

NASA-CR-196121

29P

# Relation of Motion Sickness Susceptibility to Vestibular and Behavioral Measures of Orientation

Annual Status Report  
NAGW-3782

N94-37005

Unclas

G3/52 0014091

Robert J. Peterka, Ph.D.

July 1994

(NASA-CR-196121) RELATION OF  
MOTION SICKNESS SUSCEPTIBILITY TO  
VESTIBULAR AND BEHAVIORAL MEASURES  
OF ORIENTATION Annual Status Report  
(Good Samaritan Hospital and  
Medical Center) 29 p

R.S. Dow Neurological Sciences Institute and  
Clinical Vestibular Lab, N010  
Legacy Good Samaritan Hospital  
1040 N.W. 22nd Ave.  
Portland, OR 97210  
(503)229-8154

## Grant Objective

The objective of this proposal is to determine the relationship of motion sickness susceptibility to vestibulo-ocular reflexes (VOR), motion perception, and behavioral utilization of sensory orientation cues for the control of postural equilibrium. The work is focused on reflexes and motion perception associated with pitch and roll movements that stimulate the vertical semicircular canals and otolith organs of the inner ear. This work is relevant to the space motion sickness problem since 0 g related sensory conflicts between vertical canal and otolith motion cues are a likely cause of space motion sickness.

## Experiments

### *Postural responses to a sinusoidal visual motion stimulus*

We have completed a study of the influence of visual surround sinusoidal pitch rotations on postural control in bilateral vestibular loss and normal human subjects. We have submitted a paper to Experimental Brain Research titled "Role of somatosensory and vestibular cues in attenuating visually evoked human postural sway" (a copy is attached), and the results have been presented at two conferences:

- 1) "Transient postural responses induced by visual field motion in normals and bilateral vestibular loss subjects" Peterka, R.J. and Benolken, M.S., Clinical Application of Vestibular Science, UCLA, February 1994. (A copy of the abstract & poster is attached.)
- 2) "Somatosensory and vestibular influences on attenuating visually induced postural sway in humans" Peterka, R.J. and Benolken, M.S., Neural Control of Movement meeting, Hawaii, April 1994. (A copy of the abstract & poster is attached.)

We recently developed mathematical control system models that help to explain some of the observed dynamic responses of visually induced postural sway. We anticipate that these models will help us to quantify how a subject uses various sensory cues for postural control. Dr. Peterka has been invited to present this posture data & related modelling work at the conference "Sensory Interaction in Posture and Movement Control" in Slovak Republic in September 1994 (A copy of the abstract is attached).

### *Investigation of VOR threshold phenomena*

One of the results of the posture work described above was the discovery that normal subjects apparently do not utilize vestibular sensory information for postural control if body motions remain below a specific threshold level. We have looked for evidence of a corresponding VOR threshold phenomenon in both roll and pitch rotations using variable amplitude stimuli at a fixed frequency. In previous studies we identified evidence of a VOR threshold of torsional eye

movements evoked by roll rotations. We recently looked for a threshold phenomenon in vertical eye movements evoked by pitch rotations. No indication of a VOR threshold was identified in these pitch evoked vertical eye movements. Therefore regarding this vestibular threshold phenomenon, there appears to be clear differences in how vestibular sensory information is processed and utilized for the control of different reflex functions. These results were presented at a conference this spring:

"Vestibular threshold effects in ocular and postural reflexes" Peterka, R.J., Mechanisms of Vestibular Function and Dysfunction: A Satellite of the 1994 Neural Control of Movement meeting, Hawaii, April 1994.

*Identification of VOR dumping phenomenon as a test of otolith function and canal/otolith interactions*

Recent experiments by others (primarily the work of Cohen and Raphan in monkeys) have characterized the details of the complex three-dimensional eye movements associated with the VOR "dumping" phenomenon. VOR dumping refers to the fact that the time constant of post-rotatory horizontal nystagmus decay is significantly shortened if a test subject changes head position away from an upright position while the horizontal nystagmus is decaying. In addition, the work in monkeys has shown the vertical and/or torsional eye movements develop as the "dumped" horizontal eye movements decay, indicating that eye movements are inherently organized with reference to the direction of gravity.

Some controversy exists as to whether this phenomena is present in humans. We performed experiments in one subject which did show dumping and its associated build up and decay of cross-axis eye movements. This type of experiment looks like a promising method for obtaining quantitative VOR data related to an individual's spatial orientation function. We are currently in the process of modifying our data collection/stimulus delivery program so that we can deliver the type of stimuli necessary to investigate this phenomena.

## Development

*Modification of two-axis rotation device for off-axis stimulation*

Modification of our two-axis rotation device to provide head off-axis roll and pitch stimulation is completed and functional. We now are able to deliver both head on-axis and head off-axis pitch and roll motions about an earth horizontal axis. In both cases, both the vertical canals and otolith organs are stimulated, but the otolith stimulus is different in the two cases. In the on-axis case, otoliths are stimulated due to head tilt with respect to gravity. In the off-axis case a tangential linear acceleration and a centrifugal acceleration is given to the otoliths in addition to the head tilt with respect to gravity.

*Modification of two-axis rotation device to attach eccentric arm*

We are presently building modifications to our existing two-axis rotation device using funds from the joint NIH/NASA sponsored grant "Otolith/Canal Control Mechanisms in Posture and Movement" (P60-DC02072, PI Barry Peterson, Ph.D. of Northwestern University) and contributions from this NASA grant (NAGW-3782). One of the new configurations allows stimulation of canals and otolith organs simultaneously using eccentric axis rotations. By changing subject orientation with respect to the rotation axis, the canal/otolith stimulation can be either additive or subtractive. A second new configuration includes a linear track that can be either floor-mounted or wall-mounted to provide a pure horizontal or vertical linear acceleration stimulus, respectively. These pure linear accelerations stimulate only the otolith organs. Overall, this new device will be able to produce higher frequency and higher g stimuli than most other devices currently available.

## Appendix

### *Abstracts*

Peterka, R.J. and Benolken, M.S. Transient postural responses induced by visual field motion in normals and bilateral vestibular loss subjects. Presented at Clinical Application of Vestibular Science symposium, UCLA, February 1994 (abstract and poster).

Peterka, R.J. and Benolken, M.S. Somatosensory and vestibular influences on attenuating visually induced postural sway in humans. Presented at Neural Control of Movement meeting, Hawaii, April 1994 (abstract and poster).

Peterka, R.J. Vestibular threshold effects in ocular and postural reflexes. Presented at Mechanisms of Vestibular Function and Dysfunction: A Satellite of the 1994 Neural Control of Movement meeting, Hawaii, April 1994.

Peterka, R.J. and Benolken, M.S. Dynamic interactions of visual, somatosensory, and vestibular orientation cues in postural control. To be presented at Sensory Interaction in Posture and Movement Control conference, Slovak Republic, September 1994 (abstract).

### *Papers*

Peterka, R.J. and Benolken, M.S. Role of somatosensory and vestibular cues in attenuating visually evoked human postural sway. *Exp Brain Res*, submitted June 1994.

TRANSIENT RESPONSES OF VISUALLY INDUCED POSTURAL SWAY IN  
NORMAL AND BILATERAL VESTIBULAR LOSS SUBJECTS. R.J. Peterka and M.S.  
Benolken, Clinical Vestibular Laboratory and R.S. Dow Neurological Sciences  
Institute, Good Samaritan Hospital & Medical Center, Portland, OR 97210

The purpose of this study was to determine the contribution of somatosensory and vestibular sensory cues to the maintenance of stance during visually induced postural disturbances. Visual orientation cues were altered by sinusoidal rotations of a full field visual surround about an axis near the ankle joints. Six subjects had normal vestibular function and three had a profound bilateral loss of vestibular function. Each subject performed 60 second randomized tests on both a fixed surface and sway-referenced surface (surface rotated in equal proportion to the subject's center-of-gravity displacement) with varying frequencies (0.1, 0.2, and 0.5 Hz) and amplitudes ( $\pm 0.2^\circ$ ,  $0.5^\circ$ ,  $1^\circ$ ,  $2^\circ$ ,  $5^\circ$ , and  $10^\circ$ ) of visual field motion. Vertical forces exerted on the support surface were used to calculate the torque over the course of the stimulus. The transient response to the onset of the visual surround motion was analyzed by characterizing the onset time and peak amplitude of the initial torque response for each trial. Transient responses were only characterized for visual stimuli with  $1^\circ$  and greater stimulus amplitudes.

There were qualitative differences between the transient responses induced by the visual field motion of normal and bilateral loss subjects. Bilateral loss subjects showed a consistent pattern of early onset torque responses in the direction of the initial visual field motion. The average onset time was 0.28 s (0.07 s.d.), and all initial torques produced body sways in the direction of the moving visual surround. The bilateral loss subjects' transient responses also were effected by the visual surround velocity. The peak amplitude of the initial torque response for the bilateral loss subjects increased as a function of the stimulus velocity. Larger magnitude peak torques were associated with larger stimulus velocities in both the fixed and sway-referenced conditions.

In contrast, the initial torque responses of normal subjects were less consistent than in those with bilateral loss, but generally showed greater onset delays, greater variability among subjects, and variable directional response. No relationship between the peak torque and stimulus velocity existed for the normal subjects. The average torque onset time was 1.23 s (0.81 s.d.). The direction of the initial torque response appears to be related to the direction of body sway that existed at the onset to the torque response, not to the direction of the visual surround movement. This suggests the visual field motion may have induced a general destabilization resulting in either a forward or backward body sway with the initial torque generated to counteract that sway.

(Work supported by NASA grant NAG-117)

presented at Conference on Clinical Application of Vestibular  
Science, UCLA, Los Angeles, CA, 1994

# TRANSIENT POSTURAL RESPONSES INDUCED BY VISUAL FIELD MOTION IN NORMAL AND BILATERAL VESTIBULAR LOSS SUBJECTS. R. J. Peterka and M. S. Benolken, Clinical Vestibular Laboratory and R. S. Dow Neurological Sciences Institute, Legacy Good Samaritan Hospital, Portland, OR 97210

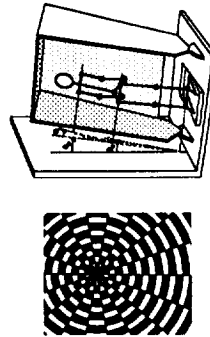
## 1. INTRODUCTION

Visual field motions have been shown to induce postural body sway which generally follows the visual stimulus. This visually induced sway is indicative of the contribution of vision to postural control. In order to understand the interactions of visual and vestibular motion cues on postural control, we characterized the transient postural responses to visual field motion in 6 normal and 3 bilateral vestibular loss subjects.

