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The Adaptive Effects Of Virtual Interfaces: Vestibulo-Ocular Reflex and Simulator Sickness

by

Mark Heider Draper

A dissertation submitted in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

University of Washington

1998

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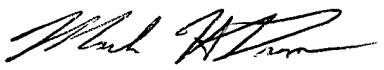
Signature 

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Abstract

The Adaptive Effects Of Virtual Interfaces: Vestibulo-Ocular Reflex and Simulator Sickness

by Mark H. Draper

Chairperson of the Supervisory Committee: Professor Thomas A. Furness III
Department of Industrial Engineering

Current virtual interfaces imperfectly simulate the motion dynamics of the real world. Conflicting visual and vestibular cues of self-motion are believed to result in vestibulo-ocular reflex (VOR) adaptations and simulator sickness, which raises health and safety issues surrounding virtual environment (VE) exposure.

Four experiments were conducted to examine the effects of conflicting visual-vestibular cues through employment of typically occurring virtual interface scenarios. Subjects were exposed for 30 minutes to a head-coupled virtual interface, completing visual search tasks using active, unrestricted head movement rotations.

It was hypothesized that commonly occurring visual-vestibular stimulus rearrangements within virtual interfaces would induce VOR gain and phase adaptation as well as increased simulator sickness. System time delays (125, 250 ms) were studied, as were virtual image scale-factor changes (0.5X, 1.0X, 2.0X magnification) generated by varying geometric field-of-view angle. A secondary hypothesis predicted a moderate correlation between VOR gain adaptation level and simulator sickness magnitude, assuming the existence of an individual 'adaptability trait'. VOR metrics included averaged horizontal VOR gain and phase estimates while simulator sickness metrics included oral ratings during exposure and a simulator sickness questionnaire (pre-exposure, post-exposure, and 20 min post-exposure).

Results demonstrated that significant VOR gain adaptation occurred only in the 0.5X and 2.0X image scale conditions. VOR gain decreased an average of 15% in the 0.5X condition and increased an average of 6% in the 2.0X condition. Simulator sickness magnitude in these two conditions was over twice that of the 1.0X condition.

Introducing time delays of 125 ms and 250 ms to the 1.0X condition resulted in significant VOR gain reductions (average 8.5%) and increases in phase lag (average 2.6 deg), though there were no significant differences between these two delay conditions. Additionally, there was no significant increase in sickness with increasing time delays above the minimum delay condition (46 ms). Only weak correlations were found between VOR gain adaptation and simulator sickness.

Other issues examined include visual-VOR performance within VEs, frequency specificity of VOR adaptation, head movement characteristics, and VOR adaptation and re-adaptation time-course. Virtual interface design guidelines are suggested to minimize unwanted interface-generated effects on the user.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

GLOSSARY

adaptability: a hypothesized individual characteristic that governs one's overall ability to adapt to sensory rearrangements (Reason & Graybiel, 1972).

adaptation: the fitting or adjustment to new conditions.

artifact: an undesired sensory distortion found in virtual interfaces.

ataxia: lack of muscular coordination; used in this document to indicate postural instability.

Cauchy-Schwarz Inequality: a mathematical relation which states that the upper limit for predictive validity is the geometric mean of the criterion reliability and the predictor reliability. In equation form: $r_{xy} \leq (r_{xx} * r_{yy})^{1/2}$.

center of projection (COP): the point, in three dimensional virtual space, through which all imaged light rays pass.

display field-of-view (DFOV): the visual angle subtended by the physical display screen during image viewing.

enhanced vestibulo-ocular reflex (EVOR): a strengthening of the compensatory oculomotor response during dark VOR testing by imagining a stationary fixation point in the distance.

frustum: the part of the viewing pyramid between the near and far clipping planes, with the side boundaries determined by the horizontal and vertical GFOVs from the COP. The frustum area defines which part of virtual space will be rendered/displayed.

geometric field-of-view (GFOV): the angle subtended, in virtual space, by the viewport at the station point. It is used to define the viewable scene boundaries for image generation on a display.

optokinetic nystagmus (OKN): an involuntary, compensatory oculomotor reflex in response to full-field image slippage across the retina. OKN helps to maintain a stable retinal image by responding to visual input while the VOR helps maintain stable vision by responding to vestibular input.

oscillopsia: a perception of instability in the visual scene.

pixel: a picture element in a visual display.

presence: a perception of 'being somewhere'; used in VR to indicate that a user is immersed in a virtual environment as opposed to his/her physical environment.

Sharpened Romberg: a stance in which a person stands with heel-to-toe, arms folded across the chest, and chin up.

Sensory rearrangement: an altered pattern of sensory signals within the human that is not expected based upon previous experience.

simulator sickness: a set of symptoms similar to motion sickness that occurs as a result of exposure to virtual interfaces and flight/driving simulators.

station point: a point in virtual space which represents the viewer's location during image generation. For monoscopic displays, the station point is coincident with the COP. For stereoscopic displays, the station point is located midway between the two COPs.

stimulus rearrangement: a pattern of stimulation differing from that existing as a result of normal interactions with the real world. Stimulus rearrangements can generate internal sensory rearrangements if the human has not recently experienced the altered stimulus pattern.

synthetic environment: similar to virtual environment, though sometimes inclusive of remote camera-generated images.

time delay: (for this dissertation) the temporal delay between when a subject moves his/her head and when the virtual image is updated to correspond with the new head position.

vection: the visually-induced perception of self-motion.

vestibulo-ocular reflex (VOR): a low-latency, compensatory eye movement reflex in response to head movements (in rotation or translation/tilt). This reflex aids in maintaining a stable image on the retina during these head movements. The VOR results from inertial stimulation of the vestibular organs within each inner ear.

video-oculography (VOG): a method of collecting eye movement data using video recording techniques. Often infrared cameras and emitters are used to facilitate the recording of eye movements in a darkened environment.

viewport: a window within the viewing frustum (in virtual space) onto which all points within the frustum are projected.

virtual environment (VE): a computer-generated space that is immersive and interactive.

virtual interface: a system of transducers, signal processors, computer hardware and software that create an interactive medium through which: 1) information is transmitted to the senses in the form of two- and three dimensional virtual images and 2) psychomotor and physiological behavior of the user is monitored and used to manipulate the virtual images.

virtual reality (VR): a synonym for virtual environment, sometimes inclusive of the hardware involved.

visual vestibulo-ocular reflex (VVOR): a low latency, compensatory eye movement reflex in response to head movements that combines the effects of the VOR with visual tracking systems (i.e., smooth pursuit and OKN).

LIST OF ABBREVIATIONS

AFOSR: Air Force Office of Scientific Research

COP: center of projection

DFOV: display field-of-view

DOF: degree-of-freedom

EVOR: enhanced vestibulo-ocular reflex

FOV: field-of-view

GFOV: geometric field-of-view

GUI: graphical user interface

HITL: Human Interface Technology Laboratory

HMD: head-mounted display

Hz: hertz

IPD: inter-pupillary distance

MAQ: MacEyeball Data Acquisition Software Program

MAP: MacEyeball Data Analysis Software Program

MB: mega-byte

MHQ: motion history questionnaire

MSQ: motion sickness questionnaire

OKAN: optokinetic after-nystagmus

OKN: optokinetic nystagmus

OMN: oculomotor nuclei

RAM:	random access memory
SCC:	semicircular canal
SSQ:	simulator sickness questionnaire
VDT:	video display terminal
VE:	virtual environment
VN:	vestibular nuclei
VOG:	video-oculography
VOR:	vestibulo-ocular reflex
VR:	virtual reality
VVOR:	visual vestibulo-ocular reflex
UW:	University of Washington

PREFACE

This dissertation is a composite work that bridges three separate research areas: human factors engineering, simulator sickness, and oculomotor physiology. Human factors engineering is the primary focus since it is my stated profession. As a result, this dissertation centers on the systematic characterization of virtual interface effects on users and the development of appropriate design guidelines. However, experiments were designed to also address several unresolved and/or unexplored issues in the research domains of simulator sickness and VOR adaptation.

Attempting to advance one area of research is a difficult task; attempting to meaningfully contribute to three areas can be positively arduous. It is sincerely hoped that the results of this undertaking have remained reasonably cohesive while contributing to the these fields.

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Dr. Erik Viirre, for introducing me to the topic of oculomotor adaptation, providing day-to-day guidance and assistance throughout this effort, and generating insightful questions and commentary on the nature of human adaptation to virtual environments. Erik participated as mentor, facilitator, pilot subject, assistant during testing, and friend.

Dr. Tom Furness, for guiding me towards the study of simulator sickness, generating a wealth of exciting ideas regarding this research, authorizing and supporting the development of the VR Effects Laboratory, and providing the necessary clout and know-how to clear various obstacles (both administrative and technical) along the way. In addition, It was solely through Tom's efforts that I was granted this opportunity to stay at the HITL to complete my Ph.D.

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Dr. Don Parker, for superb technical feedback on my ideas and the logic behind them, for allowing me use of the rotating chair and balance platform, and for personally arranging

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Any finally, to my wife Maria, who spent many evenings and weekends alone so that I could complete this work, and for contributing in a myriad of ways as needed. Her efforts are the least definable because they were by far the most encompassing.

DEDICATION

To my loving wife Maria for being by my side, supporting all I do, and for being a model of sincerity and true love which continuously enlightens me as it will our soon to be born child. And to my wonderful parents, Alfred and Bernadette Draper, for providing me a strong foundation upon which I now build.

CHAPTER 1: INTRODUCTION

1.1 OVERVIEW

> *"Engineers shall hold paramount the safety, health and welfare of the public in the performance of their professional duties."*
>
> Canon #1 of the Code of Ethics for Engineers (Tau Beta Pi: Engineering National Honor Society)

> *"There are very few topics concerning human behavior that are not relevant to the design, use, and evaluation of synthetic environment systems. The task of characterizing and modeling human responses to alterations in sensorimotor loops constitutes a major challenge. Knowledge in this area, which is fundamental to the design of essentially all synthetic systems for all types of applications, is seriously inadequate."*
>
> National Research Council Committee on Virtual Reality Research and Development (Durlach & Mavor, 1995)

A human factors engineer's primary responsibility is to evaluate the quality or 'goodness' of human-machine interfaces. While interface quality can be an ambiguous concept, it is often assessed in terms of impact on overall task performance. A different criterion for judging the 'goodness' of an interface, however, arises from an assessment of any negative side-effects that the interface may have on the user. This alternate criterion, though obviously important, has historically been disregarded by design engineers. Reasons for this neglect include a perceived lack of variance surrounding the

effects of traditional interfaces on users and a general lack of engineering knowledge in the areas of human physiology and psychology as applied to interface design.

The effects that interfaces have on users are becoming more of a concern in the information age. New 'virtual' interfaces (i.e., interfaces that connect human to immersive virtual environments) are being introduced that offer high bandwidth, interactive coupling of human to computer (Barfield & Furness, 1995; Durlach & Mavor, 1995). With multiple senses involved and a more natural means of interaction, even the mind itself can become immersed in a synthetic, computer-generated space[1]. This extensive coupling between human and computer increases the likelihood that the human will be influenced by that connection.

In spite of lofty expectations, current virtual interfaces imperfectly simulate our interactions with the real world (DiZio & Lackner, 1992; Durlach & Mavor, 1995). Examples of these imperfections include poor optical design, unintended image scale factor magnifications, system time delays, poor display resolution, limited display field-of-view (DFOV), head tracker inaccuracies, etc. These imperfections, when combined with the tight coupling between human and computer, may result in serious consequences for users. Imperfections often present stimulus rearrangements which can generate sensory pattern rearrangements within the user if these patterns were not expected based upon past experience[2]. Humans must adapt physiologically and perceptually to these sensory rearrangements and then re-adapt to normally occurring

---

[1] This concept, termed 'presence', has become a primary focus of immersive virtual interfaces (Prothero, 1998).

[2] An important distinction must be made at the outset regarding 'stimulus' rearrangements versus 'sensory' rearrangements as these terms are used repeatedly throughout the dissertation. Stimulus rearrangements refer to altered stimulus patterns existing *as a part of the interface,* while sensory rearrangements refer to the results of these stimulus patterns *within the human.* Stimulus rearrangements generate sensory rearrangements only if the altered stimulus patterns are not expected by the human's internal processes. If an existing stimulus rearrangement matches internal expectations (i.e., after adaptation to the interface), no sensory rearrangement results.

conditions upon termination of the exposure. In addition, sensory rearrangements are theorized to induce simulator sickness symptoms (Reason & Brand, 1975; also see Biocca, 1992; Kennedy, Hettinger, & Lilienthal, 1990; McCauley & Sharkey, 1992; Regan, 1995).

With virtual interfaces, one can no longer assume that system modification only occurs on the machine side of the human-computer boundary. Ill-effects of virtual interfaces can decrease performance of virtual tasks and have important health, safety, and legal ramifications. Therefore, a detailed analysis of how humans are influenced by virtual interfaces is critical before these interfaces can be properly designed and administered. Stimulus rearrangements that commonly appear in virtual interfaces need to be evaluated for their relative potential to drive adaptation processes and induce simulator sickness through the generation of internal sensory rearrangements. Only by fully characterizing the interface in this way can engineers truly understand the health and safety issues involved.

This dissertation addressed a subset of interface-generated effects on the user. The purpose of this research was to investigate the existence and extent of human physiological adaptation to virtual interfaces through a detailed study of the vestibulo-ocular reflex (VOR) and simulator sickness incidence.

The VOR is an oculomotor reflex that is vital to the maintenance of stable vision during self-motion (Robinson, 1981). Adaptive recalibration of this reflex indicates the presence of a significant visual-vestibular sensory rearrangement (Demer, Goldberg, Jenkins, & Porter, 1987) and this adaptation requires a period of oculomotor re-adaptation to normal conditions after the exposure has ended. This dissertation assessed whether virtual interfaces typically contain the necessary stimulus rearrangements to drive VOR adaptation. Specific stimulus rearrangements investigated include scale changes of the synthetic image (i.e., scene magnification factor) and time delays

between head movement input and visual scene update. The relationship between VOR adaptive activity and simulator sickness incidence was also investigated.

The general goals of this research were to: 1) better understand if and how the VOR system responds to specific stimulus rearrangements of virtual interfaces during typical interactions, 2) determine if the existence of VOR adaptation correlated with simulator sickness, and 3) compile information that would assist designers in developing virtual interfaces which minimize the occurrence of adverse side-effects.

1.2 NATURE OF VIRTUAL INTERFACES

Since virtual interfaces are the focus of this research, this term should be defined at the outset. However, the task of defining virtual interfaces often deteriorates into an extended philosophical exercise as to the true nature of VR (Furness, 1996). In an effort to avoid a detailed rehashing of such debates, I have chosen a definition of virtual interfaces that I believe is both complete enough and benign enough to placate a majority of VR theorists. Furness (1994) defined virtual interface as "a system of transducers, signal processors, computer hardware and software that create an interactive medium through which: 1) information is transmitted to the senses in the form of two- or three-dimensional virtual images and 2) psychomotor and physiological behavior of the user is monitored and used to manipulate the virtual images". Therefore, virtual interfaces attempt to interactively couple a human to a synthetic (i.e. virtual) environment in a natural manner.

Virtual interfaces are different from traditional interfaces in many ways. They (potentially) include high bandwidth, multi-sensory transfer of information involving visual, auditory, haptic, tactile, kinesthetic, and occasionally even olfactory modalities. Virtual interfaces have the ability to quickly and completely immerse the user into the virtual environment being experienced. These interfaces also offer higher interactivity

(especially naturalistic interaction), intuitiveness, and inclusiveness than traditional interfaces (Furness, 1994). These advantages have been heralded by many in the field, resulting in emerging applications in many diverse areas including the military, education, entertainment, public service, medicine, social work, space travel, and industry (Rheingold, 1991).

For all their potential benefits, however, *current* virtual interfaces are often considered to be substandard and potentially even hazardous. This dichotomy exists because of the nature of the interface, current technology levels, and poor design. As stated earlier, virtual interfaces couple tightly the user and information in an intimate and naturalistic manner. The requirements for this seamless coupling far exceed the abilities of current technology in nearly every area, including virtual image rendering, position tracking capabilities and resolutions, multi-sensory integration, etc. Given that the ideal virtual interface could reasonably be judged as one that would equal (or better) our multi-sensory interaction with the natural world, we are currently far from perfection. Therefore, designers must continually 'cut corners' and make a sizable number of tradeoffs while striving to maintain a high level of immersion and interactivity in the experience. This is almost always accomplished by engineers having only limited knowledge of applicable human physiological and perceptual mechanisms. Unfortunately, these practices result in poorly designed virtual interfaces that may effect the user through the introduction of undesired sensory rearrangements.

1.3 POTENTIAL NEGATIVE EFFECTS OF VIRTUAL INTERFACES

History is replete with examples of the negative side-effects of interfaces. Examples over time include the inevitable hammer on finger, writer's cramp, 'Nintendo thumb', VDT-induced eye strain, and carpal tunnel syndrome due to excessive keyboard use.

Similarly, virtual interfaces potentially create new consequences for the user due to their high-bandwidth, naturalistic interaction and multitude of stimulus rearrangements.

When a person enters an immersive virtual environment (VE), there are four possible outcomes concerning the effects of the VE on that user (see Table 1)[3]. The user may, or may not, experience simulator sickness while in the environment. Secondly, the user may, or may not, be forced to adapt significantly (physiologically and/or perceptually) to the VE. I argue that only one of the four potential outcomes, Cell #4, is completely acceptable from a design standpoint because no sickness occurs and no adaptations are required. The paragraphs below describe simulator sickness and adaptations in more detail and discuss why each should be avoided when designing VEs.

Table 1: Potential Adverse Effects of Virtual Interface Exposure

Physiological/Perceptual Adaptations	Simulator Sickness: Symptoms	Simulator Sickness: No Symptoms
Induced	Cell #1: maximum ill effects	Cell #2: re-adaptation issues
None Required	Cell #3: sickness symptoms	Cell #4: preferred design

[3] Note that this table assumes that simulator sickness and adaptations are independent, which is highly unlikely. Consequently, Cell #3 (the existence of simulator sickness without concurrent adaptation requirements) is highly unlikely at least for predictable patterns of stimulation.

1.3.1 Simulator Sickness

Conflicting stimuli regarding self-motion and spatial orientation exist as an unavoidable side-effect of current virtual interfaces (Peli, 1995). Although this issue is discussed in detail in Chapter 2, these conflicting stimuli are thought to generate sensory rearrangements which induce simulator sickness. These resulting sickness symptoms, which are similar to those of motion sickness[4], include headaches, nausea, dizziness, stomach discomfort, eye strain, oscillopsia, and postural ataxia (Kennedy, Hettinger, & Lilienthal, 1990). They can arise during exposure and/or immediately after terminating the experience, sometimes lingering for several hours or more (Kennedy, Hettinger, & Lilienthal, 1990). Therefore, it is obvious why designers should attempt to avoid simulator sickness when designing virtual interfaces.[5]

1.3.2 Physiological and Perceptual Adaptations

In addition to inducing sickness symptoms, these sensory rearrangements can cause other response modifications. Physiological and perceptual system adaptations may occur in an attempt to optimize sensorimotor performance in the 'distorted' synthetic environment (Welch, 1986). Examples include physiological changes in postural control, oculomotor accommodation/vergence responses, and perceptual adaptations of spatial orientation and self-motion. For each physiological and perceptual system involved, the level of adaptation attained while in the VE will necessarily be the initial condition for that system when the user exits the VE and is re-exposed to the dynamics

---

[4] Similar, but not identical. The relationship between simulator sickness and motion sickness is further discussed in Chapters 2 and 9, along with Appendix G.

[5] However, there are instances when the evocation of motion sickness symptoms is in fact desired, i.e., when attempting to accurately simulate a nauseogenic environment. However, in that case the symptom elicitation is intentional, not accidental.

of the real world. The adapted system, once again out of adjustment, must then re-adapt to the normally occurring stimulus patterns. There is concern that health and safety issues may arise during this post-exposure re-adaptation period (Kennedy, Hettinger, & Lilienthal, 1990; Kolasinski, 1995; Paige & Sargent, 1991) in addition to any sickness symptoms that this re-adaptation process induces. This concern increases with the magnitude of adaptation achieved. Therefore, by preventing (or at least minimizing) undesired adaptations from occurring, designers can avoid the potentially hazardous and nauseous re-adaptation period as well.

1.3.3 Impact

Potential ill-effects of poorly designed virtual interfaces have performance, health, safety, and legal ramifications. At the very least, discomfort experienced while exposed to synthetic environments will decrease time spent in the environment and the desire to repeat the experience in the future. This alone can be a major impediment to the widespread application of virtual interface technology. More disturbing concerns, however, involve the overall health and safety of users (Kolasinski, 1995; Peli, 1995; Viirre, 1994). Lingering negative aftereffects of adaptation to virtual interfaces can mean extended discomfort and the increased potential for mishaps while these aftereffects exist (Kennedy, Hettinger, & Lilienthal, 1990). Finally, there is no denying the potential for lawsuits, real or concocted, that can act as a critical hindrance to industry growth. Thus it would behoove designers to improve the quality of virtual interfaces by correcting those design elements that induce simulator sickness and undesired physiological/perceptual adaptations (i.e., Cell #4 of Table 1).

There is an important caveat to the discussion above. Negative effects of virtual interfaces, though demonstrated to exist in many forms through various individual studies, have not been characterized fully enough to substantiate or disprove the identified health and safety concerns. Just as sensationalism surrounds the potential

benefits of virtual interfaces, there is a growing alarmist agenda making ominous claims surrounding the effects of these same interfaces (Gross, 1995; Seymour, 1996). What is currently lacking are comprehensive, empirical investigations into the true health and safety issues surrounding virtual interfaces (Durlach & Mavor, 1995). Information must be gathered which characterizes human responses to specific sensory rearrangements found in virtual interfaces in order to increase the percentage of virtual interfaces that fall into Cell #4 of Table 1. This dissertation contributes to the body of knowledge regarding the nature of potential deleterious effects of virtual interfaces on humans.

1.4 ROLE OF HUMAN ADAPTATION

"The extraordinary adaptive capabilities of the human body are clearly involved with both causing and overcoming motion sickness"
M.J. Griffin, 1990

Humans are remarkably adaptive creatures. Human physiological and perceptual adaptations have been demonstrated in many atypical environments including the wearing of left/right image reversing spectacles and the loss of gravity during space flight (Welch, 1986). Given that virtual interfaces can also present 'abnormal' environments in terms of sensory stimulation, it is reasonable to hypothesize that various stimulus rearrangements within these environments may also activate various physiological and perceptual adaptive processes.

Both simulator sickness and negative aftereffects are rooted in human adaptation to virtual interfaces. Simulator sickness symptoms have been theorized to occur as a result of sensory rearrangement until the person adapts to this rearrangement (Reason &

Brand, 1975; Reason, 1978). In addition, Reason and Graybiel (1972) argue that an individual 'adaptability trait' largely determines motion sickness susceptibility. Negative aftereffects are also known to be the direct result of human physiological and perceptual adaptation to sensory rearrangements (Paige & Sargent, 1991; Welch, 1986).

The logical approach to the identified health and safety concerns, therefore, is to study human adaptation processes. A detailed description is needed of how the human adapts (including adaptation magnitude and time-course of adaptation/re-adaptation) for each potentially salient stimulus rearrangement in virtual interfaces. Once human responses to these rearrangements are better characterized and bounded, efforts can be made to realistically assess and respond to health and safety concerns through design guidelines or adaptation/re-adaptation strategies. This dissertation focuses on one metric of human adaptation to specific virtual interfaces: vestibulo-ocular reflex (VOR) adaptation.

1.5 VOR RECALIBRATION AS AN ADAPTATION METRIC

An issue that arose early in this exploration was whether to focus on physiological or perceptual adaptation to synthetic environments. As stated above, humans have been shown to adapt both physiologically and perceptually to induced sensory rearrangements. However, whereas motor-control systems rely on an accurate model of the environment for proper functioning, perceptual systems do not. Perception uses higher level processes to filter and differentially weight salient cues in the sensory stream to reach an interpretation (Welch, 1986). These higher level processes, though obviously critical to perceptual adaptations, are currently ill-defined in terms of structure and ambiguous in terms of effects. Therefore, I have chosen to focus on physiological adaptation in an effort to reduce extraneous variance in the data. Future

research focusing on perceptual issues might then benefit from knowledge obtained regarding relevant underlying physiological behavior.

The VOR has been identified for detailed study in this dissertation. This reflex is a compensatory eye movement response which functions to stabilize the visual image on the retina during head motion, promoting clear vision during movement (Howard, 1986b; Robinson, 1981). The VOR triggers eye movements in the opposite direction of head movements in an effort to keep the eye space-stable and minimize retinal image slip.

The rationale for selecting VOR adaptation to virtual interfaces as a main research focus is summarized below. First, VOR adaptation has been shown to be driven by sensory rearrangements of visual and vestibular cues of motion (Demer, et al., 1987; Gauthier & Robinson, 1975; Gonshor & Melvill Jones, 1976b). Given that mismatched visual-vestibular motion cues are a principal form of sensory rearrangement found in virtual interfaces, the VOR is judged to be a prime physiological reflex for study in this context. Second, its relatively rapid and consistent adaptive response to altered environments make the VOR a particularly sensitive and reliable metric of physiological adaptation to virtual interfaces. Third, much is known about VOR adaptation processes, which provides a wealth of knowledge upon which to build (Howard, 1986b; Sharpe & Barber, 1993). Fourth, VOR adaptation is often closely associated with oscillopsia (a perception of visual scene instability) which has obvious safety implications (Demer, et al., 1987; Demer, Porter, Goldberg, Jenkins, & Schmidt, 1989; Gauthier & Robinson, 1975; Leigh, Dell'Osso, & Kosmorsky, 1993; Paige & Sargent, 1991; Viirre & Demer, 1996). Finally, the VOR adaptation process is frequently marked by additional symptoms similar to the simulator sickness symptoms experienced by many VR participants such as eye-strain, headaches, and dizziness (Demer, et al., 1989; Gauthier & Robinson, 1975; Istl-Lenz, Hyden & Schwarz, 1985;

Paige & Sargent, 1991). Therefore, it is logical to study whether this association is strong and causal, or weak and coincidental.

1.6 VOR ADAPTATION AND SIMULATOR SICKNESS

This dissertation also hypothesized a relationship between VOR adaptation and simulator sickness processes. Both processes are believed to be caused by visual-vestibular sensory rearrangements. Both processes also require the existence of a functioning vestibular apparatus. In addition, as stated in the previous section, research studying VOR adaptation has often recorded the concurrent existence of sickness symptoms during the exposure period to the VOR adapting stimulus. This hypothesis is more fully detailed in Chapter 3.

1.7 VIRTUAL INTERFACES AS A CLINICAL TOOL FOR ADAPTATION

Adaptations induced by virtual interfaces have so far been presented as being negative occurrences (due to the associated aftereffects). Some researchers, however, have alluded to a potentially positive application of VE-driven adaptations. Dr. Erik Viirre (personal communication, 1996) has argued that if certain virtual interfaces are found to modify VOR gain in healthy patients (a central focus of this dissertation), they might also be effective as a rehabilitative tool for clinical patients with chronically low VOR gains. One of Dr. Viirre's ideas involves the use of virtual interfaces to incrementally increase the gain of those patients back towards normal. An underlying premise behind his hypothesis was explored as part of this dissertation.

1.8 OVERVIEW OF DISSERTATION RESEARCH

Given that many advocates wish to bring virtual interface technology into widespread use and many others promulgate the potential safety and health dangers of extended VE exposure, what is needed first and foremost is a detailed study of how humans respond to virtual interfaces. Moreover, rather than exploring human responses in an artificial, passive manner, research should concentrate on typical situations where the user is actively interacting with the VE to complete a task. This research utilized these types of situations to investigate the existence of human physiological adaptation to sensory rearrangements through a detailed study of VOR adaptation in VEs. Specific sensory rearrangements such as time delays (between head movement and virtual scene response) and image scale factor distortions (scene magnification or minification) were manipulated and their effects on VOR adaptation characterized. The relationship between these same sensory rearrangements and simulator sickness symptomology was also explored as was a hypothesized relationship between VOR adaptation and simulator sickness.

Specifically, this research had five major objectives:

1. Test General Hypothesis 1 (described in Chapter 3), which argues that certain stimulus rearrangements found in virtual interfaces cause VOR adaptation. Characterize any resulting adaptation in terms of magnitude achieved and general time-course of re-adaptation following exposure.

2. Determine whether these same sensory rearrangements modulate reports of simulator sickness and which of several existing theories of simulator sickness best fit the data.

3. Test General Hypothesis 2 (described in Chapter 3), which asserts that magnitude of VOR gain adaptation correlates with simulator sickness magnitude.

4. Given that Objective 1 is satisfied, assess the viability of a proposed rehabilitation technique for increasing VOR gain using virtual interface technology.

5. Utilize results from this research to develop a set of preliminary guidelines for virtual interface design that will minimize any occurrence of VOR adaptations and/or simulator sickness symptoms.

1.9 RESEARCH BENEFITS

The potential benefits from this research include:

- Determination of whether VOR adaptation processes can indeed be stimulated as a result of 30 minutes of active, goal-directed interaction with a head-coupled virtual interface.

- Linkage of VOR adaptive responses to specific stimulus rearrangements involved.

- Enhanced understanding of the modulating influence of system time delays and VE image scale distortions on reported simulator sickness in head-coupled virtual environments

- A potential correlation between VOR adaptation and simulator sickness symptomology. This association, if found, could offer a new objective measure of simulator sickness.

- Data to support virtual interface design guidelines that reduce VOR re-adaptation effects and/or simulator sickness symptoms through the avoidance of adverse sensory rearrangements.

- Data supporting/refuting a proposed use of virtual interfaces as a rehabilitative tool.

1.10 DISSERTATION STRUCTURE

This dissertation is structured as follows. Chapter 2 contains background information and a review of the research literature concerning the three major topics addressed in this dissertation: the VOR, virtual interfaces, and simulator sickness. An additional topic, adaptation, is discussed within the context of the VOR and simulator sickness. Those already familiar with these topics can skip this chapter without loss of continuity. Chapter 3, General Hypotheses, combines research results, existing theories, researcher assertions and supporting logic to describe and defend two major hypotheses examined in this dissertation. These arguments form the foundation of theoretical support for the research questions and experiments that follow. Chapter 4, Introduction to Experimentation, provides a summary of preliminary research performed, an overview of the main research agenda, and details of the development and configuration of an appropriate test facility in which to conduct this research. This chapter serves as an empirical foundation for the dissertation research. Chapters 5-8 describe four experiments conducted to investigate the nature of VOR adaptation and simulator sickness in VEs. A general discussion follows in Chapter 9 which integrates the results across all four experiments to address the five main objectives of this dissertation. Preliminary virtual interface design guidelines arising from this research are presented in this chapter. Lastly, some conclusions and opportunities for future research are offered in Chapter 10.

CHAPTER 2: BACKGROUND AND LITERATURE REVIEW

2.1 OVERVIEW

This chapter provides background details on the VOR (including VOR adaptation), virtual interfaces, and simulator sickness. Its purpose is to provide a foundation of knowledge regarding the major concepts and themes that are central to this dissertation. However, time and space constraints dictate that only a general summary of each topic be presented. Numerous citations are provided for those wishing more information on a particular subject or issue.

2.2 THE VOR AND VOR ADAPTATION

The VOR is a primitive eye-movement reflex that helps to stabilize visual images on the retina during movement of the head (Howard, 1986a; Robinson, 1981). To facilitate a better understanding of this reflex, a brief overview of the vestibular apparatus is presented. This is followed by a detailed description of the VOR, how it is measured, and it's characteristics. VOR adaptation is then described along with influencing factors. Literature associating VOR response with sickness is then described. Lastly, the VOR is related to the optokinetic reflex and smooth pursuit, the two visual-tracking eye movements.

2.2.1 THE VESTIBULAR APPARATUS

The vestibular apparatus is a small structure that resides in the bony labyrinth of each inner ear (see Howard, 1986a). Its function is to sense head movements and generate a response which contributes to the coordination of eye movements, posture/balance, and perceptions of motion and orientation. This signal is vital;

individuals with partial or complete loss of vestibular functioning have found it difficult to perform even the most basic of tasks (Howard, 1986a; JC, 1952; Sharpe & Barber, 1993).

The vestibular organ consists of two principle sets of structures, the semicircular canals and the otolith organs (Figure 1), which work together to provide optimum information on head movement and positioning. The eighth cranial nerve is the efferent pathway for vestibular signals.

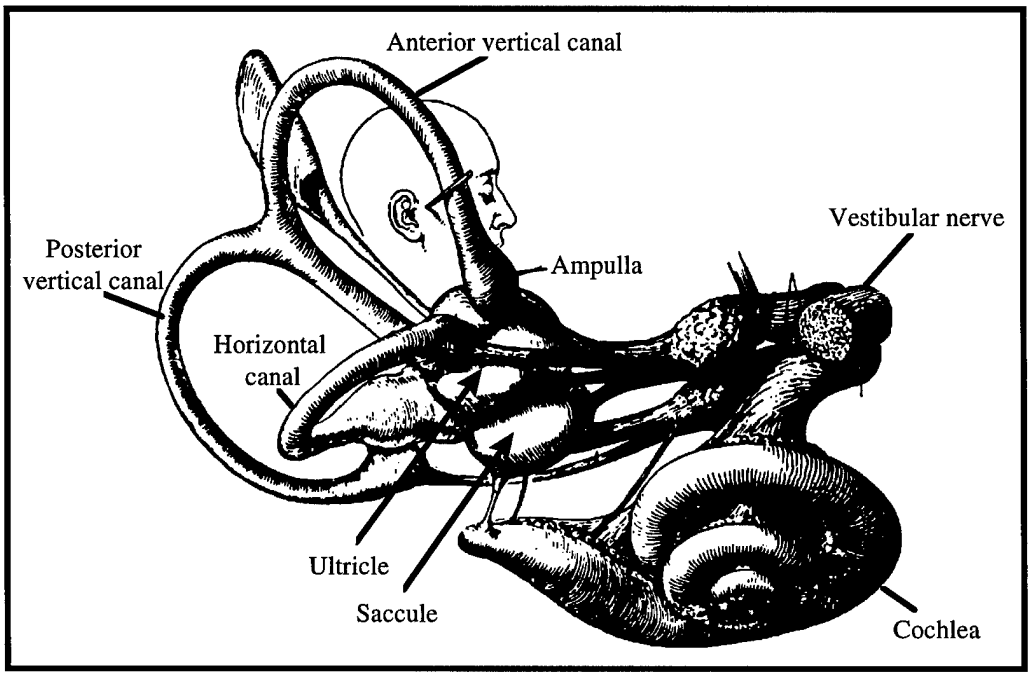

Figure 1: The Vestibular Apparatus[6]

[6] Adapted from Howard (1986a), page 11-2.

There are three semicircular canals (SCC), termed the anterior, posterior, and horizontal canals, in each vestibular organ. Their function is to detect angular accelerations of the head, acting like biological accelerometers. These canals are each bi-directionally sensitive and, when combined across the two vestibular organs, form three approximately perpendicular paired sets so as to detect angular head movement in any direction (Figure 2). Endolymph fluid within each SCC is prevented from passing through the ampulla (a widened section of each SCC) by the cupula, a thin flap that stretches across the ampulla and acts as a barrier to endolymph flow (Melvill Jones, 1993; Robinson, 1981). When the head is rotated, the force exerted by the viscosity and inertia of the fluid acts against each cupula of those SCCs that are in the plane of motion, causing it to deflect (Figure 3). This deflection bends tiny hair cells located at the base of the cupula which causes a signal to be sent to the vestibular nucleus via the eighth cranial nerve.

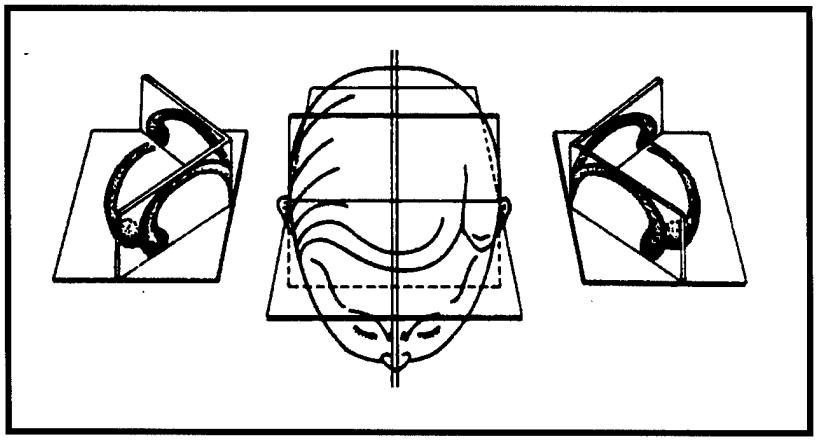

Figure 2: General Orientation of the SCCs[7]

[7] From Cullen & Scott (1997).

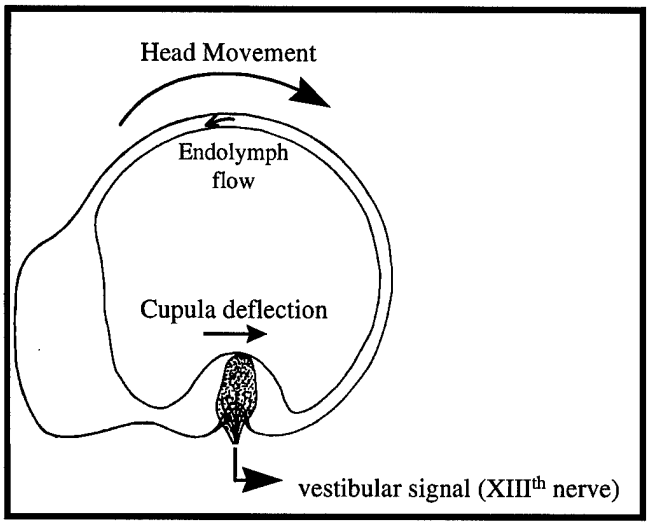

Figure 3: SCC Dynamics

For most normal head movements (i.e., moderate frequencies), the signal output of each SCC is proportional to head rotational velocity, not acceleration (Howard, 1986a; Melvill Jones, 1993). This because the extremely small diameter of the SCCs (0.3 mm mean diameter) results in viscous resistance being large compared to endolymph mass (inertia) and cupula elasticity. Using the differential equation for the torsion pendulum as a model for SCC dynamics, it can therefore be shown that head accelerations are proportional to the angular velocity of the cupula deflection (Howard, 1986a).

