normal subjects), the peak velocities ranged from 230°/s to 550°/s, the head reached peak
velocity between 35 to 70 ms after it began rotating, and the peak head accelerations ranged from $5,000^\circ/s^2$ to $20,000^\circ/s^2$.

2.2 Data Analysis

The search coils were calibrated relative to the maximum field strength values detected by the coil from the three orthogonal magnetic fields. The calibration values were then used post-recording to process the data into rotation matrices, which were then transformed into rotation vector coordinates (see Bergamin et al. 2001 for a detailed description of the calibration procedure and rotation vector calculations). The angular positions of each eye coil (relative to spatial coordinates) were then calculated from the rotation vectors. To calculate the angular velocity trajectories, a 5th order 100Hz low pass FIR filter was first applied to the rotation matrices (resulting in $M$). The resulting data were differentiated using a 5th order REMEZ (Matlab) FIR filter, designed to differentiate up to 100Hz and low-pass filter the data above 100Hz. The differentiated data were then used with the undifferentiated, filtered rotation matrices ($M$), to calculate angular velocity (Hepp 1990). The standard deviation of the velocity noise after filtering was $\pm 2^\circ/sec$. To calculate the angular acceleration trajectories, the angular velocity data were differentiated with the same 5th order REMEZ combination differentiator and low-pass filter.

We compare mean error values listed in the Table, using two-tailed, unpaired t-tests. Since we made only a few selected comparisons between the 15 groups of data, it was unnecessary to adjust the significance level with multivariate analysis methods. Five comparisons (detailed in section 3.1) were made to determine the significance of means
that were dissimilar. The resulting p-values (0.006, 0.003, 0.002, 0.0009, 0.037) if added together, still remained significant to five percent (0.0489). Prior to performing the t-tests, we calculated the F-test statistic to compare the variance of the means of the two groups. The F-values showed that those comparisons with normal subjects tested at HA and VHA had variances of the mean that were significantly different from the variances of the mean from the patient groups. For these comparisons, we used t-tests assuming unequal variances. In the other comparisons between patient groups, we used t-tests assuming equal variances.

2.3 Wavelet detection of movement transients

We used a wavelet transform technique to determine the onsets and offsets of head and eye movements, and the transient eye movements within the head thrusts. We chose this technique over other approaches, because its ease of use. Particularly with the variability in this data (both in the stimulus and response), this technique can be applied systematically to the data with minimal adjustment of parameters. Other methods, of course, can be used to achieve the same results, but likely with additional processing and parameter adjustments.

The time-varying properties of wavelet analysis allow detection of the smallest change in the unfiltered position signal, without compromising the time resolution around the transitions (Daubechies 1992). We applied the continuous “Morlet” wavelet transform (Matlab, Natick, MA, Torrence and Compo 1998) on the angular position data (without prior digital filtering), independently on each position trajectory (head-in-space (head), eye-in-space (gaze), and eye-in-head (eye)). The transform generated the
corresponding real wavelet coefficients relative to both scale (which is inversely related to frequency) and time. For the head trajectories, Morlet waveforms with scales corresponding to 1-10 Hz were used. For the eye and gaze trajectories, Morlet waveforms with scales corresponding to 5-50 Hz were used. The absolute values of the coefficients were summed across the scales and plotted over time. Each peak in the wavelet power plots marked a transition in the original position trajectory. The width of each peak defined the duration of the transition. The latter parameter established the onset and offset times used throughout the data analysis.

2.4 Gaze Plane Methodology

In order to assess the gaze strategies in each subject, we found it fruitful to focus on the behavior of the gaze components relative to its derivatives, rather than relative to time. We devised ‘gaze planes’ so that the position and velocity components of large quantities of multi-parametric gaze data can be characterized simultaneously. Note that our ‘gaze planes’ differ from ‘gaze planes’ first presented by Zangemeister and Stark (1982), which depict the eye and head components of gaze on separate axes. Our gaze planes are based on classical phase-plane methods for analysis of signals. Phase-plane plots of dependent variables and their derivatives are often used to display behavior without explicit time dependence. We found that these plots provided the clearest and most efficient means for comparing single time trajectories. As described below, large amounts of data from multiple head thrusts could be presented in a compressed format using the phase plane plots.
The time trajectories of head, eye and gaze, in position and velocity, during two example head thrusts, are presented in Fig. 1. Fig. 1A displays a response from a normal subject and Fig. 1B displays a response from a UVD patient (during a head thrust toward his lesioned side). When two time trajectories are plotted against each other, phase-plane plots are formed. We created a phase plane in which the gaze-velocity trajectories are plotted against the gaze-position trajectories (thick solid lines in the time trajectories shown in the upper panels). The corresponding phase planes of the time trajectories are shown in the bottom panels in Fig. 1. These ‘gaze planes’ of the head thrust responses characterize the dynamics of the error of the eye response. In order to represent the time behavior in the gaze plane, we formed 50ms time epochs in each head thrust response between 0-200ms, delineated by the vertical gray bars and corresponding color bars in the top panels, and coded each gaze-plane trajectory with respect to the color bar. The gaze plane was divided into quadrants to facilitate description of the responses. For the gaze plane of an under-compensatory VOR, each gaze trajectory began at the origin (circles, time = 0 ms, blue epoch) and proceeded toward the right halves of the plots for rightward yaw thrusts and toward the left halves of the plots for leftward yaw thrusts, ending at 200ms (asterisk, pink epoch). In both of the examples in Fig. 1, only rightward head thrust responses are presented. In the gaze plane, the width of the loops denotes the maximum amount of gaze-position error. The height of the loops in quadrant 1 denotes the maximum amount of gaze-velocity error during rightward head thrusts, and the height of the loops in the quadrant 3 denotes the maximum amount of gaze-velocity error during
leftward head thrusts. Quadrants 4 and 2 therefore depict the manner in which gaze-velocity and gaze-position errors were minimized, with the goal of zero gaze velocity and zero gaze position. The movement of the trajectories along the abscissa toward the ordinate (horizontal component) depicts the minimization of gaze-position error. The movement of the trajectories back toward the abscissa (vertical component) depicts the minimization of gaze velocity. The closeness of the endpoints of the trajectories (asterisks) to the origin (zero gaze position and velocity) denotes the accuracy of the final gaze. In the normal response in Fig. 1A, the gaze-position and gaze-velocity errors are small relative to that for the UVD response in 1B, with all the dynamics occurring very close to the origin. Though small, one can readily see in the gaze plane that a small amount of error is present and minimized within 100ms (red epoch). These gaze errors are less apparent in the time trajectories above. In the abnormal response in 1B, the gaze plane shows that gaze velocity increases simultaneously with gaze position, with gaze velocity reaching its maximum earlier and beginning its minimization phase sooner (in the red epoch) than gaze position (in the green epoch). In this response, the gaze-position correction, marked by a plus at the onset and a square at the offset, brings the gaze errors close to the origin. Note that the onset and offsets were marked with respect to the eye-in-head position trajectory. The onset of this correction occurred while the head was still moving towards its maximum position. The time at which the maximum head position was reached is marked by the triangle. In summary, in a two-dimensional gaze plane of the VOR, quadrants 1 and 3 depict the acquisition of gaze-position and -velocity errors, quadrants 4 and 2 depict the minimization of gaze-position and -velocity errors, during leftward and rightward head thrusts respectively. The colors and symbols in the gaze
planes allow one to view various time aspects of the response. Therefore, information from six time plots is condensed into one gaze-plane plot.

3 Results

3.1 Static analysis of gaze stabilization errors

We first describe the measurements of maximal gaze-velocity and maximal gaze-position errors that are made at static points along the eye and head movement trajectories. Then we describe the gaze errors at the end of the head thrust in terms of position. In the subsequent sections, we will compare these results with the dynamic phase-plane results. Fig. 1 shows the points (A-F) along the time trajectories that are used to calculate the gaze errors. Four selected measures are determined from each head thrust response in UVD, BVD and normal subjects and presented in the table. The ‘normalized peak gaze velocity error’ (NVE) defines an estimate of maximum gaze-velocity error during the head thrust normalized to peak head velocity (the maximum error that is found only in the direction of the head thrust). In Fig. 1, NVE = E/F. A NVE of 1.0 indicates no gaze-velocity compensation, or 100% velocity error.

As shown in the table in the UVD subjects, the NVE were less than 1.0 indicating that some residual gaze-velocity compensation existed during head thrust towards the side of the lesion (73% mean error). The velocity compensation on the intact side was much better, but still lower (44% ± 16%, mean ± SD) than that observed in both sides of
normal (HA) subjects (21% ± 8%; p = 0.006). In the BVD subjects, the NVE indicated that there was little or no velocity compensation (91% mean error). In the normal subjects, the NVE were not zero, indicating that the normal VOR is not perfect in these conditions.