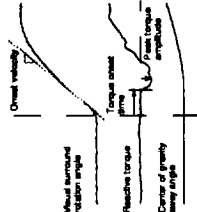
## 2. METHODS

### Stimulus

- Subjects viewed a high contrast, full field visual surround.
- Visual surround was rotated sinusoidally in a sagittal plane at various amplitudes ( $\pm 1^\circ, 2^\circ, 5^\circ, 10^\circ$ ) and frequencies (0.1, 0.2, 0.5 Hz).
- Fixed and sway-referenced support surface conditions were tested.
- Initial visual surround motion was always directed away from subjects.
- Audio cues were masked.



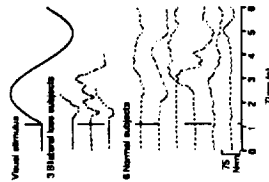
### Measurements



## 3. RESULTS

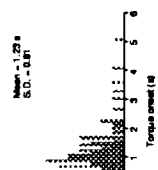
### Example Data

Torque responses at 0.2 Hz,  $\pm 5^\circ$  visual surround amplitude with sway-referenced support surface



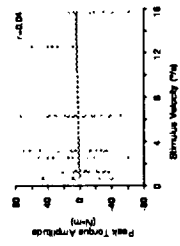
### Normal

• Torque onset times were late and had high variability.



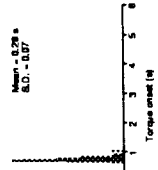
• 43% (42/97) of measurable responses had an initial negative torque direction, causing body sway to follow the visual surround motion. 57% (55/97) of measurable responses had an initial positive torque direction, causing body sway opposite to the visual surround motion.

• No obvious relationship existed between the initial peak torque amplitude and the visual surround onset velocity.



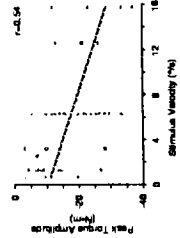
### Bilateral Loss

• Torque onset times were early and had low variability.



• 100% (55/55) of measurable responses had an initial negative torque direction, causing body sway to follow the visual surround motion.

• Initial peak torque amplitude was proportional to the visual surround onset velocity.



## 4. CONCLUSIONS

- Bilateral loss subjects showed atypical, short latency, directed transient postural responses to the onset of a visual field motion. In contrast, initial responses were delayed and appeared to be more related to corrective actions in response to a general, non-directed destabilization evoked by visual motion.
- In normals, vestibular cues may inhibit postural responses in the "sensory conflict" condition caused by visual field motion without corresponding vestibular sensed body sway. The visual motion sensitivity of bilateral loss subjects could also be enhanced by reflex changes which compensate for their vestibular loss.

Supported by NASA grant NAG 9-117

**Somatosensory and vestibular influences on attenuating visually induced postural sway in humans.** R.J. Peterka and M.S. Benolken, Clinical Vestibular Laboratory and R.S. Dow Neurological Sciences Institute, Legacy Good Samaritan Hospital & Medical Center, Portland, OR 97210

This study examined the contribution of somatosensory and vestibular sensory cues to the maintenance of stance during visually induced postural disturbances. Visual orientation cues were altered by sinusoidal rotations of a full field visual surround about an axis near the ankle joints. Support surface conditions were altered (fixed and sway-referenced support surface) to investigate the influence of somatosensory cues. Six subjects had normal vestibular function and three had a profound bilateral loss of vestibular function.

Visually induced body sway in normals showed nonlinear stimulus-response characteristics. A saturation phenomena was present in which increased stimulus amplitudes did not evoke increased body sway. This saturation phenomena was absent in subjects with bilateral vestibular loss, implying a vestibular origin. In addition, comparison of induced sway between normal and bilateral loss subjects suggests the existence of a vestibular-related threshold. That is, vestibular cues did not contribute to the attenuation of body sway which was below a frequency dependent threshold. In contrast, changes in the accuracy of available somatosensory orientation cues resulted in attenuation of visually induced sway by a factor of four in both normals and bilateral loss subjects. This attenuation was independent of the stimulus amplitude and frequency.

*(Work supported by NASA grant NAG 9-117)*

Presented at Neural Control of Movement, Hawaii, 1994



# SOMATOSENSORY AND VESTIBULAR INFLUENCES ON ATTENUATING VISUALLY INDUCED POSTURAL SWAY IN HUMANS.

R. J. Peterka and M. S. Benolken, Clinical Vestibular Laboratory and R. S. Dow Neurological Sciences Institute, Legacy Good Samaritan Hospital, Portland, OR 97210

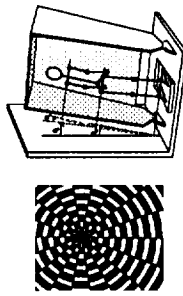
## INTRODUCTION

Visual surround motion has been shown to induce postural sway which is attenuated by the somatosensory and vestibular systems. The visually induced sway is a function of the contribution of vision to postural control. In order to understand the interactions of visual and vestibular motion cues on postural control, we observed visually induced sway in response to surround motion in 6 normal and 7 labyrinthine vestibular loss subjects.

## METHODS

### Stimulus

- Subjects viewed a high contrast, full field visual surround.
- Visual surround was varied sinusoidally in a sagittal plane at various amplitudes (0.2°, 0.5°, 1°, 2°, 5°, 10°) and frequencies (0.1, 0.2, 0.5 Hz).
- The frequency of the visual surround was varied sinusoidally at 0.1, 0.2, 0.5 Hz.
- Audible cues were masked.



### Data Analysis

- Amplitude-posture sway was measured at the hip and shoulder.
- Amplitude-posture sway was used to compare with sway,  $\theta_v$  and sway angle,  $\theta_s$ .
- $\theta_v$  and  $\theta_s$  were used to estimate the center of gravity sway angle,  $\theta_{cg}$  over time.
- Fourier analysis was used to measure  $\theta_{cg}$  amplitude and phase in the visual surround stimulus frequency.



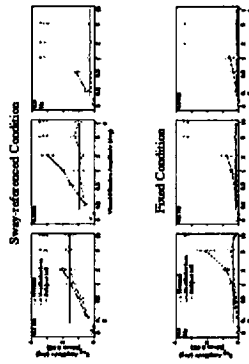
## RESULTS

Sway-related motion amplitudes induced increased sway primarily as the stimulus frequency.



Visually induced sway depended upon:

- Stimulus frequency
- Stimulus amplitude
- Platform condition
- Vestibular function



**Normal**

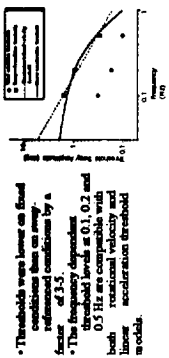
- Induced sway amplitude was similar to low stimulus amplitudes.
- Induced sway amplitude was similar to high stimulus amplitudes.
- Induced sway amplitude was similar to low stimulus amplitudes.
- Induced sway amplitude was similar to high stimulus amplitudes.
- Induced sway amplitude was similar to low stimulus amplitudes.
- Induced sway amplitude was similar to high stimulus amplitudes.

**Vestibular Loss**

- Induced sway amplitude was similar to low stimulus amplitudes.
- Induced sway amplitude was similar to high stimulus amplitudes.
- Induced sway amplitude was similar to low stimulus amplitudes.
- Induced sway amplitude was similar to high stimulus amplitudes.
- Induced sway amplitude was similar to low stimulus amplitudes.
- Induced sway amplitude was similar to high stimulus amplitudes.

1. Normal subjects apparently do not utilize vestibular information at low amplitudes of body sway.
2. Separation of sway with increasing stimulus amplitudes requires the presence of vestibular function.
3. A vestibular related threshold phenomenon is implied by the separation of sway with increasing stimulus amplitudes at low amplitudes of body sway in normal and vestibular loss subjects at low stimulus amplitudes.

Vestibular related threshold levels depended upon the platform conditions.



Threshold was lower on fixed versus sway-referenced conditions by a factor of 3.5.

The frequency dependence of the threshold levels at 0.1, 0.2 and 0.5 Hz was similar in both normal and vestibular loss subjects.

Both normal and vestibular loss subjects showed a lower acceleration threshold in both.

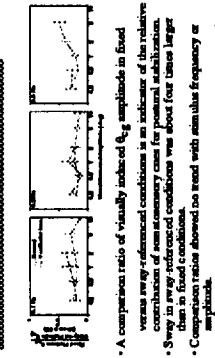
**Rational Velocity Model**

$$\text{Threshold} = \frac{1.0 \text{ Hz}}{\text{Stimulus Frequency}}$$

**Linear Acceleration Model**

$$\text{Threshold} = \frac{0.1 \text{ km/s}^2}{\text{Stimulus Frequency}}$$

Vestibular loss subjects did not utilize somatosensory cues more than normal subjects.



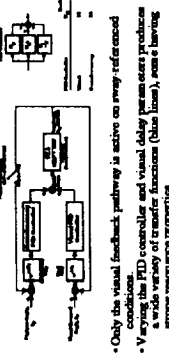
A comparison ratio of visually induced  $\theta_{cg}$  amplitude in fixed versus sway-referenced conditions is an indicator of the relative contributions of somatosensory cues for postural stabilization. Comparison ratios were about four times larger than in fixed conditions.

Comparison ratios showed no trend with stimulus frequency or amplitude.

Comparison ratios were nearly identical in normal and vestibular loss subjects, indicating that the use of somatosensory cues is compensated by altering the use of vestibular cues.

- A comparison ratio of visually induced  $\theta_{cg}$  amplitude in fixed versus sway-referenced conditions is an indicator of the relative contributions of somatosensory cues for postural stabilization.
- Comparison ratios were about four times larger than in fixed conditions.
- Comparison ratios showed no trend with stimulus frequency or amplitude.
- Comparison ratios were nearly identical in normal and vestibular loss subjects, indicating that the use of somatosensory cues is compensated by altering the use of vestibular cues.

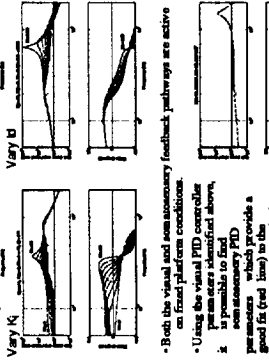
A simple model explains gain and phase responses of vestibular loss subjects to low amplitude visual surround motion.



Only the visual feedback pathway is active on sway-referenced conditions.

Varying the PD controller and visual delay parameters produce a wide variety of transfer functions (blue lines), some having strong resonance properties.

Visual PD controller parameters can be found which provide a good fit (red lines) to the experimental data (green stars), including sway gains greater than unity.



Both the visual and somatosensory feedback pathways are active on fixed platform conditions.

Using the visual PD controller parameters to fit the experimental data, the somatosensory PD parameters which provide a good fit (red lines) to the experimental data (green stars).