The receptor system of the SCC can respond to angular accelerations as low as 0.1 deg/sec^2. However, continued constant angular rotation (i.e., zero acceleration) will decrease the response from the vestibular system. The endolymph fluid decreases its inertial force on the cupula due to the lack of acceleration forces and the elasticity of the cupula which always opposes cupula deflection. The time constant for cupula deflection in humans is approximately 5 to 7 seconds (Howard, 1986a; Robinson, 1981). Therefore, SCCs encode dynamic changes in head movement only.

Also, it is important to note that each SCC transmits a tonic (resting) signal even in absence of motion. This allows the SCC to increase or decrease its response, depending upon whether the head rotation is in the direction that the SCC is tuned to or in the opposite direction. The brain then integrates information from each SCC pair that occupy the same plane of motion (termed a push-pull pair) to generate an appropriate response for motion in that plane. Between the two vestibular organs, there is a total of three of these push-pull pairs approximating three principle axes of motion.

In addition to the three SCCs, each vestibular apparatus also contains two otolith organs, the utricle and saccule, that sense dynamic changes in linear acceleration of the head and also provide information on static head position, such as head tilt (Figure 1). The receptor portion of these organs, termed the macula, contains many hair cells. The macula is covered with a gelatinous substance that contains tiny crystals of calcium carbonite called 'otoliths'. When the head is tilted or undergoes linear acceleration, the otoliths deform the gelatinous mass, which creates a shear force that bend (i.e., excite) the receptor (hair) cells in the macula. This results in a signal which is transmitted via the eighth nerve to the vestibular nuclei. The utricle's macula is located in the horizontal plane so as to be sensitive primarily to horizontal linear accelerations, while the saccule's macula is positioned vertically to be maximally sensitive to vertically directed linear accelerations, including gravity (Robinson, 1981). However, given the curved nature of both macula, it is likely that both the utricle and saccule respond at some level to all directions of linear acceleration applied to the head (Melvill Jones, 1993).

2.2.2 VOR Description and Characteristics

2.2.2.1 General Description

The greatest potential source of image slip on the retina is due to rotations of the head (Robinson, 1981). Therefore, in order to see and move at the same time, the eyes must have an ability to remain stabilized in space as the head rotates. The VOR is a fundamental eye-movement reflex that helps to keep images stabilized on the retina during head movements. Thus it helps to perform a very basic but important function, to allow stable vision during movement.

When the head begins to move in any direction, the vestibular apparatus senses this movement and sends velocity information directly to the oculomotor system. The oculomotor system responds by moving the eyes (congunctively) in an equal but opposite direction/rate to compensate for the head movement to help keep the visual image stabilized on the retina. Figure 4 presents a graphical depiction of the VOR for head movements both to the left and to the right of a center position (dashed lines represent original positions; solid lines represent final positions). This, at a top level, is the VOR. It is a very low latency oculomotor reflex in response to head movements, with compensatory eye movements beginning as early as 4 to 13 ms after head rotation begins (Sharpe & Johnston, 1993).

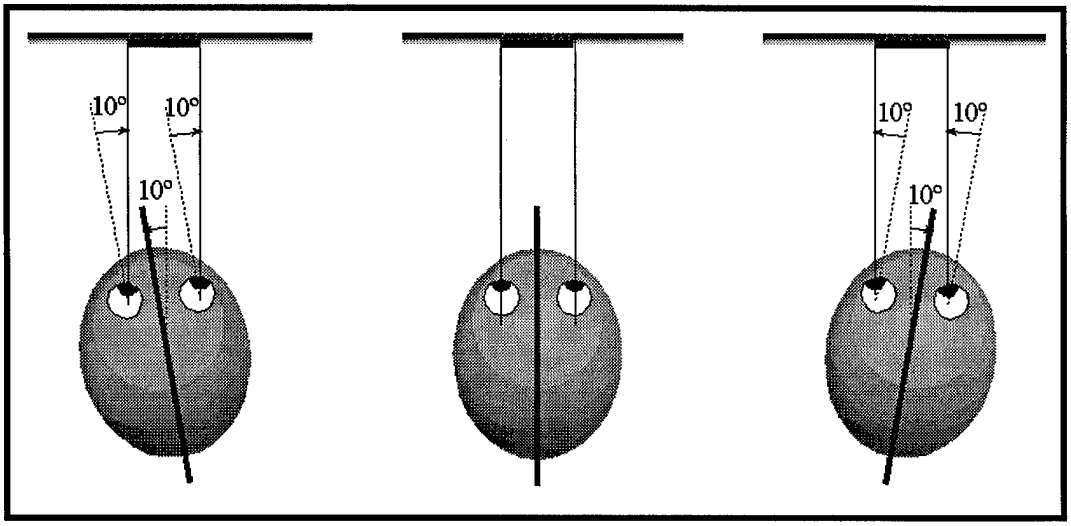

Figure 4: The VOR[8]

The VOR responds to any head rotation along any axes of movement. However, most studies have isolated VOR response to rotations along a single, standard axis of motion; either the horizontal VOR (due to yaw rotations), vertical VOR (due to pitch rotations/tilt) and to a lesser extent, the torsional VOR (due to roll movements/tilt).

Although both the SCCs and the otolith organs contribute to the VOR, most researchers agree that in many situations the otolith input is minor and transitory while the SCC input dominates the response (Robinson, 1981)[9]. This is especially true of the horizontal VOR during natural head movements. Therefore, the rest of this dissertation will only consider the SCC input to the VOR. It should be noted, however, that under certain conditions (e.g., off-axis rotation, close-in target of fixation) the otolith organs

[8] The figure depicts horizontal head movements 10 degrees left and right from a center position with resulting compensatory eye movements to maintain a stable gaze.

[9] Both vertical and torsional VORs can involve significant contributions from the otoliths. This dissertation only considers the horizontal VOR.

can have a demonstrable effect on the horizontal VOR (Merfeld, 1995; Viirre & Demer, 1996; Viirre, Tweed, Milner, & Vilis, 1986; Zee & Hain, 1993).

A more detailed description of the horizontal VOR is now described. An angular acceleration of the head will cause the appropriate SCC cupulas to deflect (creating push-pull pairs as described earlier). This deflection will cause the tiny hair receptor cells at the crista ampullaris (i.e., the base of the cupula) to send head-velocity proportional signals as either excitatory patterns (due to motion in the direction of the directionally sensitive SCC) or inhibitory patterns (due to motion opposite the direction of the directionally sensitive SCC). This signal is conveyed to the vestibular nuclei (VN) via the eighth nerve. The VN then sends the appropriate eye velocity signal to the oculomotor nuclei (ON), which in turn innervates the three complimentary pairs of muscles that govern the movements of each eye. This path is often termed the 'three-arc reflex' (Figure 5).

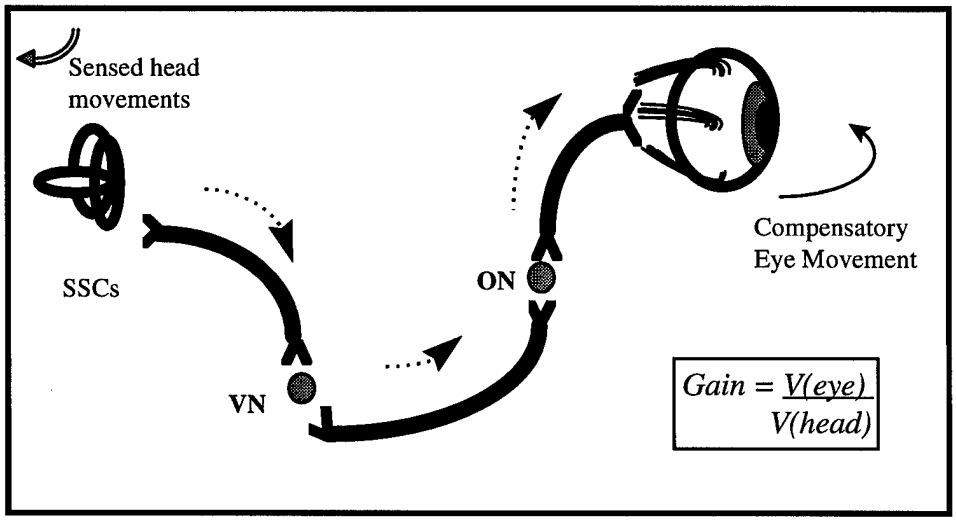

Figure 5: Simplified Schematic of the VOR Three-Arc Reflex

It is theorized that the oculomotor neurons need more than just an eye velocity command to drive the eye; an position command is also required (Robinson, 1981). This position signal functions to hold the eye at the new position so that the elasticity of the eye plant does not cause the eye to drift back to its original position after the head movement ends. Robinson (1981) and others claim the existence of a 'neural integrator' in the system that integrates the velocity signal from the VN to obtain the required position signal. This signal is transmitted along what is termed the "indirect" pathway while velocity signals are transmitted along the "direct" pathway. Goldberg, Eggers, and Gouras (1991) found that this neural integrator, although still undiscovered, requires the cerebellar flocculus, the medial vestibular nucleus, and the nucleus prepositus hypoglossi to operate. Robinson (1981) and others have postulated that this neural integrator may be common to all conjugate eye movement systems.

2.2.2.2 Vestibular Nystagmus and VOR Metrics

If the head undergoes sustained rotation in any direction, the eyes will exhibit a rhythmic oscillatory pattern called nystagmus. There are different types of nystagmus (e.g., optokinetic, vestibular, physiological, caloric), depending on what stimulus is involved[10]. If head rotation occurs in a completely dark environment, the nystagmus is thought to be due solely to vestibular input and is thus called *vestibular nystagmus*.

Nystagmus appears on a strip chart as an oscillating pattern (indicating side-to-side eye movement) that is characteristic of a "saw-tooth" (Figure 6). For instance, if the head rotates in the horizontal plane to the right, the VOR will cause the eyes to compensate by moving in an equal but opposite direction to the left. This leftward movement of the eyes will continue until the eye nears the edge of its orbit, then the eye

[10] Caloric and OKN nystagmus do not require that the head be in motion.

will rapidly reverse direction, moving back across the center of gaze. This rapid reversal of eye movement in the direction of head rotation is called the *quick phase* of nystagmus. After the quick phase, the eye will again begin to compensate for the rotation by moving to the left. This slower movement in the opposite direction of head movement is called the *slow phase* of nystagmus. This slow phase of vestibular nystagmus is often what is measured as the eye velocity component of the VOR while the quick phase acts more like a correcting saccade to reset the eye in orbit (Howard, 1986b).

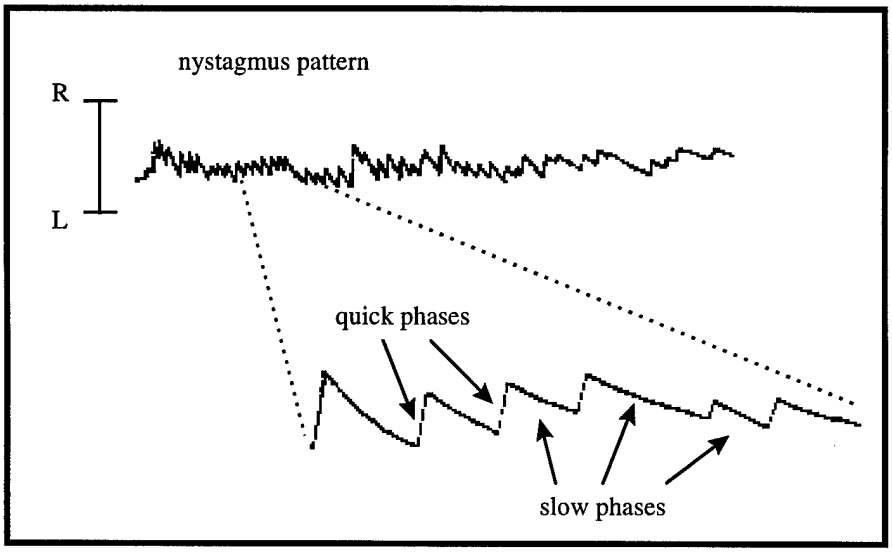

Figure 6: Vestibular Nystagmus

Vestibular nystagmus does not continue endlessly. The response decays as the SCCs habituate to a constant rotation (i.e., zero acceleration) input. The actual time constant of the SCCs is approximately 5 to 7 seconds after acceleration ceases but a central velocity storage mechanism 'extends' this time constant to approximately 25 seconds before vestibular nystagmus fully decays (Howard, 1986b; Robinson, 1981).

However, nystagmus due to constant rotation in lighted conditions will not decay, due to the added contribution of optokinetic (visual) input to the oculomotor system[11].

Under what conditions is the VOR commonly measured? The first general requirement is that measurement take place in a dark room, so that there can be no contributing affects of visually-based image-stabilizing systems[12] (i.e., optokinetic, fixation). A subject is then either oscillated sinusoidally or rotated continually in one direction. Sinusoidal oscillations can be performed either passively, though use of a rotating chair, or actively through head movements made by the subject. Subjects are also usually given a specific task to perform during testing; most commonly either mental arithmetic or fixation on an imaginary point in the distance. The movement of the eyes (vestibular nystagmus) is then recorded, the slow phase movements are extracted, and the results are compared to the associated movements of the head to determine VOR metrics. Other methods for measuring the VOR also exist (Halmagyi, 1995; Sharpe & Barber, 1993; Shelhamer, Robinson, & Tan, 1992).

Gain and phase are the two main metrics of the VOR[13]. VOR gain is calculated as the slow-phase eye velocity divided by head velocity. It is a measure of the amplitude of eye movement velocity for a given head movement. Phase indicates the relative timing of eye movements in response to head movements. Phase leads indicate that the eye responses actually precede head movements while phase lags indicate that eye movement response lags behind head movements. Phase is thus considered the angle between head movement and eye movement response.

In is very important to characterize subject activity during VOR testing. If subjects are tasked to perform mental arithmetic, their VOR gain will likely be lower than if they

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[11] As discussed in Section 2.2.5.1

[12] An exception to this requirement is a technique used by Halmagyi (1995) called a 'head impulse'.

[13] If VOR gain and phase are collected over several frequencies, the result is often graphed on Bode plots.

are told to fixate on an imaginary point in the distance (see Section 2.2.2.3). In either case, the VOR gain in the dark is rarely fully compensatory for head movement frequencies below 1 Hz, especially if mental arithmetic is performed by the subject (Robinson, 1981; Tweed, Sievering, Misslisch, Fetter, Zee, & Koenig, 1994). If the VOR was completely compensatory, it would have a gain equal to 1.0 (unity) and no phase deviation. Full VOR compensation is not normally required below 1.0 Hz, though, because of the assistance of visual tracking systems to help maintain retinal image stability in that frequency range (see Section 2.2.5).

2.2.2.3 VOR Characteristics

Previous research has shown that the VOR acts as a band-pass filter (Robinson, 1981). At very low frequency head movements (approximately 0.05 Hz or lower), VOR compensation is poor, with low gain and a small phase lead. Between 0.05 Hz and 1.0 Hz, VOR gain increases but generally remains between 0.50 and 0.85. At higher head movement frequencies (1 to 7 Hz) the VOR is very responsive with a gain close to 1.0 (unity) and eye movement in phase with head movement. The VOR should indeed operate well at these frequencies because this frequency range is most likely to be encountered in everyday activities (head movement while walking is approximately 1 Hz, while running it is approximately 4 to 6 Hz). At still higher frequencies (above approximately 7 to 8 Hz), the system rolls off with VOR gain decreasing and phase lag increasing. Therefore, VOR responses are often characterized by their gain and phase relationship with the movement of the head at a particular frequency or frequency range.

An important factor effecting the VOR is the mental activity level of the subject. It has been shown (Robinson, 1981) that if the subject is not mentally active, VOR gain is low. If asked to perform mental arithmetic during testing, VOR gain increases and averages approximately 0.65. If however, the subject is asked to imagine and fixate on an imaginary spot on the wall in total darkness, VOR gain increases to approximately

0.95 (this is sometimes called enhanced VOR, or 'EVOR'). Finally, if the subject is asked to imagine and fixate on a spot on the chair in which he/she is rotating, VOR gain drops to approximately 0.2 or lower (this is often termed VOR suppression). Apparently being alert is not enough, the subject must also be actively attending to the environment in a way that facilitates VOR functioning. A fixation system has been hypothesized that may account for these fluctuations. Other factors that may lower the VOR indirectly by decreasing alertness include fatigue, drugs, pain, and immobilization.

The VOR is also effected by target distance (Viirre, et al., 1986). For distant targets, VOR gain is close to 1.0 but gain increases as the target moves closer to the observer. This is logical because the required (theoretical) eye-movement response to prevent retinal slip also increases for a close-in target. How the VOR modulates its response for target distance is not fully understood, however. An internal estimate of target distance and location relative to the head must exist that is based upon nonvisual signals. Possible inputs include head and eye position, vergence angle and accommodation setting. Viirre, et al. (1986) provides one potential target locator model.

The literature sometimes distinguishes between the VOR of a subject that is passively moved (passive VOR) and the VOR of a subject that actively moves his/her head (active VOR). Collewijn, Martins, and Steinman (1983) demonstrated that active VORs were more robust by 3% to 13% than passive VORs in almost all conditions. The variability of active VORs was also less than passive VORs. This could be due to the addition of efference copy. A further classification in VOR measurement is between light VOR (often termed visual VOR, or VVOR) and dark VOR. VVOR entails measuring the VOR in a lighted environment and includes the influence of optokinetic stimulation (as discussed below) and fixation while dark VOR involves measuring the VOR in complete darkness and is thus more accurate in isolating the vestibular-driven VOR response.

The effect of stimulus predictability on the VOR is still undetermined. It has been shown that voluntary movement of the head can result in an improved VOR response (i.e., active VOR, see Collewijn, et al., 1983). This is probably due to an efference copy of motor commands affecting the VOR three-arc reflex and it indicates that prediction influences the VOR response[14]. McKinley and Peterson (1985), however, demonstrated that VOR gain (in relaxed state, enhanced, or suppressed) is independent of whether or not a passive rotation stimulus was predictable or not. This seems to implicate efference copy as the effective method of prediction in the VOR system.

2.2.3 VOR Adaptation

2.2.3.1 Description and Model

As described earlier, the VOR is a very low latency reflex that allows the eyes to compensate for head rotations. An important aspect of the VOR, however, is its ability to recalibrate in response to changing conditions (Robinson, 1981). Stimuli that promote adaptation create a mismatch between current VOR settings and that required to keep an image stabilized on the retina. This mismatch could be internally generated; due to the effects of age, disease, or trauma on the vestibular apparatus, circuitry, and/or eye muscles. Or the mismatch could be externally created by changing the relative movement of the visual scene in response to head movements, such as would occur when putting on prescription eye glasses or an image-magnifying scuba mask. Regardless of how the mismatch occurs, the VOR is capable of making adaptive (plastic) changes to its gain and phase settings to correct for the difference and re-stabilize the image. First a model will be presented on how this adaptation takes place, followed by some general characteristics and influencing factors of this adaptation.

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[14] The cervico-ocular reflex may also play a small role.

Ito's model (Figure 7) offers a good general description of how the VOR adapts to internal or external changing conditions (Ito, 1972). The bottom of the figure is the three-arc VOR reflex with initial gain 'α' determined by amplitude of head movement. A side branch carries this signal through the vestibulocerebellum (VC) and returns it to the main branch with gain 'β', so that the total VOR gain after the vestibular nucleus (VN) is 'α minus β'. The model states that β can be modified using retinally derived visual information on image slip, and by changing β the overall VOR gain is changed (α minus β). Retinal slip information is believed to be relayed to the VC via the accessory optic tract (aot) and climbing fibers (cf). Therefore, the instantaneous VOR retains its low latency response (three-arc pathway) but the reflex can be parametrically recalibrated over the course of several occurrences (via VC pathways).

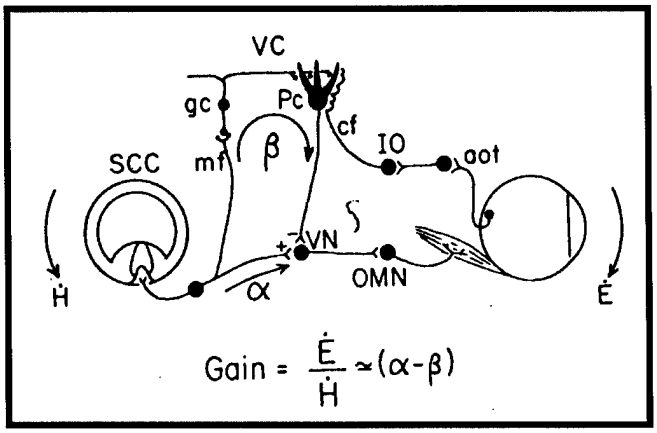

Figure 7: VOR Adaptation Model[15]

[15] From Ito (1972), as simplified by Robinson (1976, page 955).

As Ito's model shows, the VC appears to be vital to the VOR adaptation process. Robinson (1976) demonstrated that removing the VC did not significantly effect the normal VOR response but it did cause the total loss of the ability to adapt the VOR to changing conditions. Furthermore, if the VOR had already been adapted to a gain other than its natural value when the removal took place, the gain was reset to a value close to natural value. This suggests that the VC is a vital component not only in the adaptive process of the VOR but also in the maintenance of the adapted values.

2.2.3.2 Examples of VOR Adaptation

There are many examples of VOR adaptation in the literature. Below is a sample of the types of adaptation studied.

Many researchers have studied VOR gain adaptation (Cannon, Leigh, Zee, & Abel, 1985; Gauthier & Robinson, 1975; Gonshor & Melvill Jones, 1976a & 1976b; Robinson, 1976; Shelhamer, Robinson, & Tan, 1992; Viirre, et al., 1986). While most experiments have focused on the horizontal VOR, others have looked at the vertical VOR and torsion VOR as well[16]. Some researchers have investigated direction adaptation of the VOR (Khater, Baker, & Peterson, 1990; Peng, Baker & Peterson, 1994; Powell, Quinn, Rude, Peterson, & Baker, 1991). In this instance, the inertial stimulus of movement is provided along one axis (e.g., yaw) while the visual scene motion is presented along a different axis (e.g., pitch). This cross-coupling of inertial and visual movement stimuli result in a VOR gain component being generated along the axis of visual motion.

VOR phase adaptation has also been studied, though not near as often as VOR gain adaptation (Kramer, Roberts, Shelhamer, & Zee, In Press; Kramer, Shelhamer, & Zee,

[16] Torsional VOR adaptation is less often studied. An example is found in Bello, Paige, & Highstein (1991).

1995; Powell, Peterson, & Baker, 1996). Indeed, little is known about the VOR phase adaptation system.

Researchers have studied the short-term adaptation response of the VOR (Demer, et al., 1989; Gonshor & Melvill Jones, 1976a) as well as the very long-term effects (Gauthier & Robinson, 1975; Gonshor & Melvill Jones, 1976b; Paige & Sargent, 1991). Stimuli used have included magnifying spectacles (Cannon, et al., 1985; Demer, et al., 1987; Demer, et al., 1989; Gauthier & Robinson, 1975), optokinetic nystagmus (OKN) drums (Powell, et al., 1991; Shelhamer, Robinson, & Tan, 1992; Shelhamer, Tiliket, Roberts, Kramer, & Zee, 1994), and even VR HMDs (Kramer, et al., In Press).

2.2.3.3 Characteristics of VOR Adaptation

There have been many studies that examined the effects and limits of adaptation. Gauthier and Robinson (1975) were the first to demonstrate that human VOR gain could be increased (through the use of magnifying lenses) as well as decreased. Others have used image reversing prisms on humans and animals to see if the gain could in fact be reversed (Gonshor & Melvill Jones, 1976b). Although large changes in VOR gain were observed, rarely did the observed gain changes match the required changes to maintain image stability. This may have been due to the severe gain change demands that were required (commonly 100-200% or more above or below the current gain setting).

Collewijn, et al. (1983) demonstrated that if the required changes were relatively small (i.e., 36% or less), the VOR could fully adapt to the all of the new conditions within approximately 30 minutes and even within 3 to 5 minutes if the required gain change was small enough. This was an important finding because the majority of gain change requirements that the VOR system will encounter in real life and in simulated environments will be of this magnitude, not 200% or reversed vision. For example, simply putting on a pair of prescription glasses results in a 3 to 5% per diopter change in

the relative movement of the visual image (Cannon, et al., 1985; Collewijn, et al., 1983). In general, VOR adaptation has been shown to follow a decaying exponential time-course which is typical of many adaptation processes (Khater, Baker, & Peterson, 1990; Welch, 1986).

Collewijn, et al. (1983) tried to differentially adapt each eye to unequal gain change demands but found that it was not possible. If the unequal demands were close enough in size, the eyes would settle in on an intermediate adaptation level. If the discrepancy was large, however, the dominate eye's adaptation demand won out. This also makes sense, as the VOR functions as a conjunctive eye movement.

Lisberger, Miles, and Zee (1984) found that a full FOV of the visual field is not required to drive adaptation of the VOR. Using monkeys, they found that that 50-70% of full FOV VOR gain adaptation can be obtained using only a small spot of light. Shelhamer, et al. (1994) found no reduction of VOR gain adaptation between a full-field visual stimulus and a small LED set against a black background. This indicates that a full visual field is not required for VOR adaptation to take place. However, if the peripheral field is unobstructed such that regularly-occurring motion relationships exist in that area of the visual field, VOR adaptation to a centrally projected stimulus is severely impaired (Demer, et al., 1989).

There is evidence that at least two different VOR gains can exist simultaneously which can be toggled into use as the appropriate situation arises (Gauthier & Robinson, 1975). For instance, just putting on a scuba mask or a pair of glasses may immediately switch the VOR to a gain that is acceptable for that condition. The tactile feedback of putting on the goggles may be enough to drive the adaptation in some cases (Herdman, 1996; Shelhamer, et al., 1992). This is often called 'context specificity' (Herdman, 1996). In addition, transfer of VOR adaptation through context has been shown to depend on the particular context involved (Tiliket, Shelhamer, Tan, & Zee, 1993).

However, it has not been determined whether these gain values are truly stored or if the adapting mechanism simply speeds up its adaptation to conditions that have occurred often enough in the past.

Many researchers argue that VOR gain adaptation is frequency specific, which results in maximum adaptation at the adapting (i.e., testing) frequency and less adaptation at other frequencies (Lisberger, Miles, & Optican, 1983; Powell, et al., 1996; Zee, 1996). In fact, Lisberger, et al. (1983) developed a model of VOR adaptation that involves a series of overlapping frequency channels to account for his observation of frequency specificity. Others have argued, however, that adaptation at one frequency may be transferred across several frequencies (Demer, et al., 1989; Parker, personal communication, 1997). This issue will be discussed more fully in subsequent chapters.

2.2.4 Relationship to Sickness Symptoms

A functioning vestibular apparatus is required for motion sickness and simulator sickness to occur (Reason & Brand, 1975). In addition, sudden unilateral deficits in vestibular function temporarily result in the onset of motion sickness symptoms, but long-term deficits (partial as well as total) eventually decrease susceptibility to motion sickness (Graybiel & Johnson, 1963). Since the VOR also requires a functioning vestibular apparatus, it is reasonable to conjecture that simulator sickness and VOR response may be correlated.

The link between the VOR and sickness that relates most directly to this dissertation, however, is derived from VOR adaptation research. VOR adaptation is a physiological process that is directly driven by visual-vestibular mismatches. Many studies investigating VOR gain adaptation (Demer, et al., 1989; Gauthier & Robinson, 1975; Gonshor & Melvill Jones, 1976b; Istl-Lenz, et al., 1985; Paige & Sargent, 1991, to name a few) have shown that subjects (be they dogs, cats, monkeys or humans) often

experience dizziness, postural ataxia, nausea and other motion sickness symptoms during adaptation to the altered visual-vestibular stimulus. Many of these studies also revealed that these same symptoms reemerged immediately after the altered visual-vestibular stimulus was removed and existed for a time while the subjects 'readapted' to their normal environment (Gauthier & Robinson, 1975; Paige & Sargent, 1991). These re-adaptation symptoms are generally less severe and have a shorter time-course than those experienced during adaptation away from the normally occurring visual-vestibular stimulation. This correlates with speed of oculomotor re-adaptation, which is often more rapid than oculomotor adaptation.

Demer, et al. (1987) attempted to demonstrate empirically that VOR adaptation reduces visual-vestibular conflict. These researchers measured the VOR and VVOR of several subjects before exposure, immediately upon presentation of a 2.2X magnifying lens stimulus, and after 15 minutes exposure to the adapting lens. Subjects rotated passively at 0.1 Hz during stimulus presentation. These researchers found that upon initial presentation of the stimulus, VOR gain was insufficient and dynamic visual acuity was low. Subjects experienced ataxia, oscillopsia, and one experienced nausea. However, after the 15 minute exposure, VOR and VVOR had increased and dynamic visual acuity was significantly improved. Thus, when presented with a stimulus that drives VOR adaptation, the resulting adaptation will also eventually result in improved visual performance and less oscillopsia. In addition, one subject who experienced nausea also showed erratic and extra low gain VOR responses immediately after exposure to the stimulus, prompting the researchers to postulate that VOR adaptation may be an indicator of motion sickness in space.

Not all studies in this area deal directly with VOR adaptation. One particular study by Gordon, Spitzer, Doweck, Shupak, and Gadoth (1996) attempted to relate VOR gain and phase parameters to seasickness susceptibility. They found that those subjects

highly susceptible to seasickness had VOR responses that were of significantly higher gain at 0.02 and 0.04 Hz and of significantly lower phase lead at 0.01, 0.02, 0.04, and 0.08 Hz than nonsusceptible subjects. They argue that these results support the assertion the those susceptible to motion sickness have more intense vestibular response in the most provocative motion frequency range. However, since the significant differences were relatively small and the data variance was large, there was no clear-cut distinction that could be made between the vestibular response of susceptible vs. nonsusceptible subjects.

These researchers also discussed the results of an earlier study that not only showed a relationship between decreased VOR gain and reduced sickness, but also that 6 months at sea resulted in a decrease in VOR gain at 0.2 to 0.4 Hz. Therefore, susceptibility to sickness may be due in part to a naturally existing lower VOR gain at the provocative frequencies and/or the ability of seamen to adapt their VOR response to these same frequencies. As quoted by the authors, "It is quite probable that during repeated sea voyages, subjects are exposed both to unnatural low frequency motion stimulation, resulting in vestibular habituation and to visual-vestibular mismatch, which produces adaptive VOR modifications". Thus, a connection is hypothesized between VOR habituation/adaptation and sickness susceptibility.

Several researchers have looked at VOR parameters in relation to space sickness. For instance, DiZio and Lackner (1991) found sickness to be related to greater capacity for VOR velocity storage and more precipitous velocity dumping during post-rotary head tilts. Many studies have indicated that VOR gain decreases in hypogravity (Lackner & Graybiel, 1981; Vesterhauge, Mansson, Johansen & Zilstorff, 1982) though Watt (1987) argued that the effect is transitory and does not follow the same time course as space sickness. As space sickness is theorized to mainly involves a canal/otolith

conflict which is not implicated in most terrestrial simulator sickness environments, the above results may be less generalizable to this effort.

Many other researchers have attempted to relate specific VOR parameters to various forms of motion sickness with mixed success (Kennedy, Dunlap, & Fowlkes, 1990). These VOR metrics centered around the various capabilities of the vestibular apparatus (e.g., cupulogram, velocity storage) as tested using a rotating chair and caloric irrigation, and the goal of most of these efforts was to predict individual susceptibility.

2.2.5 Relationship to the Tracking Response of the Eye

The VOR does not act in isolation in attempting to keep images stabilized on the retina as the head rotates. There are also visually-based stabilizing mechanisms. These mechanisms will be described next, along with their relationship to the VOR.

The VOR is one of five major types of eye movements. The other four (i.e., optokinetic, saccade, smooth pursuit, and vergence) function with the VOR to: 1) bring visual targets onto the fovea and 2) keep them there. Optokinetic and smooth pursuit are considered visual tracking eye movements as they both use input signals for directing the eye that are derived from the retina itself and their function is essentially one of image stabilization, not image capture[17]. Therefore, this discussion examined how both of these tracking mechanisms relate to the VOR.

2.2.5.1 Optokinetic Reflex (commonly termed OKN for optokinetic nystagmus)

While the VOR compensates for head movement by using inertial input from the vestibular apparatus, the optokinetic reflex (i.e., OKN) works to maintain a stable retinal image through visual input. The OKN system uses visual information coming from the entire retina (not just the fovea) to detect if an image slip is occurring.

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[17] Saccades and vergence commands are considered to be 'image capture' eye movements.

If slippage occurs, a corrective OKN eye movement is generated to compensate for it by moving the eye with equal gain in the opposite direction of the optical flow. Therefore, while both VOR and OKN reflexes serve the same general purpose and both are considered involuntary, the VOR uses vestibular input to generate oculomotor compensation commands while the OKN uses visual input to do the same. In fact, OKN stimuli can produce optokinetic nystagmus that appears similar to vestibular nystagmus discussed earlier, but while head rotation is required for vestibular nystagmus to occur, only a large-field moving image is required to produce optokinetic nystagmus. Why would there need to be two separate systems to perform essentially the same image stabilizing task? It turns out that they both work in synergistic fashion to maximize the eye compensation response to any head movement.

First, consider the fact that even under conditions of EVOR the gain of the resulting VOR averages approximately 0.95 (not the required 1.0) to keep the eyes exactly stabilized in space[18]. Yet the same VOR measured in the light does have a gain equal to 1.0 at most natural frequencies. Visual information provided by the lighted conditions allows for the correction of VOR residual error through visual tracking mechanisms (Peli, 1995). These visual tracking mechanisms are likely a combination of OKN and possibly a fixation system. Below is a description of how the VOR and OKN combine to provide optimum image stabilization performance.

The VOR is a very fast reflex that serves to compensate effectively for head movements at frequencies in the range of approximately 1 to 7 Hz, especially if the head movement is voluntary (active) (Robinson, 1981). However, the VOR is less accurate at lower frequencies, especially those lower than 0.1 Hz where the gain drops significantly and a phase lead appears. The OKN has the opposite performance characteristics. It is longer latency (due to the fact that it uses visual input which takes

[18] Assuming head movements between approximately 0.1 and 1.0 Hz.

time to process) but at low frequencies (i.e., less than 0.1 Hz) it has near unity gain and no phase lead/lag. From 0.1 Hz to approximately 1.0 Hz, the OKN loses gain and develops a phase lag due to its processing latency (Peterka, Black, & Schoenhoff, 1987). At higher frequencies it cannot effectively compensate due to its relatively long latency and low gain compared to the VOR. Therefore, the combination of the two mechanisms allow for maximal image stabilization all the way from the lowest frequencies (governed mostly by OKN) to the highest frequencies (governed by VOR).

There is another aspect of the VOR/OKN combination that contributes to improved performance over either system alone. This aspect is a timing issue: time of onset and time of offset. Earlier it was mentioned that the VOR has a very short latency (onset time) while the OKN has a longer latency. The VOR allows for a faster reaction time even at lower frequencies. But it was also mentioned that the VOR will eventually decay during constant, zero-acceleration rotation due to the elasticity of the cupula. Although effectively extended through central processes, the time constant of pure vestibular nystagmus in humans is approximately 20 to 25 seconds (Howard, 1986a). The OKN, however, has a long latency but does not decay with repeated stimulation of the retina by an optical flow. Therefore, as the VOR decays, the OKN is building up, creating a continual seamless stabilization of most images on the retina.

Finally, it was stated earlier that VOR adaptation requires visual input from the retina regarding image slip. This information is also used by the OKN. Therefore, it may be possible similar mechanisms serve to provide this input to both processes, further coupling the two reflexes. In addition, cells exist in the VN and cortex that have the same response to either vestibular or OKN inputs.

As discussed, the VOR and OKN have different areas of specialization in latency and frequency range. Alone, neither reflex can account for all possible head movements, but together they provide an excellent means to keep an image stabilized on

the retina across the entire spectrum of head movements. A descriptive model of this relationship is shown below (Figure 8).

