Role of Orientation Reference Selection in Motion Sickness

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STATEMENT OF WORK SUMMARY

The overall objective of this proposal is to understand the relationship between human orientation control and motion sickness susceptibility. Three areas related to orientation control will be investigated. These three areas are 1) reflexes associated with the control of eye movements and posture, 2) the perception of body rotation and position with respect to gravity, and 3) the strategies used to resolve sensory conflict situations which arise when different sensory systems provide orientation cues which are not consistent with one another or with previous experience. Of particular interest is the possibility that a subject may be able to ignore an inaccurate sensory modality in favor of one or more other sensory modalities which do provide accurate orientation reference information. We refer to this process as sensory selection. This proposal will attempt to quantify subjects' sensory selection abilities and determine if this ability confers some immunity to the development of motion sickness symptoms.

Measurements of reflexes, motion perception, sensory selection abilities, and motion sickness susceptibility will concentrate on pitch and roll motions since these seem most relevant to the space motion sickness problem. Vestibulo-ocular (VOR) and oculomotor reflexes will be measured using a unique two-axis rotation device developed in our laboratory over the last four years. Posture control reflexes will be measured using a movable posture platform capable of independently altering proprioceptive and visual orientation cues. Motion perception will be quantified using closed loop feedback technique developed by Zacharias and Young (Exp Brain Res, 1981). This technique requires a subject to null out motions induced by the experimenter while being exposed to various confounding sensory orientation cues. A subject's sensory selection abilities will be measured by the magnitude and timing of his reactions to changes in sensory environments. Motion sickness susceptibility will be measured by the time required to induce characteristic changes in the pattern of electrogastrogram recordings while exposed to various sensory environments during posture and motion perception tests.

The results of this work are relevant to NASA's interest in understanding the etiology of space motion sickness. If any of the reflex, perceptual, or sensory selection abilities of subjects are found to correlate with motion sickness susceptibility, this work may be an important step in suggesting a method of predicting motion sickness susceptibility. If sensory selection can provide a means to avoid sensory conflict, then further work may lead to training programs which could
enhance a subject's sensory selection ability and therefore minimize motion sickness susceptibility.

SUMMARY OF PROJECT STATUS

Three test devices are required for the proposed experiments. They are (1) a moving posture platform, (2) a servo-controlled vertical axis rotation chair with an independently controllable optokinetic stimulator, and (3) a two-axis rotation chair for the generation of pitch and roll motions. The first two devices have been functional for quite some time and are routinely used for both clinical and research testing. The two-axis rotation device has become operational as of mid-August 1990. The development of this two-axis rotator has been a major focus of work and will be described in more detail below.

An important component associated with the two-axis rotator is a computer controlled video system for the measurement of eye movements. This video system for recording horizontal and vertical eye movements has been working for the past six months. We recently added the capability to measure torsional eye movements. The quality of the eye movement recordings are exceptional.

This new ability to record torsional eye movements should add considerable versatility in the design of experiments related to this grant. This is because torsional eye movements are closely associated with the vertical semicircular canals and otolith receptors, which in turn are implicated in the space motion sickness syndrome. In addition, very little is known about the response properties of torsional eye movements as a function of changes in body position with respect to the gravity vector. We have begun a project to characterize the dynamic response characteristics of torsional eye movements during roll rotations about an upright position.

Another experiment in progress involves the determination of the influence of visual, somatosensory, and vestibular motion cues on the control of posture.

An initial set of experiments involving the perceptual feedback technique developed by Zacharias and Young (Exp Brain Res, 41:159-171, 1981) have been completed. These experiments were designed to look for correlations between vestibulo-ocular reflex parameters and the perception of rotation. A paper describing these results is nearing completion.

Four papers describing earlier work on the VOR and posture control function in a large normal population have been accepted for publication and are currently in press in the Journal of Vestibular Research.
TWO-AXIS ROTATOR DEVELOPMENT

The two-axis rotator is a versatile, general purpose stimulator for vestibular and visual-vestibular interaction studies. It consists of two gimbals powered by rotary hydraulic actuators. A single DC torque motor is now available which is interchangeable with either of the hydraulic actuators. The inner gimbal produces yaw axis rotations of the subject. The outer gimbal rotates the subject about a horizontal axis which passes through the subject's ears.

We have completed the essential parts of 5 major projects related to the two-axis rotator in the past several months. These are (1) the installation of various mechanical, hydraulic, electronic, and computer software safety devices and procedures, (2) calibrations of the two-axis rotator motions, (3) tuning of the servo controls for optimum performance, (4) improvements in the data collection and stimulus delivery computer programs, (5) development of an improved system for the video recording and automated analysis of eye movements, including torsional eye movements.

EXPERIMENTS IN PROGRESS

Two experiments are currently being performed. One is an investigation to characterize the influence of visual orientation cues on the control of posture. The second is to measure the dynamic response properties of human ocular torsion in response to roll rotations. The results of both of these experiments will be used to develop a rating of individual subject performance in various reflex and posture control tasks so that a correlation with motion sickness susceptibility can be identified (if the correlation exists).

The Role of Vision in Posture. These experiments are performed on a moving posture platform. The subject stands facing a high contrast visual field. This visual field can be placed in motion by rotating the visual field in an anterior-posterior direction about an axis which passes through the subject's ankle joints. We have been using sinusoidal motions of the visual field at frequencies of 0.1, 0.2, and 0.5 Hz with amplitudes of 1, 2, 5, and 10 degrees presented in random order. In addition, in half of the tests the surface upon which the subject stands is "sway-referenced" in order to alter the somatosensory cues which are available for posture control. Sway-referencing involves the controlled rotation of the platform upon which the subject stands in proportion to the subject's own sway. This results in very little change in the subject's ankle joint angle even though the subject is swaying forward and backward. We record the
subject's anterior-posterior sway at waist and shoulder level. From those measures we estimate the sway angle of the subject's center of mass throughout the trial. A Fourier analysis is used to estimate the average amplitude of the center-of-mass body sway at the stimulus frequency.

Figure 1 shows typical results from a normal subject in response to 0.2 Hz sinusoidal rotations of the visual field at various amplitudes while the subject stood on a fixed surface (left column) and a sway-referenced support surface (right column). Sway-referencing of the support surface refers to a technique which alters the normal relationship between body sway and the rotation of the subject's ankle joint angle. This technique apparently reduces the somatosensory signals available for the control of body sway, and therefore forces a greater reliance on other sensory system information (visual and vestibular in particular). Sway-referencing of the support surface is accomplished by actively rotating the support surface angle in proportion to the subject's sway angle.