- Both the visual and somatosensory feedback pathways are active on fixed platform conditions.
- Using the visual PD controller parameters to fit the experimental data, the somatosensory PD parameters which provide a good fit (red lines) to the experimental data (green stars).

## CONCLUSIONS

- Visual surround motion induced sway in normal and vestibular loss subjects. The visually induced sway is a function of the contribution of vision to postural control and the accuracy of somatosensory cues. When somatosensory cues are accurate, induced sway can be greater than the visual stimulus amplitude.
- A comparison ratio of visually induced sway in normal and vestibular loss subjects is an indicator of the relative contributions of somatosensory cues for postural stabilization. Comparison ratios were about four times larger than in fixed conditions.
- Comparison ratios showed no trend with stimulus frequency or amplitude.
- Comparison ratios were nearly identical in normal and vestibular loss subjects, indicating that the use of somatosensory cues is compensated by altering the use of vestibular cues.

Prepared by Marka, given Marka 117 A, August 1988, at the good Samaritan Hospital.

**Dynamic Interactions of Visual, Somatosensory, and Vestibular Orientation Cues in Postural Control.** R.J. Peterka and M.S. Benolken, Clinical Vestibular Laboratory and R.S. Dow Neurological Sciences Institute, Legacy Good Samaritan Hospital & Medical Center, Portland, OR 97210, USA

This study examined the contribution of visual, somatosensory, and vestibular sensory cues to the maintenance of stance in humans. Visual orientation cues were altered by sagittal plane sinusoidal rotations of a full field visual surround about an axis near the ankle joints. Support surface conditions were altered (fixed and sway-referenced support surface) to investigate the influence of somatosensory cues. The vestibular contribution to postural control was investigated by comparing body sway responses to visual motion stimuli in six subjects with normal vestibular function to the responses in three subjects with profound bilateral loss of vestibular function.

Visually induced body sway in normals showed a saturation phenomenon where increasing the visual stimulus amplitude did not cause increased body sway. A vestibular origin for this saturation phenomenon was implied by its absence in subjects with bilateral vestibular loss. In addition, the similarity in the amplitude of induced sway in normal and bilateral loss subjects for sways below the normals' saturation level suggested the existence of a vestibular-related threshold. That is, vestibular cues did not appear to contribute to the attenuation of visually induced body sway when sway amplitudes were below a threshold level. Threshold amplitudes decreased with increasing frequency in a manner consistent with a constant rotational velocity threshold. A constant rotational velocity threshold might imply a semicircular canal origin. However, over the frequency range tested (0.1 - 0.5 Hz), the threshold data are also compatible with an otolithic origin.

The amplitude of visually induced sway depended upon the availability of accurate somatosensory orientation cues. The amplitude of induced sway was lower in fixed platform conditions than in sway-referenced conditions by a factor of four. This factor of four reduction was independent of the visual stimulus amplitude and frequency, and it occurred equally in normal and bilateral loss subjects. The fact that both normal and bilateral loss subjects showed a nearly identical reduction in sway implies that the bilateral loss subjects did not increase their reliance on somatosensory orientation cues to compensate for their vestibular loss.

In both normal and bilateral loss subjects under sway-referenced support surface conditions, visual stimuli of less than  $\pm 1^\circ$  amplitude consistently evoked body sways which were two to three times the stimulus amplitude. These high gain responses are predicted by a simple inverted pendulum model of body dynamics where the pendulum is stabilized by conventional negative feedback control using visual motion signals (position, velocity, and the integral of position). In this model, neither vestibular nor somatosensory cues contribute to feedback stabilization since sway amplitudes are subthreshold for vestibular detection and the sway-referenced support surface eliminates accurate somatosensory orientation cues.

*(Work supported by NASA grants NAG9-117, NAGW-3782, and NIH grant P60 DC02072)*

"Sensory Interaction in Posture and Movement Control", Slovak Republic, September 1994.

## Abstract

The purpose of this study was to determine the contribution of visual, vestibular, and somatosensory cues to the maintenance of stance in humans. Postural sway was induced by full field, sinusoidal visual surround rotation about the ankle joints at varying frequencies and amplitudes. The influences of vestibular and somatosensory cues were characterized by comparing postural sway in normal and bilateral vestibular absent subjects in conditions that provided either accurate or inaccurate somatosensory orientation information.

In normal subjects, the amplitude of visually induced sway saturated at larger stimulus amplitudes and the saturation amplitude decreased with increasing stimulus frequency. No saturation phenomena was observed in subjects with vestibular loss implying that vestibular cues are responsible for the saturation phenomenon. For visually induced sways below the saturation level, the stimulus-response curves for both normal and vestibular loss subjects were nearly identical implying that (1) normal subjects were not using vestibular information to attenuate their visually induced sway, possibly because sway was below a vestibular threshold level, and (2) vestibular loss subjects did not utilize visual cues to a greater extent than normal subjects; that is, a fundamental change in visual system "gain" was not used to compensate for a vestibular deficit.

For both subject groups, the amplitude of visually induced sway was smaller by a factor of about four in tests where somatosensory cues provided accurate versus inaccurate orientation information. These results imply that (1) the vestibular loss subjects did not utilize somatosensory cues to a greater extent than normal subjects; that is, changes in somatosensory system "gain" were not used to compensate for a vestibular deficit, and (2) the threshold for the use of vestibular cues in normals was

## Role of Somatosensory and Vestibular Cues in Attenuating Visually Induced Human Postural Sway

Robert J. Peterka, Ph.D. and Martha S. Benolken, B.A.

Legacy Good Samaritan Hospital, Clinical Vestibular Laboratory and R.S. Dow  
Neurological Sciences Institute, Portland, OR 97210, USA

Send correspondence to:

Robert J. Peterka  
Legacy Good Samaritan Hospital  
1040 NW 22nd Avenue, N010  
Portland, OR 97210  
(503)229-8154  
(503)274-4944 fax  
peterka@nsi.lhs.org

apparently lower in test conditions where somatosensory cues were providing accurate orientation information.

**Key Words:** postural control – vestibular – somatosensory – vision – human

### Introduction

Moving visual scenes have long been known to induce postural adjustments in human subjects (Berthoz et al. 1979; Brandt et al. 1986). A wide variety of moving visual stimuli have been employed to investigate this phenomenon including tilting rooms (lateral and fore-aft rotations about an axis at the level of the ankle joint (Bles et al. 1983; Bles et al. 1980)), swinging rooms (Lee and Lishman 1975), projected displays simulating a moving visual wall (van Asten et al. 1988), tunnel, floor, or ceiling (Lestienne et al. 1977; Soechting and Berthoz 1979), and visual roll rotations (Dichgans et al. 1972; Clément et al. 1985).

Direct comparisons among studies are difficult because a variety of visual stimuli have been employed and differing techniques have been used for measuring body motion. Most experiments have shown that postural adjustments were in the direction of the visual field motion (Berthoz et al. 1979; Lestienne et al. 1977; Clément et al. 1985; Dichgans et al. 1972; Bles et al. 1983; Bles et al. 1980), but oppositely directed body sways have also been reported in some subjects (van Asten et al. 1988). One consistent finding has been the existence of a saturation effect; that is, increases in the amplitude of the visual field movement cause little or no additional postural sway (van Asten et al. 1988; Lestienne et al. 1977; Clément et al. 1985; Bles et al. 1980). Below the saturation level, postural sway deviations have been shown to be proportional the logarithm of the visual motion amplitude

(Lestienne et al. 1977). The stimulus amplitude at which saturation occurs apparently depends upon the availability of accurate somatosensory and vestibular orientation cues. Standing on a compliant surface (foam), which decreases or disrupts somatosensory cues, increases the amplitude of visually induced sway at saturation (Bles et al. 1980). Patient's with loss of somatosensation due to polyneuropathy also show increased responsiveness to visual motion (Kotaka et al. 1986). In addition, loss of vestibular function results in increased responsiveness to visual motion stimuli in comparison to normal subjects, although this effect is frequency dependent (Bles et al. 1983).

Sustained, constant velocity linear translational and roll rotational visual stimuli have been shown to evoke postural readjustments in the direction of the visual field motion (van Asten et al. 1988; Lestienne et al. 1977; Dichgans et al. 1972). Although the detailed dynamics of the postural readjustments are slightly different for translational versus rotational motions (Clément et al. 1985), both stimuli evoke body sway within 1-2 seconds that shows an exponential rise to a sustained body displacement in the direction of the visual motion. The frequency response functions identified using translational and rotational sinusoidal visual field motions show low-pass characteristics (highest gains at low frequencies and phase lags at higher frequencies) consistent with the dynamic properties seen in response to constant velocity stimuli. These experimental results suggest that visual cues have their most important stabilizing and destabilizing effects at low frequencies of motion (Berthoz et al. 1979), and are involved in the estimation of orientation with respect to gravity (Brandt et al. 1986). However, other experimental results have shown that visual motion cues can also influence short latency (100-150 ms) motor responses to postural perturbations (Nashner and Berthoz 1978; Berthoz et al. 1979) indicating that visual motion cues can contribute more than static orientation information to the postural control system.

The purpose of this study was to clarify the role of vestibular and somatosensory information in human postural control by comparing visually induced sway in normal subjects and bilateral vestibular loss patients standing in environments with and without reliable somatosensory cues. The work addressed four specific questions. First, how are short latency, transient postural reactions to visual motion stimuli influenced by somatosensory and vestibular cues? Second, is the increased susceptibility of vestibular patients to visually induced sway caused by an increase in visual drive to postural control or due to a loss of attenuation of visually induced sway normally provided by the vestibular system? Third, does visually induced sway reveal the presence of vestibular threshold effects? Because the range of body sway amplitudes in quiet stance is near and sometimes below vestibular threshold levels identified in psychophysical experiments (Benson et al. 1986, 1989), the effect of visual motion on postural control may be quite different depending on whether the induced sway is above or below the vestibular threshold. Finally, does visually induced sway indicate whether semicircular canal or otolith mechanisms dominate in the control of sway in quiet stance? The frequency dependent changes of vestibular threshold effects might provide sufficient information to distinguish between a predominant otolith contribution versus a semicircular canal contribution to postural control.

#### Materials and methods (note: this section in small type)

We tested 9 subjects ranging in age from 22 to 67 years. Six subjects (aged 22–45) had normal clinical sensory organization tests of postural control (Peterka and Black 1990) and no history of balance or dizziness complaints. Three subjects (aged 50–67) had bilateral vestibular losses; two were judged to have a profound bilateral loss by the absence of a vestibulo-ocular reflex on rotation tests and severe vestibular

dysfunction pattern on sensory organization tests of postural control (Peterka and Black 1990), and 1 had a severe, but not total bilateral loss as judged by a greatly reduced vestibulo-ocular reflex on rotation tests and severe vestibular dysfunction pattern on sensory organization tests. Sensory organization test results for vestibular loss subjects were well within normal limits on test conditions which did not require the use of vestibular cues for balance.