The ‘normalized peak gaze position error’ (NPE) defines an estimate of maximum gaze-position error accumulation when normalized to peak head position (the maximum change in head position during the head thrust). In Fig. 1, NPE = A/B. A NPE of 1.0 indicates 100% position error; i.e. there is no eye in head position movement. In the UVD patients, NPE reflected the asymmetry in the responses towards the lesion side and the intact side. As shown in the table, the NPE toward the lesion side in UVD patients were lower (56% ± 14%) than the NPE from either side in the BVD subjects (76% ± 12%; p = 0.003). The NPE toward the intact side in UVD patients were higher (29% ± 10%) than the NPE from both sides in the normal (HA) subjects (9% ± 4%; p = 0.002).

The group mean of peak gaze position (absolute error) in UVD patients was 8.9±2.7° during head thrusts towards the lesion side, and 4.4±1.5° during head thrusts toward the intact side. In BVD patients the group mean of peak gaze position for head thrusts toward both sides was 10.3±1.6°. In normal subjects the group mean of peak gaze position for head thrusts toward both sides was 1.5±0.9°, during high acceleration (HA) head thrusts.

We have provided two measures for estimating the amount of correction for gaze-position error at the end of the head thrust - ‘normalized end gaze position error’ (NEE) and ‘end gaze position’. (Gaze velocity was always minimized by the end of the head thrust, because reduction of head motion reduces gaze-velocity errors.) As we will
describe in section 3.2, these measures of end gaze position depend on the variability in timing of the corrections. The ‘normalized end gaze position error’ (NEE) provided a normalized estimate of gaze-position error at 200ms or at the end of any corrective movement that is still ongoing at 200ms after the start of the head thrust. In Fig. 1, NEE = D/C. A zero NEE corresponds to perfect compensation (0% error) with the image of the target being on the fovea at the end of the head rotation. As shown in the table, the negative NEE indicate that the gaze position had moved to the other side of the zero target, on the side opposite the direction of the head thrust (overcompensation). In UVD subjects, NEE toward the lesioned side were larger (32% ± 11%) than those toward the intact side (10% ± 8%; p = 0.0009). The reduction in position error (NPE minus NEE) was comparable on the lesion side (24% ± 16%) as well as on the intact side (19% ± 9%; p = 0.48). The NEE on the intact side (10% ± 8%) were never reduced to the level observed in the normal subjects (1% ± 4%; p = 0.037). In the BVD subjects, the reduction in position error was nearly equal for head thrusts to both sides (42% ± 22%).

The ‘end gaze position’ defines the accuracy of the response to the head thrust at 200ms or at the end of any position correction that is still ongoing at 200 ms. This parameter provided an absolute measure of gaze-position error towards the end of the head thrust (point D in Fig. 1). In general the end gaze positions indicated that both UVD and BVD populations were able to reduce their gaze-position errors by the end of the head thrust, but were not able to achieve the accuracy of normal subjects. In the table, the mean values of end gaze position that were negative indicated that the eye had gone beyond the target position. In summary, we calculated values representing the maximal gaze-position (NPE) and gaze-velocity (NVE) errors during each head thrust, to
determine the worst-case performance of each subject. For each head thrust, the maximal errors may have occurred at different times after the onset of head motion. These values provide an absolute measure of overall performance independent of time, while traditional measures of gain are highly dependent on time as described in the introduction. In the table we also present measures of normalized end gaze position error (NEE) and end gaze position.

------------------------{Insert Figure 2 here}------------------------

In Fig. 2A, we show the relationship between the maximum gaze-velocity error and the maximum gaze-position error by plotting NVE versus NPE for all subjects, with mean responses from each side plotted with separate symbols. This figure shows that each subject exhibited maximum error values that were clustered according to which population they belonged. When all the populations were observed together, a gradient was formed in which gaze-velocity and gaze-position error increased linearly from normal subjects to normal subjects tested at VHA (very high acceleration), to UVD patients tested on the intact side, to UVD patients tested on the side of the lesion, to BVD patients. This linear relationship between NVE and NPE was biased, with 15% more NVE than NPE in all populations (solid line). A plot of equal errors (dotted line) is shown for comparison. The four asterisks in Fig. 2 depict the data from the normal subjects tested at VHA (see Table). In Fig. 2A, these data overlap with the UVD data from the intact side, indicating that the errors in these normal subjects were elevated above those normally observed in the normal population at HA (high acceleration). The NVE values of the normal subjects at VHA (34% ± 3%) were comparable to those in the
UVD data on the intact side (44% ± 16%; p = 0.11). Similarly the NPE values of the normal subjects at VHA (27% ± 4%) were comparable to those in the UVD data on the intact side (29% ± 10%; p = 0.65). Only two UVD subjects (CR (solid arrows) and WI) had errors from the intact side (open triangles), which overlapped with the normal population at HA. One UVD subject (HE) had errors on the lesioned side (closed triangle), which fit within the BVD error cluster. There was very little correlation between the NVE values ($R^2 = 0.2$) to the side of the lesion versus those on the intact side, or between NPE values ($R^2 = 0.1$) to each side in UVD patients. In summary, these data support our hypothesis that gaze-position and gaze-velocity errors produced by challenged vestibular systems do indeed fall along the same continuum for normal subject and patient populations.

In Fig. 2B, we show the relationship of the end gaze-position error to the maximum gaze-position error by plotting NEE versus NPE for all subjects, with mean responses from each side plotted with separate symbols. If no reduction in gaze-position error were observed, then the two errors would fall on the equal error line (dotted line). The solid line in Fig. 2B shows the slope if NPE was diminished by 50%. All normal subjects during HA head thrusts were able to reduce gaze-position error by at least 50%. All normal subjects during VHA exhibited 50% or more error, with one subject (MA – VHA, in table) having no observable reduction in gaze-position error on the right side (asterisk on dotted line). The amount of error reduction varied among UVD and BVD populations. Six of seven UVD patients during head thrusts toward their intact side (open triangles) were able to diminish their gaze-position error by 50% or more. Only UVD patient CO remained above the 50% level. Only three of seven UVD patients...
during head thrusts toward their lesioned side (closed triangles) were able to diminish their gaze-position error by 50% or more. As NPE increased, the amount of error reduction required to reduce gaze-position error below the 50% level also increased. BVD subjects (MI (×) and RO (+)) were able to reduce their gaze position by almost 70%. In summary, these results appear to reject our hypothesis that patients with more residual function (e.g., UVD) would exhibit better compensation for gaze-position errors compared to those with less residual function (e.g., BVD). Within the UVD population, some patients with less residual function (i.e., larger NVE) actually have better compensation than those with more residual function. As well, there are BVD patients who have better compensation than several UVD patients.

3.2 Range of UVD responses

The information presented so far show a static summary of selected results in all the subjects tested. From these results, it is difficult to ascertain what is actually happening during the head thrust responses in each subject. These values provide us height and width measures in the phase plane, and gives a basis from which error minimization begins in each subject. Now we will show how these results were achieved, using phase-plane analysis. We explained how the phase plane plots are related to the time response trajectories in the Methods Section 2.4.

In this section we will focus on two UVD patients. One UVD patient (CR (Fig. 2 solid arrows)) who compensated for more than 50% of his gaze-position errors on his lesioned side, and one UVD patient (BR (Fig. 2 dashed arrows)) who compensated less
than 50% of her gaze-position errors on her lesioned side. The responses from the intact and lesioned side of these two UVD patients also represent a large portion of the range of NPE values for all UVD patients (see Fig. 2).