Figure 1 shows that this normal subject's sway was only slightly influenced by the "false" visual orientation cues resulting from the sinusoidally rotating visual field when the subject stood on a fixed support surface. Sway increased when the subject stood on the sway-referenced support surface. However when the amplitude of the rotating visual field increased, the subject's sway did not correspondingly increase. This suggests that the subject's somatosensory and vestibular systems in the fixed platform case, and the vestibular system in the sway-referenced case, provided sensory cues which were used by the brain's posture control mechanisms to limit the response to the visual stimulus.

Figure 2 shows the results for a subject with complete bilateral loss of vestibular function during the same conditions. At low amplitudes of the visual field stimulus, the sway of this subject was clearly influenced by the moving visual field. At higher stimulus amplitudes with fixed platform, the bilateral loss subject consistently swayed more than the normal subject. At higher stimulus amplitudes with a sway-referenced platform to reduce somatosensory cues, the bilateral loss subject consistently fell since the subject did not have any source of sensory information which provided an accurate orientation reference.

What is not shown in these figures is that there was a wide range of sensitivities to the visual field motion among the normal subjects. At low stimulus amplitudes, some of the normal subjects showed similar sway amplitudes to bilateral deficit subjects while other normals showed much less. This suggests that there is considerable variation of the behavioral weighting of sensory orientation cues among normal subjects. As this grant work progresses, this variation will provide us with a scale
of performance against which motion sickness susceptibility can be compared.

**Ocular Torsion.** Rotations of the head about a naso-occipital axis stimulate the vertical semicircular canals and the otolith organs (depending on the orientation of the head with respect to gravity). Signals from these vestibular receptors produce torsional or counterrolling eye movements. As with other aspects of the vestibulo-ocular reflex (VOR), the presumed function is to stabilize images on the retina during head motion in order to insure clear vision. The need to torsionally stabilize eye movements during rolling head movements would seem to be less important than during pitching and yawing head movements since image motion at the eye's fovea, the region of highest acuity, is relatively small during head rolls. Therefore the results of counterrolling experiments which have demonstrated very low gains during static head positions, and relatively low gains during moderate frequency (0.1 to 0.8 Hz), actively generated head rolls seem to confirm the thought that this reflex is not very functionally significant.

Our results show that ocular torsion gains are actually quite large during head motions which resemble those which can occur during natural, everyday movements. That is, during low amplitude (<20 degrees), high frequency (>~1 Hz) head rolls, the gain of the torsional VOR is close to unity. Figure 3 shows the gain and phase responses of the three subjects tested to date. At 2 Hz, 2 of the three subjects had gains above 0.9, and phases were near zero.

As with the posture experiment results, the variability of results among individuals will be a key point of interest in determining if individual variations in reflex function relate to motion sickness susceptibility. As an example of this variability, the torsion measures from 2 subjects are shown in Figure 4 during a 0.2 Hz, ±20° roll rotation. One subject had very little nystagmus while the other had a great deal of nystagmus. In addition, the subject with the least nystagmus also was the one with the largest phase leads in Figure 3. These types of torsional VOR response differences may represent "strategy" differences among individuals in the way that they choose to use available sensory information for the control of compensatory reflexes. Perhaps these differences in strategies are also associated with either more or less successful abilities to avoid motion sickness symptoms when exposed to environments which give conflicting sensory cues to orientation.
PERCEPTUAL FEEDBACK EXPERIMENTS

In 1981, Zacharias and Young presented a method which allowed for the quantification of a subject's perception of rotation under the combined influence of visual and vestibular cues. In this technique, the subject has control over the rotational motion of the chair by adjusting a potentiometer. Subjects are seated in the vertical axis rotation test room with the potentiometer mounted on the arm of the chair. The output of this potentiometer is summed with a velocity command signal from a computer and this summed signal is delivered to the velocity command input of the chair's servo motor. The goal of the subject is to continuously adjust the potentiometer so that he feels like he is not moving. A "perfect" subject would be able to hold himself stationary in space by adjusting the potentiometer so that its output was equal but opposite to the computer's command signal. "Real" subjects do not remain stationary because of the dynamics of their motion perception and motor reaction systems, and because of presumed imbalances in the vestibular receptors.

Relation of Perceptual Feedback to VOR Test Results. The article by Zacharias and Young suggested that the drift of the subject during rotation in the dark, or with subject-referenced vision, might be due to an imbalance in the encoded motion information coming from the two halves of the vestibular system in opposite ears. This is also the interpretation which is generally given to the presence of bias, or directional preponderance observed in tests of horizontal VOR function. We anticipated that there might be a correlation between the drift observed in perceptual feedback tests and the bias recorded in VOR tests. However we have not found any obvious correlation between these two measures. It may be possible that normal subjects have too small a range of bias and drift to provide a reliable correlation. However the bias measured for a given subject does appear to remain consistent over time, as does drift. That is, the reliability of the measurement of these two parameters seems to be fairly good. This would argue that the lack of correlation between these two measures is real, and not an artifact of their limited range, at least in normal subjects. This observation suggests that there are differences between the static (very low frequency) responses of the VOR and the static properties of motion perception. We believe that exploring these differences and their possible association with motion sickness may be productive.
SCIENTIFIC PAPERS AND PRESENTATIONS

An abstract describing torsional VOR dynamics was recently submitted for presentation at the Association for Research in Otolaryngology in February 1991. A copy of the abstract is attached.

Four papers describing the results of our study of VOR, optokinetic reflex, and moving platform posturography from 200 putatively normal subjects are currently in press in the Journal of Vestibular Research. Copies of the four papers are enclosed.
Figure 1. Anterior-posterior center-of-gravity sway angle in response to sinusoidal rotation of a full-field visual surround. Results are for a normal subject standing on a fixed surface (left column of data) and a sway-referenced surface (right column) during 0.2 Hz rotations of the visual surround at amplitudes ranging from ±1° to ±10°.
Figure 2. Sway of an abnormal subject with a total bilateral loss of vestibular function responding to the same full-field visual field motions as those in figure 1. Note that the subject fell on the $\pm 5^\circ$ and $\pm 10^\circ$ trials when the platform was sway-referenced.
Figure 3. Ocular torsion response dynamics of 3 normal subjects recorded during roll rotations in a dark room while the subjects viewed a single dim fixation light located on the rotation axis. The amplitude (gain = torsional eye velocity/stimulus velocity) and timing (phase with respect to the sinusoidal rotational stimulus) of torsional eye movements changes as a function of the stimulus frequency.
Figure 4. Torsional eye movements of 2 normal subjects recorded during roll rotations in a dark room while the subjects viewed a single dim fixation light located on the rotation axis. The stimulus was a 0.2 Hz sine with ±20° amplitude (top trace). The scale applies to all three traces.
DYNAMIC RESPONSE CHARACTERISTICS OF THE HUMAN TORSIONAL VESTIBULO-OCULAR REFLEX. *R.J. Peterka. R.S. Dow Neurological Sciences Institute and Clinical Vestibular Laboratory, Good Samaritan Hospital & Medical Center, Portland, OR 97210.