Subjects were tested on a modified Equitest (NeuroCom, Clackamas, OR) moving posture platform. The visual surround on the posture platform rotates under servo control about an axis which is collinear with the ankle joint axis and is located about 10 cm above and 16 cm forward of the ankle joint position. The visual surround was modified to provide a visually provocative scene. The surface of the visual surround facing the subject consisted of a circular target pattern of concentric 6.5 cm wide rings of alternating black and white sectors. This pattern is similar to the pattern W2 of van Asten et al. (1988). The right and left sides of the visual surround consisted of a checkerboard pattern of alternating black and white rectangles 6.3 by 20.3 cm. Tests were performed in a darkened room with the visual surround illuminated by a fluorescent light attached to the visual surround so the illumination levels remained constant as the visual surround moved. Subjects were instructed to maintain an upright stance with as little sway as possible. White noise was played into headphones to limit auditory cues to visual surround motion. However, we were not able to fully mask auditory and vibration cues during the largest visual surround motions.

Subject anterior-posterior (AP) sway angle was measured by two horizontal rods attached to the subject at the hip and shoulder level. One end of each rod was attached to the subject and the other end to an earth-fixed potentiometer. The AP displacements of the subject's body at the level of the hips and shoulders was calculated using the output of the two potentiometers with appropriate

trigonometric conversions. These measures were used to estimate the displacement of the body center-of-gravity (CG) using a two part body model which assumed the subjects had an average distribution of body mass between the upper and lower body sections (Peterka and Black 1990). The motion of the body was expressed as the angular rotation (in degrees) of the CG point about the ankle joint axis with a positive sign indicating a forward body sway.

Thirty-four tests were performed by each subject in which the visual surround was sinusoidally rotated according to the equation  $s(t) = A \sin(2\pi ft)$  at 3 different frequencies ( $f = 0.1, 0.2$  or  $0.5$  Hz) and 6 different amplitudes ( $A = 0.2^\circ, 0.5^\circ, 1^\circ, 2^\circ, 5^\circ$  or  $10^\circ$ ). All combinations of frequencies and amplitudes were given except  $10^\circ$  at  $0.5$  Hz due to motor limitations. The test sequence was random. Surround rotations with a positive sign indicate movement in a forward direction (away from the subject). The sign of the stimulus amplitude,  $A$ , was always positive and therefore the initial motion of the visual surround was always away from the subject. An integer number of stimulus cycles was presented over a 60 s duration with a 1 s baseline recorded prior to the start of the stimulus. If a subject fell on a test, that test was immediately repeated up to two more times.

Each sinusoidal stimulus was presented under two different support surface conditions: fixed and "sway-referenced". Sway-referencing was performed by rotating the support surface in direct proportion to the subject's lower body sway angle as measured by the sway rod attached at the subject's hip. Sway-referencing maintains the ankle joint angle nearly constant over time (assuming no knee flexion). This greatly reduces the contribution of somatosensory cues associated with ankle joint motions which are normally well correlated with body sway when a subject is standing on a fixed surface. The effectiveness of sway-referencing in reducing the availability of somatosensory cues for postural control is demonstrated by the consistent pendular falls exhibited by the vestibular loss subjects attempting

to stand with eyes closed on a sway-referenced surface. In contrast, subjects with normal vestibular function are able to maintain stance with eyes closed under sway-referenced conditions. The sway-referencing was initiated at the start of sinusoidal visual surround motion. Two of the six sensory organization test trials served as control trials (eyes open with stationary visual surround under fixed and sway-referenced support surface conditions).

Stimulus delivery, sway-referencing, and data sampling were computer controlled at a clock rate of 50/s. Sampled data included visual surround and support surface rotational positions, vertical forces exerted on the support surface, and body sway angles at the level of the hip and shoulder. The vertical forces were used to calculate the torque exerted against the support surface over the course of the stimulus. The transient responses to the onset of the visual surround motion were analyzed by characterizing the onset times and peak amplitudes of the torque responses. Transient responses were only characterized for visual stimuli with  $1^\circ$  and greater stimulus amplitudes. The CG body sway angle time series was used for the analysis of the steady state responses to the visual stimulus. A Fourier analysis of the steady state CG sway angle and the visual surround angle time series was used to calculate the amplitude of body sway and actual visual surround motion, and the phase of body sway relative to the stimulus motion. The first cycle was not included in order to avoid transient responses. The Fourier analysis was performed at the frequency of the visual stimulus motion. Fourier spectra of spontaneous sway (eyes open with no visual surround motion) under both fixed and sway-referenced support surface conditions were also calculated. The spectral amplitudes of spontaneous sway at 0.1, 0.2, and 0.5 Hz served as control data for comparison with visually induced sway amplitudes.

## Results

### *Transient responses*

There were qualitative differences between the transient responses of normal and vestibular loss subjects (Figure 1). Vestibular loss subjects showed a consistent, stereotypical pattern of early onset torque responses with a negative sign (negative directed torques cause forward body sway which is in the direction of the initial visual surround motion). In contrast, the initial torque responses in normal subjects were less consistent than in vestibular loss subjects, but generally showed greater onset delays, greater variability among subjects, and variable sign suggesting that visual surround motion produced a general destabilizing effect that was not directionally specific in normals.

Among vestibular loss subjects, the early onset torque responses as seen in Figure 1 were clearly identifiable in 55 of 66 trials (fixed and sway-referenced conditions combined). The onset times were symmetrically distributed with a mean of 0.28 s (0.07 s.d.). The onset times under sway-referenced conditions were slightly, but not significantly, shorter than under fixed support surface conditions (0.27 s versus 0.29 s). There was no significant change in the torque onset time as a function of the stimulus amplitude or stimulus frequency.

Among the normal subjects, a torque onset time could be identified in 97 of 132 trials. The distribution of onset times was skewed toward longer times with a mean of 1.23 s (0.81 s.d.) and median of 1.06 s. Torque onsets were more difficult to identify on fixed support surface conditions compared to sway-referenced conditions (39 of 66 identifiable on fixed conditions and 58 of 66 on sway-referenced conditions). There was no significant change in the torque onset time as a function of the stimulus amplitude or stimulus frequency.

The peak amplitude of the initial torque response for the vestibular loss subjects did not change as a function of the stimulus frequency, but did change as a function of the velocity of the visual stimulus (Figure 2). Larger magnitude peak torques were associated with larger stimulus velocities in both the fixed and sway-referenced support surface conditions. Under sway-referenced conditions, the measured peak torque could be influenced by the support surface motion since a negatively directed torque produces forward body sway which produces a toe-down support surface rotation that could transiently increase the magnitude of the negative torque. Therefore all measurements of peak torque during sway-referenced trials were limited to trials where the occurrence of the peak torque was before the onset of body sway in response to the torque (peak torque time < sway onset time in 21 of 26 sway-referenced trials). A linear fit provided a reasonable characterization of the relationship between peak torque and stimulus velocity ( $r=0.54$ ). The slope of the linear regression was close to  $-1$  ( $-1.15 \pm 0.26$  s.e.) indicating that a doubling of the stimulus velocity was associated with a doubling of the torque response. However an extrapolation of the linear fit predicted a significant negative torque response at zero stimulus velocity. This prediction suggests that some nonlinear or threshold properties must apply at lower stimulus velocities.

No similar relationship between peak torque and stimulus velocity existed for the normal subjects. In normals the initial torque responses occurred in both positive and negative directions in spite of the fact that the initial visual surround movement was always directed away from the subject. This suggests that there was no direct relationship between the initial torque response and the visual surround motion. Rather, the direction of the initial torque response appeared to be related to the direction of body sway that existed at the onset of the torque response. At the time of onset of the torque response (which was usually delayed relative to the torque onset time of vestibular loss subjects), the normal subjects' body sway

position typically had deviated from the position measured prior to the onset of visual surround motion. This deviation from upright position was about equally distributed between forward and backward body movements. Of the 37 trials where a clear torque onset could be identified, the sway position and velocity were backward in 40 trials and forward in 48 trials. The remaining 9 trials showing no significant deviation from the pre-stimulus body position. In all 88 trials where a clear deviation from upright position had occurred at the time of the torque onset, the direction of the initial torque response was in the direction opposite to the sway position and velocity. That is, negative torques were generated when body position and velocity were in a backward direction and positive torques generated when body position and velocity were in a forward direction. These torques were in the appropriate direction to counteract the body sway at the time of the torque onset. Since the initial visual surround motion was always directed away from the subject, the varying signs of the initial torque responses suggest that the direction of the visual surround motion did not directly influence the direction of the initial torque response. Rather, the visual surround motion appeared to have induced a general destabilization resulting in either a forward or backward body sway with the initial torque generated to counteract that sway.

#### *Steady State Responses*

The visual surround motion induced a steady state body sway that was usually in the same direction as the visual surround motion. The power spectrum of CG sway showed that the visual stimulus produced an overall increase in the amplitude of all frequencies of body sway below about 1 Hz, but that the component of body sway at the frequency of the visual stimulus was particularly enhanced. Figure 3 shows averaged power spectra of CG body sway for both normal and

vestibular loss subjects during a 0.2 Hz sinusoidal rotation at a stimulus amplitude of  $\pm 1^\circ$ . Both the fixed support surface and sway-referenced support surface results are shown. Power spectra of control trials (fixed and sway-referenced) are shown for comparison. At this stimulus frequency and amplitude, the visual surround motion induced about twice the body sway amplitude (four times the power) in vestibular loss subjects as compared to the normal subjects.

The amplitude of visually induced sway in normal and vestibular loss subjects was dependent upon stimulus frequency, stimulus amplitude, and the support surface condition. Figure 4 shows the amplitude of CG body sway at the visual surround stimulus frequency as a function of the visual surround stimulus amplitude for different stimulus frequencies and support surface conditions. Both normal and vestibular loss subjects swayed more in sway-referenced as compared to fixed support surface conditions at any given stimulus amplitude and frequency. Some of the larger amplitude visual surround motions caused consistent falls in vestibular loss subjects. Vestibular loss subjects fell in both fixed and sway-referenced support surface conditions, with falls occurring at lower stimulus amplitudes when the support surface was sway-referenced. Normal subjects did not fall on any trial with fixed support surface conditions. Occasional falls occurred among normals during sway-referenced trials, but the normal subjects were always able to complete the trials upon repetition of the test condition.