**Better-Compensated UVD Responses**

---{Insert Figure 3 here}---

Fig. 3A shows an example of the time trajectories from a single yaw head thrust toward the intact (left) side of patient CR (a 39 year-old man tested 25 months after resection of an acoustic neuroma on the right side with associated loss of vestibular function in the right labyrinth). Fig. 3B exhibits the same time trajectories from a single yaw head thrust toward the paretic (right) side in the same patient. Note in each response, a corrective movement is produced (highlighted in yellow) that minimizes gaze-position errors. The individual gaze trajectories from all head thrusts were superimposed (and presented in 3C), so the variability within the data could be easily observed across 40-60 responses. Simply averaging the responses from multiple trials might provide an inaccurate representation of the subject’s general behavior, as head input kinematics varied between head thrusts, and the response patterns varied widely from 50 ms after the onset of head motion. The response from the intact side (3A) is, therefore, contained within the trajectories in the left half plane, while the response from the paretic side (3B) is contained within the trajectories in the right half-plane. The mean head dynamics (velocity versus position) are superimposed (thick color-coded trajectories) to show how gaze dynamics differed from head dynamics. In this patient, the individual responses are highly repeatable, showing similar patterns of behavior.
among head thrusts. Later (in Fig. 5) we will discuss some particular dynamics within this set of data. The asymmetry in gaze error in this UVD patient was clear, with less error in the responses on the intact side (quadrant 3) compared to the lesioned side (quadrant 1). In this patient, the minimization of gaze-position error (black arrows in 3C) involves significant velocity components (loops in quadrants 4 and 2). This corresponded to the large accelerations during the highlighted transients in 3A and B. The corrections on the intact side actually occurred later than those on the impaired side, after a long ‘pause’ in gaze velocity (seen in the purple highlight in the time trajectory and the green epoch in the gaze plane). Notice the onsets of the corrections (pluses) are lined up on the abscissa (zero gaze velocity), where many of the responses were reaching peak head position (triangles). The corrections on the lesioned side were, however, triggered well before the head reached peak position. No pauses were observed on the lesioned side. In summary, the relevance of the gaze planes is enhanced when individual responses are viewed simultaneously in one gaze plane and the colors and symbols are assessed together. The superimposed responses reflect the consistency of the responses in this better-compensated UVD patient. Furthermore, quadrants 4 and 2 are particularly important for identifying the distinguishing characteristics of the compensatory response. In this patient, the corrections that appear in these quadrants are very effective in reducing gaze-position error. The patterns within these quadrants will thereby aid in the evaluation of our hypotheses.

Much of the data in this study will be presented using superimposed, multiple gaze-plane presentations. When observing these phase planes, one should note the lengths of each colored band and ascertain the dynamics within each time epoch. In
general, slower movements are more rounded, while faster movements are more vertical. One should note the variability and repeatability among the superimposed trajectories. One should also note the timing of the correction transients - the onsets and offsets are denoted by the plus and square, respectively, on each phase plane trajectory - and how it relates to the time of peak head position (triangles). Finally, one should assess the accuracy of the final gaze position at the end of the 200ms trajectories (asterisks), or at the end of the corrective movement if it occurred later. In this paper, all UVD data will be presented with the responses toward the side of the lesion on the right half of the phase planes.

Less-Compensated UVD Responses

Fig. 4 displays data from the paretic side of UVD patient BR (a 49 year-old woman tested 10 days after resection of an acoustic neuroma with associated loss of vestibular function from the right labyrinth). This patient showed multiple corrections within 200ms of the response. In all the head thrust responses the first corrective eye movement (single arrowhead) resulted only in the reduction of the slope of gaze-position error. In the gaze plane this response corresponded to gaze-position error continuing to increase along the positive x-axis. In this patient, a second corrective eye movement (double arrowhead) was usually produced within 50ms of the first. In Fig. 4A the second correction was triggered as the head velocity was decreasing, thus less eye velocity was required to surpass head velocity, facilitating the reduction of gaze-position error (in the gaze plane the response was brought closer to the origin in the negative x-direction).
Fig. 4B correction 2 was generated while head velocity was increasing, and therefore was only able to reduce the slope of gaze-position error. In this patient, a third correction (triple arrowhead) was usually generated after a ‘pause’ in gaze-velocity activity (when gaze velocity remains at zero for a period of time, marked by the purple highlights). Since this third correction was generated when head velocity was close to zero, the eye velocity of this correction was able to reduce gaze-position error, similar to that of a head fixed saccade. In summary, in this less-compensated UVD patient, multiple GVCs were observed, while GPCs were less prevalent. For those corrections in which eye speed did not exceed head speed, the end effect was a reduction of gaze velocity only, this can be defined as gaze-velocity corrections (GVC). In the gaze plane, the trajectory remains in the originating quadrant (1 or 3). For those corrections in which eye velocity exceeded head velocity, the end effect was a reduction of gaze-position error; this can be defined as gaze-position corrections (GPC). In the gaze plane, these position corrections cause the trajectory to cross into the next quadrant (4 or 2). The gaze plane, therefore, facilitates our ability to distinguish between GVC and GPC, and therefore evaluate preferential reduction of gaze-position and –gaze-velocity errors.

Intra-Subject Variability

Fig. 4 also shows the variability in the pause behavior and its effects on the gaze plane. During the pauses, gaze velocity was sufficiently minimized though gaze-position error was large. From 4A to 4B the responses are presented such that the pauses became shorter and occurred earlier during the head thrust, demonstrating how the effects of GPC emerge in the phase plane. Note that the head dynamics are nearly identical in each
response. In 4A, the pause between the second and third correction caused the third correction (the second GPC of this response) to occur after the 200ms, therefore its effects are not shown in the gaze plane. In 4B, the third correction (and only GPC of this response) was triggered in the pink epoch and the speed was contained entirely in quadrant 4. Because of the variability in timing of the corrections within the 200ms gaze-plane window, the corresponding end gaze-position and end gaze-velocity values (from the table) can present an inaccurate evaluation of performance, as the pause may move large velocity GPC outside of the 200ms window. In 4C, we see that only a few high-velocity GPC occur (in the pink epoch) during all head thrust towards the lesioned side. The changes in the responses of the second corrections and the pause behavior between head thrusts result in the varied patterns in the green epoch. Responses to the intact side showed more accumulation of gaze-position and gaze-velocity error compared to the intact side in patient CR (Fig 3C), and in contrast to patient CR, no large velocity GPCs were observed in quadrant 2. In summary, quadrants 4 and 2 provide the keys for quickly ascertaining whether or not GPCs have been triggered and their effectiveness in reducing gaze-position error within the 200 ms time window of the gaze plane. The effective speed of the GPC (gaze velocity from abscissa to peak) in quadrants 4 and 2 determined the amount of gaze-position correction. Larger velocity trajectories in quadrants 4 and 2 resulted in larger degrees of correction and brought the eye to the target faster. In this less-compensated UVD patient, the GPCs are variable in time and velocity. This gaze-plane technique, therefore, allows us to ascertain the prevalence and dynamics of GPC in each subject.
Fig. 5 gives an example of how two GPC (both correction 2 responses) from two successive head thrusts can have the same gaze velocity, but different effective speeds (gaze velocity from abscissa to peak) and therefore show differences in quadrant 4. This figure demonstrates the normal variability in behavior within the 200ms window of the head thrust. Two individual responses to head thrusts directed toward the side of the lesion are shown in the left (Fig. 5A) and right (Fig. 5B) panels, from the same UVD patient CR whose results were also shown in Fig. 3. In Fig. 5, the head thrust dynamics were nearly identical (dotted lines in gaze planes, head reaches peak position at the triangles) and the NVE values were very similar (0.67 and 0.74), yet the response patterns were different. In both responses, the first correction (single arrows) was triggered around the same time after head onset and was a GVC. The second correction, however, was triggered nearly 40 ms earlier in Fig. 5B (double arrows). In both responses, the second correction became a GPC (once it crossed the abscissa). The GPC generated soon after the onset of peak head velocity (in B) was less accurate (end gaze position at 200ms = 6°) than that generated later (in A) (end gaze position at 200ms = 2°), even though the GPC triggered later had to produce a larger position correction. In the phase plane, the differences in latency are marked by the onset of the GPC (+ sign and double arrow) in the green epoch close to zero gaze velocity in (A), and in the red epoch just after peak gaze velocity was reached in (B). Thus gaze-velocity error was nearly minimized in (A) before the GPC was triggered. Both GPC in (A and B) had similar speeds (~350°/s onset to peak), but because the GPC in (A) had a larger (150°/s more) effective speed (abscissa to peak in quadrant 4), the resulting gaze-position correction
was larger and closer to zero by the end of the GPC (squares in quadrant 4). In (B) a late third correction (triple arrow) was necessary to adequately minimize gaze position. In this case, this was the second GPC that was produced in this response. There was no third correction in (A), because the GPC formed by correction 2 was adequate for minimizing gaze-position error. Notice in (B) that the second GPC began after a 50 ms pause (purple highlight), and was incomplete at end of the pink epoch (the trajectory is moving downwards and away from the abscissa in the gaze plane). Thus, because of the 200ms window, the end gaze position of 6° and end gaze velocity of 100°/s (at 200 ms, asterisk) can present a misleading evaluation of performance, if one only considers these endpoint values.