The torsional vestibulo-ocular reflex (VOR) was measured in three subjects. The stimulus consisted of controlled sinusoidal rotations with amplitudes of ±20° for 0.05 to 0.8 Hz stimuli, ±10° for 1.0 Hz, and ±5° for the 2.0 Hz stimulus. Subjects were rotated in the dark about a naso-occipital axis at the level of the intraural axis while viewing a single dim red LED located on the rotation axis about 38 cm in front of their eyes. A small bite-plate mounted video camera recorded eye movements from the right eye under infrared illumination. Each sequential video image (60/s) from a video recording was analyzed off-line by first locating the edges and center of the pupil, and then scanning the intensity of 4 to 6 concentric rings around the iris about midway between the pupil and the sclera. The peak of a cross correlation between the reference iris scan rings obtained at the beginning of each trial and the scan rings from the current video image was used to estimate the ocular torsion. The velocity of the slow phase portions of ocular torsion was calculated and compared to the stimulus velocity in order to calculate torsional VOR gain and phase. Unity VOR gain and 0° phase represent perfect compensatory response dynamics.

Torsional VOR gain generally increased with increasing frequency. Gains at 0.05 Hz ranged from 0.15-0.32 and at 2.0 Hz from 0.69-0.98. At lower frequencies, phase leads were present. Above 0.1 Hz, phases generally declined toward 0° with increasing frequency. Two of the three subjects showed more phase lead at 0.1 Hz than at 0.05 Hz. Phases ranged from 7.4°-15.4° at 0.05 Hz, 7.8°-17.6° at 0.1 Hz, and 1.2°-6.4° at 2.0 Hz.

Previous measures of torsional VOR during active head tilts at frequencies below 1.0 Hz found gains ranging from 0.3 to 0.7 (Ferman et al., Vision Res. 27:811-828, 1987). Although the results presented here were obtained using passive rotations, similar torsional VOR gains were observed at corresponding stimulus frequencies. This study extended the frequency range to 2.0 Hz for the identification of torsional VOR dynamics. At 2.0 Hz, two of the three subjects had gains greater than 0.9 and phases near 0°. This suggests that the torsional VOR can play a significant roll in stabilizing retinal image motion during low amplitude, high frequency head movements.

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Association for Research in Otolaryngology Abstract - Meeting date February 1991.
where $B$ is bias in $\gamma/s$, $A$ is response amplitude in $\gamma/s$, $P$ is response phase in degrees, and $f$ is the stimulus frequency. The recorded chair velocity data were separately analyzed to calculate stimulus velocity amplitude, $A$, and phase, $P$. The VOR gain of the reflex is defined as the ratio $A/P$, and the phase of the reflex as $P$ - $P$. Since the VOR is a compensatory reflex, the values of $P$ - $P$ were close to $-180^\circ$. For the convenience of working with smaller numbers, a value of $180^\circ$ was added to $P$ - $P$ for the VOR test. This is equivalent to inverting the horizontal eye position data.

In order to quantify nonlinear responses, the horizontal eye velocity data were shifted in time by an amount determined by the calculated phase of each period of the response. The time shift was in a direction that brought the response into phase with the stimulus. Slow phase eye velocity was then plotted against stimulus velocity to yield a scatter of points that generally lie along a negatively sloping line. An example is shown in Figure 1. The slope of the line is equal to VOR gain. A linear VOR response is consistent with equal VOR gains for rotations in opposite directions. One type of nonlinear VOR response, sometimes seen in abnormal subjects, has unequal gains for rotations to the right and left. This type of nonlinearity was quantified by separately calculating the slopes of the eye velocity versus stimulus velocity data for chair rotations to the right and to the left. The slopes were calculated by a least-squares analysis of a two-segment line to the data. One line segment was for positive and the other for negative stimulus velocities. The two line segments were constrained to intersect one another at zero stimulus velocity. The two-part linear curve fit yields three parameters: 1) the reflex gain for slow phase eye movements to the right, $G_r$, 2) the gain for slow phase eye movements to the left, $G_l$, and 3) response offset defined as the eye velocity at zero stimulus velocity. A measure of response asymmetry was calculated according to the formula $100 \times (G_r - G_l)/(G_r + G_l)$. A zero percent asymmetry is consistent with a linear system response where gain is independent of the stimulus direction.

**Caloric Tests**

Four irrigations of the external carotid canals were made using a Brucker-Gramm closed loop caloric irrigator. Subjects were in a supine position with head elevated about 30° above horizontal to assure maximal stimulation of the horizontal semicircular canals. The caloric test was not performed on subjects under 12 yrs. and complete data were not obtained on other subjects who became nauseated or simply chose not to continue the irrigations because of discomfort. Each ear was alternately irrigated for 45 s at 30 and 44°C. Horizontal and vertical eye movements were recorded with EOG techniques identical to those described for rotation tests. Eye movements were recorded during and after each irrigation for a total of 3 min. Horizontal eye movements were analyzed to calculate peak slow phase eye velocity. Caloric responses were quantified by labryinthine asymmetry (LA), directional preponderance (DP), and average response (AR) measures defined by:

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

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

$$AR = \frac{(RW + RC + LW + LC)}{4}$$

where $RW$, $RC$, $LW$, $LC$ are the absolute values of peak slow phase eye velocities recorded during right warm, right cold, left warm, and left cold irrigations, respectively. Subjects were tested throughout caloric testing to maintain alertness.

**Visualization of Trends**

In order to visualize trends in scatterplots, a robust locally weighted regression analysis (lowess fit) was used to smooth the scatterplots. This smoothing is similar to a moving average filter but is less sensitive to outlying points and allows variable amounts of smoothing. A lowest smoothing parameter of 0.5 and iteration parameter of 2 were used on all data sets.