*Saturation and threshold phenomena.* Among normal subjects tested under sway-referenced conditions (Figure 4, right column), a saturation in the amplitude of visually induced body sway occurred as the visual stimulus amplitude increased (most evident in the 0.1 Hz sway-referenced data). The saturation level decreased with increasing stimulus frequency. Below the saturation level, body sway increased in proportion to the logarithm of the visual stimulus amplitude. For the



estibular loss subjects there was no saturation effect. That is, visually induced sway increased as a function of the logarithm of the stimulus amplitude until falls occurred at higher stimulus amplitudes.

The amplitude of visually induced sway was similar in normal and vestibular loss subjects up to the point where the normal subjects reached the saturation level. Specifically, the 0.1 Hz sway-referenced data showed similar amplitudes of induced sways in normals and vestibular loss subjects for stimulus amplitudes of 0.2°, 0.5°, and 1.0°, but a clear divergence between normals and vestibular loss subjects with stimulus amplitudes of 2° and above (falls occurred in vestibular loss subjects with 5° and 10° stimuli). At 0.2 Hz, the induced sways were nearly identical for normals and vestibular loss subjects only at the lowest stimulus amplitude of 0.2°, with a clear divergence between the test groups at higher stimulus amplitudes. At 0.5 Hz, visually induced sway in vestibular loss subjects was greater than in normal subjects even at the lowest stimulus amplitude of 0.2°.

Visually induced sway under fixed support surface conditions (Figure 4, left column) showed a similar pattern of frequency and amplitude dependence as in the sway-referenced condition. However, under fixed support surface conditions the amplitudes of visually induced sway were about four times lower than in corresponding sway-referenced conditions. As in the sway-referenced condition, there was a saturation effect in the normal group at higher stimulus amplitudes. For stimuli above the saturation level at any given frequency, there was a clear divergence between the normal and vestibular loss groups in the amplitude of visually induced sway. The vestibular loss group showed increasing sway with increasing stimulus amplitude (with falls occurring at higher stimulus amplitudes). Below the saturation levels, the induced sways were similar in the two groups.

This saturation phenomenon and the correspondence between the sway amplitudes in normal and vestibular loss subjects at low stimulus amplitudes

suggest that a vestibular related threshold effect is present. That is, at sways below a threshold level, normal subjects do not attenuate visually induced sway more than vestibular loss subjects. This implies that normal subjects are not making use of vestibular motion cues at these low sway amplitudes.

The threshold amplitudes can be estimated by measuring the amplitude of body sway where the sway versus stimulus amplitude curves for normal subjects diverge from those of the vestibular loss subjects and saturate. The dotted lines in Figure 4 show that these threshold levels decrease with increasing stimulus frequency. For the sway-referenced data, the threshold estimates are 1.58°, 0.97°, and 0.29° at 0.1, 0.2, and 0.5 Hz, respectively. Under fixed support surface conditions the threshold estimates also decrease with increasing frequency, but the threshold estimates are 3-5 times lower than under sway-referenced support surface conditions. Specifically, the threshold estimates are 0.32°, 0.17°, and 0.10° at 0.1, 0.2, and 0.5 Hz, respectively.

*Response gain and phase.* Visually induced postural responses can also be expressed in terms of response gain (CG response amplitude divided by stimulus amplitude) and phase. In fixed support surface test conditions, the gains for both normal and vestibular loss subjects were less than unity at all stimulus amplitudes and frequencies tested (Figure 5). At a given stimulus frequency, gains of both normal and vestibular loss subjects generally decreased with increasing stimulus amplitude. Because of the saturation phenomenon seen in normal subjects, the gains of normals decreased more rapidly with increasing amplitude than the gains of vestibular loss subjects.

In sway-referenced support surface test conditions, the response gains of vestibular loss subjects were greater than unity at all test frequencies and amplitudes where data were obtained. The gains were greatest at the lowest test amplitude of 0.2° where the average gains were 2.4, 2.9, and 2.6 at frequencies 0.1, 0.2, and 0.5 Hz,

respectively. Normal subjects also had gains greater than or equal to unity at the lowest test amplitude of 0.2° (average gains of 1.9, 2.8, and 1.0 at frequencies 0.1, 0.2, and 0.5 Hz, respectively). The average gains of normals were less than unity at stimulus amplitudes greater than or equal to 2°, 1°, and 0.5° at 0.1, 0.2, and 0.5 Hz, respectively.

The phase of the CG sway angle relative to the stimulus angular position is summarized in Figure 6 for data from trials with stimulus amplitudes of 1° and greater. Phases for both normal and vestibular loss subjects decreased (increased phase lag) with increasing stimulus frequency. At any given frequency and support surface condition, the phases from vestibular loss subjects were advanced relative to the phases from normal subjects. For normals, average phase at 0.1 Hz showed a phase lead on sway-referenced tests and a phase lag on fixed support surface tests. This difference was significant (paired t-test,  $P < 0.05$ ). On 0.2 and 0.5 Hz tests, phases on sway-referenced conditions also showed greater phase lags relative to the fixed condition tests, but neither of these differences was statistically significant. For vestibular loss subjects, average phases from sway-referenced support surface tests were greater than phases from fixed support surface tests at all frequencies. Only the 0.1 Hz phase data difference between fixed and sway-referenced conditions was significant (insufficient data were available at 0.5 Hz to allow a comparison).

*Dependence upon somatosensory cues.* A comparison of subject performance under fixed versus sway-referenced conditions provides information on the extent to which the availability of accurate somatosensory cues decreases visually induced sway. Figure 7 shows a comparison ratio of visually induced sway under fixed and sway-referenced support surface conditions in normal and vestibular loss subjects. This ratio does not show any trend with stimulus frequency or amplitude, and is nearly identical for the normal and vestibular loss groups. The average for normals

over all test conditions was  $0.24 \pm 0.18$  and was  $0.24 \pm 0.14$  for vestibular loss subjects. That is, a decrease in the accuracy of somatosensory cues caused a degradation of postural stability by the same factor for both normal and vestibular loss subjects. This suggests that the vestibular loss subjects have not become more reliant upon somatosensory cues (i.e. have not increased somatosensory gain) to compensate for their vestibular loss.

## Discussion

### *High gains of visually induced sway*

The existence of gains greater than unity for visually induced sway was an unexpected finding. These high gain responses occurred in both normal and vestibular loss subjects under conditions where somatosensory orientation cues were inaccurate (sway-referenced support surface conditions) and at low amplitudes of visual surround motion. The high gains observed in the present study for vestibular loss subjects are consistent with the fact that these subjects would invariably become destabilized when exposed to visual surround motions of only a few degrees of amplitude. For example, all three vestibular loss subjects fell on 0.5 Hz sway-referenced trials with a 2° stimulus amplitude. This stimulus amplitude would not have been sufficient to produce a fall if the gain of visually induced sway were less than unity. The clinical implication is that vestibular loss subjects are extremely vulnerable to falls in visual motion environments, particularly when somatosensory orientation cues are inaccurate.

We are not aware of any description of high gains of visually induced sway in previous studies. This is likely due to the fact that most studies used larger amplitude visual surround motions which evoke proportionally low amplitudes of

body sway (this is consistent with our data at higher stimulus amplitudes). In addition, many studies use center of pressure energy measures which do not permit a calculation of sway gains. However one study (Lee and Lishman 1975) showed an example figure of sway velocity and visual surround stimulus velocity for a subject standing on a thick foam pad and exposed to a low amplitude, 0.25 Hz sinusoidal AP oscillations of a visual surround. Although the stimulus was a linear translational visual surround movement, for comparison purposes this movement would correspond to about a  $0.1^\circ$  visual surround tilt about the ankle joint. The induced sway velocity was clearly greater than the stimulus velocity with an estimated gain of about 1.5.

#### *Saturation and attenuation phenomena*

Body sways of normal subjects induced by visual surround motion showed nonlinear stimulus-response relations and saturation phenomenon such that increasing amplitudes of visual surround motion did not evoke increasing body sway. In subjects with bilaterally absent vestibular function, this saturation phenomenon was completely absent. In vestibular loss subjects, increasing visual surround motion induced increasing body sways resulting in consistent falls at larger stimulus amplitudes even when accurate somatosensory orientation cues were present (fixed support surface conditions). This implies that the sensory cues which cause this saturation phenomenon are of vestibular origin.

The dynamic properties of the vestibular contribution to the attenuation of visually induced sway were very different from the somatosensory contribution. For both normal and vestibular loss subjects, the availability of accurate somatosensory cues (fixed support surface conditions) resulted in a proportional decrease in visually induced sway in comparison to test conditions with inaccurate

somatosensory cues (sway-referenced conditions). This somatosensory attenuation effect was independent of the stimulus amplitude and frequency and resulted in a four fold decrease in sway in fixed versus sway-referenced conditions for both normal and vestibular loss subjects. In contrast, the saturation phenomenon associated with the availability of vestibular cues showed specific changes as a function of the stimulus frequency and amplitude (the saturation sway amplitude decreased with increasing frequency).

A saturation phenomenon has very different functional consequences than a simple gain attenuation effect. If a subject is exposed to an environment with inaccurate visual orientation cues, the availability of an additional sensory orientation cue which decreases visually induced sway gain can decrease the likelihood of a fall, but cannot prevent a loss of balance. That is, a large amplitude visual motion stimulus can always overcome the decreased gain. In contrast, a saturation effect can completely prevent a loss of balance independent of the visual stimulus amplitude as long as the body sway amplitude at saturation is within the normal stance range.

#### *Threshold phenomenon*

A comparison of the visually induced sway amplitude in normal and vestibular loss subjects showed that the induced sway in normals was indistinguishable from vestibular loss subjects until some critical, or threshold level of body sway was reached. This implies that normal subjects were not making use of vestibular motion information at low body sway amplitudes. Since head motions were not measured in these experiments, this threshold phenomenon cannot be directly attributed to the vestibular system alone. If there were changes in head position with respect to the trunk during sway, then the observed threshold effects might be

related to a more complex interplay between vestibular receptors and proprioceptive head motion information from cervical afferents. Others have shown that head orientation in space tends to be stabilized during various locomotor tasks (Pozzo et al. 1990), therefore reducing and altering the angular and linear motion components seen by the vestibular system. However, the various locomotor tasks which have been studied (Pozzo et al. 1990; Grossman et al. 1988; Grossman et al. 1989) produce head motions which are significantly larger than vestibular threshold levels derived from psychophysical measures (Benson et al. 1989; Benson et al. 1986). In contrast, the amplitude of visually induced body motions in the current study are considerably smaller and would be expected to produce head motions near vestibular threshold levels even if the head were fixed to the body.

If it is assumed that vestibular responses are the primary contributor to the observed threshold phenomenon, then the change in threshold levels as a function of frequency, and the amplitude of thresholds in comparison to psychophysically derived thresholds might suggest the receptor source responsible for the threshold phenomenon (i.e. semicircular canal vs. otolith). Figure 8 summarizes the threshold levels derived from postural data under both fixed support surface and sway-referenced conditions as a function of the stimulus frequency. In addition four different predictions of threshold levels (i.e. saturation amplitudes of body sway) are given. The predictions are extended to both lower and higher frequencies than the postural data.