**In summary,** the responses in the time domain (top panels) show temporal variability in generating the second correction, the pause and the third correction. In the gaze plane, we are able to focus on the effectiveness of the corrections and the dynamical relationships between position and velocity error. From the examples in Figs. 4 and 5, we found that later GPCs allowed GVCs to complete minimization of gaze-velocity error, and later GPCs were usually more accurate in minimizing gaze-position error. Early GPCs (generated in quadrants 1 or 3) did not allow GVCs to be complete, and usually required additional GPCs to completely minimize gaze position.

3.3 Range of BVD responses

------------------------{Insert Figure 6 here}------------------------
In Fig. 6, we present four examples of BVD responses. The top row displays patients whose GVC and GPC were unable to achieve 50% compensation, and the bottom row displays patients who were able to compensate for at least 50% of the gaze-position error. Data in Fig. 6A is from BVD patient RI (56 year-old woman tested 4 months after experiencing symptoms from gentamicin toxicity). Data in Fig. 6B is from BVD patient WU (65 year-old man tested 23 years after experiencing symptoms of bilateral deficit from unknown etiology). Patient WU had larger NPE and NVE values (NPE: 78-79%, NVE: 84-87%) than patient RI (NPE: 60-61%, NVE: 73-80%). The gaze planes in 6A show that RI had very slow (rounded) responses that contained several GVCs, but did not generate any high-velocity GPCs during leftward head thrusts. During rightward head thrusts, a few higher velocity GPCs were generated. The final responses of all the trajectories, therefore, did not end close to the origin. The gaze planes in 6B show that WU triggered several GVCs in the initial quadrants, and waited until the head reached peak position (zero head velocity, triangles) and then triggered a few GPCs during leftward head thrusts. The trajectories ended far from the origin. Neither patient was able to achieve significant gaze position compensation before 200ms.

Data in Fig. 6C display the gaze planes of a BVD patient MI (52 year-old man tested 14 years after experiencing symptoms of bilateral deficit from unknown etiology). Data in Fig. 6D is from BVD patient RO (79 year-old man tested 10.5 years after experiencing symptoms and signs of bilateral vestibular hypofunction thought to be from bilateral vestibular neuritis). From the table and Fig. 2, we find that both MI and RO had larger NPE and NVE values than both RI and WU, yet MI and RO were able to compensate for more than 70% of their gaze-position errors. In 6C, the mean head
dynamics of MI (velocity versus position) are superimposed (thick color-coded trajectories) to show how gaze dynamics differed from head dynamics. The gaze trajectories followed the head (no compensatory eye movement) until the third epoch (green, 100-150 ms), when large GPC were triggered and brought the NEE close to the origin. In the gaze plane of RO (in 5D), the head phase plane is also superimposed to show that the gaze-position and gaze-velocity errors in the initial quadrants are almost all due to the head movement, as implied by the NPE and NVE values for RO. Little or no correction is generated in the initial quadrants. Patient RO generated a high-velocity GPC during every head thrust, once the head reached peak position (triangles). Though triggered late (in the pink epoch) and completed after 200 ms, his GPC were very accurate at the end of each correction (see squares around origin).

Though (in Fig. 2) the NPE, NVE and NEE values for RO (+’s) were very close to those in MI (×’s), it is the gaze planes that help distinguish these two BVD patients. The GPC’s of MI (Fig. 6C) were triggered before the head reached peak position, in the red epoch, exhibiting a sharp drop in gaze velocity in the green epoch. Nearly all of his responses were composed of just one high-velocity GPC. The dynamics of RO’s responses were delayed, with GPC’s generated after the head stopped moving. In summary, the gaze planes provide clear dynamical information that differentiates between responses that may appear similar when only static error values are compared. In less-compensated BVD patients, gaze-velocity errors rather than gaze position errors are reduced. In better-compensated BVD patients, the ability to effectively reduce gaze-position error is reflected in the generation of high-velocity GPC.
3.4 Range of normal responses

We now consider the gaze-plane dynamics of the data within NPE from 0 to 20 percent accounting for the normal subject responses at HA (high acceleration), and NPE from 20 to 30 percent (see asterisks in Fig. 2) for the normal subject responses at VHA (very high acceleration). On a small scale, normal subjects at HA exhibited various types of idiosyncratic behavior, with the gaze plane readily showing asymmetries in head thrust directions as well as various combinations of corrective eye movements. Fig. 7 A and C show two examples of normal responses at HA. In Fig. 2 we see that because NPE is small, little compensation is required. Some subjects did not show any gaze-position error reduction in the time frames examined.

When the normal vestibular system was further challenged, during very high acceleration (VHA) head thrusts, NPE and NVE values rose above normal. When the maximal errors were normalized to the respective head movement, the VHA gain values indicated that the errors had increased by 10-20% from HA head thrusts in the same subjects (see table). In Fig. 2 we see that VHA responses were clustered with the contralesional responses in UVD, rather than the normal HA responses. Responses from two normal subjects at VHA are shown in 7B and D, and compared with responses in the same subjects at HA in 7A and C, respectively. The mean head dynamics are superimposed to compare stimulus kinematics. The gaze-plane responses during VHA head thrusts in normal subjects showed how gaze-position and gaze-velocity errors rose to reach levels observed in the UVD patients for rotation to their intact side (compare
with Figs. 3-5 left half-planes). In the gaze plane for subject MA (7B), the VHA head thrusts triggered a correction immediately after head onset, and this correction produced a constant compensatory velocity signal during the entire head movement. This behavior corresponded to a response matching the head dynamics, but of lower velocity (note low velocity GPC in quadrant 4). This correction was a primarily a GVC. Just prior to the end of the head movement (triangles), eye velocity exceeded head velocity, causing gaze position to be minimized. Gaze velocity was minimized by 100 ms after head onset, while gaze-position error was never completely minimized, indicating a limit of the capability of this correction as a GPC. In this subject, GPCs were triggered after the 200ms gaze-plane window. This subject was unable to reach the 50% compensation level within the 200ms time window (see Fig. 2). This is apparent in the gaze plane, as there are no response dynamics ending at the origin.

In the gaze plane for subject GG (7D), VHA head thrusts triggered a similar correction (to that observed in MA) immediately after head onset. After gaze-velocity error was minimized, a second correction was generated as a small GPC in the green epoch (100-150ms). Then in the final epoch (pink, 150-200ms), large GPCs were triggered to minimize gaze-position errors to levels observed at HA. This subject triggered high-velocity GPC to reduce the gaze-position error by 200ms. The timing pattern in GG resembled that in UVD patient CR during head thrusts toward his intact side. Those responses consistently produced a pause between 100-150ms (see Fig. 3A and 3C). High-velocity GPCs were triggered in the last (pink, 150-200ms) epoch and in many instances after the head had stopped moving (triangles), later than those triggered on the lesioned side. Nevertheless, the end gaze positions on the intact side were more
accurate (0.3° ± 1.8°) at the end of the 200ms trajectories, than those on the lesioned side (5.8° ± 2.9°) (see table). The data in Fig. 2 indicate that of all the VHA responses, only the rightward head thrusts in GG (7D) were able to compensate 50% of the gaze-position error.

In summary, the data from normal subjects show that the gaze errors increase as the amount of challenge is increased. The gaze errors exhibited by normal subjects at VHA are contained within the same range as those produced by UVD patients during head thrusts to their intact side. The gaze planes indicate that the strategy for compensation is also comparable to those observed in all patients. As in patients, in ‘less-compensated’ normal subjects, gaze-velocity error is preferentially reduced, while ‘better-compensated’ normal subjects develop higher velocity GPC that reduce gaze-position error. On the other hand, in patients we did not find that residual vestibular function is the primary contributor to overall compensation, when the challenge is high. Both normal subjects at VHA and patients (UVD and BVD) rely on GPC once compensation is achieved.

4 Discussion

Studies of gaze stabilization during head motion have traditionally focused on eye movement behavior examined at fixed points in time, or at fixed frequencies such that time becomes invariant. The performance of the system under controlled, repeatable conditions can be characterized by constraining the time parameter. Likewise, quantifiable measures of physiological performance under these conditions can be
compared easily between healthy subjects and patients using the same experimental protocol. Unless the experiments and analysis are all conducted under the same conditions, measures of physiological performance will differ from study to study. Furthermore, performance measured during idealized conditions does not necessarily reflect natural behavior. These issues are particularly germane to studies of patients with vestibular deficits that vary with time after lesion. In these patients, the nominal operating range of the vestibular system depends considerably on stimulus properties, because the variable pathology introduces nonlinearities (from non-constant parameters) in the system. Yet, a need exists to characterize the performance of these patients over time and during time-varying stimuli that emulate natural behavior.