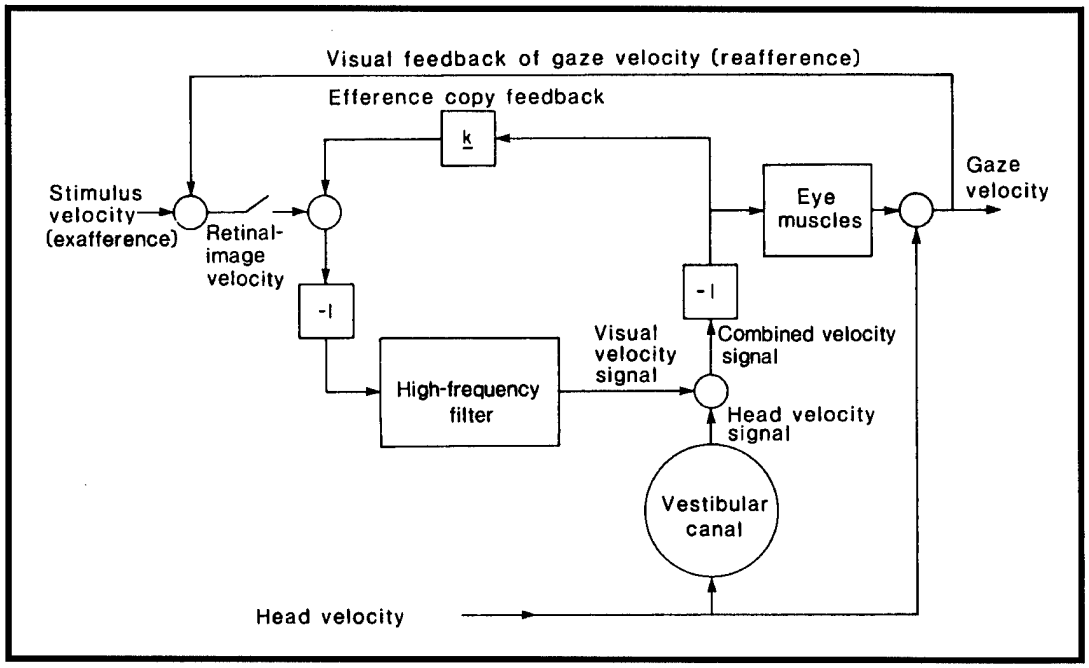

Figure 8: Descriptive Model of OKN/VOR Relationship[19]

This model accounts for how visual and vestibular signals cooperate to produce image-stabilizing eye movements (Howard 1986b; simplified from an earlier model by Robinson). Starting at the left of the diagram, stimulus (target) velocity is combined with visual feedback on current eye velocity to generate a retinal image-slip velocity signal. This signal is adjusted by efference copy (with gain 'k') which is an inner-feedback loop that provides prediction information on the current eye movement plans.

[19] From Howard (1986b, page 18-18).

The resulting visually-mediated eye-command component is phase inverted and run through a high frequency filter so that the high frequency information is rejected (due to the lags/inaccuracies of the optokinetic system at higher frequencies). This final visual-velocity signal is then combined at the vestibular nucleus with the head-velocity signal coming directly from the SCCs, and the resulting combined-velocity signal is phase inverted (due to the compensatory nature of eye movements) and sent to the eye muscles through the oculomotor neurons to stabilize the image. This final combined signal is also the efference copy that is fed back to affect future eye movements. Thus this model combines visual and vestibular signals in simple linear-additive fashion at the VN. One addition that might also be included is perhaps a low frequency filter (for frequencies below 0.05 Hz) to negate the poor low frequency response of the vestibular system.

2.2.5.2 Smooth Pursuit

Another type of eye-tracking response is accomplished by smooth pursuit eye movement. Smooth pursuit, like OKN, uses visual input to generate eye movements, but instead of functioning to keep the entire retinal image from slipping, smooth pursuit moves the eye to keep only the image that is on the fovea stabilized (Robinson, 1981). Smooth pursuit uses visual information of the foveal target to calculate its velocity so that the eyes can move accordingly. As an example, following the movement of a rabbit darting in a field would require the rabbit to always remain nearly stabilized on the fovea while the rest of the image (the field) slipped across the peripheral retina. Predictability of target motion will increase smooth pursuit performance and bandwidth (Robinson, 1981). Smooth pursuit is also often considered a voluntary eye movement (while OKN is involuntary) although some researchers (as cited by Furness, 1981) have questioned this.

So what would happen if smooth pursuit and VOR occurred at the same time? This could happen, for instance, if you rotated your head from side to side (or up and down) while following the darting rabbit in the field. Another example (quite appropriate to HMD applications) is when you attempt to pursue a target that moves with your head. Each eye movement type has different goals; the VOR strives to keep the eye stabilized in space while the smooth pursuit attempts only to have the moving target image remain on the fovea. Since their goals are conflicting in these examples, which would override the other? Benson and Barnes (1978) showed that VOR suppression by the pursuit commands occurs up to approximately 1 Hz[20]. Therefore, up to a limit, the pursuit mechanism would cancel the VOR commands (likely through central processes) and allow for the continued pursuit of the target. Above approximately 1 Hz, VOR dynamics begin to override the visual pursuit system. VOR enhancement and suppression may incorporate some pseudo-pursuit system to follow the imagined points in the dark (in the distance or on the chair respectively).

2.3 VIRTUAL INTERFACES

This section provides an overview of virtual interfaces, their theorized benefits and drawbacks, and interface artifacts imposed by current limitations in the supporting technology. Given that most readers of this dissertation will be familiar with VR and virtual interfaces, this section is intentionally brief. However, for a more detailed discussion of VR technology, issues, and applications, readers are directed to Barfield & Furness (1995), Durlach & Mavor (1995), Kalawsky (1993), or Rheingold (1991).

[20] Stimulus predictability can cause an increase in bandwidth up to approximately 2 Hz.

2.3.1 Overview

Virtual interface is defined as a system of transducers, signal processors, computer hardware and software that create an interactive medium through which: 1) information is transmitted to the senses in the form of two or three dimensional virtual images and 2) psychomotor and physiological behavior of the user is monitored and used to manipulate the virtual images (Furness, 1994). In other words, virtual interfaces interactively and naturally connect the user to information and/or a task through a VE. It is a special form of human-computer interface that goes beyond the confines of the VDT, keyboard, and mouse.

Virtual interfaces strive to provide naturalistic transfer of information between human and computer. Since humans have developed over the ages to accept information simultaneously over multiple sensory channels, virtual interfaces attempt to make use of these pathways for increased bandwidth (Barfield & Furness, 1995). Virtual interfaces involve high bandwidth, multi-sensory transfer of information involving visual, auditory, haptic, tactile, kinesthetic, and occasionally even olfactory modalities (Durlach & Mavor, 1995). They can also quickly and completely immerse the user in the synthetic environment being experienced. In addition, virtual interfaces have the ability for higher interactivity (especially naturalistic interaction), intuitiveness, and inclusiveness than traditional interfaces (Furness, 1994).

Head-coupled virtual interfaces can be more or less condensed into four major categories: immersive HMD, immersive CAVE, augmented reality, and desktop VR. The choice of a particular configuration is a function of task, immersiveness desired/required, number of participants, availability, costs, and other factors.

Immersive HMD designs involve the wearing of an HMD that displays a head-coupled VE while occluding the subject's view of the outside world. As the subject moves his/her head, the visual scene is updated for correspondence. These designs

generally offer the highest potential degree of immersiveness and currently are the most popular configuration. However the costs can be prohibitive with many limitations due to existing technology, especially for multiple participant environments.

Immersive CAVE configurations involve a room with the walls, ceiling and floor acting as display screens for rear mounted projectors (Cruz-Neira, Sandin, Defanti, Kentyon, & Hart 1992; Cruz-Neira, Sandin & Defanti, 1993). The subject moves through the room while viewing the projected images which change in accordance with these movements. These configurations have the advantage of less head encumbrance (due to that fact that displays are not worn on the head) and the potential for multiple participants to be involved simultaneously (though only one would be head-tracked). Time delays for head *rotational* motions are eliminated with this configuration (except if stereo images are displayed) but translational time delays remain. In addition, physical hardware can be placed in the room to enhance the experience (much like flight or driving simulators). However, close-in virtual objects are not displayed as accurately as in the HMD immersive designs. Costs can be high for CAVEs and a large dedicated space is required.

Augmented reality uses a semi-transparent surface to display the virtual image. The subject can see through this display to the outside world. Therefore both virtual and real images are combined into an integrated scene. This is sometimes termed 'enhanced reality'. The display is generally worn on the head and the virtual image is updated in accordance with head movements. There are significant technical issues that need to be addressed (including registration and occlusion issues) but this option offers many benefits including reduced rendering requirements, increased fidelity, lighter HMDs and reduced costs.

Lastly, desktop VR systems display a VE on a conventional VDT. Usually some provision is made for stereoscopic viewing (either through a display modification or the

use of special glasses worn by the subject). These configurations are generally less immersive, but they are inexpensive and relatively easy to implement.

This dissertation focused only on immersive HMD configurations as they are the most popular and provocative designs. However, relevant issues in adaptation can be identified in each configuration so future research should explore these areas as well.

2.3.2 Virtual Interface Design Challenges

Virtual Interfaces theoretically offer many benefits. However, actually designing a virtual interface that fully meets user expectations is nearly an impossible feat. The newness of the interface, changing technology, and multi-disciplinary design teams create certain challenges for the virtual interface designer.

Virtual interfaces are new. There is no history to draw upon regarding proper design, mistakes to avoid, etc. There are no existing industry standards on the design of HMDs, position sensing trackers, VE renderers, input devices, etc. Rather, the industry is fiercely competitive with proprietary technologies and approaches. Because of this, many sub-interfaces are additionally required to make virtual interfaces functional, such as video signal converters, special processor cards, serial adapters, null modem cables, even separate PCs. This creates a complex and suboptimal situation. Many university studies, instead of helping to focus the industry, are likewise fragmented and as such provide benefits only within the restricted domains dictated by the hardware/software and applications chosen. Standards are needed but they do not currently exist.

A related problem is that virtual interfaces employ temporary technology. Once a supporting technology has been around long enough to be fully understood, it is often replaced by a new, 'improved', technology. Sometimes this technology is a variant of

what came before but often it represents a new approach to the problem[21]. These new approaches bring new benefits and new costs. Even those in the field find it difficult to keep up with the newest 'solutions' to existing problems. Without standards to go by nor a stable technology base, designers are essentially working without a net[22].

To compound things, virtual interface design is a multi-disciplinary field often involving computer hardware and software experts, engineers, human factors professionals, graphics designers, artists, and psychologists (Durlach & Mavor, 1995). These diverse groups highlight the complex nature of advanced interfaces. The difficulty of design can be further compounded by communication difficulties and competing interests between the specialists involved.

All of these challenges will likely disappear in the long run. Industry competition and trial-and-error bring standards. Technology change will eventually asymptote, and multi-disciplinary teams will soon become a tremendous asset toward proper interface design. But until then the challenges remain.

2.3.3 Current Technology Limitations

As stated earlier, all virtual interface applications are limited by existing technology in regards to the ability to accurately simulate reality. Some systems offer more realism than others, but all fall short of perfection (as defined by human sensory capabilities) in several areas (Barfield & Furness, 1995; Durlach & Mavor, 1995; Kalawsky, 1993). Below is a partial listing of where VR systems typically are limited. Each of the listed artifacts has the potential to generate sensory rearrangements within the user.

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[21] As an example, in this research I switched from using a mechanical head tracker (the 'then' best solution, to using an inertial, sourceless tracker (the 'now' best solution).

[22] However they do have use of the internet, but that is another dissertation.

2.3.3.1 Time Latencies

Time delays exist between movements made by the user (which are tracked by some position-sensing device) and the response of the virtual interface system to those movements (Durlach & Mavor, 1995). The most common time delay discussed in head-coupled VR is the delay between when the subject moves his/her head and when the virtual scene is updated to correspond with that movement. This time delay was a focus of this research.

It would be misleading to think that the time delay between head movement and scene response is a singular entity. Rather, it is the result of several smaller time delays within the system (Silverton, 1994). The components of overall system time delay include tracker delay (sensing, filtering, and transmitting the position data), graphics computer delay (processing of position inputs and rendering of the image), and display delay (display response time and display decay time). Silverton (1994) provides a detailed analysis of each component of overall system time delay within virtual interfaces.

Much of the system time delay is fixed and determined by the particular hardware chosen. For instance, mechanical trackers have total throughput delays of approximately 3 to 7 ms while electromagnetic trackers can range from 10 to 250 ms or more (depending on choice of filtering, specific system involved, and number of sensors activated) (Meyer, Applewhite, & Biocca, 1992). With regards to graphics processors, the more powerful the computer the less processing and rendering delay is involved (Kalawsky, 1993). Finally, display lag is generally limited to LCD displays, as CRT displays have fast response time and short persistence (Silverton, 1994). However, even the refresh rate can effect system time delay (see Appendix A).

A portion of the system time delay is variable. As the visual scene becomes more complex (though increased interaction, more virtual objects, more texture mapping,

more object dynamics, etc.) the speed of rendering new images decreases (Silverton, 1994). Other factors may also change the system time delay during an exposure, such as moving too far away from the position-sensing transmitter (e.g., with electromagnetic tracking systems).

Time delays are hypothesized to be among the areas most in need of improvement in simulators and virtual interface design (Ebenholtz, 1992; Furness, 1994; Kennedy, Hettinger, & Lilienthal, 1990; Pausch, 1991; Peli, 1995). Time delays between head movements and virtual image response may generate a sensory rearrangement between visual and vestibular cues of motion. These sensory rearrangements are hypothesized to induce simulator sickness and reflex alterations (Peli, 1995; Reason & Brand, 1975; So & Griffin, 1995).

The only research related to this issue examined time delays between a subject's control input and visual scene update in a simulator (Frank, Casali, & Wierwille, 1988; Uliano, Lambert, Kennedy, & Sheppard, 1986). These researchers found no effect of increasing time lag on simulator sickness, whether the simulator was a car or an aircraft. However, the task involved (control inputs via a joystick or steering wheel) is not as intimately coupled to visual scene response as is active head movements.

Task performance is adversely effected by large time delays between control input and visual scene update (Durlach and Mavor, 1995; Frank, Casali, & Wierwille, 1988; Kennedy, Hettinger, & Lilienthal, 1990; Pausch, Crea, & Conway, 1992; So & Griffin, 1995). So & Griffin (1995) found that head tracking performance was significantly degraded at system time days of 80 ms. Lastly, time delays can effect user acceptance of the VE (Pausch et al., 1992).

Given that an ideal system with zero delay is currently unattainable, it would be beneficial to determine a specification for an acceptable minimum time delay in virtual

interface design. Furness (1994) believes that 16 ms total throughput delay is a reasonable goal and Kalawsky (1993) has indicated that 5 ms is probably necessary. The threshold value may vary with configuration employed, task performed, and individual characteristics of the user. However, more study is needed to ascertain the effects of time delays and threshold requirements.

One way to reduce lag is to improve the associated technology. Another way is through prediction of head movements. Liang, Shaw, and Green (1991) applied Kalman filters along with head movement models to predict future values of head position data. Only head rotations were modeled (filtered head position data were used for translations). Their algorithm did show some predictive value but not all lag was eliminated, new noise was introduced, and fast movements were not correctly handled. Nevertheless, prediction is a potential solution to the time delay problem (So & Griffin, 1995).

2.3.3.2 Display Field-of-View (DFOV)

DFOV is the visual angle subtended by the display screen from a given observer location (McKenna & Zeltzer, 1992). This parameter depends on screen size and screen distance from the observer's eye (Figure 9). DFOV is often described in terms of its horizontal and vertical components.

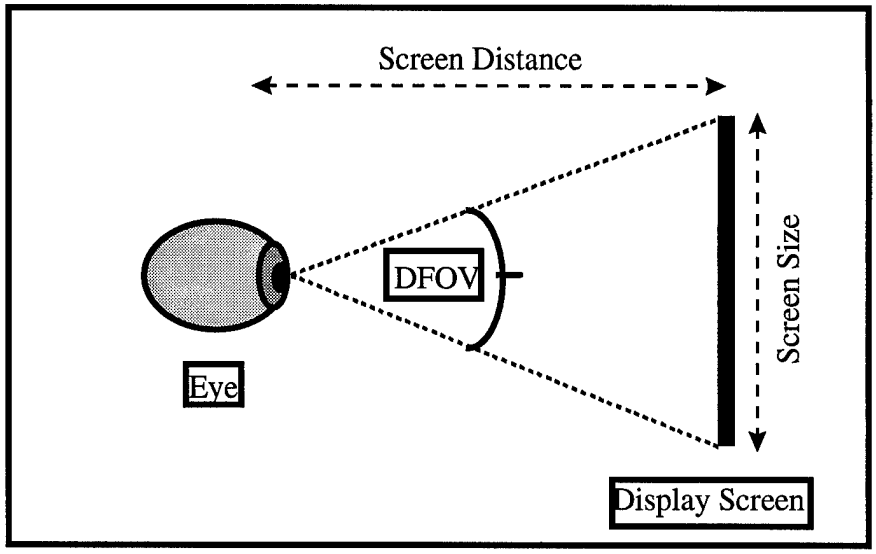

Figure 9: DFOV[23]

DFOV is often desired to be as large as possible to promote a sense of immersiveness (presence) within the VE. However there are technical limitations that preclude obtaining a DFOV on a HMD that nears a human's natural field-of-view, which is approximately 200 degrees horizontal by 120 degrees vertical (with a 120 degree overlap between the two eyes)(Kalawsky, 1993). An average DFOV on reasonably priced HMDs available today is approximately 40 degrees horizontal by 30 degrees vertical (VR News, 1995).

A tradeoff that occurs when one tries to increase DFOV using an existing display is a corresponding reduction of screen resolution. Given a fixed display size, the only way to increase DFOV is to either magnify the display using optics or move the eye closer to the screen. In either case, pixel size increases in the same proportion as screen size (since both are fixed values). As pixel sizes increase, display resolution decreases.

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[23] Horizontal or vertical DFOV, depending upon chosen perspective.

2.3.3.3 Geometric FOV (GFOV)

Before a virtual image can be displayed, the computer graphics system must determine which part of the virtual world is viewable by the observer. The virtual image must be reduced from three-dimensions to two dimensions for display on a screen. The technique most often used for this image generation is perspective projection (Figure 10). The viewable image is determined using a center-of-projection (COP), a defined viewing frustum, near and far clipping planes, and a viewport (Danas, 1995; Foley, van Dam, Feiner, & Hughes, 1996). Figure 11 graphically indicates the process of determining which image should be rendered onto the display.

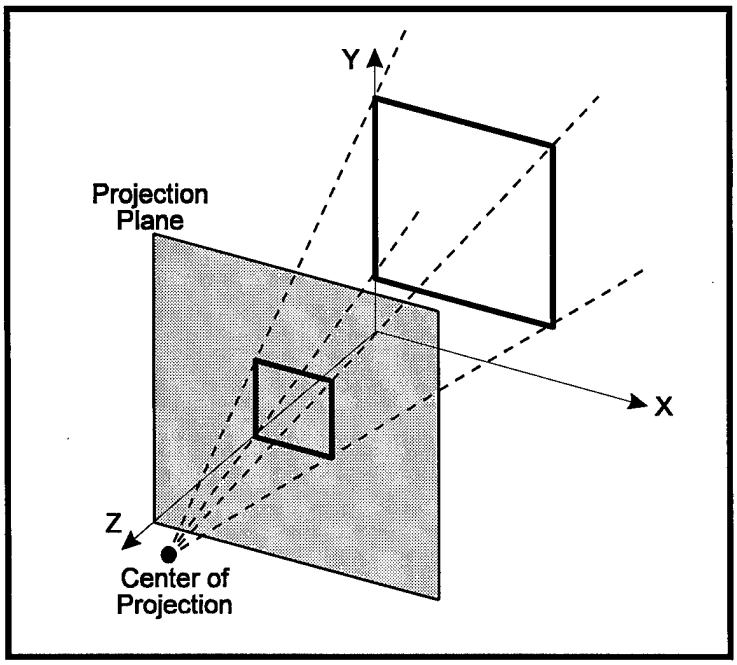

Figure 10: Perspective Projection[24]

[24] From Danas (1995, page 23).

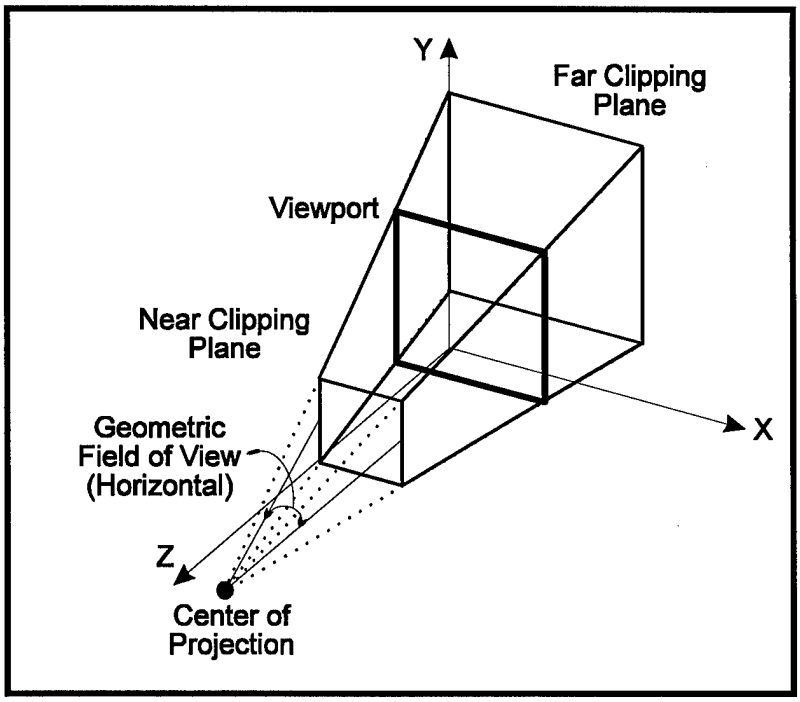

Figure 11: Viewing Frustum[25]

The COP represents the position of the observer in virtual space[26]. The viewing frustum defines an arbitrary view into the three-dimensional virtual space and determines the horizontal and vertical boundaries of the image to be rendered. The two clipping planes define the fore and aft boundaries of the image to be rendered. The viewport is a window within the viewing frustum onto which all imaged points in the VE (i.e., all points within the region defined by the frustum and clipping planes) are projected. The image projected onto the viewport will be the image rendered on the physical display.

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[25] From Danas (1995, page 24).

GFOV is the angle subtended by the viewport at the COP in virtual space (it could equivalently be defined as the angle subtended by the viewing frustum)(Figure 11). It essentially defines the viewable scene and aspect ratio for image generation. Though both horizontal and vertical GFOVs exist, this dissertation focused solely on the horizontal GFOV.

The image that is projected on the viewport will subsequently be rendered on the physical virtual interface display. This is how GFOV and DFOV interrelate, as is shown on the right side of Figure 12. If GFOV is less than the DFOV, the viewport image will appear to be magnified on the display due to the requirement for the image to fill a larger subtended angle in physical space vs. virtual space. If the GFOV is greater than the DFOV, the resulting displayed image will appear miniaturized compared to the corresponding image in virtual space (because the angle to be subtended in physical space is less than that used to create the virtual image). If GFOV is equal to DFOV, however, the image on the viewport will correctly scale onto the physical display. This magnifying or minifying effect, termed the 'virtual space effect' (Wickens, Todd, & Seidler, 1989), can result in image distortions as well as scale changes.

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[26] For monoscopic displays.

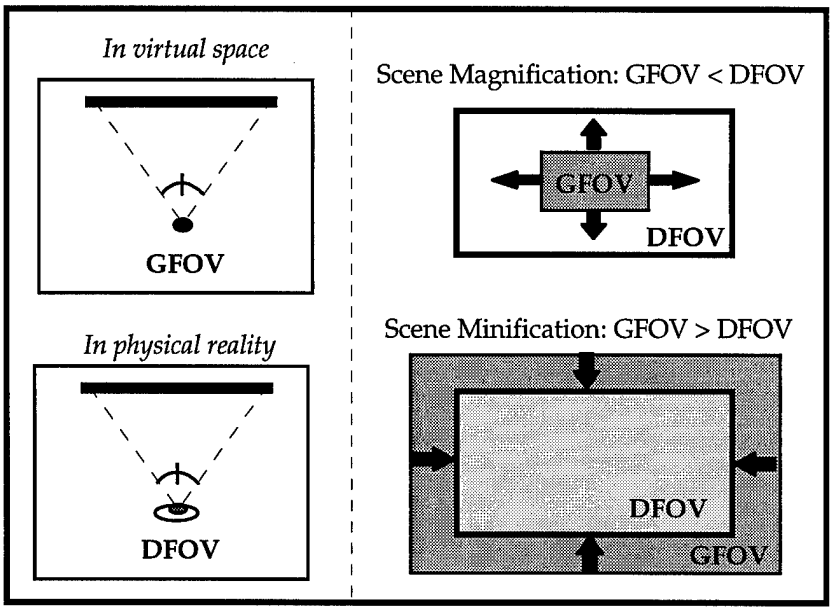

Figure 12: GFOV/DFOV Relationship

Research has investigated the effects of GFOV on performance and level of presence experienced (Boyer & Wickens, 1994; Danas, 1995; Hendrix, 1994; Neale, 1996). However, few studies have explicitly considered the relationship between GFOV and DFOV (Danas, 1995 is a notable exception). Rather, GFOV is often manipulated without mention of the existing DFOV and results imply that there is a 'best' GFOV for all DFOVs. This is not the case, as GFOV is meaningful only in terms of its relationship with DFOV. If these two values are equal, the individual will perceive a spatially accurate scene view as defined by the spatial dimensions of the virtual space model. If this ratio deviates from 1:1, the result will be change in optic flow velocity during rotational motion and either image magnification (if GFOV < DFOV) or image minification (if GFOV > DFOV) of the scene[27]. Therefore, results that describe an

[27] This is discussed further in Section 3.1.3.2

'optimum' GFOV without mention to DFOV can only be optimum for the particular DFOV used in that experiment, at least for HMD applications.

2.3.3.4 Resolution

Resolution is a measure of the level of detail available in a display (Kocian & Task, 1995). However, it can be a very misrepresented parameter in HMD specifications. Often, resolution is described in terms of number of pixels in a display. However, the size of the display and its distance from the observer also contribute to the effective resolution. As discussed earlier, increasing DFOV reduces effective resolution by enlarging each pixel in the same manner. Therefore a more effective manner in which to specify resolution is in terms of visual angle subtended per pixel, termed 'angular resolution'.

Another potential confusion that is associated with color displays is the pixel-type used for determining angular resolution. Color display pixels are formed by grouping 3 or 4 monocular pixels of different wavelengths (such as red, green, and blue). Though it would seem obvious that display manufacturers should report the number of pixels and angular resolution based upon the existing color pixels, often they report these values based upon the total number of monocular pixels available. This misleadingly increases the advertised resolution by a factor of 3 or 4.

Angular resolution is very poor in most current HMDs. The resolving capability of the human eye is approximately 1 arcmin visual angle (Kocian & Task, 1995), though some reports claim 30 arcsec and even down to 5-10 arcsec for hyperacuity measures (McKenna & Zeltzer, 1992). An average resolution of current reasonably priced HMDs is approximately 10 arcmin of visual angle (VR News, 1995). Therefore, the average resolution of virtual interfaces (using HMDs) is at least 10 times worse than normal viewing conditions. This is equivalent to a score of around 20/200 on the Snellen acuity

chart, which is near the legal limit of blindness (Zwern, 1995). This is a rather conservative number as well, considering the hyperacuity of the eye and the fact that some HMD manufacturers report specifications based on total number of pixels instead of total number of color pixels.

2.3.3.5 Tracking Resolution/Accuracy

The ability of virtual interfaces to sense, record, and transmit changes in observer position is a fundamental component of head-coupled operation. However, even the best trackers on the market today fall short of perfect accuracy, resolution and repeatability (Meyer, Applewhite, & Biocca, 1992). Resolution is the smallest change that the system can detect. Accuracy is the range within which a reported position is correct. Repeatability is the ability of the tracker to correctly sense a set position on repeated occasions. In addition, these parameters often vary with position within the working volume.

Tracking accuracy depends largely on the technology used (Meyer, et al., 1992). Often, the most accurate trackers use mechanical linkages to provide good resolution, low latency, and repeatable response. They provide a consistent response because they are relatively unaffected by errors induced by the environment. Other tracking technologies such as optical, electromagnetic, and acoustic can provide accurate readings within a limited range. As the range increases, tracking accuracy decreases. Also, these technologies are subject to distortions introduced by the environment (Meyer, et al., 1992).

2.3.3.6 Other Limitations

There are many other technological limitations of current virtual interfaces. All have limited range of operation. All involve some encumbrance by the user, though this varies with particular equipment chosen. The size and weight of the HMD is often a

major source of discomfort. There are also numerous limitations in the representation of haptic and kinesthetic stimuli and the effective use of force-feedback. Detailed accounting of each of these limitations (and more) can be found in Durlach & Mavor (1995) or Kalawsky (1993).

2.3.4 Summary

Virtual interfaces have much potential and are currently being developed for application in many different fields. Research and development is progressing in the areas of immersive VR, CAVEs, augmented VR and desktop VR. Current technology limitations create sensory distortions that are relatively unexplored as to their effects on the user. There are many stimulus rearrangements of interest in terms of their potential for driving adaptive processes. This dissertation focused on two of these rearrangements in detail, time delays and GFOV/DFOV deviations.

2.4 SIMULATOR SICKNESS

This section first discusses the various definitions of simulator sickness. The characteristics and implications of simulator sickness are then described. Several competing theories of this malady are summarized next. Influencing factors, including a person's 'receptivity' and 'adaptability', are then discussed. Finally, current metrics of simulator sickness are presented.

2.4.1 Simulator Sickness Defined

Simulator sickness is a variant of motion sickness that is commonly experienced by participants in flight simulators, driving simulators, and VR environments[28]. In fact, the

[28] Note that some researchers are investigating whether sickness that arises from simulators has a different symptom profile than sickness from virtual interfaces (Stanney, Kennedy, & Drexler, 1997).

terms 'motion sickness' and 'simulator sickness' are often interchanged in the literature, as most would agree that much of their underlying physiological mechanisms are common.

Simulator sickness can be considered as being induced by an incongruence between the real world and the simulated world. Computer simulations of a real environment are necessarily incomplete because: 1) simulations can only reproduce a subset of all the information available in the real world environment, 2) this subset of information is almost always distorted during the simulation process, and 3) new/superfluous information is often unavoidably added to the simulation that does not exist in the real world environment.

Given that distortions necessarily exist in simulated environments, simulator sickness refers to sickness symptoms that result from an inaccurate simulation, not sickness caused by a correct simulation of a nauseating experience (Pausch, et al., 1992). If the characteristics of a simulator do not match the perceptual expectations of the human, simulator sickness may be experienced which is due to those perceptual inaccuracies (i.e., sensory rearrangements per the sensory rearrangement theory). If all aspects of the simulation exactly match human perceptual expectations, any sickness symptoms that arise would thus be termed motion sickness.

There is benefit to defining terms in this way. There are many reports of individuals becoming sick as a result of a simulation of a benign environment (i.e., the environment and task is benign in the real world but becomes provocative *as a result of the simulation*). If sensory distortions of simulators and/or virtual interfaces are identified as inducing simulator sickness, they can be corrected for through redesign. This would result in an overall reduction of simulator sickness incidence without having to solve the entire motion sickness syndrome. Thus, there is applied value to differentiating simulator sickness and motion sickness in terms of the suspected sources of the malady.

However, it is unlikely that the exact cause of simulator sickness can be determined in some simulations. This is the case for simulations of environments that are borderline provocative in the real world. However, if a detailed investigation of existing simulation distortions is conducted, this knowledge can be used to reduce the effects of simulator sickness even in these borderline cases. The symptoms that remain are likely to be present in the physical manifestation of that environment as well[29].

Some researchers have argued that motion sickness requires the existence of inertial motion stimuli while simulator sickness is primarily visually-based (Kolasinski, 1995). The argument holds that if a person in a simulator receives only visual motion stimuli then simulator sickness can occur, if only inertial motion stimuli is presented then only motion sickness can occur. This, however, does not account for human movement behavior. Voluntary movement in fixed-based (visual movement) simulators result in the generation of substantial inertial and proprioceptive movement cues in addition to the visual motion input. This argument also defines sickness arising from motion-based simulators as consisting of both motion sickness and simulator sickness while sickness arising from OKN booths is considered to be only simulator sickness. Note that there is no applied 'simulation' in the latter case but there is in the former. Thus this classification scheme is not precise, independent, or delineating of simulators from other environments.

Therefore, it seems more reasonable to consider motion sickness and simulator sickness as reflecting slight variations of the same underlying physiological process involving the interactive relationship between visual, vestibular, and somato-sensory cues. Identifying provocative factors within the simulation has the added benefit of directly addressing applied simulator sickness problems. With this intent in mind, a

[29] Which can be cross-checked through the use of a Motion Sickness Questionnaire (MSQ) on those exposed to the real environment.

classification scheme was developed so that different studies on simulator sickness could be more appropriately compared and generalized (Appendix G).

Despite the above arguments, many researchers define simulator sickness as consisting of *any* sickness symptoms that arise in a simulator or virtual interface, regardless of whether the source is simulation inaccuracies or provocative experience. There is goodness in this framework as well because it allows discussion of overall sickness effects without having to assign a specific source(s) to symptoms. In addition, if the simulated task/environment is inherently benign in the real world, this definition converges with that which specifies simulator sickness as being induced strictly by simulation inaccuracies (as described earlier). Therefore, the remaining literature should be read with this more general definition in mind. This issue of defining simulator sickness is revisited in Chapter 9.

2.4.2 Characteristics of Simulator Sickness

There are many potential symptoms of simulator sickness. They include but are not limited to nausea, headaches, sleepiness, sweating, apathy, dizziness, general fatigue, lethargy, eye strain, and loss of skin color (Ebenholtz, 1992; Kennedy, Hettinger, & Lilienthal, 1990; Pausch, et al., 1992). A difference between simulator sickness and motion sickness may be in the proportion of individuals that develop certain symptoms (DiZio & Lackner, 1992). In general, motion sickness results in many more instances of nausea/vomiting than simulator sickness. Simulator sickness is more likely to cause fatigue, eye strain, and headaches. Of course there is much overlap and a great deal depends upon the specific platforms used in either case.

Simulator sickness can occur during the actual VE experience and also directly after the experience has ended. These post-effects of the virtual interface, commonly termed aftereffects, often include a number of the symptoms listed above as the participant re-

tunes his/her senses to the real world. This re-adaptation process has been shown to vary with simulator exposure time and simulator type but can require as long as 8 to 48 hours (Gower & Folwkes, 1989). The temporary reoccurrence of symptoms or simulator visual dynamics long after the exposure has also been documented, although it is rare (Baltzley, Kennedy, Berbaum, Lilienthal, & Gower, 1989; Benson, 1988). These events are often termed 'flashbacks'.

Simulator sickness often increases with exposure time (Cobb, Nichols, Ramsey, & Wilson, 1997; Garris-Reif & Franz, 1995; Howarth & Costello, 1996; Kennedy, Hettinger, & Lilienthal, 1990; Kennedy, Lanham, Drexler, & Lilienthal, 1995). It has also been shown to be reduced upon successive exposures to the same environment, especially when the time period between successive exposures is not longer than 5 to 7 days (Cobb, et al., 1996; Kennedy, Hettinger, & Lilienthal, 1990).

The existence of an working vestibular apparatus appears to be a necessary prerequisite for the occurrence of simulator or motion sickness. Labyrinthine defective individuals fail to exhibit symptoms even under the most extreme conditions and therefore are immune to simulator sickness (Ebenholtz, 1992; Reason & Brand, 1975). However, the mere existence of a working vestibular system is not sufficient for motion sickness to occur, it is simply a necessary requirement.

It is also interesting to note that visual stimulation in the absence of vestibular input can also result in simulator sickness. Hettinger and Riccio (1992) found that most individuals who do not experience vection do not experience simulator sickness. However, not all individuals who experience vection will get sick. Therefore, it appears that the ability to experience vection may also be a necessary but not sufficient requirement for simulator sickness.

The exact causes of simulator sickness are currently unknown. What makes it so difficult to decipher is that: 1) not everyone that experiences the same simulation will get sick, 2) those that do will often have a variety of different symptoms, 3) many symptoms are internal, non-observable, and subjective, 4) symptoms can arise over a period of minutes to a period of hours, and 5) some individuals may get sick one day and be fine the next (Griffin, 1990; McCauley & Sharkey, 1992). Obviously there are many variables that influence simulator sickness (Kolasinski, 1995) and the resulting variation makes it difficult to acknowledge potential contributing factors without first mentioning a number of caveats involving intervening variables. Therefore, the goal should be to concentrate on determining the major contributors to simulator sickness so that appropriate design guidelines can be developed. Not coincidentally, that was an objective of this dissertation.

2.4.3 Implications

Simulator sickness exists. Many studies have documented the severity of the problem both for simulators (Baltzley, et al., 1989; Kennedy, Lane, Berbaum, & Lilienthal, 1993; Pausch, et al., 1992) and for virtual environments (Regan & Price, 1993; Rushton, Mon-Williams, & Wann, 1994; Seymour, 1996). Susceptibility depends in large measure on the specific simulation equipment used, but commonly ranges from 10% to 60% of all participants using a particular system[30] (Kennedy, Lilienthal, Berbaum, Baltzley, & McCauley, 1989; Regan & Price, 1993).

Given that simulator sickness exists, what are its effects? For virtual environments, these effects include reduced time using a virtual interface, reduced enjoyment, creativity, learning, and productivity while in the VE, and increased negative

[30] A caveat: the above percentages denote individuals that experienced some form of simulator sickness such as eye strain, headaches, fatigue, dizziness, upset stomach, or pallor. Few were provoked to the point of emesis.

aftereffects of the virtual interface. These aftereffects can have serious safety implications for participants who try to resume normal activities too soon after the VE experience. Postural instabilities, dizziness, and altered sensorimotor control loops resulting from a virtual interface could severely effect one's ability to operate a car or other machinery, for instance. As already discussed, these aftereffects can be long lasting (i.e., from 6 to 24 hours, as cited by Pausch, et al., 1992). Therefore, there are important safety and legal issues involved in the occurrence of simulator sickness.

Also, it is important to recognize that simulator sickness can potentially effect every VR application including virtual training, medical applications, entertainment, and networked virtual worlds (Durlach & Mavor, 1995). All organizations that desire to use or sell immersive VR applications should be concerned with finding a solution to this problem. In particular, the government, military, industry, medical, and entertainment sponsors of VR have a keen interest in reducing simulator sickness along with its safety and legal ramifications.