**Data Quality**

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

**Results**

The subjects showed a wide range of responses on all measures of VOR function. Age-related changes were identified in many rotation test response measures, but the magnitude of these changes was not large relative to the variability of the data. Most changes indicated a decline in function. In contrast, no obvious or consistent changes as a function of age were found in caloric test responses. There were no significant differences in reflexes between males and females.
0.05 and 0.2 Hz, but no correlation at 0.8 Hz. There was a small positive correlation between offset and asymmetry at 0.05 Hz, no correlation at 0.2 Hz, and a larger negative correlation at 0.8 Hz.

The correlations between response bias at all test frequencies and caloric DP were about -0.4. Offset and DP showed a similar pattern but with slightly less negative correlations. Asymmetry and DP showed small negative correlations (about -0.2) at 0.05 and 0.2 Hz, but no correlation at 0.8 Hz. There was no correlation between LA and DP, or between LA and any of the rotation test symmetry measures.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Slope</th>
<th>Correlation Coefficient</th>
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</thead>
<tbody>
<tr>
<td>Gain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.05 Hz</td>
<td>-0.0030</td>
<td>-0.39*</td>
</tr>
<tr>
<td>0.2 Hz</td>
<td>-0.0038</td>
<td>-0.24*</td>
</tr>
<tr>
<td>0.8 Hz</td>
<td>-0.0022</td>
<td>-0.33*</td>
</tr>
<tr>
<td>Phase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.05 Hz</td>
<td>0.029</td>
<td>0.12</td>
</tr>
<tr>
<td>0.2 Hz</td>
<td>0.042</td>
<td>0.27</td>
</tr>
<tr>
<td>0.8 Hz</td>
<td>0.050</td>
<td>0.29</td>
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*Significantly different from zero (P < 0.05).

Caloric AR and rotation test gain and phase measures are known to covary in some vestibular abnormalities. Within our relatively normal population, there were only small correlations between these parameters. Correlations between AR and gain at the three test frequencies ranged from 0.22 to 0.31. The correlation between AR and phase was only -0.12 at 0.05 Hz, and less at the other two test frequencies.

Discussion

VOR Parameter Correlations

The pattern of correlation between caloric and rotation test parameters, and among the rotation test parameters themselves, apparently depends on rotation test frequency. The asymmetry parameter, which measures the difference in VOR gain for rotations to the right and left, showed the most complex frequency-dependent pattern. For example, the correlations between bias and asymmetry were about 0.6 at 0.05 and 0.2 Hz, but less than 0.1 at 0.8 Hz. The correlation between caloric DP and asymmetry was also higher at 0.05 and 0.2 Hz compared to 0.8 Hz. In addition, there was a shift in the correlations between offset and asymmetry from a positive value at 0.05 Hz, to a near zero value at 0.2 Hz, and then to a negative value at 0.8 Hz. Finally, the correlations between asymmetry measures made at different test frequencies were poorer than either the bias or offset parameter correlations across frequency. The poor correlation of asymmetry measures at different test frequencies and the changing relationship of asymmetry to the other caloric and rotation test symmetry measures suggests that the physiological mechanisms which control symmetry are either frequency dependent or that separate physiological factors dominate at different frequencies of head motion.

VOR Changes with Age

We were able to identify small age effects on some VOR response measures. The direction of change of VOR gains was expected. Other age-related changes were not expected. These include increased VOR phase leads with increasing age, and the fact that VOR function measured using the caloric test did not show the same trend as VOR function measured using rotation tests.

Age-related changes in VOR function identified in this study do not follow the same time course as age-related peripheral vestibular anatomical changes identified by others. Figure 4 shows the lowess curve fit to 0.8 Hz VOR gain versus age plotted along with other data for human crista nerve fibers, 1745 vestibular nerve fibers, and 1835 Scarpa's ganglion cells.

Figure 4. Comparison of age-related changes in VOR gain and peripheral vestibular anatomical data. The 0.8 Hz VOR gain fit is the same as in Figure 2. All curves fit to anatomical data are lower fits to published data. All fits are plotted on a linear scale normalized to 1.8 at age 30.2. The normalization factors are 0.97 for 0.8 Hz VOR, 0.97 for 0.2 Hz VOR, 0.97 for 0.2 Hz VOR, 0.97 for 0.2 Hz VOR, 0.97 for 0.2 Hz VOR, and 1.8 for Scarpa's ganglion cells.

The offset and asymmetry from a positive value at 0.05 Hz, to a near zero value at 0.2 Hz, and then to a negative value at 0.8 Hz. Finally, the correlations between asymmetry measures made at different test frequencies were poorer than either the bias or offset parameter correlations across frequency. The poor correlation of asymmetry measures at different test frequencies and the changing relationship of asymmetry to the other caloric and rotation test symmetry measures suggests that the physiological mechanisms which control symmetry are either frequency dependent or that separate physiological factors dominate at different frequencies of head motion.

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ratio of response amplitude to stimulus amplitude, and the difference between response phase and stimulus phase, respectively. For the convenience of working with smaller numbers, a value of 180° was added to the calculated phases for tests of the VOR reflex so that unity gain and 0° phase represented perfectly compensatory eye movements.

The algorithms for gain and phase calculations were verified by simulating reflex responses with known electronic circuits. Before analysis, segments of the simulated responses were eliminated to simulate the patterns of missing data caused by the elimination of nystagmus fast phases from real eye movement data.

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

\[ H_{av}(s) = \frac{K_{av} \cdot T_{av}}{T_{av} + s} \]

where \( T_{av} \) is an estimate of the VOR time constant (units of seconds), \( K_{av} \) is the VOR gain constant, and \( s \) is the Laplace transform variable.

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

\[ H_{av}(s) = \frac{K_{exp} \cdot T_{av}}{T_{av} + s} \]

where \( T_{av} \) is a time constant with units of seconds, \( K_{exp} \) is the OKR gain constant relating slow phase eye velocity to stimulus velocity, and \( T_{av} + 1 \) factor represents a lowpass filter which accounts for the declining gain with increasing frequency observed in some subjects. Larger values of \( T_{av} \) were consistent with gain declines beginning at lower frequencies. A value of zero for \( T_{av} \) (the transfer function reduces to \( H_{av}(s) = K_{exp} \cdot s \)) accounts for subjects whose gain did not decline with increasing frequency. \( T_{av} \) is not the time constant associated with velocity storage and optokinetic nystagmus.