In this study we developed a framework of analysis to characterize the dynamical properties of gaze stabilization during head thrusts over a large range of accelerations, in various subject populations. Through our gaze-plane analysis we were able to delineate distinguishing patterns of performance that persisted despite variation in the stimulus from trial to trial. The gaze planes provide critical dynamical information that differentiates between responses that may otherwise appear similar when only static error values are compared. In the gaze plane, we are able to focus on the effectiveness of the corrections and the dynamical relationships between position and velocity error, despite temporal variability. Our goal to characterize the dynamic function of the vestibular system over the duration of a step stimulus resulted in a multi-level (corrections, GVC and GPC outcomes, compensatory patterns), multi-variable analysis of gaze. Through this process we found the main factors to be: the number of corrective eye movements, the outcomes of the corrections (gaze-velocity correction, GVC and gaze-position
correction, GPC), the amplitude and timing of the corrections, and the pause behavior within the corrective sequence.

4.1 Gaze error continuum

We found that gaze-position and gaze-velocity errors are linearly related, and this linear relationship increased along a continuum from normal subjects to normal subjects challenged by unnatural stimuli to patients challenged by various degrees of physiological deficit. Gaze position and gaze velocity first increased from normal response levels when a novel head stimulus not experienced naturally was introduced to normal subjects. These very high acceleration (VHA) responses in normal subjects overlapped with those from the contralesional side of patients with UVD. Progressing along the continuum, gaze-position error and gaze-velocity errors increased to levels observed from the lesioned side of patients with UVD, then to the maximal levels observed from both sides of patients with BVD. The amplitude of error (the dynamics in the initial quadrants) increased in a linear manner in both gaze position and gaze velocity.

The similarity in errors between the contralesional responses in UVD and in normal responses at VHA suggests that the head stimuli had exceeded the limits in which the contralateral labyrinth normally functions. In the UVD patients, no vestibular contribution is expected from the contralateral labyrinth during contralesional rotations. In the normal subjects at VHA, the contralateral labyrinth is experiencing ampullofugal (inhibitory) stimulation. We know from Ewald’s second law that ampullopetal (excitatory) stimulation of the lateral canal has a larger firing range than that during ampullofugal (inhibitory) stimulation (Ewald 1892). It is likely that at VHA stimuli, the
inhibitory afferents and/or central vestibular neurons have reached both velocity and acceleration cutoff, therefore, contributing very little to the compensatory response.

In Fig. 2, we find that maximal gaze-velocity error is consistently larger (~15%) than maximal gaze-position error in all subjects when normalized to the respective head kinematics. In other words, the percentage of gaze error relative to head velocity is always higher than the percentage of gaze error relative to head position. If one considers gaze error minimization as a control problem, it would be more difficult to control gaze velocity than it would be to control gaze position. Gaze position can be controlled by current gaze-position and gaze-velocity values. Utilizing gaze velocity, or the slope of gaze position, the brain will be able anticipate the direction of the gaze position and minimize it. Future values for gaze velocity, however, could be predicted with gaze acceleration, but this parameter, being inherently more noisy and variable, may be more difficult for the brain to process with fidelity. Therefore, minimizing gaze velocity relies mainly on current values of gaze velocity and the functioning of the VOR.

Contralesional responses on average were 20% higher in both maximal gaze-velocity and gaze-position errors, compared to normal subjects. This relative increase in gaze-velocity error was comparable to the decrease in velocity gain found previously during yaw head thrusts in normal subjects to patients with UVD (contralesional side), 24% at 100ms after head onset (Aw et al. 1996a, b). Tabak and colleagues (1997a, b) found smaller decreases, 12-13% with manual head thrusts and 3-4% with helmet head thrusts, while Tian and colleagues (2000) found no changes between normal and contralesional responses. The absolute measures from all of the above studies were different. Two of our patients with UVD had contralesional gaze-velocity and gaze-
position errors within the upper range of the normal subject errors at high acceleration (HA) (see Fig. 2). These patients (WI and CR) are among the younger patients we studied, and they were tested more than 10 months after resection of an acoustic neuroma (see Table). These observations point out that multiple factors such as age, time after the lesion, and rate at which the deficit was acquired likely influence the degree to which responses are abnormal.

BVD responses on average were 20% higher in both maximal gaze-velocity and gaze-position errors, compared to UVD patients. This relative increase in gaze-velocity error was comparable to the decrease in velocity gain found previously during yaw head thrusts in patients from UVD (lesioned side) to BVD, 22% with manual head thrusts and 26% with helmet head thrusts (Tabak et al. 1997b). Again the absolute measures were very different among studies.

Our gaze planes readily differentiated the pattern of error dynamics between our subject populations. Set to the same error scales, contralesional UVD dynamics are clearly distinguishable from normal subject dynamics at HA. The asymmetry in the UVD responses and the symmetry of the BVD responses can be easily evaluated from the right and left sides of the gaze plane.

4.2 Compensation in the gaze plane

We found that the gaze-plane analysis revealed aspects of gaze error compensation not readily observed with traditional gain calculations. From our results, we hypothesize that the ultimate function of the vestibular system is to minimize gaze-velocity errors (retinal slip) and gaze-position errors (foveal slip) simultaneously, with a
minimal number of corrective eye movements (one GPC of the correct amplitude).

Success in achieving this goal strongly depends on the functional capabilities of the
system, the properties of the stimulus imposed on the system and the amount of exposure
each system has with those particular stimulus properties. Once this generalized
perspective was taken, we realized that the gaze planes were reflecting the same
compensation patterns for all subjects, healthy and those with lesions.

When the imposed stimulus falls within the normal operating range of the system,
small amounts of gaze-velocity and gaze-position errors are minimized with one GPC
triggered in quadrant 1. Once the limits of the system are newly exceeded by the
stimulus properties, the response deviates from this ‘normal state’. The system attempts
to achieve gaze stabilization through *multiple* corrections, *sequentially* minimizing gaze
velocity first, then gaze position. This can be exhibited by eye movement patterns that do
not converge to a single pattern during successive head thrusts. The system is in a state
of calibration, or ‘calibration state’. To return to the normal state or go to a ‘new normal
state’, the challenge needs to be reduced so that the stimulus properties fit within the
functional limits of the system (i.e. reducing the head stimulus or expanding the system
limits through compensation or both). Once the challenge is reduced, the system again
achieves gaze stabilization through *minimal* corrections, *simultaneously* minimizing both
gaze-velocity and gaze-position error.

The ‘calibration state’ was observed in the less-compensated subjects – the
normal subjects experiencing the novel VHA stimuli, and in patients who have recently
suffered their lesion, or began experiencing symptoms. These patients were not able to
compensate for at least 50% of their NPE. In these data, the gaze planes differed widely
among individual head thrusts. In each response multiple corrections were generated with various combinations of GVC and GPC, and the pause behavior tended to become more apparent. Few high-velocity GPCs were observed in these subjects before the head reached peak position, and generally were produced only after a prolonged pause. The appearance of the “pause” in the gaze plane may indicate that the first goal of that system is to stabilize gaze velocity. Once retinal slip is minimized, the secondary goal is to minimize gaze-position error. Alternatively, it may be that the functional limitations of the system only allow GPCs to be generated later, or until more compensation occurs allowing earlier, more automatic programming of GPC.

In comparing UVD patients with BVD patients, patients with UVD in the calibration state tended to have more corrections in the initial quadrant (GVC), more types of gaze-plane patterns overall, and their responses tended to be completed more quickly than patients with BVD in the calibration state. BVD patients also exhibited one type of pattern that was not observed in UVD patients, in which little or no GPCs were generated before the head reached peak head position, indicating a possible reliance on visual feedback and the generation of saccades after the head stopped moving. These differences between UVD and BVD gaze-plane patterns may be attributed to whether or not head movement information (from the contralesional side in UVD patients) is available and accurate enough to calibrate the compensatory response appropriately during the head thrust.