2.4.4 Current Theories

2.4.4.1 Sensory Rearrangement Theory

The most accepted theory of motion sickness (which has been extended to simulator sickness) is the sensory rearrangement theory. This theory, first presented by Reason and Brand (1975) and later modified slightly by Reason (1978), states that: "all situations which provoke motion sickness are characterized by a condition of sensory rearrangement in which the motion signals transmitted by the eyes, the vestibular system and the non-vestibular proprioceptors are at variance either with one another or with what is expected based up on previous experience."

Sensory rearrangements may be decomposed into two *categories*: intermodality (between the visual and vestibular systems) or intramodality (between SCCs and otolith

organs within the vestibular apparatus) (Reason & Brand, 1975). Regardless of category, there are three *types* of conflict that could occur: 1) where both signals ('A' and 'B')[31] exist and provide contradictory information, 2) where signal 'A' exists but signal 'B' is absent, and 3) where signal 'B' exists and signal 'A' is absent. The theory claims that all situations that cause motion sickness can be fit into one of the two categories and one of the three conflict types, for a total of six *conditions*.[32]

Although the sensory rearrangement theory was developed for motion sickness, it also applies to simulator sickness, with a few caveats. First, simulator sickness may include aspects of the simulation that do not fall neatly into one of the six categories described above. For instance, a non-breathable head-mounted display (HMD) or tracking bodysuit may cause sickness symptoms, although there is no sensory conflict involved in these cases. Second, it is doubtful that the second category (SCC-otolith organ conflicts) play much of a role in simulator sickness (though its role in space sickness is assumed to be central). Therefore, when considering simulator sickness it may be wise to concentrate mainly on the visual-vestibular conflicts that arise.

Sensory rearrangement theory is the most accepted theory of why motion/simulator sickness occurs, however it is not without limitations. Although it offers satisfactory explanatory value, sensory rearrangement theory offers *little* to *no* prediction regarding which cue conflicts will result in sickness symptoms and which will not (Ebenholtz, Cohen, & Linder, 1994; Griffin, 1990; Stoffregen & Riccio, 1991). Since only a small subset of potential cue conflicts induce sickness, this lack of prediction is a major impediment to a fuller understanding of the issue. Furthermore, there are compelling arguments against the very existence of internal expectations generated from continued occurrences of a single pattern of sensory stimulation (Stoffregen & Riccio, 1991).

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[31] 'A' and 'B' are arbitrary designations for the two signals that are in conflict.

[32] See Griffin (1990) for examples of each condition.

Sensory rearrangement theory also does not answer the question of *why* sensory rearrangements should be nauseogenic (Ebenholtz, et al., 1994). Indeed, several researchers have asserted that sensory rearrangement theory, as it currently exists, is essentially untestable (Ebenholtz, et al., 1994; Griffin, 1990; Stoffregen & Riccio, 1991).

2.4.4.2 Evolutionary Theory

Treisman (1977) attempted to answer the question of why sensory rearrangements cause sickness from an evolutionary point-of-view. He claimed that motion sickness is simply a byproduct of an evolutionary adaptive mechanism that serves to protect the organism. Treisman argued that the ingestion of various toxins can effect movement coordination control. The eye-head movement system would be one of the first systems to be effected by certain toxins due to its high sensitivity (required to maintain visual stability under a variety of changing conditions) and the thin blood barrier that exists in the vestibular apparatus. Thus, alterations in eye-head movement control would likely be the first indication that toxins had been ingested, causing an increased potential of emetic response to rid the body of the toxin. Also, other accompanying motion sickness symptoms such as upset stomach, lethargy, headache, and dizziness would serve as adaptive stimuli to stop eating the current food and avoid it in the future. Therefore, certain mismatches between visual and vestibular inputs that occur in simulators could accidentally trigger this evolutionary toxin-identification mechanism, causing the same symptoms to occur. Money (1990) has strongly supported the evolutionary explanation for why motion sickness exists but Crampton (1990) cited data that are incongruent with the theory.

2.4.4.3 Subjective Vertical Theory

A variant of the sensory rearrangement theory has recently been proposed which attempts to better define which sensory rearrangements are nauseogenic and which are not. This theory, unofficially termed the 'subjective vertical' theory, states that "all situations which provoke motion sickness are characterized by a condition in which the sensed vertical (as determined on the basis of integrated information from the eyes, the vestibular system and non-vestibular proprioceptors) is at variance with the subjective vertical" (Bles, de Graf, Bos, & Groen, 1997). Thus, yaw rotations of the head or of the visual surround are not considered provocative stimuli, only those motions that impact the sensed or subjective vertical are. These researchers have presented data from centrifuge tests to support this theory (de Graf, Bles, & Groen, 1997; de Graf & Bos, 1997). This theory still needs to be reconciled with studies that have induced simulator sickness as a result of car simulators and OKN drums that presented only horizontal visual flow.

2.4.4.4 Reflex-Response Theory

Griffin (1990) presented his reflex-response theory to complement and extend sensory rearrangement theory. According to Griffin, a problem with sensory rearrangement theory is that the only dependent variables available are the symptoms of motion sickness, which do not provide clear visibility as to possible causal factors. He believed that inappropriate or conflicting reflexes may be associated with simulator sickness. Griffin argued that reflex responses to motion (including the VOR) should be included as additional dependent variables to 'provide greater visibility of causal factors and associated mechanisms'. Reflexes offer more visibility into the subconscious 'interpretations' of sensory responses to motion and may be incorporated into a theory or model of motion sickness (i.e., inappropriate reflexes in response to motion may have a causal role in the elicitation of symptoms).

2.4.4.5 Postural Stability Theory

A true alternative to the sensory rearrangement theory is the postural instability theory of Riccio and Stoffregen (1991). This theory states that sensory conflict cannot occur because its basic premise of intermodal stimulus pattern redundancy does not exist (Stoffregen & Riccio, 1991). If redundancy of stimulation does not occur, no standard template or expectancy within the central nervous system can be developed in which to compare with incoming stimulation patterns. The authors maintain that the postural behavior of the individual will determine if simulator sickness occurs. Specifically, they argue that environments which create an extended period of postural instability within the individual will produce sickness symptoms. Therefore, this theory focuses on the behavior of the individual instead of the patterns of sensory inputs. However, it is important to note that this theory does not describe why certain environments cause postural instability while others do not.

2.4.4.6 Extraocular Afference Hypothesis

Ebenholtz, et al. (1994) offered an alternative to sensory rearrangement theory as well. His hypothesis is that afferent signals that emanate from extraocular muscles during inappropriate combinations of oculomotor movements directly induce motion sickness. A direct connection between vestibular function and vagal responses is theorized to exist through extraocular muscle afference. Vestibular signals trigger the VOR, which links the vestibular system with extraocular afference. Extraocular traction during strabismus surgery can cause both emesis and the occurrence of the oculocardiac reflex (which in turn can cause bradycardia and possibly cardiac arrest). This is the link between oculomotor response (i.e., extraocular afference) and vagal responses. Therefore, he claimed that a direct link between vestibular function and sickness symptoms exists through inappropriate combinations of oculomotor movements which cause abnormal extraocular muscle traction. Ebenholtz also discussed the results of

Stern, Hu, Anderson, Leibowitz and Koch, (1990) which demonstrated a reduction of motion sickness with reduced nystagmus.

2.4.4.6 Status of Motion Sickness Theories

Given the above, it would appear that there does not currently exist a single viable theory of motion/simulator sickness. That would be an accurate assertion. However, sensory rearrangement theory does provide explanatory value that is currently unmatched by the other theories. Stimulus rearrangements can be identified under nearly all conditions that evoke sickness symptoms and this theory includes a significant role for adaptation to influence subsequent exposures to the same environment. So efforts progress toward correcting the deficiencies in the sensory rearrangement theory (i.e., subjective vertical, evolutionary, reflex-response).

There had been little written on the postural-stability theory for a few years, but recently proponents have presented data that corroborate its basic premise that postural stability appears to precede conscious perception of sickness (Stoffregen & Smart, 1997). This research is performing a valuable service beyond the examination of postural stability theory. It also serves as a reminder to those in the field that sensory rearrangement theory is incomplete. It also emphasizes the role of meaningful behavior and environmental interaction in the elicitation of symptoms.

Likewise, there has been no reported updates on the extraocular afference hypothesis. However, Ebenholtz himself described the critical experiment needed: anesthetize extraocular muscles by retrobulbar injection so as to block all afference signals and then expose the subject to a provocative environment (Ebenholtz, et al., 1994). If motion sickness occurs the hypothesis is refuted; if not than Ebenholtz's hypothesis gains support. It is not known whether this research was ever conducted.

In some ways the reflex-response theory could be considered as a bridge between the sensory rearrangement theory and either the extraocular afference theory and/or the postural control theory[33]. The reflex-response theory advocates looking at inappropriate reflex responses (including postural stability mechanisms as well as combinations of eye movements) as a way to better understand provocative sensory rearrangements. However, its description in Griffin (1990) is ambiguous and the theory has not been formally advanced since (Griffin, personal communication, 1997).

2.4.5 Influencing Factors

Many factors can effect the occurrence and level of severity of simulator sickness. Three powerful variables appear to be past experience with the simulated environment, exposure time, and individual factors. There is some discrepancy regarding exposure time, however, because recently some researchers have indicated that the majority of sickness occurs during the first few minutes of exposure. Interested readers can refer either to Pausch, et al. (1992) or Kolasinski (1995) for detailed descriptions of influential factors and their potential modulating effects. Kolasinski divided her list of factors into three categories: individual factors (e.g., age, gender, concentration level, past experience), simulator factors (e.g., binocular viewing, field-of-view, motion platform, position-tracking errors, time lags, scene content, update rate), and task factors (e.g. degree of control, duration, head movements, rate of acceleration), although there is overlap among many of the factors.

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[33] Comparison with the postural-stability theory ends at common investigation of postural control reflexes. The two theories are conceptually opposing; reflex-response is based upon an underlying theory of perception and action involving *unconscious inference* while postural stability theory is based on the ecological approach of *direct perception.*

2.4.6 Receptivity and Adaptability

In an effort to account for individual variation in sickness susceptibility, Reason and Brand (1975) hypothesized the existence of two relatively stable individual traits, receptivity and adaptability. These two traits are theorized to govern the strength and duration of the sensory rearrangement signal.

Receptivity is defined as the individual's sensitivity to the initial intensity of the stimulus rearrangement. Individuals can be classified according to the way in which the central nervous system as a whole codes stimulus intensity (Reason & Brand, 1975). Thus, some individuals may be more sensitive to rearrangements than others. Those who are more receptive to sensory mismatches are more likely to suffer sickness symptoms (Reason & Brand, 1975). Research exists which supports this hypothesis (Reason, 1968; Reason, 1972).

Adaptability refers to the rate at which an individual typically adjusts to conditions of sensory rearrangements (Reason & Brand, 1975). It is hypothesized to be a relatively stable trait within individuals, though it can vary widely between individuals. Since sensory rearrangements can induce sickness, the rate at which individuals can adapt to sensory rearrangements may predict sickness susceptibility. Studies have shown adaptability to be consistent within subjects and a significant predictor of motion sickness (Reason & Graybiel, 1972) and there is advocation for continued research in this area (Kennedy, Dunlap, & Fowlkes, 1990).

The relationship between receptivity and adaptability should also be considered. Reason and Graybiel (1972) presented results indicating that these two traits are independent. Adaptability was also shown to account for much more of the individual variation in susceptibility. However some combination of these traits may maximally predict sickness potential, as shown in Figure 13. Note that, in each graph, it is the percentage of area above the adaptation curve and below the stimulus rearrangement

magnitude that likely indicates relative sickness susceptibility (more area equals more chance of sickness onset). The width of this area at any point in time is considered to be the strength of the sensory rearrangement signal while the length of this area indicates signal duration.

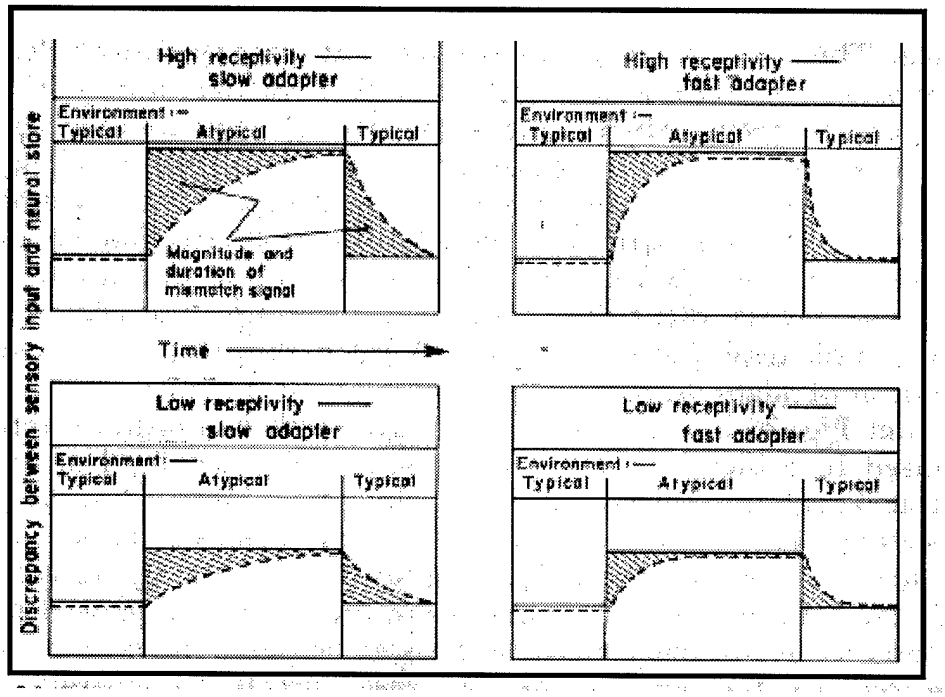

Figure 13: Receptivity and Adaptability Combinations[34]

[34] From Reason & Brand (1975, page 205).

2.4.7 CURRENT METRICS

The most widely used measures of simulator sickness are subjective ratings. These metrics address the polysymptomatic nature of simulator sickness, are easy to administer and score, and have strong face validity. Subjective ratings can take the form of verbal reports or questionnaires. Verbal reports are optimum for recording sickness levels during an exposure while multi-item questionnaires are best completed before and after exposure (due to timing and distraction concerns). One disadvantage of subjective measures is the potential for priming effects (i.e., asking 'are you sick?' may in itself increase sickness symptoms by alerting and focusing the individual on health issues) (Kirkner, 1949).

Verbal reports can take many forms. One verbal reporting system involves rating of overall discomfort on a 1 to 4 scale, with 1 signifying no effects, 2 indicating initial symptoms, 3 indicating mild nausea, and 4 signifying moderate to strong nausea (Golding, Phil, & Markey, 1996). There are several minor variations of this scheme found in the literature. An alternative verbal reporting system involves the reporting of various symptoms during exposure for later quantification using Graybiel's scale (Graybiel, Wood, Miller, & Cramer, 1968).

The most commonly used questionnaire is the simulator sickness questionnaire (SSQ) developed by Lane and Kennedy (1988)[35]. The SSQ is a revised version of the Pensacola Motion Sickness Questionnaire (MSQ) that was developed by NASA to quantify motion sickness symptoms (Kellogg, Kennedy, & Graybiel, 1965). The SSQ entails rating the severity of 16 simulator sickness symptoms on a 4 point scale. These symptoms are grouped into three partially independent subscales identified as Nausea, Oculomotor Discomfort, and Disorientation. These subscales provide potential diagnostic value in interpreting which aspects of a simulation may provoke specific

[35] The SSQ is described in a more concise manner in Kennedy, Lane, Berbaum, & Lilienthal, (1993).

sickness symptomology (assuming large numbers of subjects are tested to reduce variance). In addition, there is a total severity score which combines the results of these subscales into a singular rating of overall sickness level.

Postural stability measures have also been used to assess simulator sickness, specifically its aftereffects (Cobb, Nichols, Birchall, & Clifford, 1997; Hamilton, Kantor, & Magee, 1989; Kennedy, Berbaum, & Lilienthal, 1997; Kennedy, Fowlkes, & Lilienthal, 1993; Kennedy, Lanham, Drexler, & Lilienthal, 1995; Kennedy & Lilienthal, 1995). Simple posture tests include the sharpened Romberg, stand on preferred leg, stand on non-preferred leg, and walking a line, all of which are performed with either eyes open or closed.

More recently, devices originally developed for clinical postural assessment and treatment of vestibular-deficit patients have been incorporated to help quantify the postural instability effects of sickness-producing VEs (Prothero, Draper, Furness, Parker, & Wells, 1998). Neurocom and Chattecx are two such systems. These devices are more sensitive than simple stance metrics because they accurately measure and track subtle changes in a body's center-of-gravity over a period of time. These systems also allow for systematic manipulation of visual and inertial cues through a tilting visual scene and a motion platform. Although the simple postural measures have been described as lacking sensitivity and having ceiling effects (Hamilton, et al., 1989), these new postural assessment devices hold promise for increased sensitivity and diagnostic value. However, the downside of all postural stability measures is their relative obtrusiveness. They require that the individual stand and perform various postural stances over a period of time.

Various attempts have been made to correlate motion/simulator sickness with physiological measures such as heart rate, blood pressure, skin conductance, electrogastrogram (EGG) activity, pupil size, etc. (Casali, & Wierwille, 1986; DiZio &

Lackner, 1992; Harm, 1990; Hu, Grant, Stern, & Koch, 1991; Stout, Toscano, & Cowings, 1995). However, only limited success has been achieved outside of laboratory settings, even when combinations of measures were tried (Durlach & Mavor, 1995). Recently, however, additional support has been documented for the use of heart rate and salivary cortisol levels as indexes of simulator sickness (Cobb, et al., 1996; Ramsey, 1997).

CHAPTER 3: GENERAL HYPOTHESES

This chapter summarizes the two major hypotheses under investigation. Each hypothesis is described and defended using previous research results, supporting logic, and researcher assertions. Additional aspects and related issues are then considered. The intent of this chapter is to specifically describe the theoretical framework underlying the research performed. Additional details regarding virtual interfaces, virtual interface stimulus rearrangements, the VOR, VOR adaptation processes, and simulator sickness can be found in Chapter 2.

3.1 HYPOTHESIS 1: VIRTUAL INTERFACES MAY DRIVE VOR ADAPTATION

3.1.1 DESCRIPTION AND SUPPORT

The first hypothesis states that stimulus rearrangements found in virtual interfaces can induce VOR recalibrations during typical, unrestricted, goal-driven interactions with the VE. This hypothesis is straightforward, as illustrated in Figure 14. It is an abbreviation of more detailed and complete neurophysiological models of VOR adaptation presented by Ito and Robinson (See Section 2.2). The novel aspect of this hypothesis lies within the shaded area.

Virtual interfaces are believed to induce visual-vestibular sensory rearrangements regarding self-motion (DiZio & Lackner 1992; Furness, 1994). These sensory rearrangements may result in the occurrence of retinal image slip during head movements (Peli, 1995). The function of the VOR is to help maintain a stable image on the retina during head movements through the initiation of involuntary compensatory eye movements (Robinson, 1981). Detected retinal slip during head movements may be interpreted as an error signal, activating VOR recalibration processes (Robinson, 1981).

VOR gain and phase responses are then recalibrated to reduce existing retinal slip. Thus, sensory rearrangements arising during head movements are theorized to drive VOR adaptation processes. Given the importance of active head movements (Leigh, Dell'Osso, & Kosmorsky, 1993) and the potential for adaptation to occur after short exposure periods (Collewijn, et al., 1983), it is logical to hypothesize that VOR gain and/or phase adaptation can occur as a result of short-term, active head-coupled interaction with virtual interfaces (if the stimulus rearrangements are predictable and of sufficient magnitude).

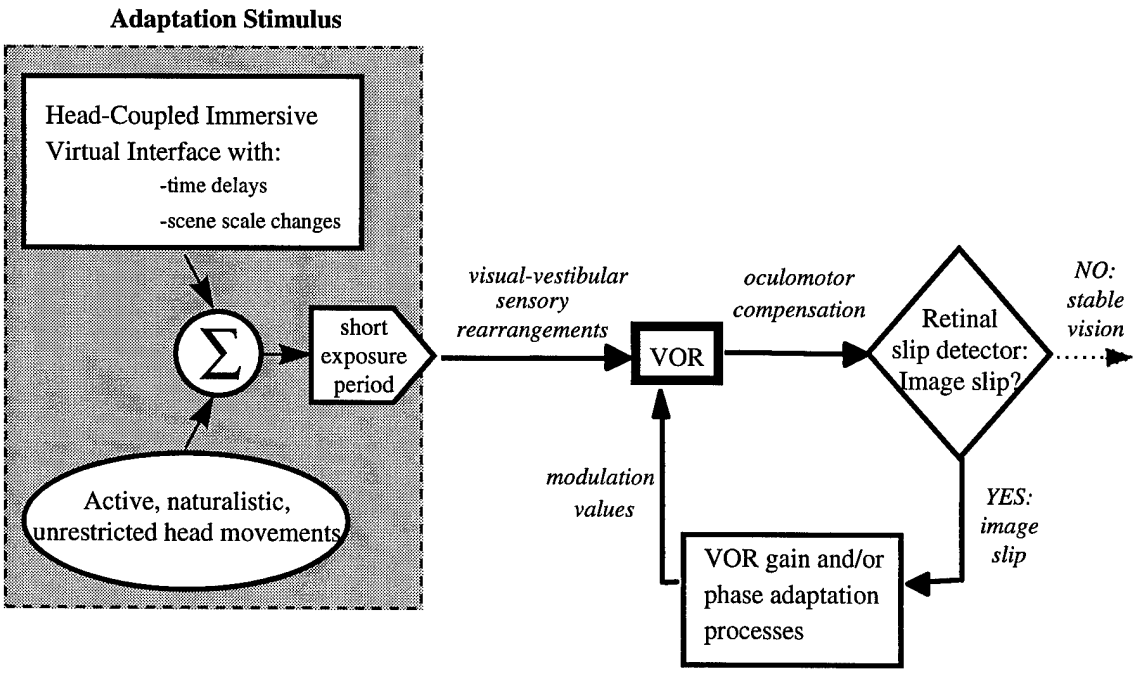

Figure 14: VR-VOR Adaptation Hypothesis

Kramer, et al. (In Press) demonstrated that a VR HMD can be used to drive VOR adaptations both in gain and phase. These researchers provided a prerecorded virtual OKN stimulus (horizontally oscillating vertical stripes) through a HMD that was fixed to a rotating chair. The subject used a chin rest to steady the head. Through various visual and inertial manipulations, these researchers were able to achieve VOR adaptations similar to those attained using a physical OKN drum. This research was among the first to provide evidence that virtual images can drive VOR adaptations.

The virtual interface in Kramer's et al. (In Press) work was designed exclusively to evoke VOR adaptation. The experiment was constrained by the following factors: 1) no active movements were performed by subjects[36], 2) system time delays were minimized, 3) a highly artificial visual scene was used, and 4) subjects had no meaningful task to perform. In contrast, this dissertation focused on the potential for VOR adaptation to occur in typical VEs with normally occurring stimulus rearrangements (e.g., time delays) while subjects made active, unrestricted head movements in the performance of a meaningful task. In other words, the question in this dissertation was whether virtual interfaces unintentionally induce VOR adaptation when these interfaces are used in an intended application.

VOR research has documented the occurrence of VOR adaptation through natural head movements (Gauthier & Robinson, 1975; Paige & Sargent, 1991). However, most of these studies were for moderately long durations and none used virtual interfaces. This research centered on short duration exposure (i.e., 30 minutes) to typically occurring stimulus rearrangements generated by virtual interfaces.

No other previous research investigating the relationship between virtual interfaces and VOR adaptation has been discovered. As a result, Dr. Viirre and I performed a

[36] The HMD was fixed to the chair, not the subject.

small pilot study to explore this issue with a commercially available head-coupled VR system (this study is explained in more detail in Section 4.2.4). VOR and VVOR measures were recorded before, during, and after a 12 minute exposure to an interactive head-coupled virtual interface. The subject's task was simply to play a virtual reality game which required repeated yaw head rotations. Eye and head position data were collected with a magnetic search coil system for maximum spatial and temporal resolution (Fuchs & Robinson, 1966; Robinson, 1963). Although only one subject was tested, the analysis indicated recalibration of the VOR to the virtual interface.

Research has shown that virtual interfaces can alter other oculomotor movements. Mon-Williams, Wann, and Rushton (1993) demonstrated that poor HMD optics, stereoscopic image quality, and poor interpupilary calibration resulted in a measurable change in visual function in 50% of subjects after only 10 minutes exposure to a virtual interface. These changes included decompensated heterophoria (i.e., deviation from perfect binocular positioning) and altered near point of convergence. A follow-on study (Rushton, et al., 1994) indicated that a higher quality HMD, better image quality, and bi-ocular viewing (instead of stereoscopic viewing) reduced but did not completely eliminate these visual changes.

Kotulak and Morse (1995) investigated oculomotor adaptations as a potential source of eyestrain in a monocular HMD used operationally by the US military. These researchers found that accommodation and vergence responses significantly differed between asymptomatic and susceptible pilots in three different circumstances that often occur in flight. Although the specific issues underlying their research differ from those of this dissertation, it is revealing that oculomotor adaptations occurred and were capable of differentiating symptomatic from asymptomatic subjects with regards to specific sickness symptoms (which supports the second hypothesis of this dissertation as well). As a final example, Kennedy and Murry (1993) found that increasing the

update rate on a computer display altered saccadic eye movements in a reading task. They subsequently hypothesized that this alteration may be a source of visual fatigue and eyestrain while reading computer generated text.

Many researchers have asserted that virtual interfaces likely induce changes in the VOR and other oculomotor movements. Ebenholtz (1992) stated that "VR can drive adaptive changes in the VOR". Peli (1995) argued that the VOR may need to adapt to virtual environments (especially head-coupled systems with long latencies or low update rates) and lamented the lack of research into this issue. He also questioned which environment the VOR would tune to in the case of augmented (see-through) displays. Viirre (1996) not only believed that VR can adapt the VOR unintentionally but that it can be made to do so intentionally to facilitate clinical treatment of patients with vestibular disorders.

3.1.2 Type of Head Movements under Consideration

As stated earlier, the focus of this research was on VOR adaptation resulting from volitional head movements by the subject to complete a meaningful task. Both head translations and head rotations can trigger VOR adaptations in the presence of significant stimulus rearrangements (Viirre, et al., 1986). This is because both canal and otolith signals are involved in the vestibular signal that triggers compensatory oculomotor responses (Robinson, 1981; Tiliket, Shelhamer, Tan, & Zee, 1993). However, this research focused solely on head rotations (canal input) due to the relatively minor contributions of the otoliths to the VOR in the specific test conditions used in these experiments (i.e., horizontal rotational oscillations with no close target fixations and no off-axis rotations).

3.1.3 Stimulus Rearrangements under Investigation

This research concentrated on those stimulus rearrangements typically found in virtual interfaces that result in visual-vestibular sensory rearrangements during head rotations. Three commonly occurring stimulus rearrangements that meet this requirement are system time delays, image scale distortions, and limited DFOV.

3.1.3.1 System Time Delays

Normal physiologic latency between the beginning of a head rotation and beginning of a VOR compensatory response is approximately 4 to 11 ms (Sharpe & Johnston, 1993). This results in little to no VOR phase offset between eye and head motion over the range of natural head movements.

Any system time delay in a virtual interface that extends beyond this threshold. constitutes a stimulus rearrangement[37]. This time delay introduces a phase lag between the head movement and the resulting optic flow (of the virtual image) in the opposite direction. This phase lag demand is hypothesized to stimulate VOR phase adaptation.

Figure 15 demonstrates this hypothesis. The four frames illustrate a top-down view of a subject wearing a HMD and viewing a virtual scene that is coupled to head movements. The subject is performing a VVOR[38]. Frame 1 shows a stationary subject focusing on a spot in a center of the virtual image. Frames 2 and 3 indicate a head movement to the right. Signals from a head tracking system, represented by squiggly lines, report the change in head position to the computer that updates the visual scene. In Frame 2, the signal still has not resulted in an update in the visual scene (due to the existence of a system time delay). In Frame 3 the visual scene has been updated in response to earlier head movements but it lags behind the current movement of the

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[37] System time delays associated with virtual interfaces are fully described in Section 2.3.3.1

[38] VVORs are described in Section 2.2.2.3

head. In Frames 2 and 3, the dashed lines indicate gaze direction assuming zero time delay and the solid lines indicate actual gaze. In Frame 4, the head has stopped moving while the motion of the visual scene continues to completion.

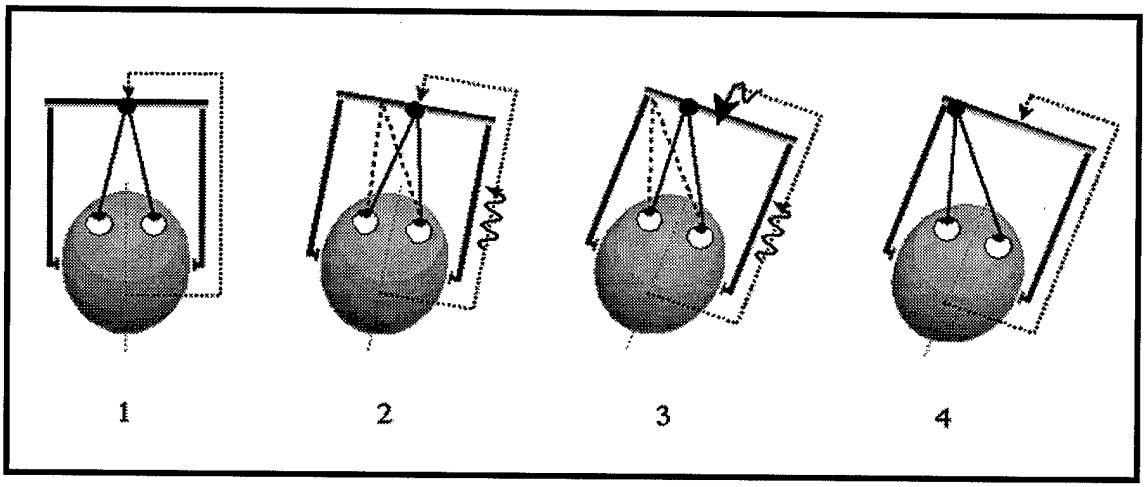

Figure 15: Effects of Time Delay on VVOR Response.

Fixed system time delays produce a variable phase-lag demand on the VOR adaptation system with specific phase lag demand increasing with increasing head movement frequency. Figure 16 demonstrates how a 100 ms time delay results in different phase lags depending upon head movement frequency.

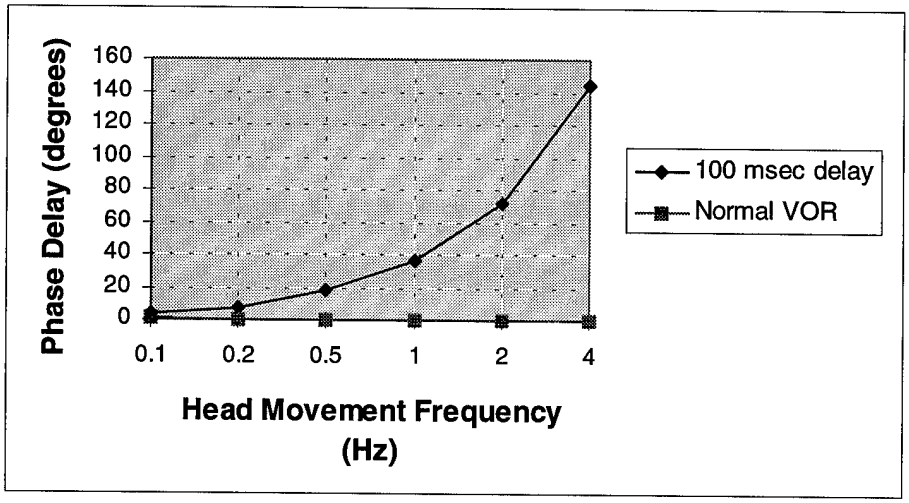

Figure 16: Fixed Time Delay Equals Variable Phase Lag Demand

Some studies indicate that VOR adaptation is fairly specific to the particular frequency(s) used during exposure to the stimulus rearrangement (Khater, Baker, & Peterson, 1990; Lisberger, Miles, & Optican, 1983; Peng, Baker, & Peterson, 1994; Zee, 1996). However, other studies have not come to this conclusion (Demer, et al., 1989). Research has not been uncovered which describes the effects of a specific time delay (i.e., a variable phase lag demand) on VOR gain and phase adaptive response across several frequencies.

If VOR phase recalibration occurs in response to a fixed time delay, it could follow one of two courses. It might result in a variable phase adaptation of the VOR across all natural head movement frequencies, as shown in Figure 17. This is unlikely if one assumes a frequency-domain model of the VOR adaptation process. The VOR would seemingly require an extended period of time at each frequency to reliably establish the nature of the stimulus rearrangement, recalibrate the phase for that frequency, then store this value for extremely rapid retrieval when the same frequency head movements are

made in the future at this time delay. Nonetheless, it is reasonable to speculate that various internal deficits to the vestibular system might result in a fixed time delay demand being placed upon the VOR adaptation system. For example, changes in transmission time would create a variable phase change demand across frequency. If the VOR was designed to recalibrate to these types of deficits, the phase response may appear similar to Figure 17.

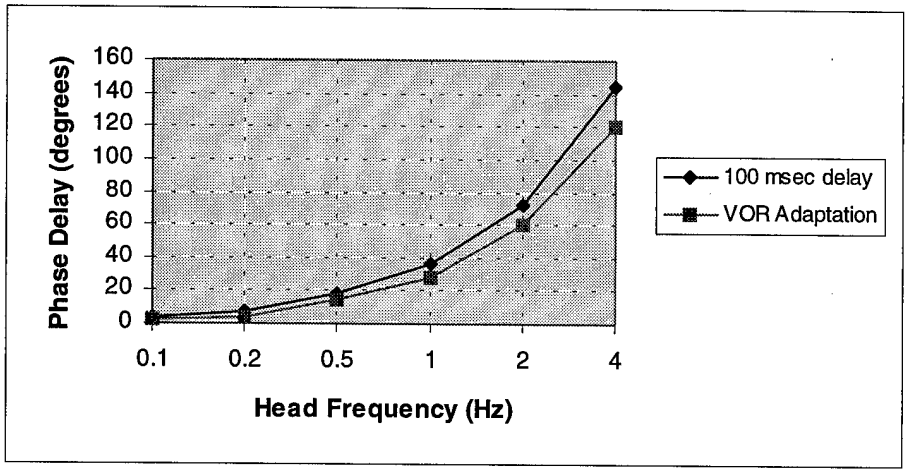

Figure 17: VOR Phase Adaptation Strategy 1

A second scenario is the following. Head movements may be naturally constrained to a small range of frequencies when a person is confronted with a stimulus rearrangement such as a system time delay. Therefore, VOR adaptation may occur for this reduced frequency set only, thus making the phase adaptation 'frequency-specific' (Figure 18). This approach, involving fewer frequencies and a more stable/predictable stimulus rearrangement, would also seemingly result in faster adaptation. Research has provided indications that head movements are indeed constrained while using certain

virtual interfaces (Pausch, Snoddy, Taylor, Watson, & Haseltine, 1996; So & Griffin, 1995).

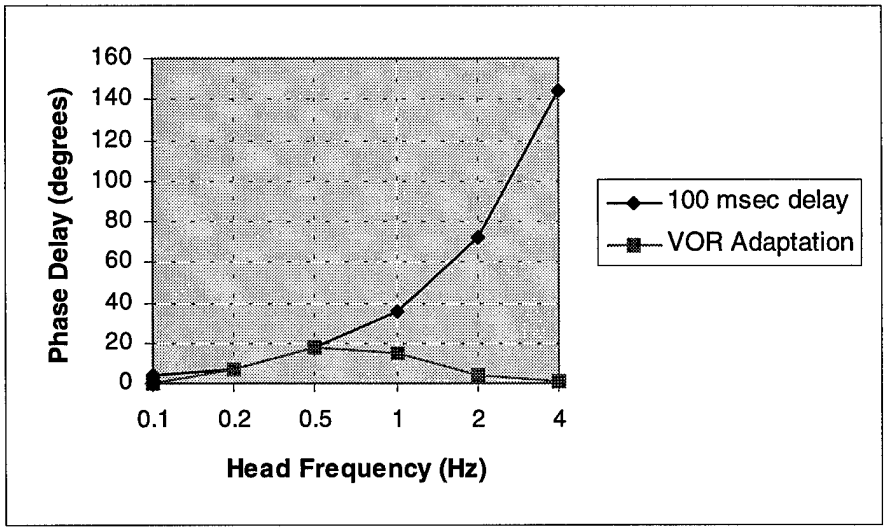

Figure 18: VOR Phase Adaptation Strategy 2

Few studies have investigated VOR phase adaptation and even fewer are directly relevant to this dissertation. Kramer, Shelhamer, and Zee (1995) demonstrated that a pure phase change demand might modify the VOR in gain as well as phase. They found that a phase lead demand resulted in gain decreases but they did not find an effect of phase lag demand on gain. Powell, et al. (1996) also investigated phase adaptation and found a complex relationship between gain and phase adaptation magnitudes, but this research used a direction adaptation protocol, cats for subjects, and passive rotations.

Time delays may have other effects on the user, besides the potential for inducing VOR phase adaptation. Research has shown that time delays decrease performance of

control tasks and can reduce satisfaction with the interface (Durlach & Mavor, 1995; So & Griffin, 1995).

3.1.3.2 Scale Changes

Virtual interfaces may often contain image scale distortions, whether intentional or accidental. Image scale factor distortions are easily induced by creating an inequality between GFOV and DFOV angles[39]. These scale changes result in virtual magnification or minification of the virtual scene with a corresponding increase or decrease in optic flow velocity that corresponds to the magnification level. This change in optic flow rate generates a gain adaptation demand. Similar rearrangements using magnifying or minifying spectacles as stimuli have resulted in VOR gain adaptation, if the rearrangement was of large enough in magnitude and predictable (Collewijn, et al., 1983; Gauthier & Robinson, 1975; Robinson, 1981). Therefore, image scale distortions resulting from GFOV/DFOV inequalities in virtual interfaces are hypothesized to drive VOR gain adaptations.