Visualization of Trends

In order to visualize trends in scatterplots, a robust locally-weighted regression analysis (lowess fit) was used to smooth the scatterplots (4). This smoothing is similar to a moving average filter, but is less sensitive to outlying points and allows variable amounts of smoothing. A lowess smoothing parameter of 0.5 and iteration parameter of 2 were used on all data sets.

Data Quality

The overall quality of each rotation test for each subject was subjectively given a rating of good, fair, or poor. Only good and fair quality data are included in the data summaries in the results section. Quality judgments were based on the consistency of the responses throughout the duration of the stimuli, and on the accuracy of the eye movement analysis in the separation of slow and fast phases of nystagmus. The actual values of response parameters were not used in judgment of data quality. The test results from about 4% of subjects were rated poor for each test. Poor quality data for one subject on a given test was not used to disqualify other data from the same subject from other tests.

Results

The subjects showed a wide range of responses on all measures of VOR and OKR function. Age-related changes were identified in almost all rotation test response measures, but the magnitude of these changes was not large relative to the variability of the data. Most changes indicated a decline in function. There were no significant differences in reflexes between males and females.

VOR Responses

A sample of a typical response to a pseudorandom VOR stimulus is shown in Figure 1. The pseudorandom stimulus evokes a complex eye movement pattern (Figure 1C). However, separation of slow and fast components, and calculation of slow phase eye velocity reveals the underlying compensatory motion (Figure 1B). A spectral analysis of slow phase eye velocity and the recorded stimulus velocity provides measures of response gain and phase as a function of stimulus frequency. Examples of gain and phase data from three subjects are shown in Figure 2. Typically, the gain is lower at the lowest stimulus frequency and increases with increasing frequency. In some subjects the gain monotonically increases over the frequency range tested and in others it appears to reach an asymptote. The solid lines through the data points represent curve fits of a two parameter transfer function model (eqn 2) to the data of each subject. The patterns of VOR gain and phase data of most subjects was similar in form to the data in Figure 2A, and was well characterized by the two parameter transfer function model (eqn 2). There were deviations from this pattern which are exemplified by the data from two other subjects in Figures 2B and 2C. The low frequency data in 2B were fit well by the two parameter model but the higher frequency data showed increasing phase lead with increasing frequency. The phases of VOR responses to 0.05, 0.2, and 0.8 Hz sinusoidal rotations were 10°, 2°, and 6.9°, respectively, for this subject, and therefore confirmed the general pattern. A more accurate curve fit to this data would require a higher frequency lead term in the transfer function. A transfer function of this form would be similar to the one used to describe the dynamic responses of phasic canal afferents in the squirrel monkey (6).

The VOR phase data in Figure 2C were fairly flat and greater than zero across all test frequencies. The phases of responses to sinusoidal stimuli were 3.7°, 2.8°, and 2.1° at 0.05, 0.2, and 0.8 Hz, again confirming the general pattern but with less phase lead than the pseudorandom data. The curve fit identified a long VOR time constant of 44.9 s. However, the two parameter model does not
The table below compares the performance of various VOR systems and their corresponding errors in navigation.

<table>
<thead>
<tr>
<th>System</th>
<th>Error (m)</th>
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<tbody>
<tr>
<td>System A</td>
<td>0.80</td>
</tr>
<tr>
<td>System B</td>
<td>0.95</td>
</tr>
<tr>
<td>System C</td>
<td>1.10</td>
</tr>
<tr>
<td>System D</td>
<td>0.60</td>
</tr>
</tbody>
</table>

The chart illustrates the trend of VOR error over time, showing a decrease in error as time increases.
Age Effects in VOR and OKR

OKR Responses

Typical OKR test results from pseudorandom stimulation for two subjects are shown in Figure 3. Response gain was less than unity in all subjects. The gains of most subjects were approximately flat across the bandwidth of frequencies tested (0.02 to 1.5 Hz) as in Figure 3A. Phases were near 0° at the lowest frequencies and showed monotonically increasing phase lag as frequency increased. Since perfect tracking of the visual stimulus is represented by unity gain and zero phase at all frequencies, subjects demonstrated imperfect tracking in terms of both amplitude (gain) and timing (phase). The major variation on the typical OKR result was the presence of declining gain with increasing frequency in some subjects. Figure 3B shows the OKR transfer function data from one such subject.

The means, standard deviations, and ranges of OKR gain constant, time constant, time delay, and bias are given in Table 4. OKR response bias was near zero for all subjects. Both the gain constant and time delay had approximately symmetric distributions. In contrast, the OKR time constant had a highly skewed and possibly bimodal distribution with about 40% of the values near zero. OKR time constants near zero reflect the fact that OKR gains for these subjects were approximately constant over the frequency range tested.

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

Age-Related Changes in VOR and OKR

Several VOR and OKR response parameters changed with age (Figures 4 and 5), while the absolute values of VOR and OKR bias did not. Many of the age-related changes showed roughly linear trends. Linear regression slope, intercept, and correlation coefficients are summarized in Table 5. Both VOR time constant (Figure 4B) and OKR gain constant (Figure 5A) increased slightly in subjects up to about 30 y and then decreased with increasing age. The OKR time delay parameter increased with increasing age and showed the clearest age-related trend (r = 0.53 and slope = 1.2 ms/y) of all VOR and OKR parameters.

The age-related change in the OKR time constant was clearly not linear. Data in Figure 5B shows a large proportion of subjects between about 20 and 60 y had OKR time constants close to zero, indicating that their OKR gains were constant across frequency. In contrast, there were very few subjects under 20 y of age and proportionally fewer subjects over 60 who had zero OKR time constants, indicating that on average their OKR gains declined with increasing frequency. The lowest curve fit indicates that age-related trends were minimal for subjects between 20 and 60 y. Subjects under 20 showed an age-related decline in their OKR time constant with increasing age. Subjects over 60 showed an age-related increase in their OKR time constant with increasing age.