The first threshold prediction (dotted line in Figure 8) assumes a constant rotational velocity threshold independent of frequency. For a constant velocity threshold, the predicted threshold amplitude is given by:

$$A_{p1} = K_1 / \omega$$

where  $\omega$  is the radian frequency of a sinusoidal movement. A constant velocity threshold level of  $K_1 = 1.03^\circ/s$  provides the best fit of this equation to the data obtained under the sway-referenced conditions.

This threshold amplitude of  $1.03^\circ/s$  amplitude ( $2.06^\circ/s$  peak to peak) is compatible with angular whole-body movement threshold levels identified using psychophysical methods. The results of Benson and coworkers are among the most comparable to the results of this paper since they identified thresholds for rotations about various body axes using sinusoidal waveforms. Using a raised cosine stimulus with 3.3 s duration, Benson and coworkers identified threshold levels of  $2.07^\circ/s$  (Benson et al. 1989) and  $2.25^\circ/s$  (Benson and Brown 1992) for pitch axis rotations about an earth vertical axis in the dark. Similar threshold levels have been identified for yaw and roll rotations (Benson et al. 1989). Mergner and coworkers (1991) using continuous sinusoidal yaw axis rotations identified an average threshold of  $1.3^\circ/s$  ( $2.6^\circ/s$  peak to peak) at 0.4 Hz. Both groups have demonstrated that the threshold levels, when expressed in terms of angular velocity, are relatively constant over the range of test frequencies used in this study ( $0.1-0.5$  Hz) and increase slightly at lower stimulus frequencies. This pattern of threshold shifts are compatible with the dynamic properties of the semicircular canals.

A velocity threshold with an amplitude compatible with psychophysically derived rotational velocity thresholds provides a good prediction of the posturally derived thresholds under the sway-referenced condition, but not under the fixed support surface conditions. The constant velocity threshold amplitude would need to be reduced by a factor of about 4 in order to provide a good match to the fixed support surface threshold measures. However, a factor of 4 reduction would make the threshold amplitudes derived from the fixed support surface data incompatible with psychophysically derived rotational velocity threshold measures. In addition,

extending the constant velocity threshold prediction to lower stimulus frequencies leads to the prediction that very large amplitude body sways would be induced in normal subjects by low frequency visual surround motions. If the constant velocity threshold applies to low frequencies, one would predict that falls would occur under sway-referenced conditions with  $\pm 5^\circ$ – $10^\circ$  visual surround motions below about 0.05 Hz.

The second threshold prediction (dashed line in Figure 8) assumes a linear acceleration threshold which is independent of frequency. If the otolith organs were the sensors of the threshold linear acceleration, otolith afferents with polarization vectors oriented in a nasal or occipital direction would be most sensitive to the linear accelerations produced by the AP body sways. The linear acceleration components acting along these polarization vectors are the tangential acceleration  $a_t = R\alpha$  and the linear acceleration due to change in head orientation with respect to gravity,  $g \sin\theta$ , where  $\alpha$  is the angular acceleration of the body,  $R$  is the distance of the otolith organs from the rotation axis (presumably the ankle joint),  $\theta$  is the rotation angle, and  $g$  is the acceleration due to gravity. For small amplitude sinusoidal rotations, the peak linear acceleration amplitude is  $A\omega^2R + gA$  where  $A$  is the peak angular displacement of the sinusoidal sway and  $\omega$  is the sinusoidal radian frequency. The predicted threshold sway amplitude (in radians) as a function of frequency is given by:

$$A_{p2} = K_2 / (\omega^2R + g)$$

A value of  $K_2 = 0.20 \text{ m/s}^2$  provides the best fit of this equation to the data obtained under the sway-referenced conditions. However the threshold predictions given by  $A_{p2}$  (dashed line in Figure 8) do not provide a good match to the thresholds derived

from the sway-referenced data since the predicted  $A_{p2}$  thresholds show less decline with increasing frequency than the posturally derived threshold data.

Benson and coworkers (1986) showed that psychophysically derived linear acceleration thresholds decreased with increasing frequency at a rate of 5.1 dB/decade (0.26 log units/decade) in a manner consistent with the dynamic response properties of irregular otolith afferents (Fernandez and Goldberg 1976). The predicted threshold sway amplitude which accounts for this change in threshold with frequency is given by:

$$A_{p3} = K_3 (\omega/\omega_r)^{-0.26} / (\omega^2R + g)$$

where  $\omega_r$  is a reference frequency of 2.07 rad/s (0.33 Hz) selected for convenience to facilitate comparisons with psychophysical results (Benson et al. 1986). A value of  $K_3 = 0.18 \text{ m/s}^2$  provides the best fit of this equation to the data obtained under the sway-referenced conditions (solid line in Figure 8). The  $A_{p3}$  threshold prediction provides a good fit to threshold measures derived from the sway-referenced posture data.

It is not possible to distinguish which is the better fit; the constant velocity threshold prediction,  $A_{p1}$ , or the frequency corrected constant acceleration threshold prediction,  $A_{p3}$ , to the sway-referenced data over the frequency range of 0.1 to 0.5 Hz. Therefore the available data cannot be used to differentiate between a possible semicircular canal versus an otolithic origin for the observed threshold phenomena. However the two predictions projected to lower test frequencies predict very different results. At frequencies lower than 0.1 Hz, the linear acceleration sensed by the otolith organs is dominated by the gravitational component which is proportional to head tilt. Therefore the predicted body sway amplitudes induced by visual surround motion show only a small increase with

sources including inherent variability of vestibular afferent activity (Goldberg and Fernandez 1971), vestibular responses to head motions due to the inherent instability of the head-neck system (Goldberg 1992), and vestibular responses to spontaneous body sways. The levels of spontaneous body sway are lower under fixed compared to sway-referenced support surface conditions (Figure 3). In addition, the introduction of a moving visual stimulus increases body sways at frequencies other than the stimulus frequency. Vestibular "noise" is therefore greatly increased in conditions where visual and somatosensory orientation cues are inaccurate. If active control were initiated only when a vestibular signal rose above the level of vestibular noise, then one would expect to identify larger effective thresholds in conditions of increased noise (sway-referenced versus fixed conditions).

Other evidence of threshold effects in postural control have recently been observed. Collins and De Luca (1993) analyzed center of pressure time series data recorded during quiet stance. Their results suggested that short term postural fluctuations were not controlled by closed-loop mechanisms until some systematic threshold was exceeded. The existence of a threshold for the use of vestibular information might contribute to the open-loop/closed-loop control strategy identified by Collins.

#### *Compensation for Vestibular Loss*

One might expect that well compensated vestibular loss subjects would adjust to their vestibular loss by altering the way in which somatosensory and/or visual cues are used for postural control. That is, the appropriate strategy for using somatosensory and visual sensory information for balance control might be different when vestibular cues are not available, and central mechanisms might

decreasing frequency (this small increase is due principally to the 5.1 dB/decade increase in threshold levels with decreasing frequency). In contrast, a constant angular velocity threshold predicts a large increase in sway with decreasing frequency.

A linear acceleration threshold value of  $K_3 = 0.18 \text{ m/s}^2$  is 2.9 times larger than the psychophysically derived threshold of  $0.0625 \text{ m/s}^2$  at 0.33 Hz for AP linear accelerations (Benson et al. 1986). The predicted threshold sway amplitudes based on the  $0.0625 \text{ m/s}^2$  linear acceleration threshold, and corrected for a 5.1 dB/decade threshold decrease with increasing frequency, is shown in Figure 8 (dash-dot line). These predicted thresholds are similar to, but slightly larger than the threshold levels derived from the fixed support surface posture data.

What is the cause of the four fold threshold shift associated with different support surface conditions? One could speculate that the central postural system actively adjusts these thresholds to optimize balance control under varying environmental conditions. For example, when visual and somatosensory orientation cues are absent or inaccurate, it may be advantageous to increase vestibular thresholds in order to avoid vestibular initiated control actions caused by small imbalances or asymmetries of peripheral vestibular function. When other accurate sensory orientation cues are present, these other cues might serve as a reference for vestibular signals so that vestibular initiated control actions could occur at lower levels of body sway. This could possibly be analogous to the observation that angular motion perception thresholds are lower in experimental situations which evoke the oculogyral illusion in comparison to perceptual thresholds measured in complete darkness (Benson and Brown, 1989; Clark and Stewart, 1968).

Alternatively, the apparent vestibular threshold shifts might be due to changes in the signal-to-noise ratio of vestibular signals. Vestibular "noise" has several

If one were to compare the sway amplitudes in normal and vestibular loss subjects during larger amplitude visual motion stimuli, one might conclude that visual motion sensitivity was increased in vestibular loss subjects. This increased sensitivity might be the result of central compensatory adjustments for the loss of vestibular function. However the data in Figure 4 indicate that low amplitude visual surround movements induced approximately equal body sway amplitudes in both normals and vestibular loss subjects. This close correspondence between the levels of induced sway in normal and vestibular loss subjects at low stimulus amplitudes suggests that the apparent increase in visual sensitivity in vestibular loss subjects is likely due to the absence of vestibular suppression of postural sway rather than to a fundamental increase in sensitivity to visual motion.

Vestibular suppression might also explain the less robust transient postural responses seen in normals in comparison to vestibular loss subjects. As with visual-vestibular interactions associated with the control of eye movements, one would expect there to be a tight coupling between the postural influences of visual and vestibular motion cues since both motion receptors reside in the same rigid structure. The onset of visual surround motion results in conflicting visual and vestibular motion cues in normal subjects because the visual system indicates body motion is occurring while the vestibular receptors do not. The discord between the visual and vestibular cues could cause suppression of the visually induced postural responses.

If vestibular cues are not available, then visually induced sway is not inhibited, particularly when somatosensory orientation cues are inaccurate. In the experimental conditions tested, this unsuppressed visual influence was destabilizing for vestibular loss subjects. However in most common situations where the visual world is not in motion, a larger effective visual influence could provide some degree of substitution for missing vestibular information. The time

adapt to achieve a more optimal utilization of the available visual and somatosensory sensory cues.

Bles et al. (1983) tracked visually induced lateral body tilts over time in one patient following a bilateral loss of vestibular function. The results generally showed decreases in the amplitude of induced sway over time which suggested that somatosensory cues were becoming more effective in attenuating the visually induced sway. However the attenuation was frequency dependent with the greatest attenuation changes over time occurring at the lowest test frequency (0.025 Hz), and no attenuation occurring at the highest (0.2 Hz). Our test frequencies corresponded to the upper range of frequencies used by Bles et al. (1983). At these higher frequencies we also were not able to identify compensatory changes in the use of somatosensory cues in vestibular loss subjects.