Figure 19 demonstrates the effect of different image scales on the eye motion required to maintain fixation on a distant, stationary target (i.e., for a VVOR task). In each of the image scale conditions (shown as 200%: magnification, 100%: normal, and a 50%: minification), the subject's head is shown moving to the right 10 degrees. The resulting optic flow varies with image scale factor. Therefore, the eye movement rotations required to maintain fixation on a visual target also varies with scale factor. Dashed lines indicate starting positions of head and eyes and the solid lines indicate final positions. These differences in eye movements for the same head movement illustrate the differing gain demands placed on the VOR system in the different image scale conditions.

[39] As discussed in Section 2.3.3.3.

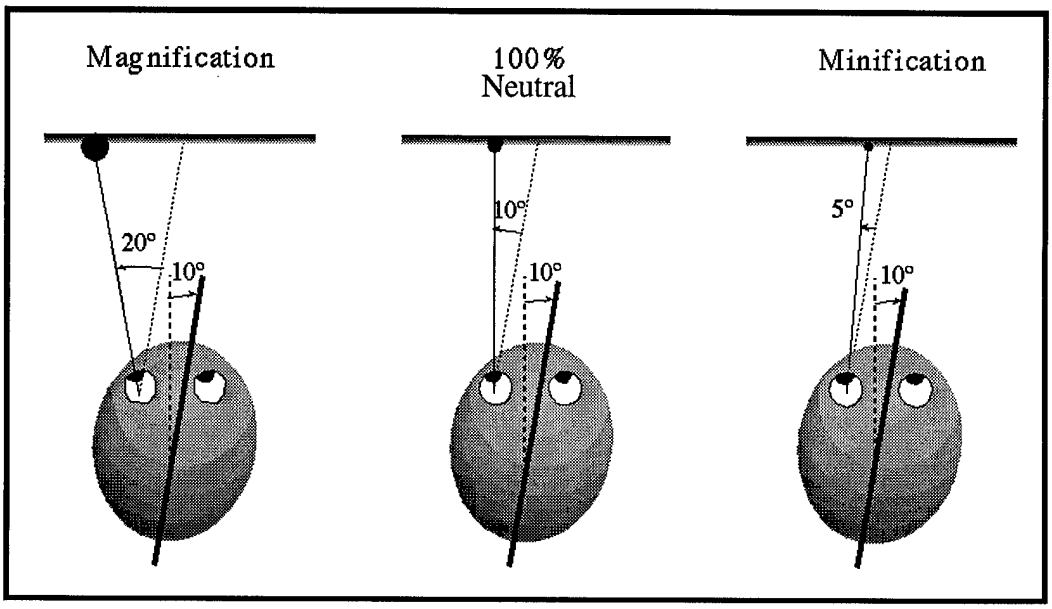

Figure 19: Effect of Image Scale on Compensatory Eye Movements during VVOR

Previous VOR adaptation research investigating short-duration exposures often restricted exposure interaction to passive head movements at a specific frequency of rotation. In contrast, this dissertation considered active head movements at unconstrained frequencies of motion. Active head movements are theorized to enhance VOR adaptation (due in part to the addition of efference copy signals) but unrestricted head movements across a range of frequencies in a limited exposure period may decrease overall adaptation if frequency specificity is correct. Therefore the results of these dissertation experiments are not easily predicted by the literature.

3.1.3.3 DFOV

DFOV may modulate the magnitude of any oculomotor adaptation that occurs in virtual interfaces. Although Lisberger, Miles, and Zee (1984) found that a full visual field is not required for adaptation to occur, their results suggest that full FOV motion did result in much greater adaptation taking place. This indicates that large FOV displays drive adaptation to a higher level than small FOVs. However, Shelhamer, Tiliket, Roberts, Kramer, and Zee (1994) found no difference in VOR gain adaptation whether the stimulus was full-field or a single LED, which presents a compelling argument against influence of DFOV on VOR adaptation. Thus more research is needed.

There are data suggesting that for maximum adaptation to occur, a small DFOV should not allow concurrent viewing of the normal, real-world environment. Research by Demer, et al. (1989) demonstrated that VOR gain adaptation was minimal when the periphery of the magnifying spectacles was not occluded[40]. However, as DFOV increases and the peripheral stimulation decreases, VOR adaptation has been shown to reliably occur (Cannon, et al., 1985).

3.1.4 The Relative Movements of Head, Eye, and Gaze

This section characterizes the relative motions of head, eye, virtual target, and gaze (i.e., eye in space) as a result of added time delays and changes in image scale within a VE. For simplification, these equations assume that the eye perfectly tracks a distant, 'fixed' virtual target[41] (V_{target}) within the VE while the head undergoes passive sinusoidal rotation at a single frequency (as in the case of VVOR testing). All defined motions (H, E, V_{target} and G) are velocities.

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[40] This suggests that augmented reality applications may be safe from undesired VOR adaptations.

[41] As will be shown, 'fixed' is a relative term within VEs.

The Head motion is given by:

$$H = A\cos(wt)$$

where A is the amplitude of head motion (1/2 peak-to-peak) and w is the oscillation frequency in radians. The VE is directly driven by head movements (i.e., it is head-coupled) such that it moves (with respect to the head) in equal and opposite direction to the head motion in order to maintain the apparent space-stability of virtual targets. If there is no image scale change and no system time delay, the motion of the virtual fixation target (V_{target}) with respect to the head can be described by:

$$V_{target} = -A\cos(wt)$$

However, changes in image scale would change the amplitude of V_{target} motion in response to head movements, such that the equation would become:

$$V_{target} = B\cos(wt)$$

where 'B' is the new amplitude of V_{target} that is equal to the image magnification factor (S_{image}) multiplied by '-A':

$$B = -A*S_{image}$$

In addition, an existing system time delay would act as a fixed phase lag component to the V_{target} in response to head movement[42], such that the equation for V_{target} becomes:

$$V_{target} = B\cos(wt + P)$$

where P is the added phase change in degrees[43].

---

[42] Given a single frequency of rotation w.

[43] If the phase change 'P' is negative a phase *lag* exists, if it is positive a phase *lead* exists. This equation also shows a change in image scale as 'B'. If there is only a system time delay involved, then B = -A.

So far, the motion of the head (H) and virtual target with respect to the head (V_{target}) have been described. Assuming that the eye perfectly tracks the V_{target}, the eye-in-head motion (E) necessarily equals V_{target} motion. Therefore, eye motion can also be described by:

$$E = B\cos(wt + P)$$

Thus, E should respond to changes in image scale by changing its gain response to amplitude B and it should respond to changes in system time delay through increases in phase lag to P.

For completeness, gaze motions under the different conditions will be described. The equation for gaze (G) is:

$$G = H + E$$

Assuming no changes in image scale and no system time delays, E perfectly opposes H and therefore G equals 0. This is an ideal but unreasonable goal of virtual interface design, given current technology limitations. In reality, both image scale factor distortions and system time delays often exist which cause sinusoidal movement of the V_{target} *with regards to space*. If only a system time delay exists, gaze becomes (after substitutions and reduction)

$$G = 2A\cos(P/2)\cos(wt - P/2)$$

As can be seen, a phase change in the V_{target} with regards to the head results in a net movement of G with a amplitude equal to 2Acos (P/2) and a defined phase relationship to head motion equal to (P/2). If both image scale distortions and time delays exist, the equation for gaze becomes more complex. After multiple substitutions and reductions, the general equation for G is:

$$G=SQRT(A^2 + 2ABcos(P) + B^2)cos[wt + arctan((BsinP)/(A + Bcos(P)))]$$

In this case, the amplitude of gaze equals $SQRT(A^2 + 2ABcos(P) + B^2)$ and the phase lag is the result of arctan((BsinP)/(A+BcosP)). Therefore, it is obvious that the V_{target} and G moves with regards to space, as a function of image scale changes and existing system time delays.

3.1.5 Stimulus Predictability Concerns

A general requirement of adaptation is that stimulus rearrangements must be predictable (Welch, 1986; Welch & Cohen, 1991). Therefore, an issue of importance in this dissertation is the relative stability of the virtual interface stimulus rearrangements explored. Scale changes generated by GFOV/DFOV inequalities are thought to be nearly as stable as the wearing of magnification or minification spectacles, but system time delays can be more variable in nature.

As described earlier, time delays are generally a function of tracking hardware and computing power. These are relatively stable factors of a virtual interface[44]. However, system time delay also is effected by complexity of the VE, computational load from parallel processes, and distance from transmitter for emitting tracking devices (if an emitting type of head tracker is a part of the interface).

It is preferable to first obtain the influences of stable time delays before attempting to understand the influences of additional variability. Therefore this research was specifically designed to generate relatively fixed time delay stimuli.

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[44] This is not necessarily true for all virtual interfaces (e.g., networked VR environments mediated through a UNIX operating system).

3.1.6 SUMMARY

In summary, there is support from a pilot study, existing literature, and researcher assertions for the hypothesis that virtual interfaces can drive VOR recalibration. Time delays and image scale distortions occurring in virtual interfaces may generate sensory rearrangements which drive VOR gain and/or phase adaptation.

3.2 HYPOTHESIS 2: ADAPTABILITY GOVERNS SICKNESS SUSCEPTIBILITY

3.2.1 LOGIC AND SUPPORT

This hypothesis[45] assumes that Hypothesis 1 (that virtual interfaces can induce VOR adaptation) is confirmed. This hypothesis also assumes that the sensory rearrangement theory of simulator sickness is valid. Figure 20 provides an overview of the hypothesized relationship between VOR adaptation and simulator sickness.

[45] This hypothesis is a revised version of earlier presented work (Draper, Viirre, Furness, & Parker, 1997).

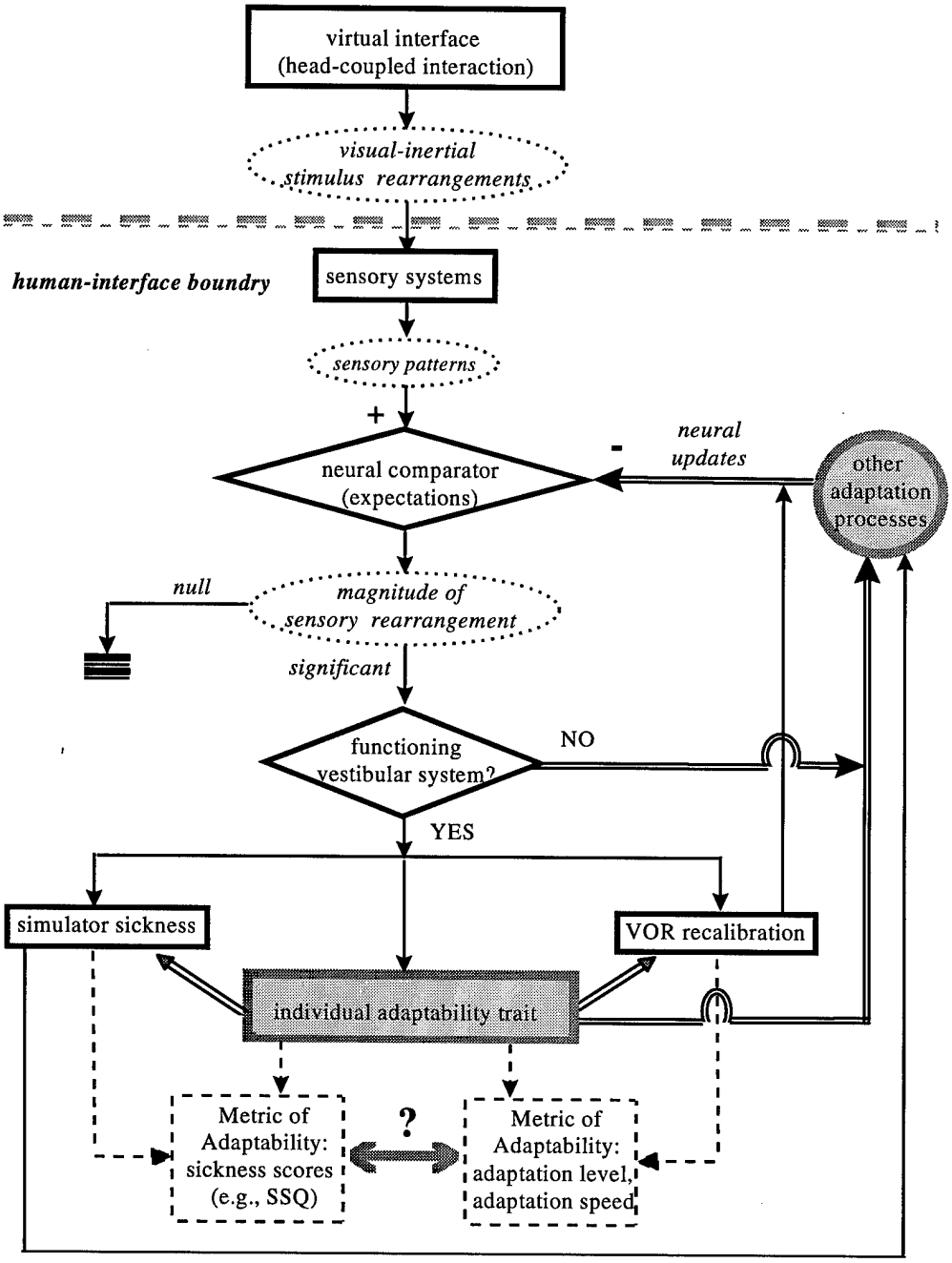

Figure 20: Adaptability Hypothesis

Figure 20 is described as follows. Head-coupled virtual interfaces containing system time delays and/or image scale deviations from 1.0X magnification will present visual-vestibular stimulus rearrangements. The user's sensory systems transduce these stimuli and generate internal sensory patterns. These sensory patterns are compared (via a 'neural comparator') to expected sensory patterns (held in a 'neural store') obtained from recent experience. The result of this comparison is the generation of a sensory rearrangement signal. If the magnitude of this signal is very small, the information flow terminates and no adaptation takes place. However, if the magnitude is significant and if there is a functioning vestibular apparatus, this sensory rearrangement signal will trigger VOR recalibration activities, a build-up of effect on simulator sickness, and other adaptation processes[46]. These adaptation processes then update the 'neural store' which reduces the magnitude of the sensory rearrangement. This, in turn, reduces VOR recalibration requirements and the build-up of effect on simulator sickness.

Generalized adaptation processes are represented by gray boxes and double lines in Figure 20. The hypothesized individual 'adaptability' trait serves to govern the overall rate of adaptation. Reason and Brand (1975) theorized that this stable adaptability trait reflects the rate at which a person typically adjusts to conditions of sensory rearrangement (see Section 2.4.6). In sensory rearrangement theory terminology, adaptability is the time it takes for the 'neural store' of expected combinations of motion signals to be updated once a sensory rearrangement occurs. A person with high adaptability would rapidly adjust to sensory rearrangements and would therefore avoid motion (or simulator) sickness. A person who had low adaptability would be prone to more sickness symptoms due to the increased duration of the mismatch before the neural store was updated.

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[46] If the vestibular system is not functional, no simulator sickness or VOR recalibration will occur but other adaptation processes will still be stimulated, as is shown in the diagram.

There is some experimental evidence for the existence of an individual adaptability trait. Studies have shown that those most prone to motion sickness tend to adapt more slowly to new combinations of motion (Reason & Graybiel, 1972). However, empirical successes have been modest because of the complexities of predicting a syndrome such as motion sickness. Still, some investigators consider differences in adaptability to be the single most important determinant of inter-subject difference in susceptibility to motion sickness (Griffin, 1990; Guedry, 1991; Kennedy, Dunlap & Fowlkes, 1990; Reason & Graybiel, 1972).

If an fixed adaptability trait exists within individuals that indicates relative adaptive ability to sensory rearrangements, than an objective measure of this adaptability trait would likely predict individual susceptibility to simulator sickness. VOR adaptation response is suggested as an objective measure of this adaptive ability to altered visual-vestibular motion cues. The remainder of this section defends the appropriateness of this metric by first describing the commonalties between VOR adaptation and simulator sickness and then discussing the rationale behind specifically investigating VOR adaptation time-course as the adaptability measure. Additional support for the hypothesized relationship between the VOR and simulator sickness can be found in Section 2.2.4 and Section 2.4.4.6.

Virtual interfaces often generate visual-vestibular sensory rearrangements during head movements, which are theorized as capable of driving VOR adaptation (Hypothesis 1). VOR adaptation is often marked by the occurrence of sickness symptomology that is similar to simulator sickness (Demer, et al., 1987; Demer, et al., 1989; Gauthier & Robinson, 1975; Gonshor & Melvill Jones, 1976b; Istl-Lenz, et al., 1985). According to the sensory rearrangement theory, visual-vestibular sensory rearrangements regarding motion and orientation are also strongly implicated as inducing simulator sickness (Reason & Brand, 1975). Given that the same catalyst is

involved for both processes, it is reasonable to examine if a correlation exists between simulator sickness and VOR adaptation.

Additionally, a functioning vestibular apparatus is a fundamental requirement for both VOR adaptation and simulator sickness (Reason & Brand, 1975). The role of the vestibular organ in the VOR and VOR adaptation is obvious, but it also has an essential role in simulator sickness (and motion sickness) as well. No bilateral labyrinthine-defective subject (either through birth, disease, or surgical interference) has ever been made to experience any form of motion or simulator sickness. VOR adaptation is also impossible in bilateral labyrinthine-defectives. This further implies a relationship between simulator sickness and the VOR.

If VOR adaptation and simulator sickness are correlated, the association is not a perfect correspondence, nor is it a cause-and-effect relationship. VOR adaptation can occur without the onset of simulator sickness symptoms and simulator sickness can occur without concurrent VOR adaptation. Therefore, one must look deeper to uncover an objective measure of adaptability.

Prior to VOR adaptation, maximum retinal slip of the visual scene exists. Retinal slip during head movements can result in oscillopsia and it also generates a signal that the visual response is inappropriate for a given vestibular stimulus. Thus at the onset of VOR adaptation, the visual-vestibular sensory rearrangement is likely at its greatest magnitude (Guedry, 1991). This suggests that fast VOR adaptation would reduce the exposure time to the maximal stimulus mismatch while slow VOR adaptation would prolong it. As simulator sickness is often characterized by a build-up of response over time, longer adaptation periods allow more build-up of response to occur to the initial, maximally-provocative sensory rearrangement.

Both time-course of adaptation and level of adaptation achieved after a fixed exposure period may provide meaningful correlation with sickness likelihood. The relationship is hypothesized to be:

$$[\text{sickness level}] \Leftrightarrow f(\delta(\text{VOR})/\delta t\ ,\ \max(\text{VOR}))$$

where the likelihood of experiencing sickness symptoms is related to the speed of VOR recalibration to a sensory rearrangement along with the maximum level of adaptation achieved.

In summary, there are commonalties that exist between VOR adaptation processes and simulator sickness. There is also reason to believe the VOR adaptation time-course may be predictive of simulator sickness, with fast adapters being less prone to sickness than slow adapters. Therefore, a reasonably objective metric of adaptability to investigate is speed of VOR adaptation. The specific metric used for these experiments is level of adaptation achieved after 30 minutes.

The last few paragraphs may inadvertently suggest to some that VOR recalibration processes are the only adaptation processes at work during sensory rearrangements. This is obviously not the case, as was addressed in Figure 20. Many physiological and perceptual adaptations are involved. For instance, if retinal slip still exists after VOR adaptation processes asymptote (i.e., if the magnitude of the rearrangement is too large to result in complete VOR adaptation compensation), perceptual adaptations can further compensate to reduce or eliminate the perceptual effects of the remaining retinal slip. Therefore, a physiological adaptation process such as VOR recalibration cannot fully determine a complex psycho-physical syndrome like simulator sickness. However, if the adaptability hypothesis is correct, speed of VOR adaptation may provide partial but meaningful predictive value in determining simulator sickness susceptibility.

3.2.2 Prediction Issues

The ability to predict individual susceptibility to simulator sickness has real benefits. These individuals can be identified prior to exposure so that they can avoid provocative environments. Also, countermeasures can potentially be developed for these individuals. Lastly, prediction provides insight into the meaningful covariates of the syndrome.

However, identifying simulator sickness predictors has been a very difficult task (Kennedy, Dunlap, & Fowlkes 1990). Pausch (personal communication, 1996) has compared this process to 'pinning jello to a wall'. Below are some issues to consider when attempting to predict simulator sickness.

Simulator sickness is affected by factors of the simulator (in this case, virtual interface), task, and individual (Kolasinski, 1995). In a technical report, Kolasinski (1995) identified 40 factors thought to influence sickness incidence. Given so many potentially influencing factors, it is highly doubtful that one or two variables can be found that fully predict sickness onset. In response to this challenge, this research assessed an individual's adaptability while holding the task constant and systematically controlling simulator factors.

Other proposed metrics of adaptability have met with mixed success in predicting sickness onset (Kennedy, Dunlap, & Fowlkes, 1990). This could be due to the particular adaptation metric chosen for comparison. Some metrics, though valid indicators of adaptation, may not be directly relevant to the process being studied. In addition, as suggested by Kennedy, et al. (1990), the mixed success of adaptability metrics in predicting sickness might also be a related to flaws in statistical analyses.

Another issue is the degree to which one can generalize adaptability from results obtained using one specific sensory rearrangement. Reason and Graybiel (1972) offered

support for generalized adaptability through the finding that individual susceptibility to motion sickness in flight could be predicted by brief but severe exposures to cross-coupled angular acceleration.

Finally, the Cauchy-Schwarz Inequality applies. This mathematical relation states that the upper limit for predictive validity is the geometric mean of the criterion reliability and the predictor reliability. In equation form:

$$r_{xy} \leq (r_{xx} * r_{yy})^{½}$$

The implication of this is that the both the predictor and the criterion must be able to predict itself (measurement reliability) adequately for the predictor to successfully predict the criterion. VOR adaptation to a rearrangement is assumed to be relatively stable within subjects but simulator sickness is known to be more variable.

CHAPTER 4: INTRODUCTION TO EXPERIMENTATION

4.1 OVERVIEW OF EXPERIMENTATION

This chapter provides an empirical preface to the dissertation research discussed in Chapters 5 through 8. Early research is summarized, marking a progression from peripheral concepts to central focus. This chapter also presents is a detailed description of the research facility that was constructed to accomplish these and other similar research agendas. Lastly, an overview of the main dissertation research is provided.

4.2 EARLY RESEARCH

The following studies illustrate the empirical journey towards the main research questions. Initial efforts primarily involved assessments of postural adaptation while later work focused on oculomotor adaptation. Some research on simulator sickness was also conducted. These experiments provided a foundation upon which to develop a better understanding of the issues surrounding adaptation, simulator sickness and oculomotor research.

4.2.1 THE SIT-STAND STUDY

This study arose from an incidental observation that I had regarding postural stability. I noticed that a fellow graduate student and I experienced more ataxia while performing the Sharpened Romberg stance (eyes closed) after prolonged sitting than after standing[47]. A hypothesis was formed theorizing that postural control mechanisms

[47] This observation actually occurred while we were preparing to conduct a study involving vection.

adapt to extended periods of sitting, which results in temporary low-magnitude negative aftereffects when postural position is suddenly and radically changed.

The eyes-closed Sharpened Romberg is a very sensitive stance for identifying changes in postural stability (Hamilton, et al., 1989) and the Chattecx Balance Platform is well equipped to detect small postural changes (see Section 4.3.1 for a description of the platform). Therefore, to detect these hypothesized low-magnitude postural effects, the Sharpened Romberg stance was employed while the subject stood on the balance platform.

A pilot study of four subjects (counterbalanced, within-subjects design) provided an indication that the hypothesis may be supported. Subjects either sat or stood for 10 minutes prior to being tested on the Chattecx Platform (in the eyes-closed Sharpened Romberg position). The platform test recorded postural changes over a 10 second period. A paired t-test (assuming unequal variances) indicated a strong trend towards significance ($p < 0.06$) for position prior to balance test, with more ataxic effects occurring after 10 minutes of sitting vs. 10 minutes of standing (Figure 21).

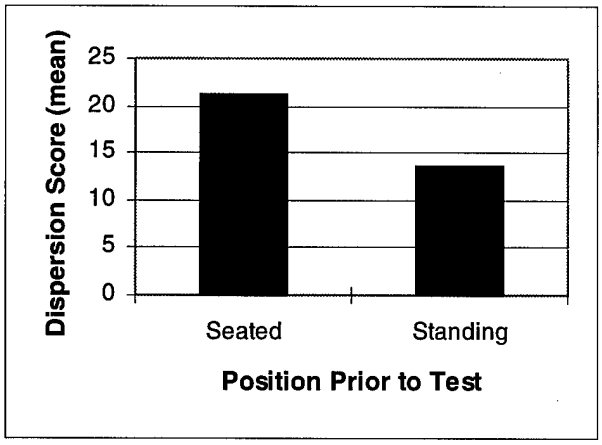

Figure 21: Pilot Results (Sit-Stand Exp)

A more rigorously controlled evaluation with 10 subjects failed to replicate the results of the pilot study, due in part to excessive variance in the data (the eyes-closed Sharpened Romberg stance is potentially a very sensitive metric for ataxia but it is also extremely variable). This experiment, however, was my first exposure to physiological adaptation research and as such it provided a good introduction to the complexities of the area.

4.2.2 Ataxia Experiment #1

A set of ataxia experiments was conducted with a fellow graduate student to determine the effects of HMD display transparency (completely occluded versus partially see-through) on ataxia, vection, and simulator sickness. A circular vection stimulus (in yaw) was presented to the subject though a Virtual i/O HMD (this HMD is described in Section 4.3.1). In one HMD condition, the subject could partially see the laboratory room superimposed with the virtual rotating scene when the subject viewed the display (the see-through display condition). In the occluded HMD condition, only

the virtual vection scene could be seen. In both cases, the edges of the display were occluded to prevent peripheral viewing. It was hypothesized that the occluded condition would result in more ill-effects (in the form of ataxia and simulator sickness) due to a switching of internal reference frames (Prothero, 1998). A paper fully describing this research has been submitted for review in anticipation of publication (Prothero, Draper, Furness, Parker, & Wells, submitted). Below is a summary of the experimental details most relevant to this dissertation.

The first ataxia experiment (Ataxia #1) was my first experiment investigating simulator sickness. A total of 15 subjects were tested using a counterbalanced, within-subject, one-factor design. The factor manipulated was HMD transparency (see-through, occluded). The yaw vection stimulus rotated at 60 degree/sec so as to be maximally provocative for inducing both vection and sickness (Griffin, 1991)[48]. Kennedy's Simulator Sickness Questionnaire (SSQ) was administered to assess sickness symptoms both pre- and post-exposure to each condition. Ataxia was measured during the 3 minute stimulus exposure while the subjects tried to maintain balance in the Sharpened-Romberg (eyes-open) position. Stance breaks, defined as the number of times subject broke the Sharpened Romberg stance, were used as the measure of ataxia. Stance breaks were recorded during the first and third minute of exposure, then totaled to provide an overall measure of ataxia for that condition.

One subject's data were not analyzed due to his excessive difficulty in maintaining balance during the test; the results of the remaining 14 subjects are discussed here. Data were analyzed using a 2-tailed paired t-test (for stance measures) and a non-parametric, 2-tailed paired Wilcoxon (for SSQ data). The occluded display resulted in significantly

[48] I created this vection stimulus by continually walking in a circle on campus while pushing the handle of a video camera as my partner incessantly called the proper cadence. Sadly, we had to repeat this several times before we got a proper image, which gave me some long lasting self-induced ataxic effects.

more sickness symptoms (Figure 22) and more ataxia (Figure 23) than the see-through display. There was also an increase in stance breaks over the 3 minute exposure when pooled across display condition (Figure 24), which suggests that ataxia increased with exposure time.

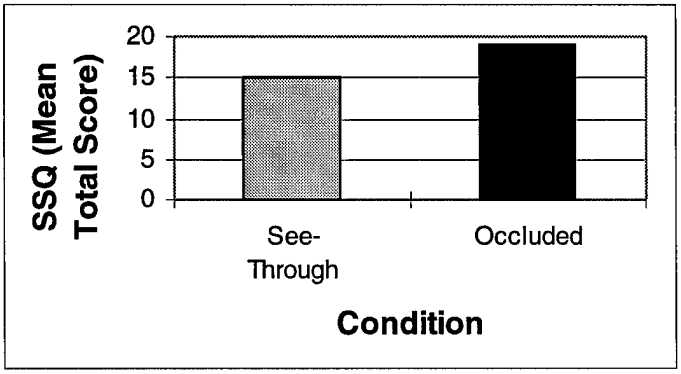

Figure 22: Simulator Sickness Results (Ataxia #1 Exp)

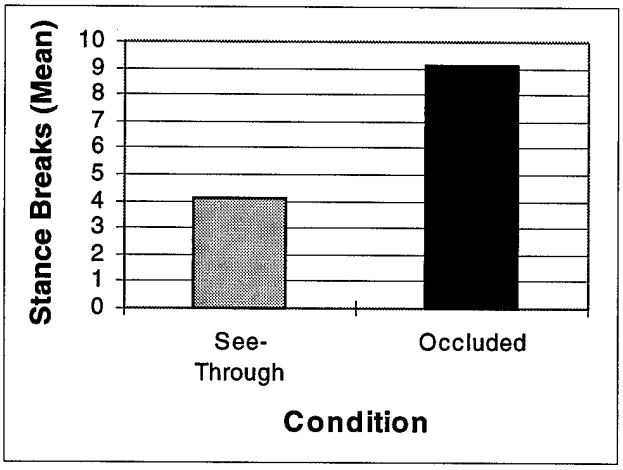

Figure 23: Mean Stance Breaks (Ataxia #1 Exp)

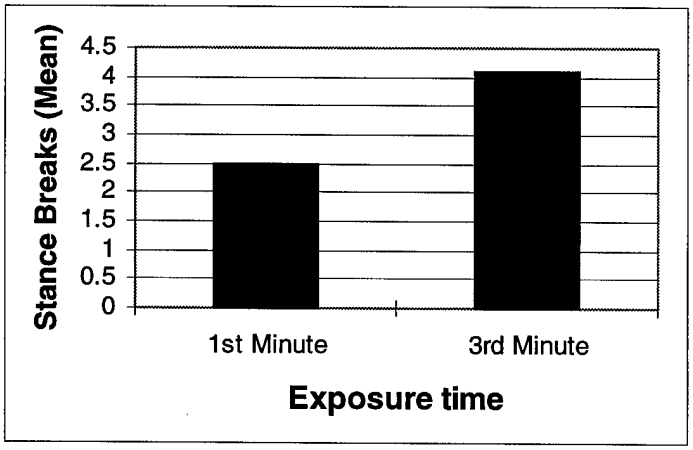

Figure 24: Ataxia by Exposure Time (Ataxia #1 Exp)

This experiment provided an empirical introduction to the syndrome of simulator sickness. It also demonstrated that specific characteristics of a virtual interface could modulate sickness incidence as well as essential physiological control processes. Furthermore, the decrease in postural control over exposure time confirmed the importance of the visual motion cues in the maintenance of postural stability, even when those cues are at odds with the inertial cues. Visual-inertial stimulus rearrangements have definite physiological effects. The results of this experiment led to a more detailed follow-on effort, aptly named Ataxia #2.

4.2.3 Ataxia Experiment #2

Ataxia #2 addressed issues raised in Ataxia #1 and it added a pre- and post-exposure balance test (using the Chattecx system described in Section 4.3.1) to study ataxic aftereffects. Other changes included the addition of a visual search task to maintain subject attention within the virtual vection scene, a removal of head tilts performed by the subject during exposure[49], and increasing the exposure time from 3 to 4.5 minutes. A total of 21 subjects were tested in a counterbalanced, within-subject, one-factor design.

The data presented below were analyzed using the same statistical tests as in Ataxia #1. The results again showed a reduction in ataxia during exposure for the see-through display ($p < 0.04$) (Figure 25) and there was a slight trend toward less sickness reports for that same condition. The overall strength of the signal declined, however, most probably due to the removal of head tilts in this study. This probably also accounted for the failure of the sickness data to reach statistical significance. An interesting finding regarding aftereffects also occurred. Post-exposure ataxia did not differ significantly between display conditions (likely because of the low overall response magnitudes

[49] These head tilts were accomplished twice per treatment in Ataxia #1 in an attempt to increase stimulus effect, but were found to be too variable in execution by subjects.

generated). When pooled, however, the post-exposure values were significantly worse than pre-exposure values ($p < 0.04$) (Figure 26). Thus, a 4.5 minute exposure to a virtual interface resulted in negative aftereffects of a fundamental physiological control system. This further solidified the assertion that virtual interfaces can alter essential physiologic processes, even after short duration exposures. However, it should be noted that this virtual interface was highly geared towards vection at the expense of naturalistic interaction (though some VR entertainment applications may strive to achieve this same effect).

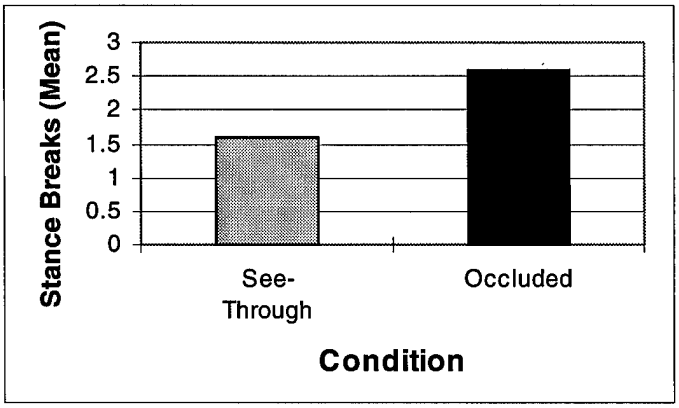

Figure 25: Mean Stance Breaks During (Ataxia #2 Exp)

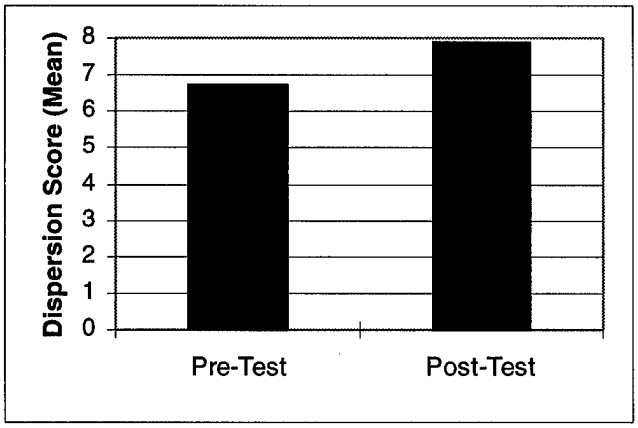

Figure 26: Ataxic Aftereffects (Ataxia #2 Exp)

4.2.4 Pilot Study on Oculomotor Adaptation

Given that virtual interfaces could effect postural control both during and after exposure, a pilot study was conducted to determine if these interfaces could also alter oculomotor response. The hypothesis was that short-term exposure to a immersive virtual interface could result in altered oculomotor responses associated with the VOR and eye-head gaze shifts.

This experiment was conducted at the Jules Stein Eye Institute on the UCLA campus. The reason for the remote test site was to utilize a magnetic search coil eye tracking system. The search coil technique is a high resolution (spatially and temporally) system for measuring eye and head movements.

The virtual interface consisted of a Virtual i/O HMD, its associated head position tracking system, and VE software. The HMD was full color, bi-ocular, and collimated

to approximately 3.35 m. with a DFOV of approximately 19 degrees vertical by 25 degrees horizontal (see Section 4.3.1 for a more detailed description of the HMD). The head tracker, sold by Virtual i/O along with the HMD, utilized a compass (for yaw movements) and liquid-based tilt sensors (for pitch and roll movements) in order to track head rotations at a 250 Hz update rate (head translations were not tracked). The VE was a virtual reality game provided with the HMD called 'Ascent'. This game, which ran on a Pentium PC, required many yaw head rotations by the subject in order to successfully advance.

Due to equipment malfunction, data were collected from only one subject. The subject's VOR was measured before and after a 12-minute exposure to the immersive virtual interface. Results indicate that both gain and phase of the VOR changed from baseline levels (Figure 27). This suggests that oculomotor adaptations may occur from using an immersive virtual interface in a naturalistic fashion. Interpretation of these results is restricted given that only one subject was run. However, this study provided a basis for more rigorously exploring oculomotor adaptation to virtual interfaces.