Discussion

Pseudorandom Testing

There are both advantages and disadvantages to the use of pseudorandom stimuli for VOR and OKR testing. An advantage is the considerable savings of response dynamics over a large frequency bandwidth. The total test

Table 4. OKR Response Parameters for Pseudorandom Stimuli; Mean, SD, and Parametric Values from Parameter Distributions (N = 179 subjects)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean</th>
<th>SD</th>
<th>Bias</th>
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</thead>
<tbody>
<tr>
<td>Gain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time delay</td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

Figure 3. Examples of OKR gain and phase data from two individuals derived from responses to pseudorandom optokinetic stimulation. Solid lines show transfer function curve fit to data. Equations of the curve fits are inset. Gain and frequency scales are logarithmic.
Both younger (<15 y) and older (>65 y) subjects were relatively less responsive to the higher frequency components of the stimulus. The lower OKR responsiveness at higher frequencies could have functional consequences, particularly for older subjects. While it is generally appreciated that visual tracking reflexes improve visual-vestibular generated compensatory eye movements during low frequency head movements, visual motion information is apparently used to improve the dynamics of compensatory eye movements at higher stimulus frequencies associated with natural head movements (13). This would be particularly important for individuals who had VOR phase leads at higher frequencies (Figure 2B).

Since older individuals had larger VOR phase leads on average than younger subjects (13), we might expect that the older subjects would need more help from their visual tracking reflexes to correct their imperfect VOR dynamics. However, the sensitivity to optokineti

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   ocular reflex in the pigeon. J Neuropathol. 1982:19:
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Results

General Response Pattern

Figure 1 shows typical EMG, CP, CPV, and \( f_{CP} \) response patterns for one subject during a 3 cm backward translation. The backward support surface translation results in forward body sway with respect to the platform. In the first 100 ms, the CP movement away from baseline is probably the result of passive properties of body mechanics combined with artifacts of the platform force recording system. Approximately 110 ms following the start of the translation, the distal leg muscles (gastrocnemius) that oppose the forward body sway begin to contract as evidenced by EMG recordings. The proximal leg muscles (hamstrings) begin to contract about 20-30 ms following the distal muscles. The dorsal leg muscle contractions generate torque about the ankle joint that causes a forward displacement of CP. The onset of the active torque generation (CP) begins about 130 ms after the start of platform translation. CP reaches a peak displacement amplitude (CPV) at about 230 ms (CP). Sway (\( f_{CP} \)) reaches a peak (\( f_{CP} \)) at about 260 ms (\( f_{CP} \)) and then returns toward an upright position. The time (CPV) of the CPV peak amplitude (CPV) occurs between CP and CP.

Forward support surface translations causing backward sway with respect to the platform initiate contractions of the \( T \) and \( Q \) muscles. The patterns of sway and changes in CP are similar to those for basic translations, but have opposite sign.

The population statistics describing EMG onset times, CP, CPV, \( f_{CP} \), CPV, and \( f_{CP} \), are given in Table 1. The values of all EMG onset, CP, CPV, \( f_{CP} \), and CPV, were symmetrically distributed about their means. CP and CPV, distributions were slightly skewed toward larger values. Most values of \( f_{CP} \), for both forward and backward conditions, were tightly grouped around 260 ms but 15% of the population had values of \( f_{CP} \) of 375 ms. \( f_{CP} \), for backward translations also included a scattering of times shorter than 260 ms.

CP, for both forward and backward translations, showed bimodal distributions. For backward translations, 81% of the subjects had both right and left CP, centered about a mean of 245 ms, 11% had both right and left CP, centered about 350 ms, and the remainder of the population had one leg's CP, <300 ms and the other leg's CP, >300 ms. For forward translations, 4% of the subjects had both right and left CP, centered about a mean of 260 ms, 34% had both right and left CP, centered about 350 ms, and the remainder of the population had one leg's CP, <300 ms and the other leg's CP, >300 ms.

For both forward and backward translations, subjects with shorter CP, (<300 ms) had larger mean values of CPV, and smaller

\[
\text{Table 1. EMG, CP, CPV, and } f_{CP} \text{ Parameters (mean } \pm 1 \text{ SD)}
\]

<table>
<thead>
<tr>
<th></th>
<th>Backward Translation</th>
<th>Forward Translation</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>( G_C ) ms</td>
<td>115 ± 12.9</td>
<td>162 ± 22.5</td>
</tr>
<tr>
<td>( H_C ) ms</td>
<td>145 ± 22.5</td>
<td>108 ± 14.3</td>
</tr>
<tr>
<td>( S_C ) ms</td>
<td>127 ± 13.1</td>
<td>211 ± 23.1</td>
</tr>
<tr>
<td>( T_C ) ms</td>
<td>137 ± 23.1</td>
<td>211 ± 137 ± 24.5</td>
</tr>
<tr>
<td>( CP ) ms</td>
<td>167 ± 17.2</td>
<td>211 ± 180 ± 36.7</td>
</tr>
<tr>
<td>( CPV ) ms</td>
<td>261 ± 46.5</td>
<td>211 ± 287 ± 46.5</td>
</tr>
<tr>
<td>( f_{CP} ) ms</td>
<td>298 ± 56.5</td>
<td>211 ± 290 ± 53.5</td>
</tr>
<tr>
<td>( f_{CP} ) cm/s</td>
<td>1.31 ± 0.42</td>
<td>108 ± 1.24 ± 0.14</td>
</tr>
<tr>
<td>( f_{CP} ) cm/s²</td>
<td>0.46 ± 0.13</td>
<td>108 ± 0.46 ± 0.14</td>
</tr>
<tr>
<td>( f_{CP} ) degrees</td>
<td>1.02 ± 0.37</td>
<td>108 ± 1.24 ± 0.14</td>
</tr>
<tr>
<td></td>
<td>1.02 ± 0.37</td>
<td>108 ± 1.24 ± 0.14</td>
</tr>
</tbody>
</table>

*SD = standard deviation.

mean values of CPV and CPV (all significant at \( P < 0.01 \)) than subjects with longer CP (\( (> 300 \text{ ms}) \) ). For backward but not forward translations, mean CPV were also significantly larger for the short CP group. There was no clear relation between the bimodality of the \( f_{CP} \) and CP, distributions. That is, many subjects with larger CP had smaller \( f_{CP} \) and other subjects with smaller CP had larger \( f_{CP} \).

The response pattern from three subjects during backward translation and two subjects during forward translation did not allow for accurate estimation of the various center of pressure and sway parameters. In all these cases there appeared to be little or no active torque generated by the subjects.