The ‘normal state’ or the ‘new normal state’ was observed in the better-compensated subjects - the normal subjects at HA stimuli, and in chronic patients who have properly calibrated their systems. These subjects were able to compensate for at
least 50% of their NPE. The surprising finding is the similarity in gaze planes between better-compensated UVD (lesioned side) and BVD patients. We expected from our original hypotheses that UVD patients (having residual vestibular function) would exhibit better compensation than BVD patients. In better-compensated patients from both groups, however, high-velocity GPCs were equally observed before the head reached peak head position (triangles), and completed the corrections within 200ms. In some patients successive small GPCs were triggered to correct for the gaze-position error in a step-wise manner. One of these patients also exhibited hypometric responses during saccades to head-fixed targets suggesting some common neural processing for controlling saccade amplitude for visually and vestibularly triggered corrective saccades. In the cases where the NEE values from the lesioned sides were similar between UVD and BVD patients (e.g. Fig. 2 arrows and ×’s), this usually indicated that the BVD patient compensated more (in this case 70% compensation) than the UVD patient (in this case 30% compensation), because the NPE started at higher levels. This implies that even with little or no head velocity information from the vestibular system, the system is still capable of predicting gaze-position error. Certainly in our paradigm there is likely information about head motion, even in the BVD patients, either from residual labyrinthine function (though we do not know how faithful it could transduce high-acceleration, high-frequency stimuli) or from neck proprioception. In either case, just the detection of head motion – without any specific information about its speed or acceleration – might still be used by the brain to generate a compensatory response based on its ‘best guess’ of what the head might be doing.
4.3 Adaptation

Our data suggest that a ‘new normal state’ can be achieved, even when gaze-position and -velocity errors are initially at higher levels, by reducing the challenge. One BVD patient who was tested twice actually produced higher-velocity GPC in quadrants 4 and 2 after one year. This patient may have promoted more head movements in his daily activities, allowing his system to be challenged across a wide range of head stimuli, developing more reliable and consistent patterns of corrective eye movements for the purpose of gaze stabilization at those stimuli. We thereby propose that repeated exposure to challenge trains the system to adapt to a novel stimulus. This premise is not new, but falls under the basic definition of vestibular adaptation. In classical adaptation experiments, the system is trained by unusual stimuli to achieve a new performance state (altered gain, phase, direction, etc.). In our study, patients are trained by challenging stimuli to achieve a new normal state – the fundamental performance criterion for the system. Bloomberg et al. (1991) found that normal subjects, who were recovering from adaptation to visual VOR suppression, exhibited varied combinations of slow phase and saccadic eye movements. Long-term exposure to challenge has been shown to convert these varied responses into ones that are patterned. Melvill Jones et al. (1988) documented how subjects exposed to optically reversed vision adaptively modified their ocular motor strategy within six hours. In this complex task, the eye movements were initially variable containing large saccades. By the end of the six hour period, head braking studies produced consistent responses, reflecting an attenuated VOR and smooth eye movements that had taken the place of saccades. Melvill Jones et al. (1988) suggest that novel behavioral demands increase the potential for adaptive modification, drawing
new movements from the available repertoire of sensorimotor functions. Istl-Lenz et al. (1985) also showed alterations in adaptive strategies in normal subjects during 2× (magnifying glasses) vestibular adaptation. They found that early on, saccades supplemented inadequate VOR slow-phases but became progressively less necessary as slow-phase gain increased with adaptation. This decrease in the number of corrections closely reflects what we observed in our data between compensated and less compensated subjects.

4.4 Outcomes of the corrective movements

We found that reducing one of the errors did not imply that the other error would be reduced as well. In the analysis of individual time responses, gaze-position errors are reduced only when eye (relative to head) speed exceeded head speed. Success in achieving this condition depended on the time at which the correction was triggered relative to the head velocity profile. Corrections triggered around peak head velocity required more eye velocity than those triggered on the rising or falling phases of peak head velocity, to reduce gaze-position error by the same amount. In other words, two corrections with the same amount of eye velocity could result in only a generation of a GVC or varying amplitudes of the GPC, depending on what the head was doing at the time of the trigger. This implies that if the VOR slow phase response was suppressed during a GPC, the total amount of gaze-position error compensation achieved during the correction would be attenuated. Indeed there is evidence that the VOR is at least partially inhibited during gaze saccades in the direction opposite of the head movement in humans (e.g., Tabak et al 1996, Roy and Cullen 1998). The triggering of the corrections was not
rigidly related to the kinematics of the head stimuli, as two head thrusts of identical
kine matics could result in two different corrective movement patterns. We also
determined that the variability in gaze performance at the end point in time (200ms,
asterisks on the gaze planes) is due to the variable onset of the last GPC. The amount of
error at this arbitrary end point is therefore not related to the maximum amount of gaze-
position and gaze-velocity error during the head thrust, and therefore not directly related
to the level of slow-phase contribution in the subject (Fig. 2B).

Because the abscissa defines zero gaze velocity, GVCs are only triggered in
quadrant 1 (and 3 for leftward head thrusts). A GVC triggered in quadrant 1, therefore,
will be completed in quadrant 1 (exhibiting a reduction in gaze velocity only), while a
GPC triggered in quadrant 1 will end in quadrant 4 closer to the origin (exhibiting both a
reduction in gaze-position and gaze-velocity error). A GPC triggered in quadrant 4 is
generated to reduce gaze-position error. From our study we found that classical
characteristics of the responses to head thrusts defined by slow phase and corrective
saccades describe a small minority of the population, and most responses present a
complex mixture of the two properties.

The corrective eye movements form a repertoire of responses, of which only a
subset can be defined as saccades. GPCs may resemble saccades in that they correct for
gaze-position errors, however, GPCs generated early also act to minimize gaze-velocity
errors quickly. Studies have shown that saccades are generated during a head step to
make up the gaze-position error created by deficient vestibular slow phases (Segal and
Katsarkas 1988a,b; Bloomberg et al. 1991; Tian et al. 2000). In these studies the
investigators identified saccadic eye movements using selection criteria based on typical
saccade profiles and compared saccade and slow phase contributions to minimizing gaze-position error. In our study we showed that such synergistic behavior exists in minimizing both gaze-position and gaze-velocity error. Throughout the duration of the head thrust, corrections were generated for gaze-position and gaze-velocity error that was uncompensated by preceding corrections, ultimately bringing the gaze-plane trajectories back to the origin. We found, however, that the order of the corrections and therefore the outcomes depended on the amount of compensation and perhaps the amount of exposure to the challenge (see section 4.3 - adaptation). In the Tian et al. (2000) study, the vestibular slow phase and only the first saccadic correction were taken into account in terms of gain, so the overall dynamic response was not evaluated. In the Segal and Katsarkas (1988a and b) and Bloomberg et al. (1991) studies, cumulative saccadic contributions were subtracted from the final eye position to estimate slow-phase contribution, so the overall data was taken into account, but not in relation to time. In the Kasai and Zee (1978) study, other types of compensatory eye movements were observed in patients with vestibular deficits. Eye movements such as preprogrammed compensatory slow phases and slow phases driven by the cervico-ocular reflex could also account for some component of the GVCs we observed.

4.5 Summary and Implications

We have shown that the during head motion gaze-position and gaze-velocity errors increase linearly along a continuum as we have hypothesized, depending upon the degree of challenge, and this applies to both patients with compromised vestibular function and to normal subjects who are challenged by unnatural patterns of head motion.
The gaze-plane analysis allowed us to discern patterns of velocity and position correcting movements not easily observed with traditional gain measurements, and relate them to the degree of vestibular loss and compensation. As we have hypothesized, phase-plane analysis clearly shows strategies, which preferentially reduce gaze-position or gaze-velocity errors. Contrary to our hypothesis, patients with more residual vestibular function did not exhibit better compensation for gaze-position errors than those with less residual function. Both groups could achieve better compensation with the generation of high-velocity gaze-position corrections (GPC). As we have hypothesized, patients with BVD relied more on saccade-like GPC. With the framework from this study, and especially the gaze-plane analysis, we can now expand the nature of our questions and explore the dynamics within the gradients of vestibular challenge. How does residual vestibular function contribute to the establishment of a new adapted state? What is the minimum amount of exposure (to challenge) required to cause a response to be less than compensatory, and how much exposure to challenge induces training and adaptation? Would training at a challenged stimulus create more compensated patterns? How much exposure to challenge causes the pause to disappear? How much does age affect gaze performance within the challenge gradients?