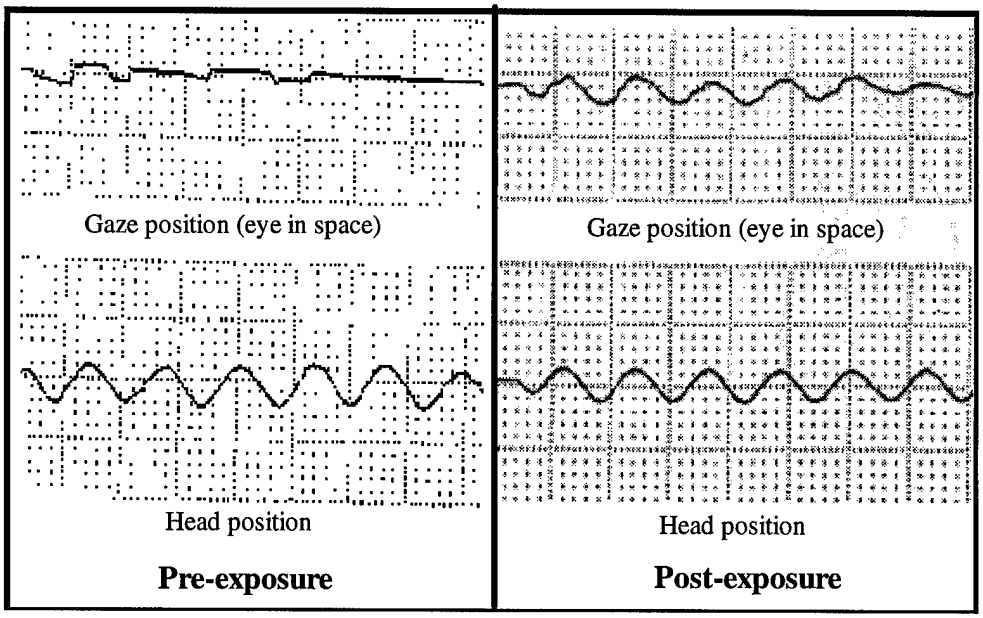

Figure 27: VOR Response: Pre- and Post-Exposure
(UCLA Exp)

4.2.5 Missteps, Roadblocks, and Should-Have-Workeds

Lest it be presumed based upon the orderly nature of this presentation that early research proceeded smoothly and quickly from step to step, it should be made clear that the 'steps' appear orderly because the 'trips' were omitted from detailed discussion. For instance, the selection of an appropriate eye tracking system was an adventure to say the least. From early troubles in getting an antiquated BioMuse to track eye movements, to trouble with a malfunctioning EOG eye tracking system[50] (Figure 28), to continually jerking the head of a dissertation committee member wearing an inexpensive VOG system as I attempted to collect pilot VOR data without use of sinusoidal oscillations

[50] I became fairly nauseous spinning around in an OKN drum while three M.D.s haggled over the output.

(the technique[51], which relied on corrective saccades, is shown in Figure 29, though all we ever obtained were sore necks), to the mistake of trying an ISCAN beta-version VOG camera system (which we returned after two months of strange behavior), the road was indeed a circuitous one. Also not included are the numerous failed simulation/optimization attempts, pilot study miscues, homemade VOR analysis programs, manual de-saccading techniques that gave horrendous results, a mid-stream HMD switch, multiple lab reconfigurations, etc. But with each failure and misstep came increased understanding and experience which resulted in an improved dissertation program overall.

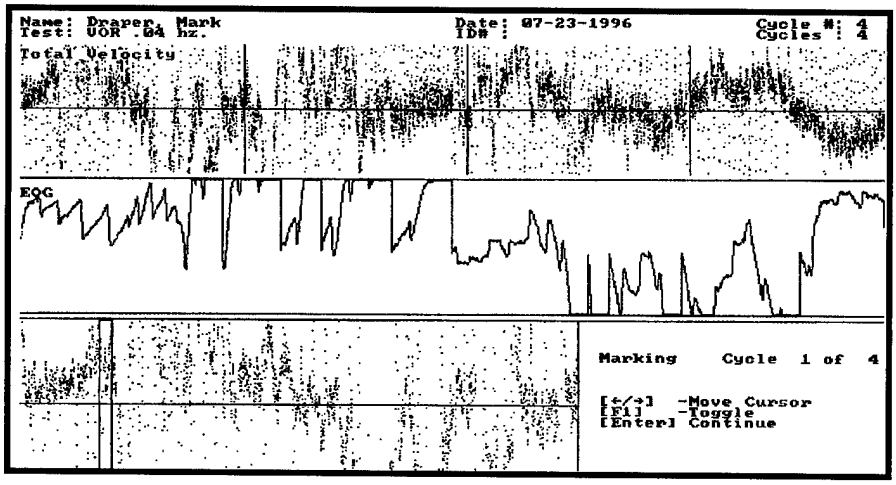

Figure 28: An Unsuccessful Attempt at EOG Eye Tracking

[51] This technique was more successfully employed by Jones, Berthoz & Segal, (1984) and Shelhamer, et al. (1992).

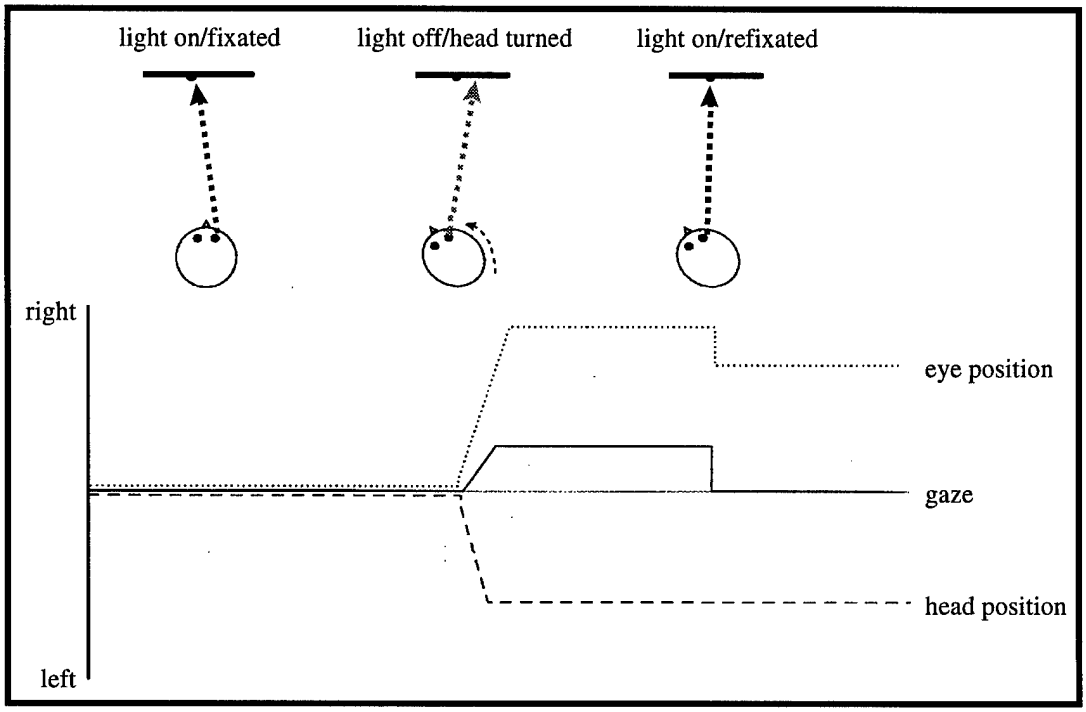

Figure 29: VOR Analysis - Corrective Saccade Technique[52]

4.2.6 Summary of Preliminary Research

Early research advanced this dissertation in two ways: experience and relevant findings. First, these studies provided an empirical introduction to the topics of human physiological adaptation, oculomotor research, and simulator sickness. Knowledge was gained, issues were raised, and lessons learned. Second, results from this research provided logical stepping stones towards the major questions asked in this research. Physiological recalibration processes (in the form of postural stability) were observed

[52] This example demonstrates the response when VOR gain is greater than 1.0, as evidenced by the initial eye movement overshoot followed by a corrective saccade back to the target after the light was turned on.

during and after exposure to a virtual interface. In addition, modifications to the virtual interface modulated physiological response characteristics. Finally, VOR changes were observed in one subject after a fairly short exposure to a commercially available immersive virtual interface.

4.3 FACILITY DEVELOPMENT

Prior to initiation of a research agenda into the physiologic effects of virtual interfaces, a facility had to be constructed with the specialized capability to explore these issues. With the full backing and support from the HITL Director, a physiologic mini-laboratory was assembled at the HITL. This lab, titled the VR Effects Lab, has the capability to research a range of oculomotor, postural stability, and psychophysical responses to virtual environments. Below is a detailing of the lab's equipment along with its current configuration.

4.3.1 THE EQUIPMENT

Visual image: WARP TV software provides a low latency virtual image in response to head movements. A 360-degree cylindrical image is pre-computed and pre-rendered into memory (RAM) at program initiation (Figure 30). When a subject moves his/her head, the portion of the image that corresponds to the new head position is drawn on the display directly from RAM (shown as the square insert in Figure 30) via a memory address computed from head position sensor data. The displayed image does not have to be rendered in real time, reducing total system latency. The measured update rate is 65 fps in isolation and approximately 45 fps when integrated with the head tracker. Only rotations (pitch, yaw, and roll) are registered; linear translation through the VE is not an option with the current configuration. WARP TV has a selectable time-delay buffer so that a desired time delay can be directly input into the system. The minimum system

time delay (from head tracker movement to visual scene update) is 48 ms and the maximum attainable is approximately 500 ms.

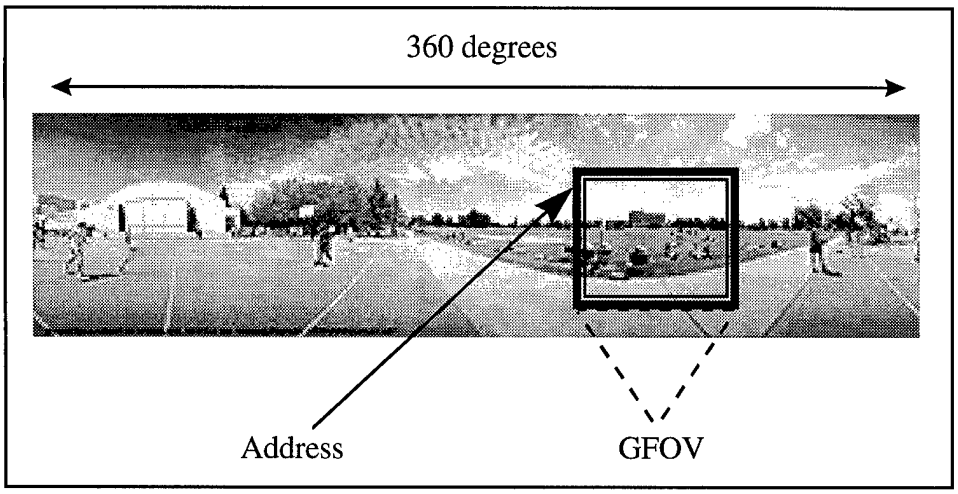

Figure 30: WARP TV Image Presentation

WARP TV runs on a Pentium 166MHz PC with an ATI Technologies Mach 64 Pro Turbo graphic accelerator with 4 MB of video memory. The PC has 16 MB of RAM and a PCI bus.

HMD: A Virtual i/O HMD was used as the virtual interface display (shown in Figure 31 with the eye tracker mounted) for these experiments. This HMD has 2 full-color, 1.78 cm active matrix liquid crystal displays (each with 180,000 addressable pixels) and the unit accepts VGA input at 60 or 70 Hz (Real Time Graphics, 1995). Angular resolution is 6.84 arc min/color pixel. This HMD has a 25 deg horizontal by 19 deg vertical DFOV, with 100% overlap between the two eyes (the virtual images were not presented in stereo). The Virtual i/O HMD has a fixed focus at 3.35 m in order to minimize eye-strain. Though the HMD can be worn with glasses, it was not an option

in these experiments because the spectacle lenses would interfere with the attached infrared eye-tracking system. The weight of the HMD is approximately 228 grams.

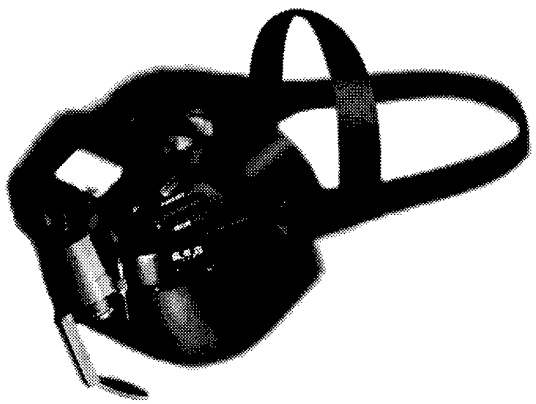

Figure 31: Virtual i/O HMD (Shown with VOG Eye Tracker mounted)

Head Position Tracking: An InterSense IS-300 system is used to track head movements. 'sourceless' technology (a miniature, solid-state, drift-corrected inertial measurement unit with angular rate sensor, gravitometer, and compass), the system has essentially unlimited range. It also minimizes 'jitter' in the visual scene and is scarcely impacted by electromagnetic interference. The sensor unit's dimensions are 2.54 x 3.3 x 3.0 cm and it weighs 60 grams. When used in the current configuration, the system updates at 250 Hz (system latency of approximately 4 ms) with an angular resolution of 0.02 deg RMS, a dynamic accuracy of 1 deg RMS and a static accuracy of 3 deg RMS.

The tracker was mounted directly above and centered on the subjects head via an aluminum bar attached to the HMD (Figure 32).

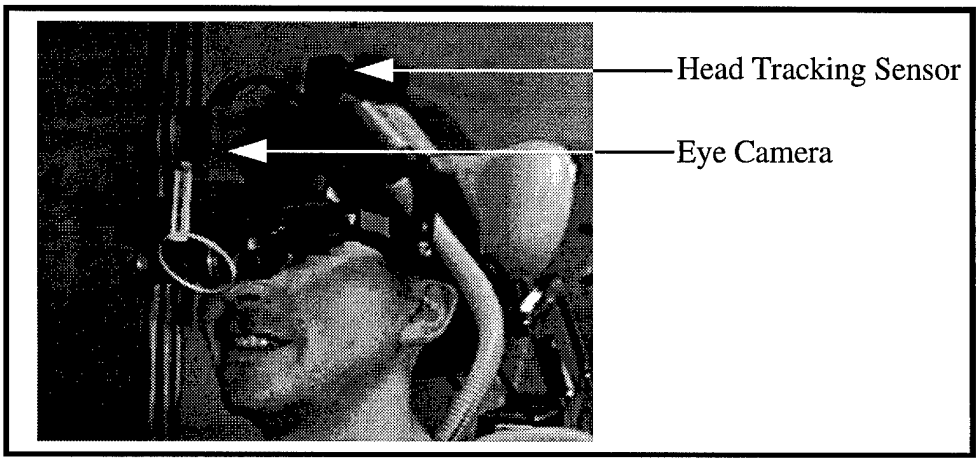

Figure 32: HMD with Head Tracking Sensor

The InterSense system was replaced the Shooting Star ADL-1 (a mechanical head tracking system based on high precision potentiometers) because it performed as well without the safety implications of a mechanical linkage coupled to the subject's head[53]. However, the ADL-1 remained as a back-up unit and was useful for calibration purposes (see Appendix A). Therefore, its specifications are included for completeness. The ADL-1 provides good resolution (0.15 to 0.30 deg), repeatability (less than 2.5 mm) and low system latency (3 to 5 ms). The ADL-1 has a limited working volume due to the mechanical linkages (half cylinder, approximately 1 m diameter, 0.5 m high).

[53] This especially became an issue during VOR tests when the chair oscillated.

Eye Position Tracking System: An ISCAN video-oculography (VOG) system is used to measure eye position relative to the subject's head (Figure 33). This system utilizes a high-speed CCD camera, a real-time digital image processor, and an IR LED emitter. The camera and emitter are mounted on the Virtual i/O HMD (Figure 32). The camera records a video image of the left eye as reflected through the HMD optics and off a dichroic mirror. The IR LED provides infrared illumination (not coaxial with the eye imaging camera) so that the pupil and a corneal reflection can be identified in software. The system uses 'dark pupil' processing, with the pupil acting like a sink and the surrounding area of the eye reflecting the IR back towards the camera. The IR illumination also allows for the unit to be used in complete darkness. Software detects and tracks the pupil centroid as well as a corneal reflection even in the presence of shadows and/or eyelashes in the image. The system has a selectable update rate of 60, 120, 180, or 240 Hz and an average horizontal resolution of 0.5 deg. For these experiments, the ISCAN system update rate was fixed at 180 Hz. The ISCAN system is controlled via a Pentium P-166 PC with 32 MB of RAM.

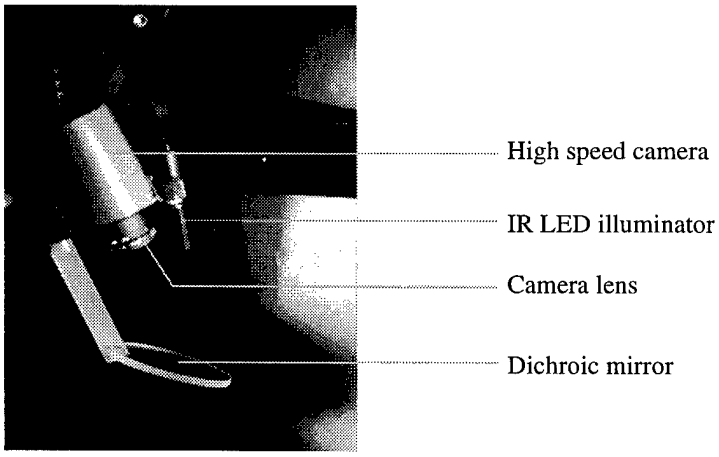

Figure 33: ISCAN eye-tracking system
(shown not mounted on the HMD)

Rotating Chair: A rotating chair (Contraves Direct-Drive Rate Table; Series 800; Figure 34) was obtained via a loan from NASA (through Dr. D.E. Parker). A Neurokinetics Servo Controller and Stanford Research Systems function generator (Model DS-345) are used to control the driving signal to the chair. For these experiments, the chair was always commanded to perform sinusoidal oscillations between 0.2 and 0.8 Hz at no more than 50 deg/s peak velocity.

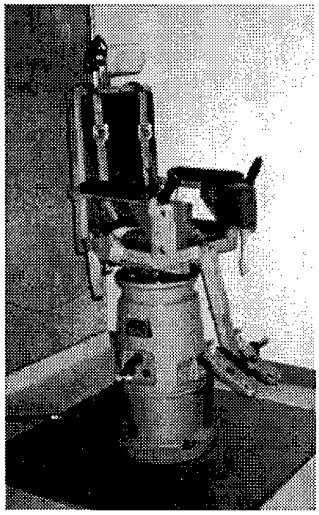

Figure 34: Rotating Chair

Balance Platform: A Chattecx Balance Platform is used to assess balance (Figure 35). The subject stands with a foot on a each of two force-sensing plates that rest on the platform base. The force plates detect shifts in the subject's center of gravity over a 10 or 25 s test. The main measure of a subject's balance is an 'index of dispersion', which

is the standard deviation of the subject's center of gravity (in cm) over the period of the test. The platform has an update rate of 100 Hz. The experimenter controls the balance test via an integrated PC (286 Intel processor) running Chattecx DOS-based software. Subjects can stand in many different stances, including the sensitive Sharpened Romberg stance, simply by moving the two force-sensing plates. In addition, the base of the platform can be made to move in sinusoidal or impulse fashion in order to explore dynamic postural control. There are safety rails around the platform base and an optional harness can be installed for further protection. This platform is also on loan from NASA (through Dr. D.E. Parker).

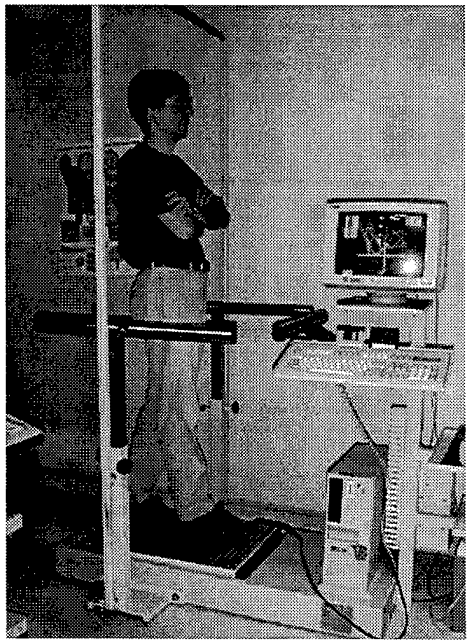

Figure 35: Chattecx Balance Platform

Acquisition/Analysis software: NI-DAQ software and data acquisition cards from National Instruments control transmission of data from the two Pentiums to a Macintosh computer for storage. MacEyeball software, developed by researchers at UCLA, controls test file configuration, storage, and analysis.

There are two separate MacEyeball programs, one for data acquisition (MAQ) and one for data analysis (MAP). The MAQ allows the experimenter to set the parameters of a VOR test (including experimenter comments), calibrate eye and head position data, determine the sampling rate for data collection, and initiate VOR testing. After VOR test completion, MAQ saves the collected eye and head position data in a proprietary format prior to the beginning of a new test trial. The MAQ needed to be slightly modified in order to function correctly using the equipment configuration in the VR Effects Lab. Some extraneous functionality was removed and the program was modified to read from the appropriate A/D DAQ card.

The MAP reads files stored by MAQ and performs VOR analysis using three methods: XY Analysis, Fourier, and Varant. However, only the latter two methods were used by this research. The specially coded MAQ files contain the relevant information needed for automated VOR analysis including sampling rate and testing frequency. A detailed description of the overall capabilities of MAP can be found in Demer, et al., (1989) and Demer (1992). A summary of its operation is described in Section 5.6.1.

The MAP had to be altered to better link with the capabilities of the specific equipment used to collect eye and head position information. Specifically, the saccadic removal subroutine and the low-pass frequency cut-off were adjusted after many simulations to achieve optimal performance.

Computers: Two PCs (Pentium P166 MHz processors) and one Macintosh IIfx computer exist in the lab. The WARP Pentium (which controls the virtual image) has 16MB of RAM and an ATI Mach 64 graphics accelerator card while the VOG Pentium (which controls the ISCAN system) has 32 MB of RAM. The Macintosh (used for data acquisition and storage) has 32 MB RAM and includes a ZIP Drive and CD ROM player.

4.3.2 VR Effects Laboratory Configuration

The physical layout of the VR Effects Lab is described first, followed by the specific equipment configuration utilized for these experiments.

4.3.2.1 Physical Layout

The physical layout of the VR Effects Lab is shown in Figure 36. There are three areas: an experimenter control shed, a subject area, and a reception area.

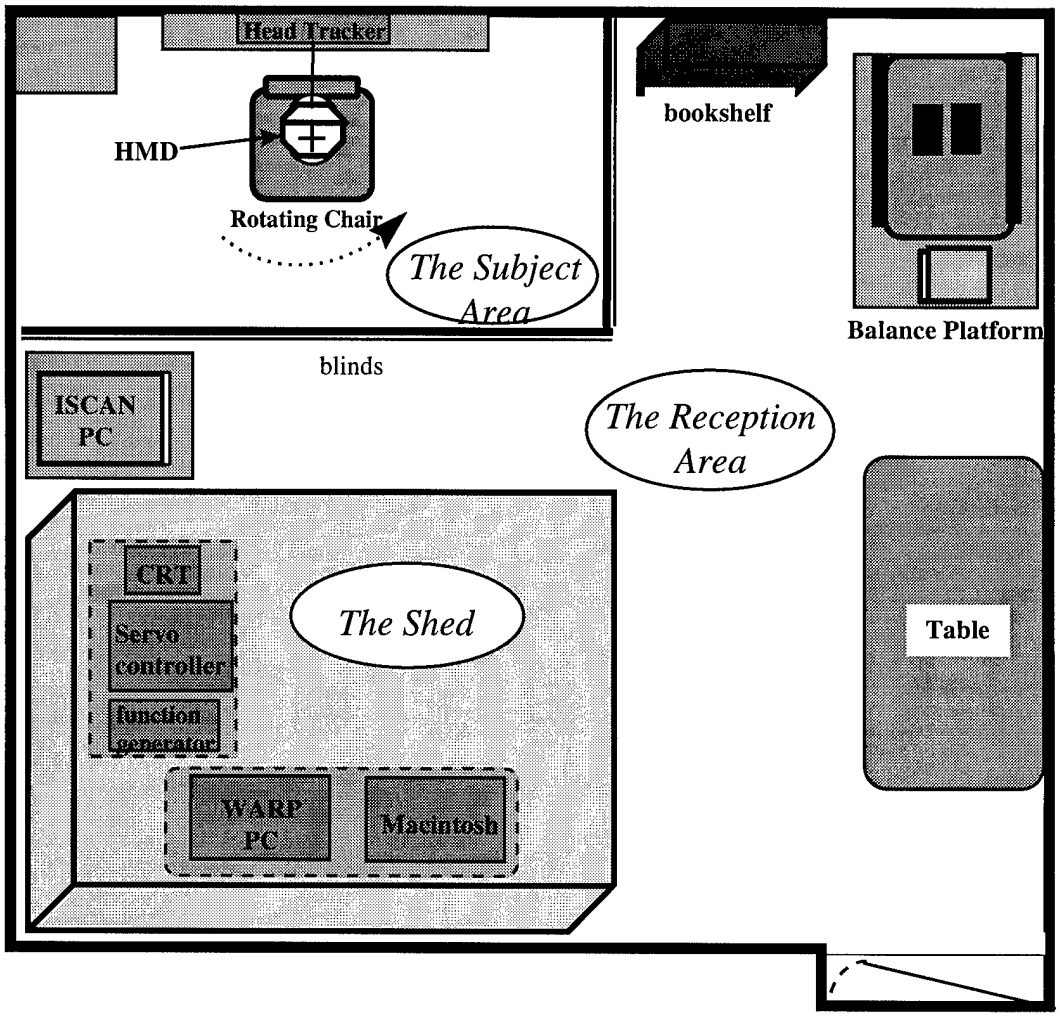

Figure 36: Physical Layout (VR Effects Lab)

The experimenter shed contains all the equipment that needs to be operated during a VE exposure and VOR test. The experimenter is enclosed in the shed with the necessary equipment, and the shed is made light tight[54] with respect to the other two

[54] Foam and duct tape were wonderful in this regard. So much so that the lab bequeathed the name 'Duct Tape' to the PC that resides in the shed.

areas[55]. Equipment in the shed includes the Macintosh data acquisition computer, the WARP Pentium PC, the servo-controller, the function generator, and a CRT displaying the subject's eye image as seen through the ISCAN camera. Using this equipment, the experimenter can monitor what the subject sees in the VE (using the WARP PC monitor), change VEs, monitor eye image quality, start, stop and alter chair oscillations, and control data collection for during each test trial (see Figure 37 for an internal view of the shed). The experimenter communicates with the subject using the sophisticated method of talking loud enough to be heard from within the shed.

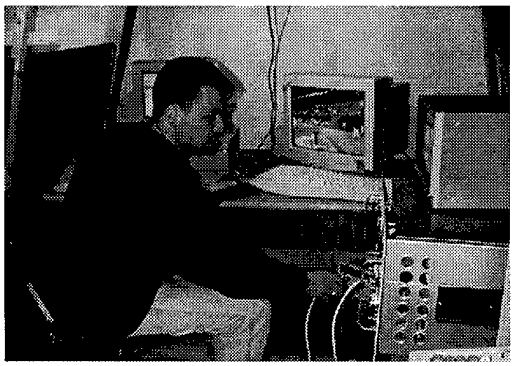

Figure 37: Shed: Internal View (with experimenter)

The subject remains in the subject area for the entire VE exposure and VOR testing periods. This area contains the rotating chair, the HMD/VOG unit, the head tracker, two fans for heat dissipation (one of which is attached to the chair for masking of auditory cues), an area light, and a mini flashlight. A research assistant also remains in the subject area to help conduct testing. Though the room was light-tight with regards

[55] In addition, the shed sports 4 "sunroofs", a large entrance area, and a fan to facilitate heat dissipation between test sessions.

to the exterior and the shed was light-tight with regards to the reception area, there was still a slight possibility that small stray light sources could exist. Therefore, the subject area was further made light-tight by a wall of drawn occluding blinds which isolated this area from the reception area.

The reception area is used for subject briefings, questionnaire completion, VOG set-up and calibration, and balance testing. The Chattecx Balance Platform is located in this area as is the VOG computer. The eye calibration markers are located just above the experimenter shed, approximately 4.1 m from the subject.

4.3.2.2 Equipment Configuration

For these dissertation experiments, the equipment was configured as depicted in Figure 38. The virtual scene was generated on the WARP PC and presented to the subject via the Virtual i/O HMD. Head position changes were tracked by the InterSense system which sent this information via serial connection to the WARP Pentium PC for image updates. Since the entire 360 degree image had already been pre-rendered and stored in RAM, the minimum system time was very small (approximately 48 ms). Changes to the GFOV (which influences image scale) and changes to system time delay could be made using the WARP computer.

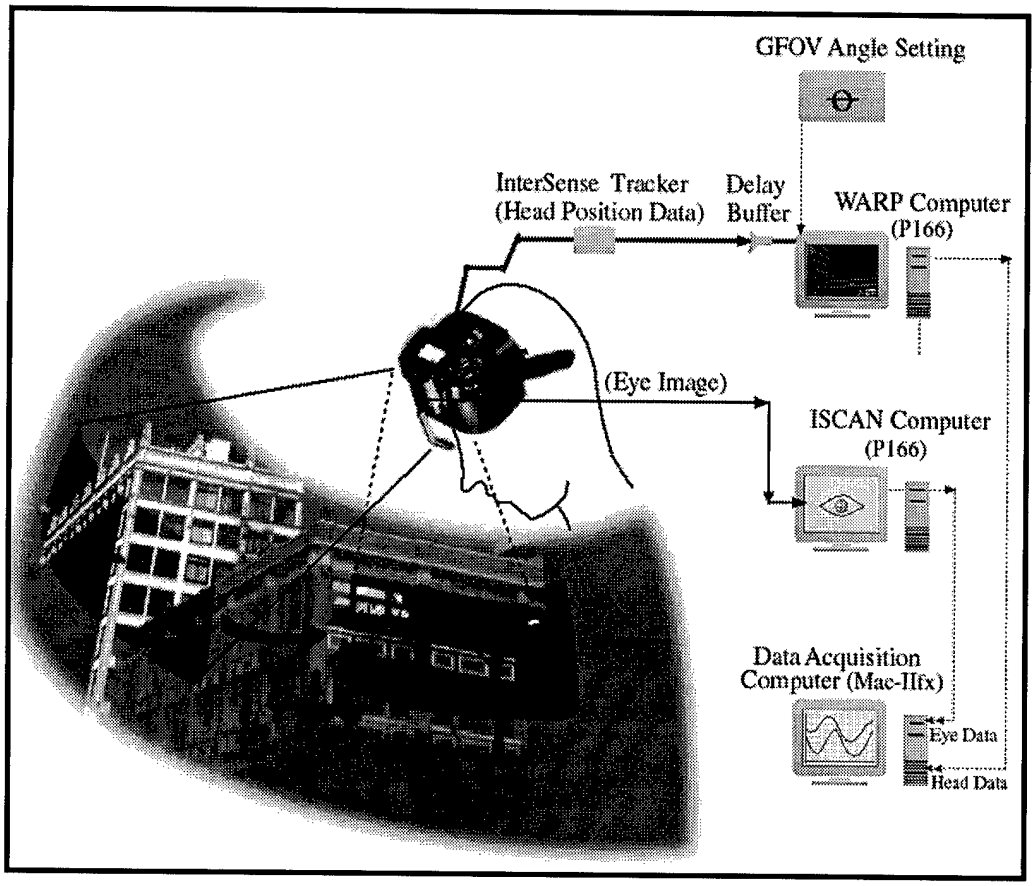

Figure 38: Equipment Configuration (VR Effects Lab)

During VOR testing, the subject was sinusoidally oscillated on the rotating chair in the dark. The ISCAN head-mounted camera recorded video images of the eye which were sent to the VOG PC for processing of eye position data. The InterSense tracker provided head position data via the WARP PC. Both head and eye data were automatically converted from digital to analog input using digital-to-analog cards (National Instruments LAB PC+ DAQ Cards) residing in each PC and the resulting

analog signals were sent via cables to the data acquisition computer. Upon entering this computer, the data were re-digitized (National Instruments Lab NB DAQ Card). MAQ software synchronized and stored the combined eye-head data file along with the necessary test configuration information for later analysis. MAP software was used to analyze the data (including filtering, digital differentiation, fast component removal and calculation of best curve fit) to obtain VOR gain and phase metrics.

4.3.3 System Calibrations

This section summarizes the calibrations performed for this research. First, head and eye tracking system calibrations are discussed, followed by GFOV calibration. System time delay calibration is discussed in Appendix A.

4.3.3.1 Head and Eye Tracking Calibrations

The InterSense head position sensor was calibrated both statically and dynamically. The static tests consisted of reading raw tracker angular position outputs in response to sensor placement at predefined angles. Several trials were accomplished at each angle to verify acceptable repeatability. Dynamic accuracy in the yaw direction was verified by oscillating the sensor on the rotating chair at predefined amplitudes. In addition, amplitude data were recorded from the ADL-1 mechanical tracker as a cross-check. The InterSense tracker was found to perform according to its specifications with no recalibrations required.

Horizontal eye position was calibrated for each subject at the beginning and end of each experimental session. Three markers (center, 10 deg left, 10 deg right) were placed at approximate eye-height on a wall 4.1 m away from the subject. The exact separation of these markers was determined first by a small laser pen positioned on the chair (at approximate eye-height; see Figure 39) and later confirmed through geometry.

Eye calibration values were determined by having the subject fixate on each of the three markers in succession while the output of the eye tracker was recorded.

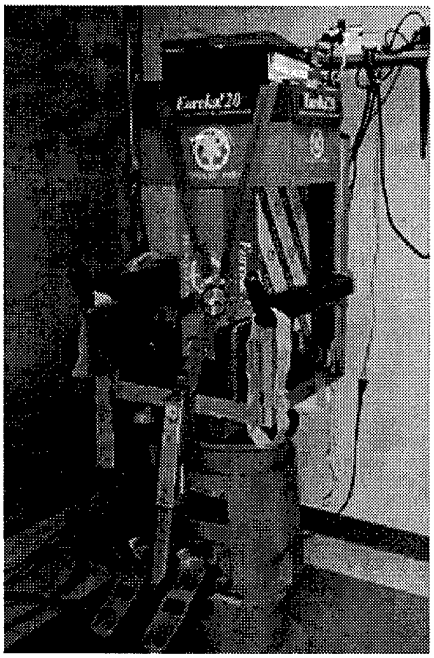

Figure 39: Rotating Chair with
Laser Mounted at Eye Level

4.3.3.2 GFOV and System Time Delay Calibration

Though GFOV already existed as an adjustable parameter of WARP software, angle accuracy needed to be verified. GFOV was calibrated by using a virtual compass. A model of a virtual compass was developed such that the station point (i.e., observer location in virtual space) was at the center of the compass. Markers identified each five-degree increment of the compass (in yaw only). When rendered using WARP

software, the number of marker segments spanning the horizontal DFOV of the HMD was an empirical estimate of the GFOV. Figure 40 illustrates this procedure for the setting GFOV = 25 deg (five post segments, each five degrees separation). Note that this figure represents an image scale of 1.0X because the HMD horizontal DFOV was also 25 deg. This calibration procedure verified the accuracy of Warp TV'S GFOV settings.

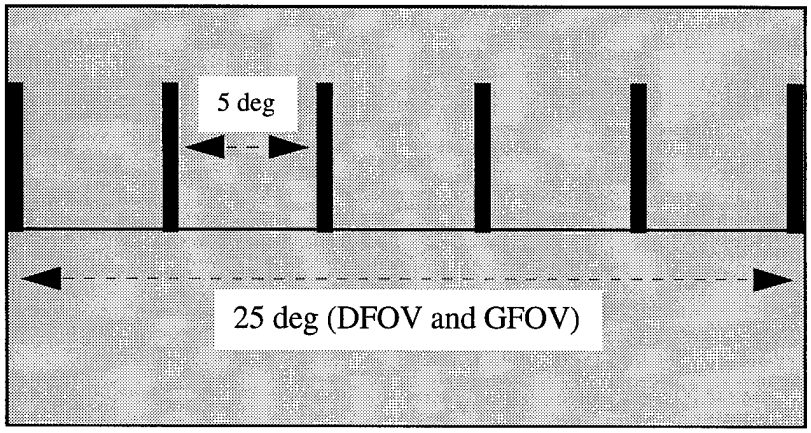

Figure 40: Virtual Compass (GFOV calibration)

Calibration of system time delay was a more complex undertaking. Therefore, a description of this process is presented in Appendix A.

4.4 RESEARCH OVERVIEW

The following research (Chapters 5 through 8) investigated the physiologic effects of virtual interfaces through a detailed study of the VOR. Four studies were conducted

to address the five main objectives presented in Chapter 1 (including the two hypotheses described in Chapter 3).

The first experiment (Image Scale Experiment: Chapter 5) investigated VE image scale changes caused by GFOV/DFOV inequalities in order to determine if the resulting magnification or minification of the VE could drive VOR gain changes. GFOV is often misunderstood or ignored when generating VEs, potentially resulting in an incorrect setting and subsequent stimulus rearrangement being generated. This experiment comes the closest to matching the stimuli used in previous VOR adaptation research, since the visual manifestation of a GFOV/DFOV inequality is similar to that obtained using telescopic spectacles. Therefore, it was the most straightforward way to directly assess the effects of virtual interfaces on VOR recalibration mechanisms. Simulator sickness data also were collected for independent analysis and to correlate with any occurring VOR gain changes.

The second experiment (Time Delay Experiment: Chapter 6) explored the effect of system time delay on VOR gain and phase adaptation as well as on simulator sickness. Time delays are inherent in current virtual interfaces and they are one of the major sources of stimulus rearrangements regarding self-motion. As detailed in Chapter 3, system time delays create a variable VOR phase change demand. Since there have also been many claims circulating in the literature that time delays cause simulator sickness, these data were also collected.

Having demonstrated statistically significant VOR adaptation in the first two experiments, the third experiment (Longitudinal Experiment: Chapter 7) was an investigation into the time-course of adaptation to virtual interfaces. Two subjects had their VOR tested at 0, 10, 20 and 30 min of a 30 min VE exposure. These data provided insight into the influence of exposure time on VOR gain changes.

The fourth experiment (Step Experiment: Chapter 8) explored an underlying premise behind a potential technique for increasing the VOR gain of patients with chronically reduced vestibular function. The technique, proposed by Viirre (1996), utilizes virtual interfaces to incrementally increase the VOR gain change demand over a series of steps instead of a single large gain-change demand. This experiment investigated the relative benefits of incremental vs. single step gain changes on VOR adaptation.