If vestibular loss subjects compensated by increasing their sensitivity to somatosensory cues for balance control (i.e. if they had a higher gain than normals), then the loss of somatosensory cues or a decrease in accuracy of those cues should have a proportionally larger effect on their balance than it does on normal subjects. Figure 7 shows that this was not the case since both normal and vestibular loss subjects had nearly identical factor of four reductions of sway in the fixed versus the sway-referenced condition. This suggests that the vestibular loss subjects have not experienced a change in sensitivity to somatosensory cues as a result of their loss of vestibular function over the frequency range tested. As mentioned above, Bles et al. (1983) have identified compensatory changes at lower test frequencies and in more dynamic settings. Specifically Bles and coworkers (1984) showed increased amplitudes of nystagmus during "apparent stepping around" stimuli in subjects with bilateral vestibular loss compared to normals. Perhaps compensatory changes in somatosensory sensitivity are only necessary for specific types of postural or locomotor activities.

delays in the processing of visual information might partially limit the effectiveness of this substitution. However since postural movements are relatively slow due to body mechanics, a substitution of visual for vestibular motion information could be more effective in postural control than in eye movement control.

*Acknowledgements.* This study was supported by NASA grants NAG9-117 and NAGW-3782.

#### References

- Benson AJ, Brown SF (1989) Visual display lowers detection threshold of angular, but not linear, whole-body motion stimuli. *Aviat Space Environ Med* 60:629-633
- Benson AJ, Brown SF (1992) Perception of liminal and supraliminal whole-body angular motion. In: Berthoz A, Graf W, Vidal PP (eds) *The head-neck sensory motor system*. Oxford University Press, New York, pp 483-487
- Benson AJ, Hutt ECB, Brown SF (1989) Thresholds for the perception of whole body angular movement about a vertical axis. *Aviat Space Environ Med* 60:205-213
- Benson AJ, Spencer MB, Stott JRR (1986) Thresholds for the detection of the direction of whole-body, linear movement in the horizontal plane. *Aviat Space Environ Med* 57:1088-1096
- Berthoz A, Lacour M, Soechting JF, Vidal PP (1979) The role of vision in the control of posture during linear motion. *Prog Brain Res* 50:197-210
- Bles W, Kapteyn TS, Brandt T, Arnold F (1980) The mechanism of physiological height vertigo II. Posturography. *Acta Otolaryngol (Stockh)* 89:534-540
- Bles W, de Jong JMBV, de Wit G (1983) Compensation for labyrinthine defects examined by use of a tilting room. *Acta Otolaryngol (Stockh)* 95:576-579
- Bles W, de Jong JMBV, de Wit G (1984) Somatosensory compensation for loss of labyrinthine function. *Acta Otolaryngol (Stockh)* 97:213-221
- Brandt T, Paulus W, Straube A (1986) Vision and posture. In: Bles W, Brandt T (eds) *Disorders of posture and gait*. Elsevier Science Publishers B.V., New York, pp 157-175
- Clark B, Stewart JD (1968) Comparison of sensitivity for the perception of bodily rotation and the oculogyral illusion. *Perception & Psychophysics* 3:253-256



- Clément G, Jacquin T, Berthoz A (1985) Habituation of postural readjustments induced by motion of visual scenes. In: Igarashi M, Black FO (eds) Vestibular and visual control on posture and locomotor equilibrium. Karger, Basel, pp 99--104
- Collins JJ, De Luca CJ (1993) Open-loop and closed-loop control of posture: A random-walk analysis of center-of-pressure trajectories. *Exp Brain Res* 95:308--318
- Dichgans J, Held R, Young LR, Brandt T (1972) Moving visual scenes influence the apparent direction of gravity. *Science* 178:1217--1219
- Fernandez C, Goldberg JM (1976) Physiology of peripheral neurons innervating otolith organs of the squirrel monkey. III. Response dynamics. *J Neurophysiol* 39:996--1008
- Goldberg J (1992) Nonlinear dynamics of involuntary head movements. In: Berthoz A, Graf W, Vidal PP (eds) The head-neck sensory motor system. Oxford University Press, New York, pp 400--403
- Goldberg J, Fernandez C (1971) Physiology of peripheral neurons innervating semi-circular canals of the squirrel monkey. I. Resting discharge and response to constant angular accelerations. *J Neurophysiol* 34:635--660
- Grossman GE, Leigh RJ, Abel LA, Lanska DJ, Thurston SE (1988) Frequency and velocity of rotational head perturbations during locomotion. *Exp Brain Res* 70:470--476
- Grossman GE, Leigh RJ, Bruce EN, Huebner WP, Lanska DJ (1989) Performance of the human vestibuloocular reflex during locomotion. *J Neurophysiol* 62:264--272
- Kotaka S, Croll GA, Bles W (1986) Somatosensory ataxia. In: Bles W, Brandt T (eds) Disorders of posture and gait. Elsevier Science Publishers B.V., New York, pp 178--183
- Lee DN, Lishman JR (1975) Visual proprioceptive control of stance. *J Human Movement Studies* 1:87--95

- Lestienne F, Soechting J, Berthoz A (1977) Postural readjustments induced by linear motion of visual scenes. *Exp Brain Res* 28:363--384
- Mergner T, Siebold C, Schweigart G, Becker W (1991) Human perception of horizontal trunk and head rotation in space during vestibular and neck stimulation. *Exp Brain Res* 85:389--404
- Nashner LM, Berthoz A (1978) Visual contribution to rapid motor responses during posture control. *Brain Res* 150:403--407
- Peterka RJ, Black FO (1990) Age-related changes in human posture control: Sensory organization tests. *J Vest Res* 1:73--85
- Pozzo T, Berthoz A, Lefort L (1990) Head stabilization during various locomotor tasks in humans. *Exp Brain Res* 82:97--106
- Soechting JF, Berthoz A (1979) Dynamic role of vision in the control of posture in man. *Exp Brain Res* 36:551--561
- van Asten WNJC, Gielen CCAM, Denier van der Gon JJ (1988) Postural adjustments induced by simulated motion of differently structured environments. *Exp Brain Res* 73:371--383

## Figure Legends

Fig. 1. Representative transient corrective torque traces at the start of visual surround motion in normals and vestibular loss subjects. Stimulus is a 0.2 Hz,  $\pm 5^\circ$  sinusoidal visual surround rotation initially moving away from the subjects who were standing on a sway-referenced support surface. Reactive torques generated by the 3 vestibular loss subjects and the 6 normals are shown for this particular stimulus. Downward deflection of the torque traces indicates increased force exerted on the heel of the foot relative to the toes. Histograms show the distribution of onset times of the initial torque transient in vestibular loss subjects (top right) and normals (bottom right) measured from the start of visual surround motion.

Fig. 2. Peak transient torque amplitude as a function of peak visual surround velocity for the 3 vestibular loss subjects. Data is from both fixed and sway-referenced support surface conditions.

Fig. 3. Power spectra of CG sway induced by visual surround motion in vestibular loss (thin solid lines) and normal subjects (dotted lines) under fixed support surface conditions (left) and sway-referenced conditions (right). Visual surround motion was  $\pm 1^\circ$  at 0.2 Hz. Power spectra of control trials (stationary visual surround) with both fixed and sway-referenced support surface conditions are shown for comparison (thick solid lines).

Fig. 4. Average CG sway amplitude as a function of visual surround amplitude under fixed (left column) and sway-referenced (right column) conditions at three frequencies of visual surround motion. The +s indicate the number of vestibular loss subjects who fell during a given trial. Data points (mean  $\pm 1$  standard error) are only plotted if all 3 vestibular loss subjects were able to complete a given trial. Dashed lines show the average amplitude of saturated sway in the normal subjects.

Fig. 5. Average gain (ratio of CG sway amplitude to visual surround amplitude) of visually induced CG sway as a function of visual surround amplitude at three frequencies of visual surround motion.

Fig. 6. Average response phase (CG sway angle relative to visual surround angle) for normal and vestibular loss subjects in fixed and sway-referenced conditions. Phase data are from responses to completed trials with visual surround amplitudes of  $1^\circ$  and greater with the exception of the 0.5 Hz, sway-referenced data from vestibular loss subjects which are from trials with  $0.5^\circ$  amplitude.

Fig. 7. Ratio of average CG sway obtained on fixed and sway-referenced trials as a function of visual surround amplitude.

Fig. 8. Threshold levels derived from normal subjects' body sway saturation amplitudes for fixed (solid circles) and sway-referenced (solid squares) conditions as a function of stimulus frequency. Curves Ap1, Ap2, and Ap3 are least squared error fits of three different models to the sway-referenced threshold data. Curve Ap4 is the predicted threshold sway amplitude based upon psychophysically derived linear acceleration detection thresholds.