Age-Related Changes in EMG Onsets

With the exception of the quadriceps, EMG onset times generally increased with increasing subject age (Figure 2A-D). Linear fits to the data (Table 2) showed that the rate of change of EMG onset times with age were 0.021 ms/yr for \( G_C \), 0.020 for \( H_C \), 0.10 for \( T_C \), and \(-0.87 \) for \( f_{CP} \), with linear correlation coefficients of 0.335, 0.267, 0.158, and \(-0.07 \), respectively. However, the lowest fits to \( G_C \), \( H_C \), and \( T_C \), suggested that there may be an infection point at roughly age 55 with a larger rate of change for subjects older than 55 yr. To compare the ages for younger and older subjects, two parallel lines were fitted to \( G_C \), \( H_C \), and \( T_C \) for subjects younger than 55 yr. The slopes of these lines are given in Table 2.

The difference between the EMG onset times for the \( H \) and \( G \) muscles (\( G_C \), \( H_C \)) during backward translations, and between \( Q \) and \( T \) muscles (\( Q_P \), \( T_P \)) during forward translations is plotted as a function of subject age in Figure 2E and F. There was a small increase in the \( H_C \), \( G_C \) delay with increasing age (0.17 ms/yr with \( r = 0.183 \)). For the \( Q_P \), \( T_P \) delay, subjects younger than 20 y tended to have larger \( Q_P \), \( T_P \) delays (mean 22.2 ms ± 2.20 SD) than subjects older than 20 y (mean 9.6 ms ± 18.0 SD). The difference in mean \( Q_P \) = \( T_P \), between these two groups is significant \( P < 0.01 \). The larger \( Q_P \), \( T_P \) delays for younger compared to older subjects is the result of: (1) larger \( Q_P \) values for younger subjects (Figure 2D), and (2) the upward
were between CPa and CPd. The largest correlation between any EMG and CP parameter was CPd and CPa for forward translations. All correlations with other response time parameters were smaller than most other comparisons.

The interpretation of this correlation analysis is potentially problematic since different subsets of the population contributed to different correlation measures. However, a correlation analysis that included only subjects with no missing values gave similar results.

**Forward-Backward Translation Comparison**

Table 1 shows that CPa, CPd, CPd', and $\dot{a}$ response time parameters were larger for forward than backward translations even though $T_c$ and $Q_c$ times were similar, and even slightly shorter than $\dot{Q}_c$ and $\dot{Q}_c'$ times. In addition, the timing difference between forward and backward translations increases for parameters that occur later in the normal sequence of motion. This is, EMG timing is similar, $CP_d$ is 10 ms later, CPV is 15 ms later, and CP and $\dot{a}$ are -30 ms later for forward compared to backward translations.

Response amplitude measures also differed between forward and backward translations. Both CPa and CPd were significantly larger for backward translations, and $\dot{a}$ was larger for forward translations (all $P < 0.0001$).

**Discussion**

Most of the results of motor tests of postural control showed a wide range of what must be considered normal function. In spite of the large variances, age-related changes in function were identified in some response parameters. Although these changes were statistically significant, they were small in magnitude. In particular, the latency of EMG onsets, with the exception of quadriceps, increased with increasing age. In addition, there was evidence that the rate of increase of EMG onset with age was larger for subjects older than 55 years. This increased rate was more evident in the tibialis muscle. Studies of muscle strength in the elderly have also shown proportionally larger losses in tibialis strength compared to other leg muscles. The loss of strength combined with the slowing of the tibialis muscle response to body perturbations would diminish an individual's ability to control backward sway.

The distal before proximal muscle contraction synergy was observed in most subjects. However, during forward translations, $Q_c$ preceded $T_c$ in $25\%$ of the subjects. This pattern is consistent with the generation of less corrective torque on forward translations compared to backward, resulting in larger peak body sway from forward platform motions.

**Figure 3**

Various motor response amplitude (A, C, E) and time (B, D, F) parameters as a function of subject age. All plots are from backward translation responses from the right leg. Solid lines through data are least squares fits with $t = 0.2$. The results of a paired t-test. This pattern is consistent with the generation of less corrective torque on forward translations compared to backward, resulting in larger peak body sway from forward platform motions.

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Body Sway Analysis

Sway data were summarized by two measures: average rectified sway (ARS) and peak-to-peak sway. Both measures were calculated over the final 20 s of the 21 s trials (the visual field and support surface were always earth-referenced during the first second of each trial). For ARS calculations, sway data was normalized by subtracting the average sway values recorded in the first second from the entire sway record. Sway data samples < zero were then rectified (inverted), and the new sway trace was averaged over the final 20 s.

ARS often did not reflect how close a given individual was to a fall since, for example, a subject who leaned forward by a few degrees and stayed in that position throughout the remainder of the trial could score the same as a subject who oscillated back and forth during the trial with the peak of the oscillations close to the threshold of a fall. The peak-to-peak sway measure was more indicative of the closeness of sway to fall thresholds.

Movement Strategies

Subjects typically use one of two body motion strategies to maintain upright stance without moving the feet. A hip strategy consists of \( \theta_h \) and \( \theta_p \) motions that are out of phase. Subjects can be forced to use a hip strategy by asking them to stand on a narrow beam that limits the amount of torque that can be exerted at the ankle. A pure ankle strategy occurs when all motion is about the ankle joint (AP sway angles measured at the hip and shoulder are equal and \( \theta_a \) is zero). A less pure ankle strategy occurs when there is some motion about the hip joint, but \( \theta_h \) and \( \theta_p \) are in phase with each other. In order to quantify the type of body motion, a strategy measure was calculated according to the following formula:

\[
\text{strategy score} = \frac{\theta_a}{\theta_p - \theta_h} (\theta_h - \theta_p)
\]

where the bars over the various terms indicate the average values over time. The strategy score is the average product of zero-meaned \( \theta_a \) and \( \theta_h \) calculated over the duration of the trial. The strategy score is negative if \( \theta_h \) and \( \theta_p \) are out of phase indicating that the trunk and legs move in opposite directions, positive if they are in phase and the body moves like a whip, and zero when the body moves like an inverted pendulum with no bending at the waist. Since this measure is an average over the entire trial, changes in strategy during the trial would not be correctly characterized by this single measure. In practice, this was not a problem since this putatively normal population did not show marked strategy changes within trials.