Rather than passively expose the subject to a VE via forced rotation, these four experiments attempted to determine the physiologic effects of virtual interfaces during active, unrestricted, head-coupled interaction with the VE. This method was chosen to specifically address the *applied* health and safety questions of virtual interfaces.

CHAPTER 5: IMAGE SCALE EXPERIMENT

5.1 OBJECTIVES AND HYPOTHESES

Inequality between DFOV angle and GFOV angle often exists in virtual interfaces, due either to software limitations or the inability of the user to properly set this variable. This disparity can be quantified by examining the ratio DFOV/GFOV. Deviations of this ratio from 1.0 result in image scale changes (i.e., magnification or minification) of the visual scene (see Section 2.3.3.3). These scale changes mimic the visual stimuli experienced in traditional VOR gain adaptation studies that require subjects to wear magnifying/minifying spectacles. Therefore, it is reasonable to infer that certain DFOV/GFOV ratios may induce VOR gain adaptations as well as sickness symptoms in users of immersive head-coupled virtual interfaces. The following experiment examined this possibility by systematically manipulating GFOV while holding DFOV constant.

This experiment, however, did not employ a traditional VOR adaptation protocol. Classic VOR adaptation research often entails exposure to a visual-vestibular rearrangement through passive rotation of the subject at specific frequencies with the subject's head immobilized with respect to the body. The subject would stare at a meaningless visual scene (often random dots or vertical stripes) with no task to perform. This traditional protocol attempts to amplify the effects of the stimulation over a very limited and highly specified set of conditions.

The interesting question of this experiment, however, was not simply whether DFOV/GFOV deviations could drive VOR gain changes but whether they could do so while subjects *actively* moved their heads in a *natural, unrestricted manner*, across *several frequencies*, while they *performed a meaningful task* in a VE. In other words,

would DFOV/GFOV deviations from 1.0 cause VOR gain adaptations in realistic VE user scenarios?

Given the five main objectives of this dissertation identified in Chapter 1, this experiment addressed objectives 1, 2, 3, and 5. Specifically, this experiment was designed to ascertain: 1) if VOR gain adaptation could occur in virtual interfaces that involved natural head-coupled interaction with meaningful visual scenes over a short exposure period, 2) if this adaptation could be directionally modulated through systematic variation of the ratio DFOV/GFOV, 3) if any occurring VOR adaptation was frequency specific or if it generalized across the tested frequencies, 4) if simulator sickness reports were also effected by DFOV/GFOV deviations, and 5) if VOR adaptation covaried with sickness reports. In addition, preliminary data were to be collected on post-exposure re-adaptation time-course information for any occurring VOR adaptation and sickness incidence.

Henceforth, the ratio DFOV/GFOV is termed 'image scale' to simplify matters. It was hypothesized that an image scale of 0.5 (corresponding to a 0.5X visual scene magnification, which is thus termed a minification) would reduce the gain of the VOR, an image scale of 2.0 (corresponding to a 2.0X scene magnification) would result in a VOR gain increase and an image scale of 1.0 (i.e., no scene magnification) would result in no VOR gain changes. Given that the VOR can be a noisy signal and that the gain adaptation achieved rarely matches stimulus demand, a threshold for determining adaptation was required. A threshold of 5% change in VOR gain was chosen, partially from a review of short-term VOR adaptation research results, partially from its potential implications on retinal image stability (if not compensated for by visual tracking mechanisms), and partially on 'scientific intuition' as to what amount of gain change would be considered meaningful after a 30 minute exposure period. Therefore, the

hypotheses presented above specified adaptation as a statistically significant VOR gain change of at least 5% from baseline values.

In addition, it was hypothesized that VOR adaptation magnitude would be the largest at the lowest test frequencies since several VOR adaptation experiments have shown frequency specificity and subjects tend to reduce amplitude and frequency of head movements while immersed in virtual environments (Pausch, et al., 1996). Sickness incidence was hypothesized to be the highest in the scale magnification condition (due to the sensory rearrangement and increased optic flow velocity), less but still significant in the minification condition (due to the sensory rearrangement), and least in the neutral (correctly scaled) condition. Lastly, it was hypothesized that there would be a moderate correlation (approximately 0.40 to 0.60) between sickness reports and VOR adaptation.

5.2 SUBJECTS

A total of 11 adult subjects (6 males/5 females, mean age 28.5, age range 19 to 39) volunteered to participate in this experiment. All subjects reported to be in good health with no history of epilepsy or vestibular medical problems. Subjects were tested for normal or corrected visual acuity of 30/20 or better. Five subjects wore contacts for corrected visual acuity and three of these reported some level of diagnosed astigmatism. Only contacts were acceptable for vision correction during the experiment due to incompatibilities between the eye tracking system and spectacles. Subjects voluntarily abstained from drugs and alcohol for 12 hours prior to participating in each session. Nine subjects reported that they had experienced motion sickness in the past and two subjects reported experiencing simulator sickness in the past. Two subjects claimed to be nonsusceptable to motion sickness, six subjects reported slight susceptibility and three reported moderate susceptibility. At the onset of testing, all subjects were new to

the particular virtual environment used in this experiment and had not experienced any head-coupled virtual interface in the previous 30 days (seven subjects had never before been exposed to head-coupled virtual interfaces).

5.3 EXPERIMENTAL DESIGN

The overall experiment was conducted as a 3 x 2 x 3 within subjects design. Three levels of image scale (SCALE: MIN [0.5X], NEU [1.0X], and MAG [2.0X]) were crossed with two levels of testing time (TESTTIME: PRE-exposure, POST-exposure) and three levels of oscillation frequency (FREQ; 0.2, 0.4, 0.8 Hz). Additionally, VOR measurements were recorded 10 minutes post-exposure on several subjects. These data, obtained only on a subset of subjects, were analyzed separately. Each subject participated in a total of three, 1.5 hour sessions (i.e., one session per SCALE condition). Each session was separated by a minimum of 6 days to minimize carryover effects. Sessions were counterbalanced across SCALE and SUBJECT.

The dependent variables for VOR gain were averaged VOR gain estimates and percent gain adaptation. Percent gain adaptation was a derived variable of adaptation using the formula: ((POST-PRE)/PRE)*100. VOR phase data were also collected, averaged and analyzed.

Sickness reports were also collected before, during, and after VE exposure. These dependent variables included: 1) oral reports of sickness during exposure and 2) SSQ scores. The oral reports were collected at specified times during VE exposure (10, 20, and 30 minutes). Each report was a number on a scale of 0 to 3, in which a '0' indicated no discomfort, feeling fine, '1' indicated slight but noticeable discomfort, '2' indicated moderate discomfort, and '3' indicated very strong discomfort such that the subject wished to take a break or end the experiment. SSQ data were collected pre-exposure, immediately post-exposure, and 20 minutes post-exposure.

In addition, postural stability was measured pre- and post-exposure as a safety check prior to releasing the subject. The metric of postural stability utilized in this experiment was the number of stance breaks by the subject over a 30 second period while in a Sharpened Romberg stance with eyes open.

5.4 EXPERIMENTAL SET-UP AND APPARATUS

This experiment was conducted in the VR Effects Laboratory, a component of the HITL on the University of Washington campus. The apparatus and experimental set-up was as described in Section 4.3. Head and eye position data were collected at 165 Hz. The minimum system time latency (mean: 48 ms; SD: 8 ms) was fixed for this experiment, as was DFOV (25 deg horizontal by 19 deg vertical). Only GFOV was systematically varied. GFOV was set by adjusting the appropriate variable in the WARP TV software.

5.5 PROCEDURE

This experiment required two experimenters, myself and an assistant. I was the main communicator with the subject, controlled all equipment, conducted all eye tracking calibrations and VOR testing, collected sickness data, called out search targets during VE exposure, and managed the conduct and flow of each session. As I was sealed in a light-tight shed during VOR testing and VE exposure, the assistant was by the subject's side during this time. The assistant aligned the chair, issued mental tasks for the subject to perform during VOR testing, administered the first post-exposure SSQ as well as the controlled post-exposure eye-head movement task, acted as a safety monitor and handled miscellaneous problems that would occasionally arise during VOR testing or the exposure period. A copy of the experimental protocol appears in Appendix B which more explicitly details this teaming arrangement.

5.5.1 Preliminaries, Calibrations, and Baseline Measures

Each experimental session began with a pre-briefing, subject consent (Appendix C), a pre-exposure SSQ (Appendix D), and a test of visual acuity (using a Snellen Chart). Two pre-exposure balance trials were then conducted to obtain baseline values for use in assessing the subject's post-exposure postural stability (a safety precaution prior to releasing the subject after the experiment). In each balance trial, the subject stood on the lab floor in a Sharpened Romberg position with eyes open for 30 seconds. The number of stance breaks per trial was recorded and averaged across trials for comparison with post-exposure values.

After the balance tests, the subject was seated in the rotating chair and secured with a 5-point safety harness and foot straps. The HMD/Eye tracker unit was positioned on the subject's head such that the subject felt comfortable and a optimum eye image was obtained by the VOG camera (see Figure 41). In actuality this required some effort because in order to prevent slippage the HMD had to be snug, but not so tight as to cause subject discomfort. Lastly, a chair headrest was positioned to minimize potential decoupling of head from chair during chair oscillations.

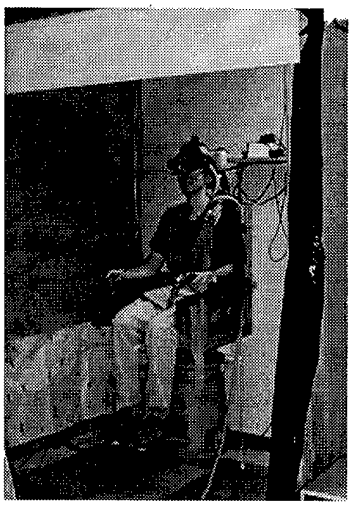

Figure 41: Subject in Chair

Once subject comfort and eye imaging were maximized, the eye tracking system was calibrated for horizontal eye movements. Subjects were instructed sit erect, place his/her head against the headrest (to prevent any head movements), and to only move the eyes towards each of three calibration dots. These dots (located at approximate eye-height on a wall 4.3 meters from the subject) consisted of a center dot directly in front of the subject, along with a left and a right dot which were each horizontally separated from the center dot by 10 deg visual angle from the subject's position. Calibration was accomplished manually with the aid of the MacEyeball data acquisition system. The results from a minimum of three successful calibration sequences were averaged to obtain the final calibration factor for horizontal eye position data.

After calibration of eye movements, the subject's VVOR in the real world was measured. The subject oscillated at 0.8 Hz (32 deg/sec peak velocity) while he/she attempted to fixate on the center calibration dot. Two separate trials (8 sinusoid cycles each) were recorded. Upon completion, the room was darkened for dark VOR baseline

measurements. Final room darkening consisted of sealing the shed entrance, turning off all lights and lowering occluding blinds around the subject area.

Baseline VOR data were collected at each sinusoidal test frequency (0.2, 0.4, & 0.8 Hz), with a peak velocity of approximately 50 deg/sec. Two trials were collected at each test frequency. For each trial, four sinusoidal cycles were collected at 0.2 Hz, and eight cycles were collected at 0.4 and 0.8 Hz The subject was given mental alerting tasks to perform during the oscillations[56]. The subject was not instructed to fixate on an imaginary point in the distance, as that behavior (EVOR) may involve contributions from a separate visual fixation system (Fuchs, personal communication, 1997).

5.5.2 VE Exposure

After collecting baseline VOR data, the subject's VVOR in the VE was recorded. These virtual VVOR data provided information on the saliency of the VE for stimulating oculomotor adaptation processes. The HMD was turned on so that the subject could view the virtual image (at the specified image scale for that session) and the subject was instructed to fixate on a point within the VE near the center of the display[57]. The subject was then oscillated at 0.8 Hz (approximately 30 deg/sec peak velocity) while he/she maintained fixation on this point in the VE. Data from two trials were collected, with eight sinusoidal cycles recorded per trial.

Upon completion of the virtual VVOR tests, the subject began 30 minutes of active, task-driven interaction with the VE. A total of five different QuickTime VR 360 degree cylindrical images[58] were presented to the subject during the exposure period

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[56] Examples of mental alerting tasks used appear in Appendix B.

[57] The initial movement of the chair required subjects to fixate on a target between the center an left edge of the visual display so as to keep the target within the DFOV throughout the entire oscillation cycle.

[58] All images had equivalent resolution and were approximately equal in size (around 400kB). Appendix E contains the images used along with one image's list of search target items.

(approximately six minutes of exposure to each image). For each VE image, the first minute of the exposure was designated as exploration time for the subject to memorize the spatial arrangement of objects within the scene. The subject performed several head rotations during this time in order to view as much of the visual scene as possible. Since all scenes were images from around the Seattle area, subjects enjoyed guessing 'where they were at' during this exploration time. The remaining five minutes consisted of a series of visual search tasks. The subject began each search task looking directly ahead. The experimenter then called out a target and the subject responded by finding the target, fixating on it, and verbally identifying that the search was completed. The experimenter verified the response by monitoring a display that mirrored the subject's visual scene. After verbal confirmation by the experimenter that the target was located, the subject would again look straight ahead and a new search target was identified. The main purpose of the search task was to continually have the subject interact with the VE using active, unrestricted head movements. Therefore, performance metrics on the task were not recorded. After this five-minute search period, the subject was presented with a new VE image. This procedure was repeated until all five images had been presented, resulting in a 30-minute exposure duration.

It is important to note that during the VE exposure period, no specific instructions were given to the subject regarding the speed or type of head movements. The subject moved his/her head as desired to explore the scene and successfully complete the search tasks. In order to gain knowledge on particular head movement patterns, head position epochs (yaw angle only) were collected. These epochs, 80 seconds in length, were collected at the beginning of the VE exposure, halfway through the exposure, and near the end of the exposure (they corresponded with the presentation of the first, third, and fifth VE images).

Simulator sickness data were collected during the exposure period through oral reports. At 10, 20, and 30 minutes, the subject was prompted to report his/her comfort level on a scale of 0 to 3, as described in Section 5.3. If a subject reported a '3', the display was immediately turned off and the subject was prompted to describe his/her symptoms. The subject was also prompted (and often encouraged) to end the experiment. Only those subjects who insisted on continuing were allowed to do so, under more stringent observation and with breaks taken as often as desired by the subject.

5.5.3 Post-Exposure Testing and Re-adaptation Protocol

At the end of the 30-minute exposure period, the subject's VOR response was again tested at 0.2, 0.4 and 0.8 Hz. The procedure was the same as for the baseline VOR tests. In some cases the HMD had to be adjusted to re-acquire a valid eye image. This readjustment was accounted for by a post-test eye tracking calibration.

At the end of these post-exposure VOR measurements, the room lights were turned on and the subject completed the first post-exposure SSQ (Appendix D). The subject was then asked to remain in the chair so that VOR data could be collected at 10 minutes post-exposure. Between the completion of the SSQ and the '10 minutes after' VOR tests, the subject performed controlled eye-head gaze shifts in the real world by searching for letters and numbers on a large white poster paper that was placed approximately one meter in front of him/her on the occluding blind. The subject kept the HMD on during this entire period, viewing the real world through the now transparent display optics. Although the subject remained seated, there was no restriction on head movements. At 10 minutes post-exposure, the room was once again darkened and VOR data were collected in the same manner as before. Lastly, the lights were turned on and a post-calibration of the eye tracking system was accomplished.

At this point the HMD was removed, the chair's harnesses were released, and the subject got out of the chair. The subject was provided with refreshments (soda and cookies) as he/she completed a general post-test questionnaire (Appendix F). The subject then completed 2 balance trials (in the same manner as pre-exposure) to verify that no obvious balance instability was created by the environment. If any was noted, the subject was required to wait 5 minutes and perform the balance tests again[59]. Lastly, at approximately 20 minutes post-exposure (5 minutes post-release from the chair) the subject filled out a second SSQ. Then, upon assurances that the subject was 'feeling fine' with no observable aftereffects, the subject was released.

5.6 DATA ANALYSIS

Specific details regarding the analysis of VOR and VVOR data is presented first. Simulator sickness data and head position analyses are then described.

5.6.1 VVOR AND VOR DATA

The following is a summary of: 1) how the MacEyeball Analysis Program (MAP) obtained VOR gain and phase estimations and 2) how the final estimates used in the analyses were determined. A more detailed description of the complete MAP can be found though Demer, et al. (1989) or Demer (1992). This research utilized only a portion of the capabilities of the MAP.

The head and eye position signals were digitally low-pass filtered (8 pole Bessel, 0 to 22 Hz) and differentiated using the two-point central difference method. Large quick

[59] Only one subject needed to wait the additional five minutes and repeat the test.

phases were removed using conventional velocity and duration criteria[60]. The de-saccading algorithm did not attempt to remove all saccades, only those that were large and well defined. Smaller saccades and artifacts were removed though the elimination of statistical outlying velocity points during the regressions. The data were further analyzed by two separate methods, both of which used Fourier analysis techniques.

In the first method, termed 'Varant', the remaining data (after removal of quick phases) were fit cycle-by-cycle to a sinusoidal function using the method of least squares. The first response cycle was always discarded due to the potential presence of transient components. A Fourier transform was then performed at the fundamental frequency. Head velocity, eye velocity, phase and gain were computed for each cycle, and outlying cycles indicating low gain artifacts were automatically removed[61] The gain and phase estimates recorded were the averages of the remaining cycles from that trial. This method also provided an estimate of within-trial variability by calculating VOR data for each valid cycle in a trial.

In the second method, termed 'Fourier', the data points removed by the saccadic removal algorithm were replaced by velocity values computed from linear regressions and instantaneous head velocities. Fourier spectral analysis was then performed to compute gain and phase values at each of a range of frequencies. Phase lags were assumed negative, phase leads positive. This analyses was only accepted as reliable if the coherence at the frequency in question exceeded 0.80. Typical coherence values in this research were above 0.98 and nearly all of the trials were above 0.95. Gain and phase estimates at the test (i.e., peak) frequency were computed.

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[60] The MAP had to be altered to better link with the capabilities of the specific equipment available in the VR Effects Lab. Specifically, the saccadic removal subroutine and the low-pass frequency cut-off were adjusted after many simulations to achieve optimal performance.

[61] The statistical criterion by which outlying cycles are removed is described in Demer, et al. 1989. Excluded cycles had gain estimates that were greater than 1.3 standard deviations below the trial mean.

The above describes the automated determination of VOR gain and phase by two different methods. However, a few additional (manual) steps were required to determine the final values recorded first for each trial and then for each cell[62]. These steps are discussed below.

Since MacEyeball calculates gain and phase estimates using two different methods (Fourier and Varant), the final gain and phase values recorded per trial were obtained by averaging these two estimates. These estimates were in most cases within 1 to 3 percent of each other for gain estimates and for phase estimates were within a degree of each other. In addition, two trials were collected for each cell. For cells with 2 valid trials (144 of 162; 89%) the two estimates were averaged to get the final VOR value for that cell. This was the case for most cells, but a few cells had trial values that were deemed unacceptable due to excessive noise/distortion in the eye position data (i.e., lack of coherence between eye and head velocity data as determined by MAP), incomplete data collection, unwanted head movements by the subject, spurious head tracker output, etc. For cells where there was only one acceptable trial (14 of 162; 9%) that trial was used as the final value. For the cells that had invalid values for both trials (2 of 162; 1%), there was no meaningful information for what occurred in that cell (both of these cells occurred during POST tests of the MAG condition). I omitted those 2 cells and their associated PRE cells from the analysis, reducing the N from 27 to 25 for the MAG condition.

In most cases (22/27), the post-exposure eye calibration value was within two percent of the pre-exposure value. However, if the post-exposure value deviated by three percent or more, the post-exposure and 'after ten-minute' VOR gain values were adjusted using the appropriate correction ratio. The highest deviation found between pre- and post-exposure calibration values was seven percent (one case).

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[62] Note that a cell is the cross between FREQ and TESTTIME, within each SCALE.

A note about the experimental design: though the experiment was originally designed to be analyzed using one omnibus ANOVA, I decided to break the design into smaller sub-experiments for the following reasons. The primary question of this experiment was whether or not gain adaptation occurred, pre- vs. post- exposure, for each image scale. The levels of TESTTIME (PRE, POST) were individually meaningless but could be reduced to a single, meaningful 'difference' statistic. Additionally, the effects of FREQ, though interesting, were decidedly less pertinent to the focus of this dissertation. It is quite possible, given that subject attrition occurred between sessions (discussed below) along with the small number of subjects involved, that a three-factor between-subjects analysis of variance statistical procedure could mask true effects of adaptation. Therefore, it was deemed more reasonable to consider this experiment as consisting of three separate sub-experiments, one for each level of SCALE. Identification of adaptive effects is made much more directly and clearly with the sub-experiment approach while the risk of increases in family-wise error is no greater, given the multitude of post-hoc analyses required to glean specific findings from the omnibus ANOVA alternative. Finally, this approach to statistical analysis is consistent with much of the VOR adaptation literature. It should also be stated that this decision was made prior to any statistical analyses being performed, and no omnibus procedure was ever attempted.

Since the dependent variables utilized (e.g., VOR gain estimates, percent gain adaptation) were found to be normally distributed with equal variances, parametric statistics were used in the majority of cases. However, when these assumptions were not met, appropriate non-parametric statistics were employed.

5.6.2 Simulator Sickness Data

Almost all SS data are positively skewed (i.e., not normally distributed). This is true with the data collected in this experiment as well. In addition, the sickness reports

during exposure were ordinal, not interval, data. Therefore, only non-parametric tests were used in the analyses which in turn obviates the need for normality or homogeneity of variance testing.

As discussed below, 8 of 9 subjects participated in all sessions. The remaining subject position was filled by 3 different subjects, one for each SCALE. Because of this change, no 'related' or 'paired' statistics were used. The result is that all tests are correspondingly more conservative than would normally be expected for a design so heavily weighted towards pure repeated measures.

5.6.3 Head Position Data Analysis

Head position epochs (80 seconds each, yaw angle only) were collected at the beginning, middle, and end of each VE exposure session. Using LABVIEW software, each position epoch was passed through a autopower spectrum function to get the power spectral content of the head movements during that epoch. Data recorded from each spectral distribution included peak frequency, peak amplitude (in RMS deg), and the cutoff frequencies at which the power falls below 10 deg RMS and below 5 deg RMS (for estimates of bandwidth). Since the precise conditions under which these epochs were collected were not controlled, only descriptive statistics were calculated.

5.7 RESULTS

Eight subjects successfully completed the three 1.5 hour sessions. Two subjects withdrew after the first session: a female after becoming sick in the MIN condition and a male after the NEU condition, claiming disinterest[63]. Rather than discard their data,

[63] A review of his post-exposure sickness scores indicated nominal sickness for that condition.

one subject (a male) was run only in the MAG condition so that each of the three SCALE conditions would have a within-subjects design with nine subjects each.

This section addresses VVOR data, adaptive changes in VOR response, changes in sickness reports, head position analyses as related to changes in the VOR and sickness reports, the relationship between VOR changes and sickness reports, and re-adaptation data. To facilitate matters, some subsections will be parsed into the relevant sub-experiment involved (MIN, NEU, MAG).

The balance data collected pre- and post-exposure were not analyzed due to the complete lack of variance in the data.

5.7.1 VVOR DATA

To verify that the VE stimuli were appropriate to promote VOR gain adaptation, average real-world VVOR was compared to average virtual VVOR. These data are shown in Table 2 and graphically in Figure 42 and Figure 43.

Table 2: VVOR Gain and Phase Information
(Image Scale Exp)

Condition	Gain (SD)	Phase (SD)
Real	*0.94 (0.05)*	*-1.1 (2.0)*
MIN (VR)	*0.56 (0.04)*	*-8.5 (5.5)*
NEU (VR)	*0.90 (0.06)*	*-14.0 (3.0)*
MAG (VR)	*1.45 (0.06)*	*-20.3 (4.0)*

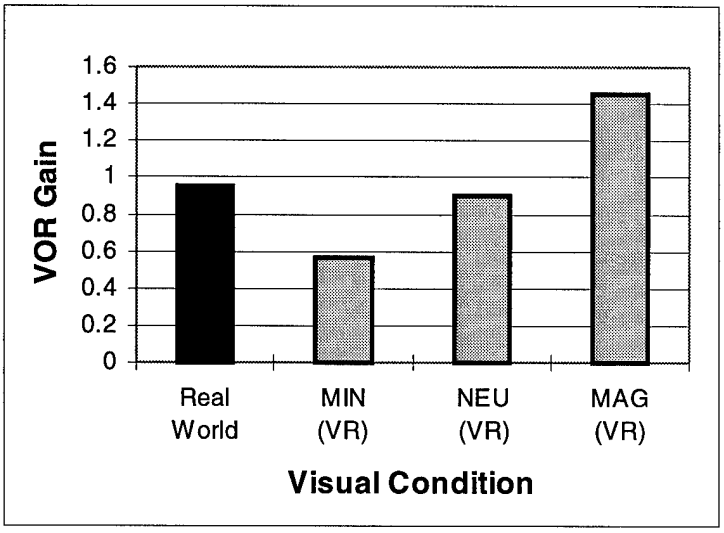

Figure 42: VVOR Gain by Visual Condition
(Image Scale Exp)

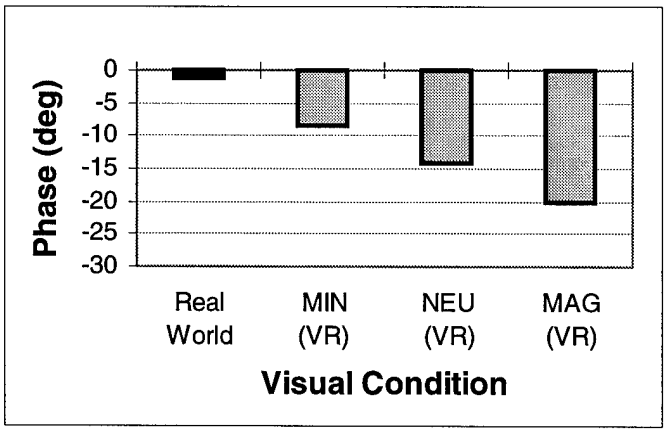

Figure 43: VVOR Phase Lag by Visual Condition
(Image Scale Exp)

The VVOR gain data suggests that each condition provided the expected gain adaptation stimulus. Real-world VVOR and NEU VVOR data were near the perfectly compensatory gain of 1.0. Phase data indicated an increasing VVOR phase lag (lag is denoted by negative values of phase) with increasing OKN stimulation.

5.7.2 VOR Gain Adaptation

A summary of the VOR gain adaptation data across the three sub-experiments is shown in Table 3 and Figure 44. Each sub-experiment's statistical results are then individually presented.

Table 3: Summary Data (all sub-exps)

Condition	N	Scale DFOV/ GFOV	Pre Gain	Post Gain	Difference (Post-Pre)	% Change	# of subj w/ gain decrease	# of subj w/ no gain change	# of subj w/ gain increase
MIN	*9*	*0.5*	*0.635*	*0.539*	*-0.096*	***-15.1***	*8*	*1*	*0*
NEU	*9*	*1.0*	*0.602*	*0.584*	*-0.018*	***-3.0***	*3*	*5*	*1*
MAG	*9*	*2.0*	*0.608*	*0.644*	*0.036*	***+5.9***	*2*	*0*	*7*

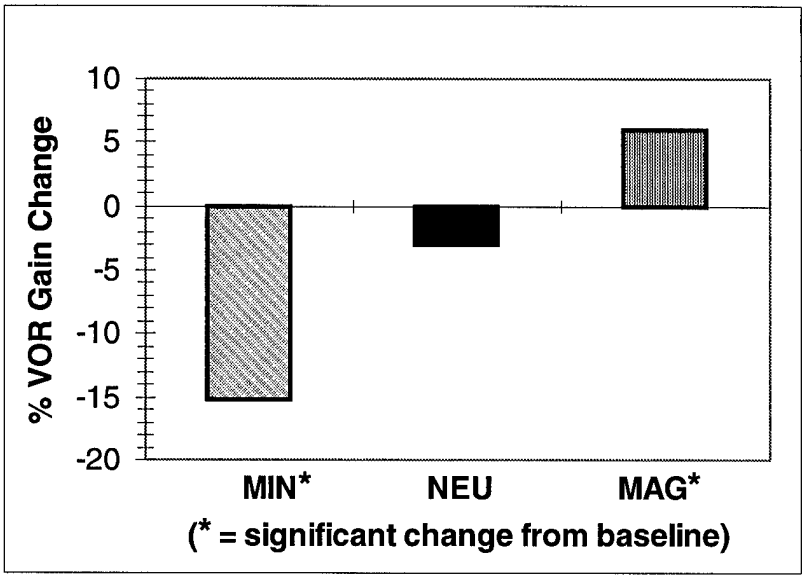

Figure 44: Summary of VOR Gain Change by SCALE

5.7.2.1 MIN Sub-Experiment

Summary data from the MIN sub-experiment are shown in Table 4. The main focus was to determine if a VOR gain reduction occurred after a 30-minute exposure to a VE with a image scale of 0.5X. Both the PRE and POST data were normally distributed (Shapiro-Wilks) with equal variances (F-test) and the PRE/POST data were significantly correlated ($r = 0.77$; $p > 0.001$)[64]. A paired t-test (POST - PRE) revealed a significant reduction of gain in the POST tests ($t(26) = 6.19$; $p < 0.0001$).

[64] Significant correlation is a prerequisite to performing a paired t-test

Table 4: VOR Gain Summary Data
(MIN sub-exp)

Test	N	Mean	Median	Variance	SD	SE	% Change
Pre	27	0.635	0.65	0.016	0.12	0.02	
Post	27	0.539	0.55	0.010	0.10	0.02	**-15.1**

Percent adaptation was also found to be normally distributed (Shapiro-Wilks). A one-way ANOVA indicated no effect of FREQ on percent gain adaptation (Figure 45), and a visual inspection of percent gain adaptation by session indicated no systematic effects of repeated exposure such as learning (Figure 46).

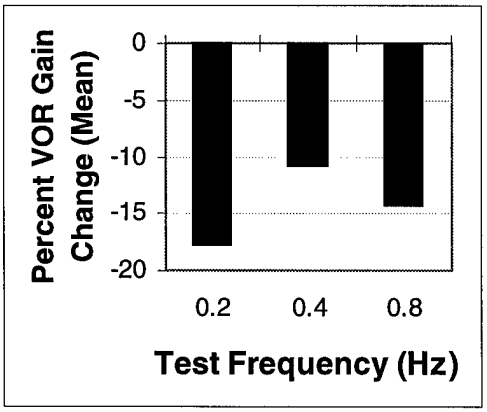

Figure 45: Percent VOR Gain Adaptation by FREQ
(MIN sub-exp)

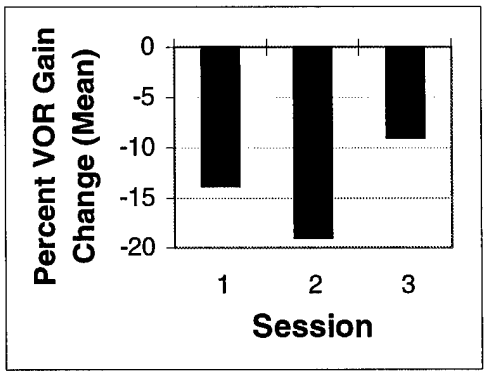

Figure 46: Percent VOR Gain Adaptation by Session (MIN sub-exp)

5.7.2.2 NEU Sub-Experiment

Summary data from the NEU sub-experiment are shown in Table 5. Both the PRE and POST data were normally distributed (Shapiro-Wilks) with equal variances (F-test) and the PRE/POST data were significantly correlated ($r = 0.85$; $p > 0.001$). A paired t-test (POST - PRE) was nonsignificant ($t(26) = -1.18$; $p > 0.24$).

Table 5: VOR Gain Summary Data (NEU sub-exp)

Test	N	Mean	Median	Variance	SD	SE	% Change
Pre	*27*	*0.602*	*0.62*	*0.023*	*0.15*	*0.03*	
Post	*27*	*0.585*	*0.59*	*0.016*	*0.13*	*0.02*	***-3.0***

Percent adaptation was also found to be normally distributed (Shapiro-Wilks). A one-way ANOVA indicated no statistically significant effect of FREQ on percent gain adaptation (Figure 47) and a visual inspection of percent gain adaptation by session indicated no systematic effects of repeated exposure.

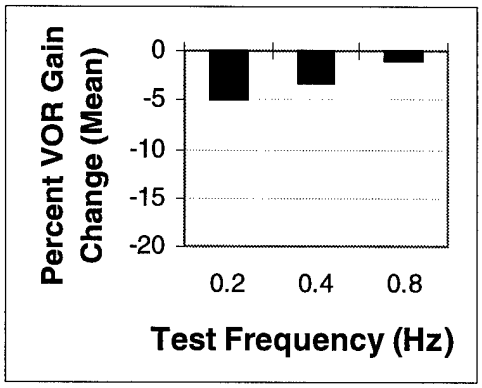

Figure 47: Percent Gain Adaptation by FREQ
(NEU sub-exp)

5.7.2.3 MAG Sub-Experiment

Summary data from the MAG sub-experiment are shown in Table 6. PRE data were normally distributed (Shapiro-Wilks) but the POST data were only marginally so (passing the K-S test but failing the Shapiro-Wilks). Invoking the Central Limit Theorem, a test of normality was conducted on the (POST - PRE) data. These data were normally distributed, allowing for the paired t statistic to be used. The PRE/POST data were significantly correlated ($r = 0.68$; $p > 0.001$). A paired t-test (POST - PRE) was significant ($t(26) = 2.24$; $p > 0.04$), indicating an increase in VOR gain as a result of VE exposure.

Table 6: VOR Gain Summary Data
(MAG sub-exp)

Test	N	Mean	Median	Variance	SD	SE	% Change
Pre	27	0.608	0.61	0.01	0.10	0.02	
Post	27	0.644	0.66	0.01	0.10	0.02	+5.9

Percent adaptation was also found to be normally distributed (Shapiro-Wilks). A one-way ANOVA indicated no main effect of FREQ on percent gain adaptation (Figure 48), and a visual inspection of percent gain adaptation by session indicated no systematic effects of repeated exposure such as learning, though there was no net adaptation in the third session.

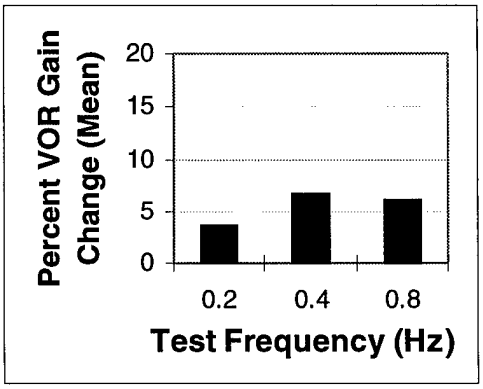

Figure 48: Percent Gain Adaptation by FREQ (MAG sub-exp)

5.7.3 Sickness Reports

As stated earlier, only nonparametric statistics were used for the sickness data given the lack of normality involved. Two of nine subjects had to terminate a VE exposure session early. These subjects both withdrew from the MIN condition after approximately 15 minutes due to sickness. However, both of these subjects completed

post-exposure VOR testing, completed the remaining two SSQs, and one returned for her remaining sessions. However, due to the remaining subject attrition, only between-subject statistics were employed. First SSQ results are presented, followed by the sickness ratings during exposure.

5.7.3.1 SSQ Data

Mean and median SSQ total scores over time are shown in Figure 49. A Kruskal-Wallis test revealed that simulator sickness was induced as a result of VE exposure (collapsed across SCALE), ($X^2(2) = 23.07$; $p < 0.001$). Nonparametric Scheffe post-hoc tests indicated that POST1 sickness reports (those collected immediately after VE exposure) were significantly higher than PRE sickness reports ($S_2 = 4.73$; $p < 0.001$). POST2 reports (collected 20 minutes post-exposure) were also higher than PRE reports ($S_2 = 3.14$; $p < 0.01$), indicating that sickness aftereffects, though reduced, were still present after 20 minutes.

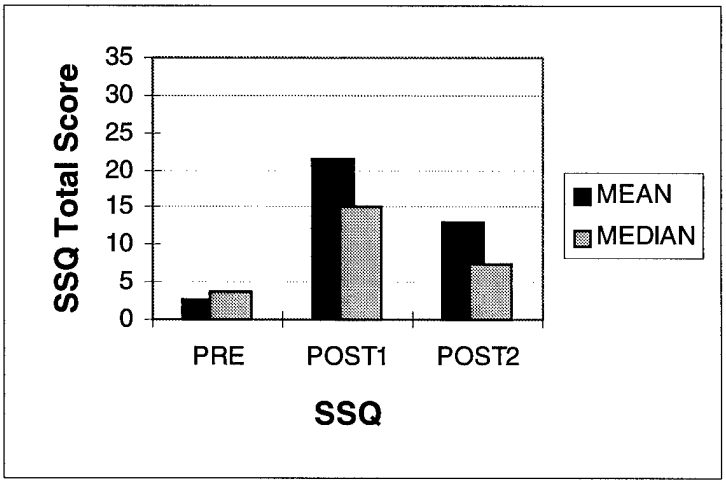

Figure 49: SSQ Total Score by Time (Image Scale Exp)

Mean and median POST1 SSQ total scores by SCALE are presented in Figure 50. A Kruskal-Wallis test indicated a significant effect of SCALE ($X^2(2) = 5.99$; $p < 0.05$). Nonparametric Scheffe post-hoc tests indicated that sickness reporting was less in the NEU condition than in the altered scale conditions (MIN & MAG) ($S_2 = 2.45$; $p < 0.05$). There was no statistical difference between the MIN and the MAG condition.