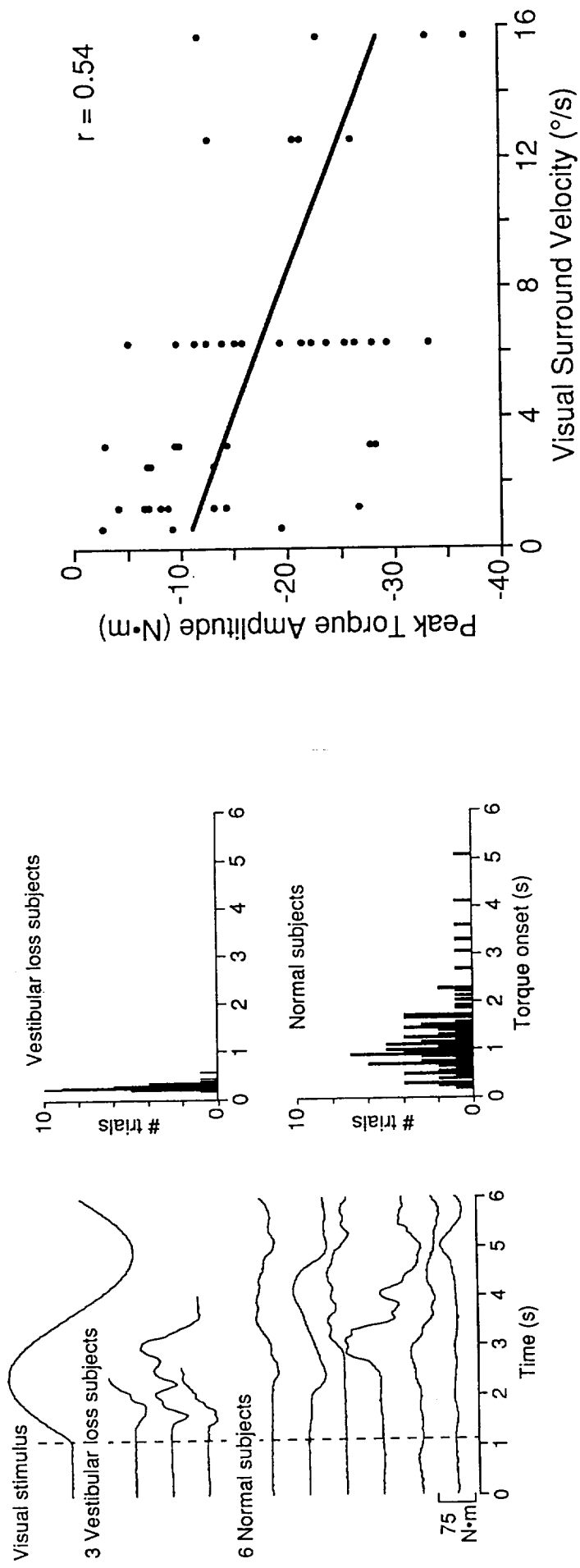


Figure 1

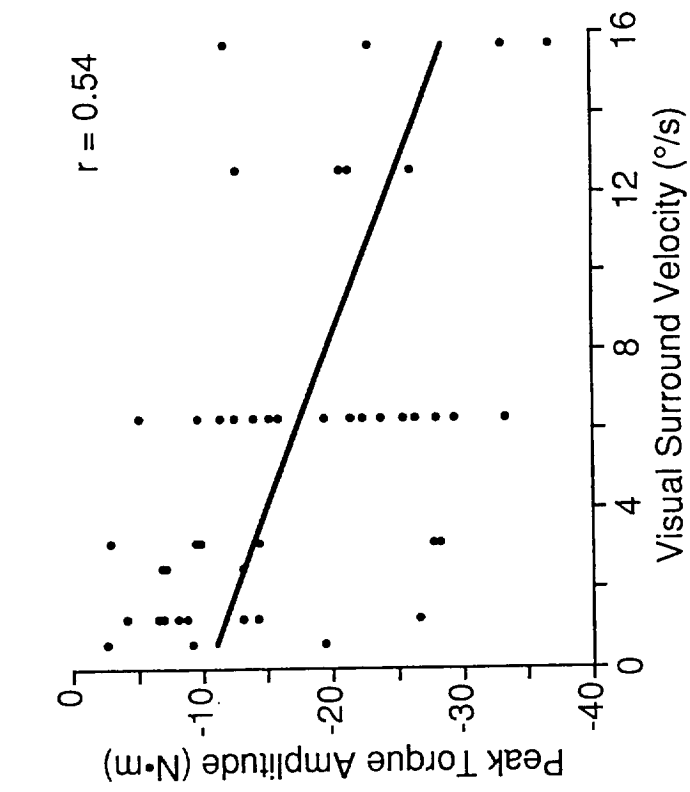


Figure 2

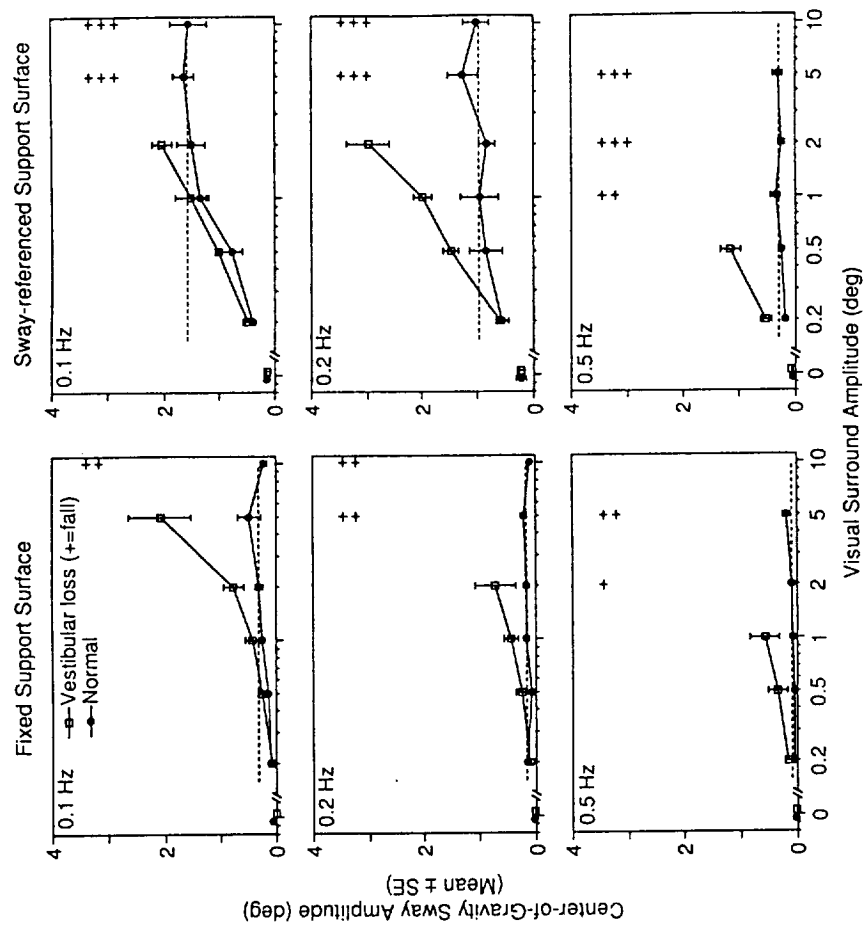


Figure 4

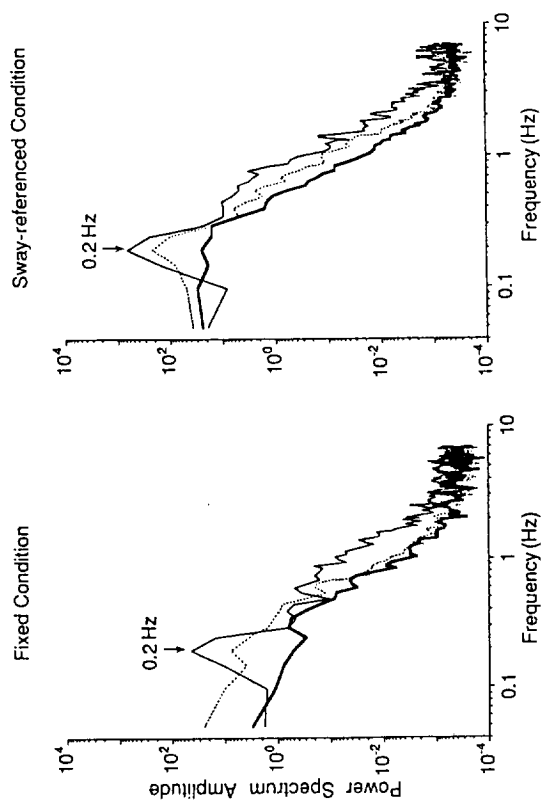


Figure 3

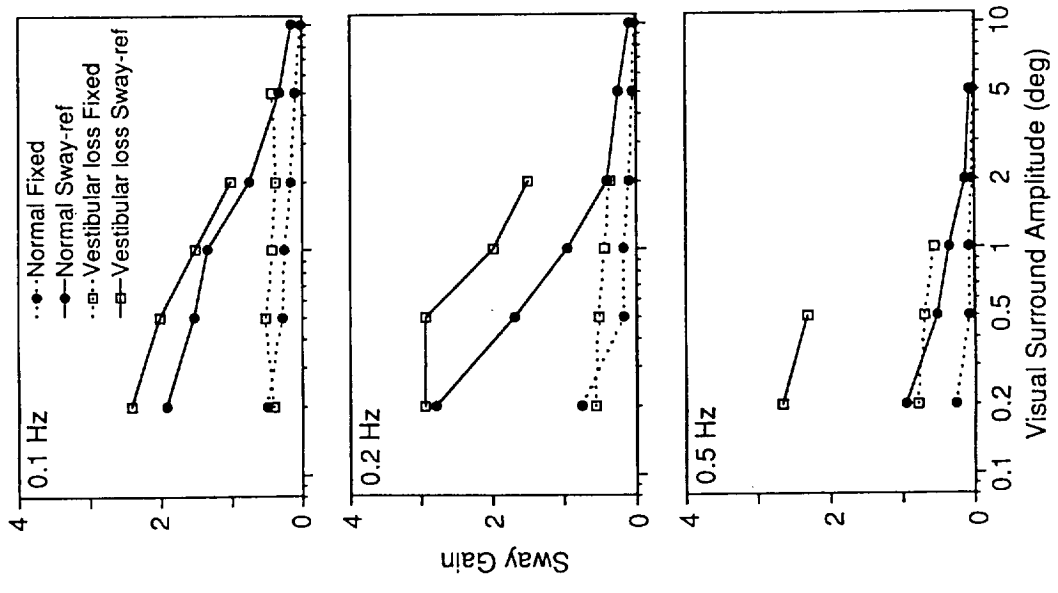


Figure 5

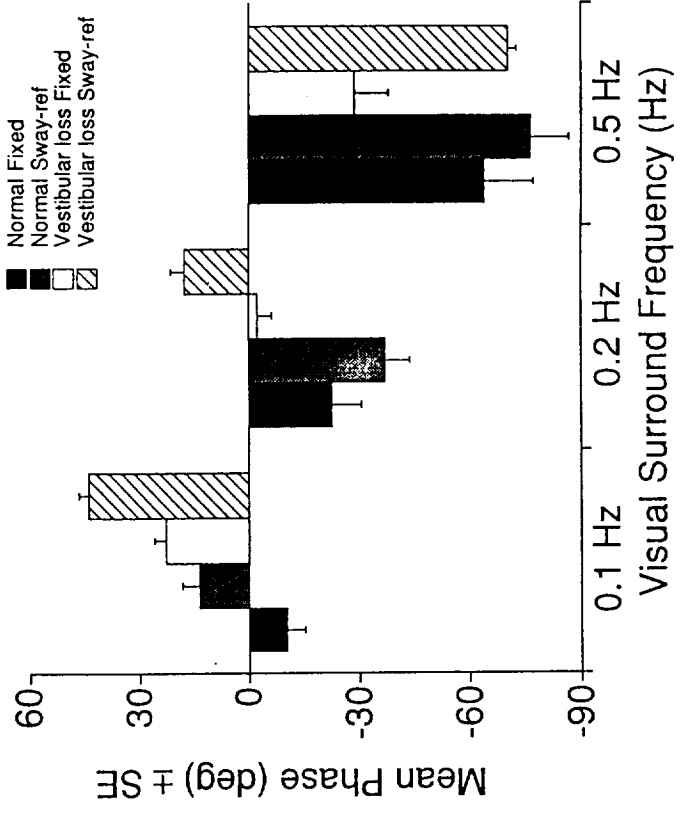


Figure 6