The multivariate nature of the gaze stabilization requires a careful mapping of the critical characteristics of eye movement sequence, outcome, amplitude and timing, within the continuum. The challenges within the continuum reflect the functional limits of the system, the properties of the stimulus and the amount of exposure to the stimulus. The gaze-plane framework can be ideally utilized to assess patient performance and begin to build relationships between physiological function and functional rehabilitation.
Acknowledgements

We wish to thank Mark Shelhamer for critically reviewing an earlier draft of this manuscript, and to Adrian Lasker and Dale Roberts for technical assistance with the experiments and equipment. This work was supported by NIH Grants DC00979 and DC05040, and by the Clare Boothe Luce Foundation.
References


Ewald J.R. *Physiologische Untersuchungen uber das Endorgan des Nervus Octavus*. Bergmann, Wiesbaden, Germany, 1892


Figure Legends

**Figure 1.** Comparison of plots of time versus position and versus velocity, and phase-plane trajectories, normal and abnormal examples. (A) Normal response and (B) UVD response. The eye-in-head, or eye, trajectories are inverted to allow comparison to the head-in-space, or head, movement. The eye-in-space, or gaze, trajectories (thick solid lines) depict the error during the head thrust. With a normal VOR, gaze should not change during head rotation. Static error measures listed in the Table are: Normalized peak Position Error (NPE), Normalized peak Velocity Error (NVE) and Normalized End position Error (NEE), where A = peak gaze position, B = peak head position, C = head position at 200ms, D = gaze position at 200ms (or end of correction), E = peak gaze velocity, F = peak head velocity. The time epochs (under the top panels) from the start of each trajectory are coded in color: 0-50ms (blue), 50-100ms (red), 100-150 (green), and 150-200ms (magenta). The start of each phase-plane trajectory is marked by a circle and the end (at 200ms) by an asterisk and gray bar. Triangles mark the time of peak head position (zero head velocity). Each GPC (gaze-position correction) is marked with a plus at onset and a square at offset. Since only responses to rightward head thrusts are presented, in the phase plane this corresponds to gaze trajectories occupying the right half of the plot - beginning at the origin, moving through quadrant 1 then quadrant 4 and heading back towards the origin. The normal and UVD response patterns have the same shape though the amount of error is about 10 fold greater in the patient.

**Figure 2.** Gaze error relationships. (A) NVE (Normalized peak gaze Velocity Error) versus NPE (Normalized peak gaze Position Error) for all subjects. UVD patient responses (triangles) are shown for head thrusts directed towards the side of the lesion (solid triangles), and for head thrusts directed towards the intact side (open triangle). Solid arrows highlight UVD patient CR responses, and dotted arrows highlight UVD patient BR responses (double triangles, solid (lesion-side) and open (intact side)). BVD patient responses (squares) are shown for rightward head thrusts (solid squares) and leftward head thrusts (open squares). BVD patient MI (×, highlighted (rightward) and plain (leftward)) and patient RO (+, highlighted (rightward) and plain (leftward)) responses are shown separately. Normal subject responses (circles) are shown for rightward head thrusts (solid circles) and leftward head thrusts (open circles). The
normal responses from VHA head thrusts (asterisks, highlighted (rightward) and plain (leftward)) are shown separately. The dotted line denotes equal NVE and NPE; the solid line denotes 15% greater NVE than NPE. Note there is a continuum of responses from normal subjects performing relatively well to BVD patients performing quite poorly. (B) NEE (Normalized End gaze position Error) versus NPE (Normalized peak gaze Position Error) for all subjects. Symbols are defined as those in (A). The dotted line denotes no change in NPE; the solid line denotes a 50% reduction in NPE. Note the lack of a consistent relationship between the severity of deficit as reflected in velocity error, NVE in (A), and the ability to compensate as reflected in reduction of end position error, NEE in (B).

**Figure 3. UVD Responses, better-compensated.** Position, velocity and acceleration time response trajectories in unilateral patient CR, during a single, yaw head thrust towards his intact side (A), and a single, yaw head thrust towards his side of the lesion (B). Multiple gaze responses from the same patient are displayed in (C). Right eye responses are displayed. In (A) there is a momentary increase in gaze error, around 40 ms after head onset. The effect is more prominent in the velocity and acceleration plots. In (B) this momentary increase in gaze error occurs around 20 ms after head onset. The lightly shaded bars in (A) and (B) highlight the occurrence of a transient gaze-position correction in each response. This gaze-position correcting movement, or GPC, brings the image of the fixation light back toward the fovea. The purple bar in (A) highlights a ‘pause’ in gaze-velocity activity (constant gaze velocity). (C) All of the head thrust data from the same subject are plotted in the form of multiple gaze-plane plots of gaze velocity versus gaze position. Responses to head thrusts toward the side of the lesion are on the right half-plane and responses to head thrusts toward the intact side on the left half-plane. Mean head thrust dynamics are superimposed in the thick curves (intact side: 264 ± 35°/s, 7371 ± 1257°/s² and lesion side: 272 ± 39°/s, 6454 ± 1456°/s²). The right halves of each plot describe responses to rightward head thrusts (side of lesion) from quadrant 1 to 4, and the left halves describe responses to leftward head thrusts (intact side) from quadrant 3 to 2. The arrows in (C) correspond to the amount of gaze-position correction for leftward (dashed arrow) and rightward (solid arrow) head thrusts. The pause in (A) is represented by the gaze activity in the green epoch, in the left half-plane of (C). The patterns of response in this patient (prominent and effective gaze-position correction) reflect relatively good compensation.
**Figure 4. UVD Responses, less-compensated.** (A, B) Individual position and velocity time trajectories and the corresponding phase planes from unilateral patient BR, during yaw head thrusts toward the side of the lesion. Left eye responses are displayed. Only the pause intervals are highlighted. (C) total gaze-plane responses during yaw head thrusts towards the lesioned and intact side for patient BR. The other notations are as in Fig. 1. In the gaze planes, the plus and square denote the start and end of the second correction. The phase plane of the head (dotted lines) show that the dynamics are nearly identical in (A) and (B). In this patient, large variability existed among individual head thrust responses, exhibiting multiple GVC, GPC, and pause patterns. Compared with the better-compensated UVD response in Fig. 3, responses to the lesioned side are slower and minimize gaze-velocity, rather than gaze-position error. Hence there is a considerable residual position error 200ms into the head movement.

**Figure 5. UVD Responses, GPC effects.** (A) The same time trajectories from Fig. 3B and the corresponding phase planes below, (B) another set of time trajectories during a single head thrust towards side of the lesion in patient CR and the corresponding phase plane plots below. Right eye responses are displayed. The other notations are as in Fig. 1. In the gaze planes, the plus and square denote the start and end of the second correction. The phase plane of the head (dotted lines) show that the dynamics are nearly identical in (A) and (B). The purple shaded box in (B) highlights a 50ms pause in gaze-position activity (zero gaze velocity) between the first (single-headed arrow) and third (triple-headed arrow) correction. The later onset of the second (double-headed arrow) correction in (A) results in more accurate and earlier reduction in gaze position compared to (B). The earlier onset of the second correction in (B) results in earlier reduction in gaze velocity (at the end of the red epoch, 100ms) compared to (A). For (A), NVE = 0.67, NPE = 0.59, NEE = 0.13; for (B), NVE = 0.74, NPE = 0.48, NEE = 0.27. These plots show that relatively later-occurring gaze-position corrections are a usually more effective strategy at reducing gaze error 200 ms after the onset of the head rotation.

**Figure 6. BVD Responses.** Total gaze planes across NPE gradient for BVD patients. Gaze-plane trajectories of multiples head thrust trials. The axis scaling, colored time epochs, and symbols are as in Fig.
1. Data are from (A) BVD patient RI, (B) BVD patient WU, (C) BVD patient MI, and (D) BVD patient RO. Responses to rightward and leftward head thrusts are shown in the respective half-planes. In (C) and (D) mean head thrust dynamics are superimposed in the thick curves (in (C): rightward thrusts: $161 \pm 45^\circ/s$, $4306 \pm 1603^\circ/s^2$ and leftward thrusts: $182 \pm 29^\circ/s$, $4889 \pm 1024^\circ/s^2$; in (D): rightward thrusts: $148.45 \pm 37.97^\circ/s$, $4163.21 \pm 1514.33^\circ/s^2$ and leftward thrusts: $157.18 \pm 16.29^\circ/s$, $4090.75 \pm 638.42^\circ/s^2$).

Top row represents dynamics that resulted in less than 50% gaze-position error compensation, and the bottom row represents dynamics that resulted in more than 50% gaze-position error compensation. These plots show that in less-compensated BVD patients (e.g., BVD patient WU, panel B), gaze velocity errors rather than gaze position error are reduced while in better-compensated BVD patients (e.g., BVD patient MI, panel C), high-velocity, gaze position correction improve performance relatively early during the head movement. These results follow the same gaze-plane patterns for less-compensated and better-compensated UVD patients, respectively.

**Figure 7. Challenged normal subjects.** The same plot types, axis scaling, colored time epochs, and symbols used in Fig. 1 are used here. (A) Normal subject MA during HA (high acceleration) head thrusts, (B) Normal subject MA during VHA (very high acceleration) head thrusts, (C) Normal subject GG during HA head thrusts, (D) Normal subject GG during VHA head thrusts. Vestibular challenge is presented to normal subjects in the form of VHA head thrusts. These responses in normal subjects to very high accelerations exhibit gaze-plane patterns similar to those of patients at much lower speeds and accelerations, in both error accumulation and error compensation.
Table. Gaze correction characteristics in UVD, BVD and normal subjects during HA head thrusts.