Visualization of Trends

In order to visualize trends in scatterplots, a robust locally weighted regression analysis (lowess fit) was used to smooth the scatterplots (4). This smoothing is similar to a mo-
tions. Falls during sensory test conditions were not random occurrences, but rather were associated with the inability of some subjects to obtain and/or coordinate the sensory information available for the control of posture. Consider subjects who fell in two of the four conditions that presented them with sensory conflict situations. There are six possible combinations of paired falls within the grouping of the four more difficult conditions. If paired falls occurred randomly they would be evenly distributed across the six possible combinations. This was clearly not the case since four of the six combinations of paired falls either were not observed or were rare. That is, no subjects fell in 3-4 and 4-5 paired conditions, and only two subjects fell in the 3-5 and one in the 4-6 combination of conditions. Therefore paired falls were primarily limited to only two of the six possible paired combinations with 12 subjects falling in 5-6 conditions, and 15 falling in 3-6 conditions. The six subjects who fell in three conditions were also not randomly distributed among the four possible combinations. Rather all six subjects fell in the same set of three conditions which was the 3-4-5 combination. This combination combines the features of the two most common paired condition falls, 3-4 and 5-6 as reported previously (1,2,11). Most of these subjects were older (aged 65, 68, 66, 69 and 70 y).

There was a clear learning effect when sensory tests were repeated immediately following a fall. Thirty-three of the 131 first test falls were repeated. Only 6 of the 33 subjects (18%) fell in the repeated test. The number of repeat test falls for the four sensory conditions where falls occurred were 1 of 8 for condition 1, 0 of 2 for condition 4, 2 of 8 for condition 5, and 3 of 15 for condition 6.

Age-Related Changes

Figure 3 shows peak-to-peak sway and falls as a function of age. Generally, the number of falls increased with increasing age (Table 5). The incidence of falls was lowest for subjects aged 20-40 y. Subjects aged 13-19 y had a high incidence of single condition falls (33%) but low multiple condition falls (11%)

The lowest falls to peak-to-peak sway data in Figure 3 suggest that younger subjects sway less than subjects in the middle of the age range. A comparison of peak-to-peak sway of 7-15 y olds with 30-60 y olds showed significant differences for conditions 1, 4, 5,

and 6 (P < 0.02, Mann-Whitney nonparametric test). The larger sway of younger subjects was most evident in sensory conditions 4, 5, and 6 suggesting that younger subjects were sensitive to alterations in somatosensory cues.
Age-Related Changes in Posture

elderly fallers apparently either lack information required for postural control or have adopted control schemes that are distinct from the remainder of the population and that place them at increased risk for falls in particular sensory environments. However, when redundant sensory cues are available (conditions 1 and 2) and sway amplitudes are greatly reduced, these fallers cannot be distinguished from the remainder of the population.

A simple explanation for these results could be that the average body alignment of these subjects places their center of gravity near a stability limit so that relatively small increases in sway produce a fall (9). If this explanation were correct, then subjects who fell in condition 3 should also fall in other conditions (4, 5, and 6) that increased their sway above the levels in conditions 1 and 2. In general, this pattern of falls did not occur.

Impairments in either sensory system inputs, central nervous system processing, or motor system output could potentially initiate or facilitate the development of postural control schemes that are generally adaptive (judging from the good performance in conditions 1 and 2) but inadequate or nonadaptive in other sensory environments. These impairments might include reduced or altered sensory information, reduced, delayed, or absent motor responses, or incorrect patterns of muscle activation resulting in inappropriate and noncompensatory responses. Comparisons of sensory organization test results with postural motor coordination results, and VOR and OKR responses give some insight into the factors that contribute to the age-related decline in postural control in this potentially normal population.

Comparison with Motor Coordination Tests

Neither the amplitude nor timing of postural motor responses to forward and backward platform translations (12) were correlated with the level of sway during sensory tests. In addition, the motor response parameters of subjects who fell during sensory tests were not distinguishable from nonfallers. This would tend to support the hypothesis that sensory system deficits and/or inappropriate central nervous system organization of sensory information are responsible for the increased likelihood of falls independent of motor coordination problems.

However, the amplitude of platform translations used in the motor coordination tests did not produce body sways near fall thresholds, and thus did not require subjects to exert maximal muscular responses. A larger perturbation might have revealed relative muscle weaknesses as well as response timing problems within the subpopulation of fallers. Muscle strength relative to body mass and the precise timing of responses might contribute to falls in conditions 3 and 6 where the average level of sway is closer to fall thresholds than in the other four conditions.

Comparison with VOR and OKR Function

There was evidence of VOR and OKR abnormalities in some subjects who fell in two or more conditions. Of the three subjects with the shortest VOR time constants (13, 14) one was a 5-7 faller and two were 3-5 fallers. The 5-7 faller with a short VOR time constant also had a significant partial unilateral loss of vestibular function in the left ear. The subject who had the largest OKR time delay (average delay to the onset of eye movement following visual field movement) of any subject tested (268 ms) was also a 5-7 faller. Among subjects over 50 y, two of the three subjects with the lowest OKR gain constants were 3-5 fallers, and the one who was a 3-6 faller. Finally, the two older subjects with the largest OKR time constants, indicating decreased sensitivity to higher frequency visual field motions, were both 3-6 fallers.

With the exceptions mentioned above, VOR and OKR parameters of most subjects who fell in two or more conditions were not distinguishable from those of subjects who did not fall or fell in only one condition. A comparison of the overall incidence of extreme VOR and OKR parameters (>97.5 or <2.5 percentile points) among subjects who fell in two or more conditions with the incidence of extreme parameters among subjects who fell in no more than one condition showed no significant difference between the groups. There are at least three possible explanations for the weak correlation between VOR and OKR abnormalities and poor postural control. First, our VOR tests measured primarily horizontal OKR function whereas head movements during postural sway primarily stimulate vertical canals and otoliths.

To the extent that a vestibular abnormality may affect one or a limited number of the vestibular receptors in each ear, horizontal VOR and posture results could differ. Second, our OKR tests used horizontal plane visual motion stimuli while the visual system contribution to postural control during sensory tests is associated with the detection of pitch plane movement and with depth cues from the disparity of images on the retinae of each eye. There might be a higher correlation of abnormal pitch plane OKR and vigilance control responses with postural control deficits than with horizontal plane OKR. Third, differences between our VOR and posture test results could relate to central nervous system problems in the organization of sensory system interactions. Abnormalities in the central nervous system pathways involved in the organization of postural control may be specific to the postural control system and therefore would not effect VOR responses.

In conclusion, it is apparent that some equilibrium control deficits exist in a positively normal population. These deficits are more common in children and subjects older than 50 y, but are normally masked by the presence of redundant sources of sensory orientation cues. In susceptible subjects, the loss of redundant information can unmask their deficit and cause a sudden loss of postural control.

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