<table>
<thead>
<tr>
<th>Test status</th>
<th>NVE</th>
<th>NPE</th>
<th>NEE</th>
<th>end gaze position (°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UVD</td>
<td>lesion</td>
<td>intact</td>
<td>lesion</td>
<td>intact</td>
</tr>
<tr>
<td>Group MEAN</td>
<td>0.73</td>
<td>0.44</td>
<td>0.56</td>
<td>0.29</td>
</tr>
<tr>
<td>BR (an)</td>
<td>±0.12</td>
<td>±0.16</td>
<td>±0.14</td>
<td>±0.10</td>
</tr>
<tr>
<td>49 F 10 days</td>
<td>±0.12</td>
<td>±0.17</td>
<td>±0.08</td>
<td>±0.06</td>
</tr>
<tr>
<td>EN (an)</td>
<td>0.73</td>
<td>0.63</td>
<td>0.57</td>
<td>0.46</td>
</tr>
<tr>
<td>39 F 37 days</td>
<td>±0.10</td>
<td>±0.15</td>
<td>±0.06</td>
<td>±0.08</td>
</tr>
<tr>
<td>ST (an)</td>
<td>0.56</td>
<td>0.49</td>
<td>0.41</td>
<td>0.28</td>
</tr>
<tr>
<td>67 F 7 months</td>
<td>±0.09</td>
<td>±0.14</td>
<td>±0.08</td>
<td>±0.07</td>
</tr>
<tr>
<td>WI (an)</td>
<td>0.70</td>
<td>0.25</td>
<td>0.57</td>
<td>0.15</td>
</tr>
<tr>
<td>30 F 10 months</td>
<td>±0.04</td>
<td>±0.13</td>
<td>±0.10</td>
<td>±0.08</td>
</tr>
<tr>
<td>HE (md)</td>
<td>0.97</td>
<td>0.65</td>
<td>0.86</td>
<td>0.36</td>
</tr>
<tr>
<td>67 F 18 months</td>
<td>±0.14</td>
<td>±0.10</td>
<td>±0.09</td>
<td>±0.09</td>
</tr>
<tr>
<td>CR (an)</td>
<td>0.71</td>
<td>0.29</td>
<td>0.52</td>
<td>0.18</td>
</tr>
<tr>
<td>39 M 25 months</td>
<td>±0.06</td>
<td>±0.04</td>
<td>±0.07</td>
<td>±0.04</td>
</tr>
<tr>
<td>CO (md)</td>
<td>0.73</td>
<td>0.39</td>
<td>0.53</td>
<td>0.27</td>
</tr>
<tr>
<td>37 F 37 months</td>
<td>±0.09</td>
<td>±0.06</td>
<td>±0.07</td>
<td>±0.04</td>
</tr>
<tr>
<td>BVD</td>
<td>right</td>
<td>left</td>
<td>right</td>
<td>left</td>
</tr>
<tr>
<td>Group MEAN</td>
<td>0.91</td>
<td>0.92</td>
<td>0.76</td>
<td>0.76</td>
</tr>
<tr>
<td>RI (gent)</td>
<td>0.80</td>
<td>0.73</td>
<td>0.60</td>
<td>0.61</td>
</tr>
<tr>
<td>56 F. 4 months</td>
<td>±0.09</td>
<td>±0.14</td>
<td>±0.60</td>
<td>±0.45</td>
</tr>
<tr>
<td>LO (?)</td>
<td>0.93</td>
<td>0.87</td>
<td>0.59</td>
<td>0.66</td>
</tr>
<tr>
<td>20 M. 9 months</td>
<td>±0.07</td>
<td>±0.06</td>
<td>±0.09</td>
<td>±0.06</td>
</tr>
<tr>
<td>WH (gent)</td>
<td>0.85</td>
<td>0.83</td>
<td>0.81</td>
<td>0.74</td>
</tr>
<tr>
<td>85 M 18 months</td>
<td>±0.08</td>
<td>±0.07</td>
<td>±0.08</td>
<td>±0.12</td>
</tr>
<tr>
<td>LO2 (?)</td>
<td>0.83</td>
<td>0.90</td>
<td>0.68</td>
<td>0.60</td>
</tr>
<tr>
<td>21 M. 21 months</td>
<td>±0.05</td>
<td>±0.03</td>
<td>±0.14</td>
<td>±0.10</td>
</tr>
<tr>
<td>GR (gent)</td>
<td>1.00</td>
<td>1.06</td>
<td>0.80</td>
<td>0.79</td>
</tr>
<tr>
<td>46 F. 45 months</td>
<td>±0.04</td>
<td>±0.05</td>
<td>±0.13</td>
<td>±0.20</td>
</tr>
<tr>
<td>RO (vn)</td>
<td>1.01</td>
<td>1.09</td>
<td>0.93</td>
<td>0.95</td>
</tr>
<tr>
<td>79 M. 10yr, 6mths</td>
<td>±0.04</td>
<td>±0.04</td>
<td>±0.04</td>
<td>±0.09</td>
</tr>
<tr>
<td>MI (?)</td>
<td>0.98</td>
<td>1.01</td>
<td>0.89</td>
<td>0.92</td>
</tr>
<tr>
<td>52 M. 14 years</td>
<td>±0.03</td>
<td>±0.02</td>
<td>±0.09</td>
<td>±0.07</td>
</tr>
<tr>
<td>WU (?)</td>
<td>0.87</td>
<td>0.84</td>
<td>0.78</td>
<td>0.79</td>
</tr>
<tr>
<td>65 M. 23 years</td>
<td>±0.09</td>
<td>±0.02</td>
<td>±0.10</td>
<td>±0.10</td>
</tr>
<tr>
<td>Normal</td>
<td>right</td>
<td>left</td>
<td>right</td>
<td>left</td>
</tr>
<tr>
<td>Group MEAN</td>
<td>0.19</td>
<td>0.23</td>
<td>0.10</td>
<td>0.09</td>
</tr>
<tr>
<td>10 subjects (HA)</td>
<td>±0.06</td>
<td>±0.10</td>
<td>±0.04</td>
<td>±0.05</td>
</tr>
<tr>
<td>MA (HA)</td>
<td>0.20</td>
<td>0.15</td>
<td>0.10</td>
<td>0.07</td>
</tr>
<tr>
<td>38 M</td>
<td>±0.11</td>
<td>±0.11</td>
<td>±0.04</td>
<td>±0.02</td>
</tr>
<tr>
<td>MA (VHA)</td>
<td>0.32</td>
<td>0.37</td>
<td>0.31</td>
<td>0.24</td>
</tr>
<tr>
<td>39 M</td>
<td>±0.05</td>
<td>±0.05</td>
<td>±0.05</td>
<td>±0.04</td>
</tr>
<tr>
<td>GG (HA)</td>
<td>0.14</td>
<td>0.14</td>
<td>0.09</td>
<td>0.09</td>
</tr>
<tr>
<td>32 F</td>
<td>±0.03</td>
<td>±0.03</td>
<td>±0.02</td>
<td>±0.02</td>
</tr>
<tr>
<td>GG (VHA)</td>
<td>0.31</td>
<td>0.34</td>
<td>0.23</td>
<td>0.28</td>
</tr>
<tr>
<td>32 F</td>
<td>±0.06</td>
<td>±0.05</td>
<td>±0.06</td>
<td>±0.04</td>
</tr>
</tbody>
</table>
**Table Legend.** The first column from the left defines the population, the subjects included in the calculations, their age and gender. In the second column, the cause of vestibular deficit is shown in parentheses ((an) = surgery for acoustic neuroma, (md) = surgery for Meniere’s disease, (gent) = gentamicin toxicity, (vn) = bilateral vestibular neuritis, (?) = unknown etiology). The time after loss, or experiencing symptoms is noted below the cause. In the next four columns, the mean of all the parameters calculated from each the head thrust within each trial is noted (in bold), and the standard deviation is noted below the mean. For each population, the group mean from all the subjects is shown in the first row (shaded), before the individual subject listings. Individual data for normals are only shown for those that participated in the VHA studies. Four characteristic parameters are displayed. Within each parameter, the data are divided between responses toward the lesion side and intact side for UVD subjects, and between right and left side for BVD and normal subjects. Normalized gaze-velocity error (NVE) is the value of peak gaze velocity divided by the value of peak head velocity. Normalized gaze-position error (NPE) is the value of peak gaze position divided by the value of peak head position for each head thrust. Normalized end gaze error (NEE) is the value of gaze position at the end (either at 200ms after head onset, or at the end of a GPC that is incomplete at 200ms) divided by the value of head position at the same endpoint. The “end gaze position” is the value of gaze-position error at the point that end position gain is measured.