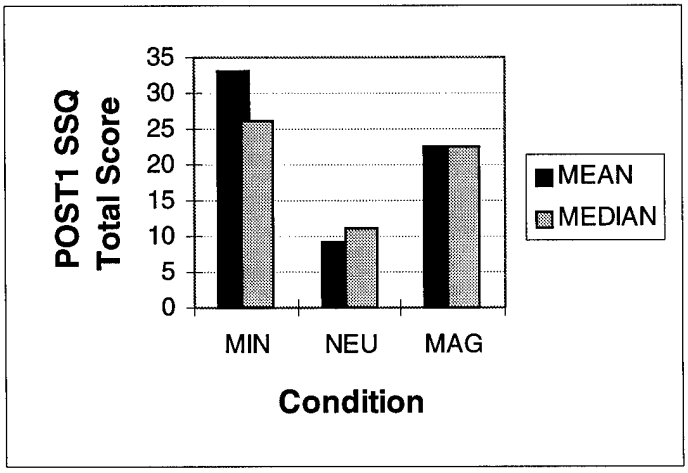

Figure 50: POST1 SSQ Total Score by SCALE

Mean and median POST2 SSQ total scores by SCALE are presented in Figure 51. A Kruskal-Wallis test was not significant but indicated a slight trend towards an effect of SCALE ($X^2(2) = 4.29$; $p < 0.12$).

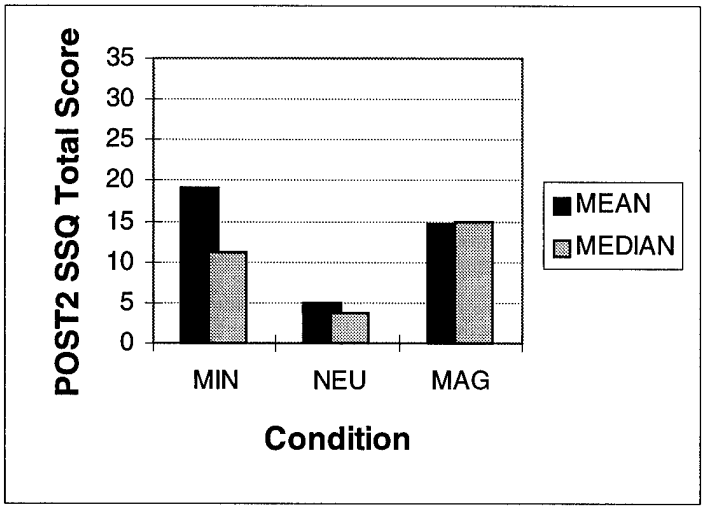

Figure 51: POST2 SSQ Total Score by SCALE

Figure 52 presents POST1 SSQ data across sessions. A Kruskal-Wallis was not significant (X^2 (2) = 3.04; $p > 0.22$), indicating that subject's reports of sickness were not effected by prior exposures. POST2 SSQ data were also not significantly different across sessions.

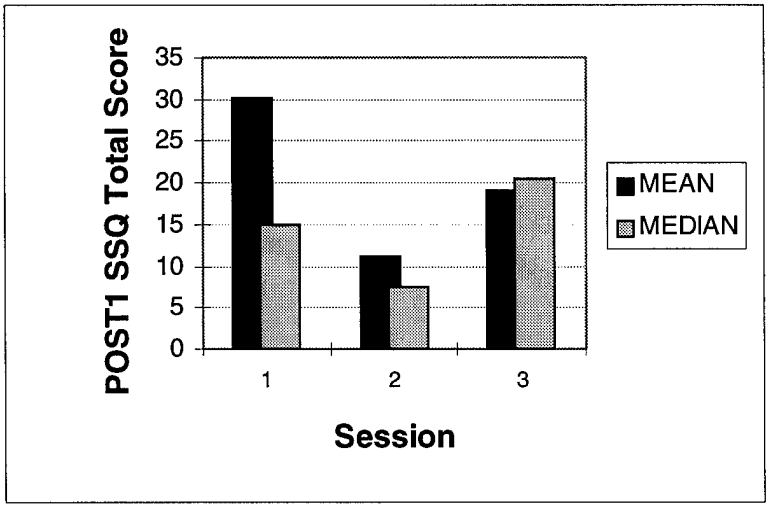

Figure 52: POST1 SSQ Total Score by Session (Image Scale Exp)

Though mean SSQ scores were lower for males than females, a Mann-Whitney nonparametric test indicated that there was no significant effect of Gender on either POST1 SSQ scores ($U = 88.5$; $p > 0.90$) or POST2 SSQ scores ($U = 91$; $p = 1.0$).

5.7.3.2 Sickness Reports During Exposure

Reported sickness during the exposure were influenced by SCALE ($X^2 = 9.48$; $p < 0.009$) and a nonparametric Scheffe revealed that reports in the NEU condition were lower than in the deviated conditions (MIN and MAG) ($S_2 = 3.07$; $p < 0.01$). Figure 53 presents average reports of sickness during exposure by SCALE. In addition, there was a trend for sickness to increase with exposure time ($X^2 = 4.85$; $p < 0.09$) (Figure 54). There was no effect of Gender on sickness during ($U = 65$; $p > 0.22$).

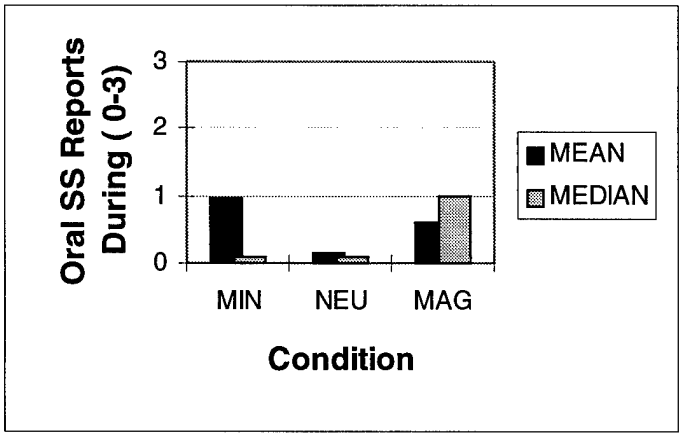

Figure 53: Sickness During Rating by SCALE

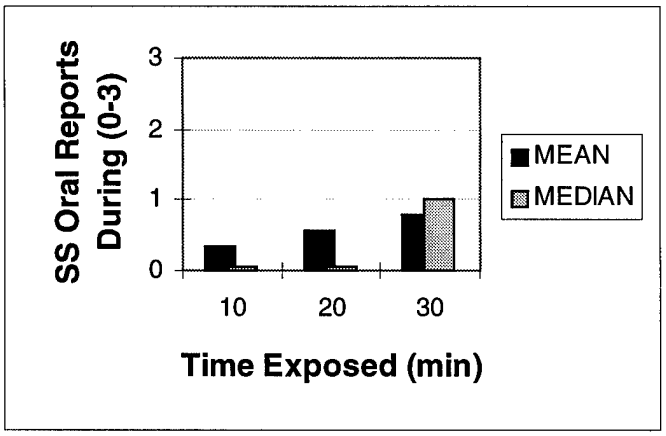

Figure 54: Sickness During Rating by Time Exposed
(Image Scale Exp)

5.7.4 Head Position Analyses

A total of 68 of 81 potential head position epochs were collected during the experiment (nine subjects, three sessions each, with three epochs per session). Each

head position epoch (80 s) was analyzed using LABVIEW software's auto-power amplitude spectral analysis program to obtain a range of frequencies over which most head movements occurred and the associated power (deg RMS) at those frequencies. A typical example of an epoch's autopower spectrum is shown in Figure 55.

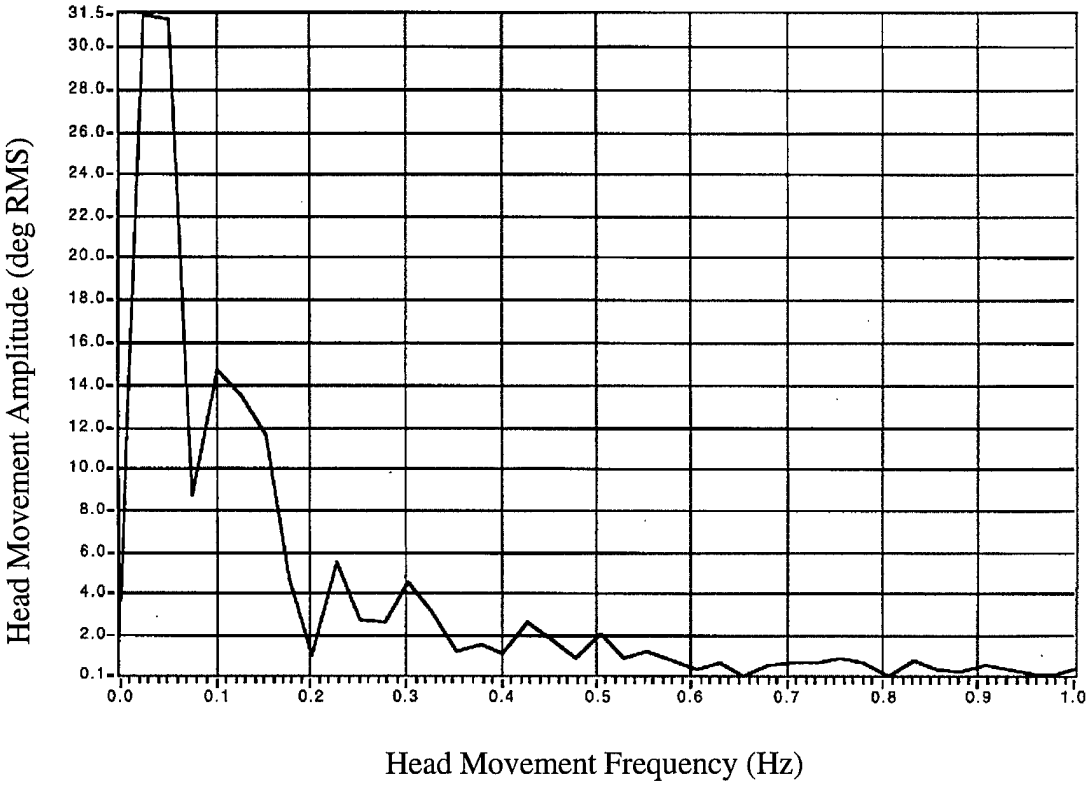

Figure 55: Typical Autopower Spectral Data
from Head Position (Image Scale Exp)

Four metrics were derived from each epoch: peak amplitude, frequency at which the peak amplitude occurred, frequency at which the power dropped below 10 deg RMS, and frequency at which the power dropped below 5 deg RMS. The last two metrics were rough estimates of bandwidth. Table 7 summarizes data across all conditions.

Table 7: Head Movement Summary Data (Image Scale Exp)

	peak amplitude (deg RMS)	**peak freq** (Hz)	**BW** (10 deg RMS)	**BW** (5 deg RMS)
Mean	*38.26*	*0.06*	*0.25*	*0.43*
SD	*12.01*	*0.04*	*0.12*	*0.18*
Median	*35.80*	*0.05*	*0.23*	*0.39*

Table 8 compares the head movement data for each scale condition. There does not appear to be any meaningful difference in head movements across scale. Table 9 presents head movement data collapsed across scale but divided by time of recording (beginning, middle, or end of a VE exposure). Head movements did not seem to change appreciably from the beginning to end of the session. The head data were also no different between sessions.

Table 8: Head Movement Data by SCALE

	peak amplitude (deg RMS)	**peak frequency** (Hz)	**BW** (10 deg RMS)	**BW** (5 deg RMS)
MIN				
Mean	*37.78*	*0.06*	*0.24*	*0.43*
SD	*10.33*	*0.04*	*0.11*	*0.18*
Median	*36.35*	*0.07*	*0.21*	*0.41*
NEU				
Mean	*36.31*	*0.07*	*0.24*	*0.40*
SD	*11.29*	*0.05*	*0.08*	*0.09*
Median	*33.30*	*0.05*	*0.23*	*0.41*
MAG				
Mean	*38.86*	*0.05*	*0.26*	*0.42*
SD	*9.55*	*0.03*	*0.22*	*0.29*
Median	*37.75*	*0.05*	*0.21*	*0.33*

Table 9: Head Movement Data by Exposure Time
(Image Scale Exp)

	peak amplitude (deg RMS)	peak frequency (Hz)	BW (10 deg RMS)	BW (5 deg RMS)
Beginning				
Mean	*37.29*	*0.06*	*0.28*	*0.47*
SD	*11.68*	*0.03*	*0.18*	*0.24*
Median	*33.70*	*0.05*	*0.25*	*0.41*
Middle				
Mean	*39.38*	*0.06*	*0.22*	*0.41*
SD	*10.30*	*0.04*	*0.07*	*0.12*
Median	*39.75*	*0.06*	*0.21*	*0.41*
End				
Mean	*38.38*	*0.06*	*0.25*	*0.42*
SD	*13.22*	*0.05*	*0.10*	*0.16*
Median	*35.55*	*0.06*	*0.23*	*0.41*

5.7.5 Gain Adaptation / Sickness Relationship

Correlation tests were performed between percent VOR gain adaptation and each of three sickness variables: final sickness during rating (at 30 minutes), the POST1 SSQ total score, and POST2 SSQ total score. A total of 27 pairs of data were compared (nine subjects with three sessions each) in each correlation. Due to the ordinal nature of the sickness data, Spearman's rho statistic was chosen over the Pearson statistic (which requires interval data). All correlations were examined using two-tailed tests.

The results of all correlation pairings indicate only a weak association between percent VOR gain adaptation and sickness reports. With POST1 SSQ, $r = +0.283$ ($p < 0.15$), with POST2 SSQ, $r = +0.24$ ($p < 0.25$), and with the third sickness during rating, $r = +0.282$ ($p < 0.15$). Figure 56 shows the scatterplot of percent gain adaptation and

final sickness rating during exposure. Note the slight but noticeable positive relationship between these variables. The other two scatterplots also did not reveal any nonlinear relationships.

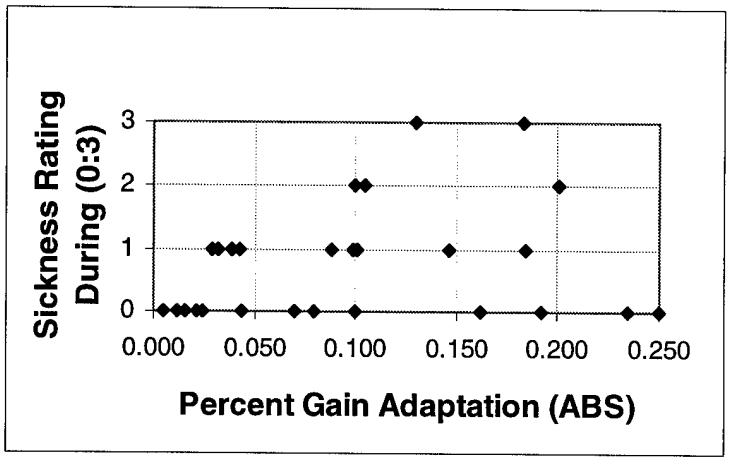

Figure 56: Scatterplot VOR Adaptation/Sickness During

5.7.6 VOR Gain Re-adaptation

Gain re-adaptation data are presented separately because not all cells in the PRE-POST analysis contained re-adaptation information and because the question of re-adaptation time-course is only relevant after establishing and detailing the nature of any occurring adaptation. Incomplete data were a result of subjects exiting early due to sickness and cells that had two unacceptable trials at a particular frequency. A total of nine additional data lines were removed from the analysis, leaving 70 analyzable data lines.

Figure 57 shows all VOR gain data (PRE, POST, and AFT10: after 10 minutes) for each SCALE. In the deviated scale conditions, VOR re-adaptation was incomplete after

10 minutes of re-exposure to the real world. Only 20% of the gain change dissipated in the MIN condition and only 50% of the gain change in the MAG condition. The PRE and AFT10 gain data were almost identical in the NEU condition.

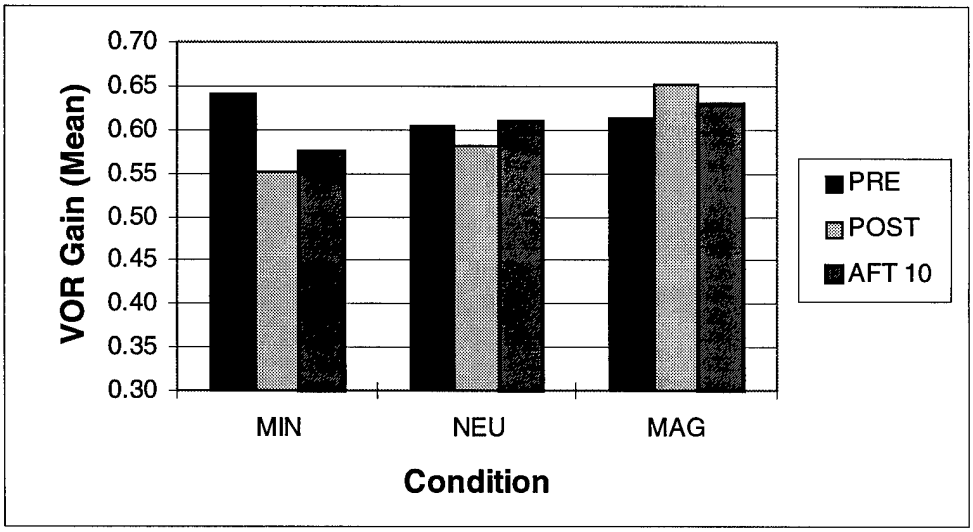

Figure 57: VOR Gain Re-adaptation by SCALE

5.7.7 Phase Adaptation

Phase data were also collected and analyzed with the results shown in Figure 58, Table 10, and Table 11. A paired t-test revealed that POST phase showed slightly more lag (1.6 deg) than PRE phase, when collapsed across conditions and frequencies ($t = 2.37$; $p > 0.03$). Much of this phase adaptation dissipated within 10 minutes post-exposure. The most substantial phase changes occurred in the NEU condition. Also note that the phase change at 0.2 Hz (2.6 deg phase lag) was 80% of the required change at that frequency given the minimum system time delay used in this experiment (48 ms).

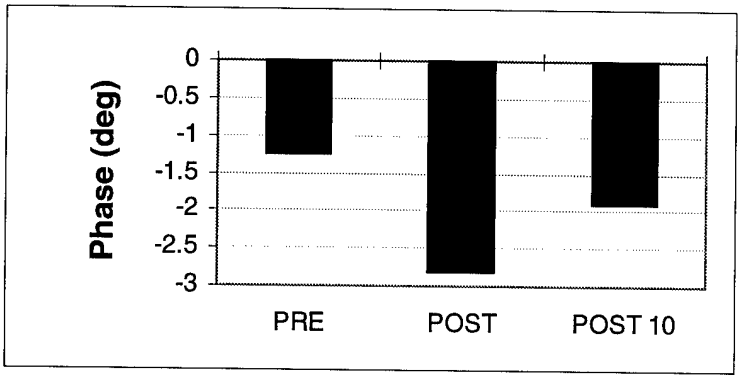

Figure 58: Mean VOR Phase Lag by Time of Test (Image Scale Exp)

Table 10: Average Phase (deg) by SCALE

Condition	Pre-phase	Post-phase	Difference (post-pre)	Post-phase 10min. after	Difference (post10-pre)
MIN	-2.2	-3.3	(-1.1)	-1.7	(0.5)
NEU	-0.9	-4.0	(-3.1)	-2.5	(-1.6)
MAG	-0.6	-1.1	(-0.5)	-1.5	(-0.9)
TOTAL	-1.3	-2.8	(-1.5)	-1.9	(-0.6)

Table 11: Average Phase (deg) by FREQ (Image Scale Exp)

Freq. (Hz)	Pre-phase	Post-phase	Difference (post-pre)	Post-phase 10min. after	Difference (post10-pre)
0.2	+1.0	-1.6	(-2.6)	-0.9	(-1.9)
0.4	- 1.3	-1.9	(-0.6)	-1.2	(0.1)
0.8	- 3.7	-5.3	(-1.6)	-4.2	(-0.5)

5.8 DISCUSSION

This section first addresses the findings regarding the VOR including VVOR, VOR adaptation, and re-adaptation data. Simulator sickness issues are then discussed, followed by the head movement information that was obtained. Lastly, the weak correlation between VOR gain adaptation and simulator sickness is considered.

5.8.1 VVOR DATA

VVOR gain data indicate that the expected VOR gain direction demand existed in each visual condition. Relative to the real world condition, eye movement amplitude increased in the MAG condition, decreased in the MIN condition, and was essentially unchanged in the NEU condition. This confirms a fundamental premise that magnification and minification effects can be simulated in virtual interfaces.

VVOR gain did not fully match the stimulus demand in the MAG condition however. This finding has also occurred in previous research (Demer, et al., 1987), though an explanation remains elusive. One obvious possibility, that the visual target may have moved outside of the DFOV during the VVOR test, did not occur in this experiment and I would expect that most experimenters would also prevent this error from occurring.

Another possible explanation involves a differential ability to track virtual targets across image scale conditions. The VVOR task in the MIN and MAG condition required a continual sinusoidal displacement of gaze (eye in space) in order to perfectly

track the virtual target[65]. Given that the amplitude of the gaze stimulus varied with SCALE and that head movements were identical across all conditions, differing gaze requirements brought about by the different scale factors must be accomplished solely through altered eye movements. Eye movements have vestibular (VOR) and visual (OKN, smooth pursuit, saccadic) components, though it is reasonable to assume that during these initial VVOR tests the visual components provided the majority of the additional compensation in the MIN and MAG conditions. Pursuit movements likely dominate the compensation due to the nature of the task. Failures in smooth pursuit need to be corrected for by saccadic movements towards the target. For instance, a lag in tracking (which would most likely occur in the MAG condition) would result in corrective saccades to 'catch up' to the target. Since these saccades were eliminated from the eye velocity data automatically[66] in this experiment, the remaining slow phase eye movement velocity was reduced below that required. Therefore, incomplete compensation for gain demand occurred and this effect was more pronounced in the upward direction (i.e., with increasing optic flow).

The VVOR phase data support this interpretation. In the NEU condition, the obtained phase delay was as predicted by the minimum time delay in the experiment (at 48 ms time delay, the theorized lag at 0.8 Hz is 13.8 deg and the actual phase lag of eye movements averaged 14.0 deg). However, there was less phase lag in the MIN condition and more phase lag in the MAG condition, even though WARP TV responds a bit more quickly in the MAG condition than the MIN condition (by approximately 5-8 ms). Previous research on smooth pursuit explains this outcome (Matin, 1986). Pursuit movements often begin to lag appreciably behind the stimulus at around 40

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[65] This was less of an issue in the NEU condition, where the virtual target generally appeared to be space-stable (notwithstanding the effects of the minimum time delay). See Section 3.1.4 for a detailed discussion.

[66] By MAP automated analysis. See Section 5.6.1.

deg/s. Though the VVOR tests were performed at approximately 30 deg/s, in the MAG condition the optic flow moved at 60 deg/s due to the 2X magnification. At such speeds, there is often an inability to perfectly track the target combined with an increasing phase lag, both of which occurred in this experiment. Therefore, this increased phase delay in the MAG condition indicates difficulty in maintaining perfect pursuit. In the MIN condition, however, it is easier to maintain fixation because the target velocity is reduced in response to the head motion to approximately 15 deg/s. It is possible that if subjects were given a period of practice on the tracking task, the resulting gain and phase would approach calculated values (due to the activation of prediction mechanisms).

5.8.2 VOR Adaptation in VEs

This experiment demonstrated that VOR gain adaptation (and to a small extent phase adaptation) can occur during natural interaction with head-coupled virtual environments, confirming Hypothesis 1. This is quite an achievement for the oculomotor system given the method and duration of exposure. Subjects moved their heads in a natural, unrestricted fashion as they performed visual search activities for 30 minutes. At no time were the subjects instructed to move their heads more quickly or at specific frequencies; in fact the power spectral distributions of head movements clearly indicate that the largest concentration of head movement power was at or below 0.2 Hz (in most cases below 0.1 Hz). However, VOR tests conducted at only three pre-defined frequencies captured the existence of VOR gain adaptation in the MIN and MAG conditions. Active head movements during the exposure period may have supported VOR gain adaptation (Demer, Oas, & Baloh, 1993).

VOR gain changes were not fully compensatory for stimulus demand. This was expected given previous research (Collewijn, et al., 1983). The adapting mechanism may have limits that have been ecologically determined over the millennia, and seldom

have there been such artificial visual-vestibular demands occurring in nature. Smaller demands, which are more likely to appear inadvertently in VEs, are much more likely to be fully compensated for in a short period of time (Collewijn, et al., 1983).

VOR gain adaptation direction and magnitude were clearly influenced by changing GFOV. This is an important finding because virtual interface systems are often designed with little or no regard to the proper setting of this variable. There are potentially many virtual interface systems that are inadvertently stimulating VOR gain adaptation through DFOV/GFOVs that do not equal 1.0. To reduce the potential for unwanted oculomotor adaptation, designers should attempt to equate GFOV with the DFOV used, as in the NEU condition where no significant adaptation took place. If that is not possible, it is better to error on the side of image magnification, as these results indicate that gain adaptation may be less modifiable in the upward direction.

Why was VOR gain more readily reduced than increased? There are five potential explanations: 1) VOR adaptation processes have direction asymmetries, 2) influence of spectacles to correct for myopia, 3) influence of the system time delay, 4) perceptual effects of visual displays, and 5) subject fatigue. These explanations are clarified below.

Past VOR adaptation studies have indicated that VOR adaptation processes may be biased towards gain reduction (Bello, Paige, & Highstein, 1991). This could be due to evolutionary conditioning, though it seems more intuitive that VOR gain increases would be likely to be demanded naturally due to the effects of disease, trauma, or age. Second, myopics who wear corrective spectacles are as a result pre-conditioned to reduce their VOR gain[67] (Cannon, et al., 1985). One diopter of visual correction in spectacle optics results in approximately a 2-3% VOR gain change demand (Cannon et

[67] This is not true for those who only wear contacts, however, because the line of sight always moves with the eye in this case.

al., 1985; Collewijn, et al., 1983). Since the two subjects who wore spectacles for visual correction were both myopic, there may have been an increased propensity, based upon past experience, for those subjects to reduce VOR gain. The third potential explanation, further discussed in Section 6.8, is that the minimum time delay inherent in the virtual interface produced a gain reduction stimulus across conditions. This time delay may have further reduced the gain in the MIN while inhibiting gain increases in the MAG condition. Fourth, previous research suggests there is often a perceived minimization of a spatial environment when viewed through a HMD (Roscoe, 1991). Roscoe argued that this minimization of the virtual image is due in part to 'positive miss-accommodation' of the eyes, i.e., the eyes do not focus at infinity when viewing collimated virtual images, but rather lapse towards dark focus (which is around an arm's length). This perceptual minification could contribute to the resulting VOR gain adaptation downward bias through cognitive inputs to adaptation processes. Lastly, subject fatigue may have played a part in driving VOR gain downward over the three conditions (Demer, et al., 1987).

As a general note, it is not image scale per se that stimulated VOR gain adaptation but relative velocity of optic flow in response to head movements in concert with other hypothesized factors (Shelhamer, et al., 1994). As discussed earlier, changing image scale is simply one direct way to modulate optic flow velocity with regard to head movements. Another way would be to decrease or increase the gain on head tracker rotations. Reducing rotational gain on a head tracking sensor would result in less OKN in the visual response to head motion (a MIN condition) while increasing the gain would result in increasing OKN (a MAG condition). In either case, it is the amount of retinal slip detected during head movements that contributes to the error signal. Thus, additional design guidance for the reduction of unwanted oculomotor adaptation must include the importance of accurately calibrating the head tracker's recording of rotational head motion.

When collapsed across SCALE, VOR gain adaptation did not appear to systematically change over the three sessions. This implies that the session separation was adequate to prevent long term sensory-motor learning between sessions. It would be interesting to examine the effects of more tightly spaced sessions, as some research indicates that VOR gain adaptation carryover effects can occur (Gonshor & Melvill Jones, 1976a). Multiple exposures within a limited period of time could result in the long term storage of gain change parameters for that stimulus (Shelhamer, et al., 1992). This has been termed 'adaptation set' by Parker (personal communication, 1997).

Lastly, a statistically significant phase lag adaptation occurred in this experiment. The phase lag increased by 1.6 deg as a result of the 30 minute exposure to the VE (collapsed across conditions). The most significant phase lag adaptation (2.6 deg) occurred at 0.2 Hz, with less adaptation occurring at the higher test frequencies. This could be interpreted as minor support for frequency specificity in phase adaptation, especially given that the phase lag change at 0.2 Hz was nearly fully compensatory for phase change demand (3.2 deg at 0.2 Hz). However, given the small adaptation magnitudes involved, it seems a somewhat tenuous conclusion.

5.8.3 Frequency-Specific vs. Generalized VOR Gain Adaptation

An interesting finding resulted from the comparison of VOR gain change with test frequency. There has been some debate in the VOR literature on the issue of frequency specificity and VOR adaptation. Many researchers have argued that VOR adaptation is frequency specific (i.e., tuned to the peak frequency experienced during the stimulus exposure period) while others have found that it may be more generalized across head movement frequencies. These data support generalized adaptation across frequencies, given that VOR gain changes did not vary across test frequency while the head movements performed by subjects in the VE had peak power below 0.2 Hz.

These results suggest the importance of active, unrestricted head movements in generalized adaptation of VOR gain. The concept of frequency specificity was developed using passive head oscillations at one set frequency (Lisberger, et al., 1983). It is quite possible that more natural head movements provide an effective stimulus for the adaptation processes to extrapolate gain compensation beyond dominant frequencies of motion during exposure. This issue is further discussed in Chapters 6 and 9.

5.8.4 VOR Gain Re-adaptation

The results of this experiment suggest that VOR gain re-adaptation following VE exposure is incomplete after 10 minutes. Though gain values recovered 20% to 50% of changes within the 10 minute period, the gains were still off pre-exposure levels. This finding was not expected, given that adaptation processes often follow a decaying exponential with the majority of adaptation (or in this case, re-adaptation) occurring quickly (Collewijn, et al., 1983; Welch, 1986). This, combined with the magnitude of gain changes experienced in this experiment and the hypothesized existence of adaptation sets, all point to more complete re-adaptation after 10 minutes. Some explanations for the obtained results follow.

One explanation involves the influence of subject fatigue or lack of alertness. In the MAG condition, 50% of the gain change was recovered in 10 minutes whereas only 20% percent of the gain change was recovered in the MIN condition over the same time period. Given the observation by experimenters that subjects quickly grew tired of the post-exposure controlled eye-head movement task and often showed signs of boredom during this recovery period, perhaps this contributed to the directional recovery asymmetry. Since the gain levels after 10 minutes recovery from the MAG condition were within 3% of pre-exposure levels, the remaining difference could very well be within the signal variance of the reflex.

A second explanation involves a lack of interaction with the real world. Since subjects were still strapped in the chair and looking through the HMD during this period, full interaction with the real world was not possible by the subject. The remaining contextual information provided by the chair and HMD may have contributed towards either a decrease in rate of re-adaptation or perhaps a minor relapse towards the post-exposure gain levels once the lights were turned off. This relapse is not unheard of in the literature (Gauthier & Robinson, 1975; Shelhamer, et al., 1992).

5.8.5 Simulator Sickness

The overall findings regarding simulator sickness are discussed, followed by the influence of uncontrolled factors, re-adaptation, and miscellaneous issues.

5.8.5.1 Overall Findings

This experiment induced significant levels of simulator sickness as a result of a 30 minute exposure to the virtual interface. However, the magnitude of simulator sickness was related to the image scale of the VE such that the NEU condition was approximately half as provocative as either the MIN or MAG condition. The data were consistent whether the metric examined was sickness reports during, SSQ total score immediately post-exposure, or SSQ total score 20 minutes post-exposure. This finding also reinforced what the experimenters had informally noticed throughout the experiment: the NEU condition least adversely effected the health of the subjects.

These results support the sensory rearrangement theory of motion sickness which argues that sensory rearrangements provoke the occurrence of simulator sickness. Both the MAG and MIN conditions had visual-vestibular sensory rearrangements involving visual motion velocity in response to head movement velocity, whereas the NEU condition did not (save the minimum time delay in the system which was shared across all conditions).

Other theories of motion sickness were not supported by these data. The emerging subjective vertical theory (de Graf, Bles, & Groen, 1997; de Graf & Bos, 1997; Bles, de Graf, Bos, & Groen, 1997) is a modification of the sensory rearrangement theory. It argues that only those sensory rearrangements that stimulate otolith responses are provocative. However, this experiment involved very few, small-amplitude head movements in pitch and none in roll, yet significant simulator sickness was elicited. Furthermore, the subjective vertical theory cannot explain the differential sickness experienced across conditions, given that the tasking across exposures were identical.

The proponents of an optic flow model of simulator sickness will also be disappointed by the outcome of this experiment. These researchers hypothesize that increased optic flow is a prime factor in the generation of sickness symptoms. However, both the MIN and MAG conditions resulted in similar amounts of sickness even though the optic flow differed by a factor of four between these conditions.

Lastly, though this experiment does not specifically address the postural control theory of motion sickness, it is evident that although the subjects were firmly strapped into a chair using a five-point harness and Velcro straps for the feet, simulator sickness symptoms were still elicited.

5.8.5.2. Other Influencing Factors

It is important to note that significant simulator sickness *was* reported in the NEU condition, only less so (by a factor of two) than in the MIN and MAG condition. This indicates that there are other provocative factors of motion sickness that were present but not controlled for in this experiment[68]. A prime suspect, system time delay, is investigated in Chapter 6. Other potential contributors include accommodation-

[68] This is an obvious statement given the number of factors thought to influence motion and simulator sickness.

vergence mismatch, reduced resolution, display flicker, optical distortions, and form and fit of the HMD.

In fact, those who totally discount the potential contribution of poor HMD fit on SSQ ratings have probably been out of an HMD for too long. Current technology HMDs can be heavy, hot and have pressure points where they contact the surface of the head. Though the specific HMD used in these experiments was the lightest and most comfortable on the market, there was likely a contribution of poor HMD fit on SSQ results (most likely in the 'headache' category). However, this contribution was stable across sessions and does not account for the obtained variance in sickness reporting.

5.8.5.3 Simulator Sickness Recovery

Simulator sickness was still present in the MIN and MAG condition after 20 minutes post-exposure, though its magnitude was decreased. Evidently the time constant of re-adaptation is not much shorter than the time constant of adaptation, at least for this virtual interface-tasking combination. The sickness stemming from the NEU condition, however, was completely absent after 20 minutes. This further indicates that GFOV setting is an important modulating factor in the occurrence of simulator sickness.

5.8.5.4 Miscellaneous Issues

Two more issues merit discussion. First, simulator sickness increased throughout the exposure period. This corresponds with other research, indicating a build-up of effect over time (Regan, 1995). This also correlates with ataxic effects experienced in the postural ataxia studies (Chapter 4) which also increased over exposure time. Though data were not collected on VOR adaptation onset time-course, such data would allow for time-course comparisons between these two processes. Second, there was no effect of gender on simulator sickness incidence. This is most readily explained by the

small sample size involved (six males and five females), as there is a preponderance of data in the literature supporting the claim that females are more susceptible than males. Even in this study, the only subject to discontinue due to sickness was a female.

5.8.6 Head Movement Analyses

The most striking aspect of the head movement data was the lack of variance involved. Subjects did not appreciably change head movement frequencies or amplitudes with the changing scale conditions. Evidently, the task involved was the major determinant of head motion and it remained consistent across conditions. Subjects were required to search for a target, fixate on it, then return to the starting head position. There was no time pressure on the subject to find objects as quickly as possible so subjects often moved at a leisurely pace. Also, it is important to keep in mind that changing the image scale does not change the angular movement required to center each target in the display[69]. Therefore, subjects were required to make similar head movements during the exposure periods regardless of the scale condition, which may also account for the lack of variance obtained.

However, a common finding in simulator and motion sickness is that head movements are often curtailed when subjects begin to experience simulator sickness. This would predict a reduced set of head movements in the MIN and MAG condition versus the NEU condition, which did not occur. In addition, there was no effect of exposure time on head movement amplitude or frequency, which does not correspond with the increase in simulators sickness reported with time exposed. There are two

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[69] Those with doubts only need grab a camera with a highly adjustable lens. If, after positioning the camera straight ahead you center an eccentric target in the viewfinder, the camera will cover the same angular extent whether the camera is in telephoto or wide-angle mode. However, the apparent position of targets seen through *but not centered in* the viewfinder will vary with magnification factor. To test this, center a target in the viewfinder, then fixate on an eccentric target. As you progress from wide-angle to telephoto lens settings, the eccentric target will appear to move inward or outward but the centered object will remain stationary.

possible explanations for this discrepancy. The most likely reason is that the power of head motion was concentrated at such low frequencies due to task requirements (discussed further below) that a floor effect existed. Subjects may not have been able to move their heads more slowly and still complete the search tasks in a reasonable length of time. Another explanation points to the lack of control surrounding the collection of these data. There was no rigorous attempt to standardize the collection periods across subjects other than to test each at the beginning, middle, and end of the 30 min exposure period. Subjects were not given the exact same items to look for in the exact same order, for instance. This potentially allowed extra variance in the data that could mask small effects, though the 80 second duration of data collection probably minimized these small differences between subjects.

These data indicate surprisingly low peak head movement frequencies. As stated above, this can partially be explained by a lack of a time constraint in task performance. In addition, during each 80 second epoch, there were several periods of time where the subject did not move his/her head at all (i.e., while awaiting a new target to search for or for confirmation that a target was correct). This stationary head data served to pull the peak power frequency towards 0 Hz. Finally, virtual interfaces have been shown to decrease head movements of VR participants (Pausch, et al., 1996).

5.8.7 VOR - Sickness Relationship

At first glance there appeared to be a rather significant relationship between VOR gain adaptation and simulator sickness. VOR gained changed significantly in the MIN and MAG conditions but not in the NEU condition. Simulator sickness was significantly worse in the MAG and MIN conditions versus the NEU condition. However, the correlations between VOR gain change and simulator sickness metrics were never larger than 0.30. This implies that maximally only 9% of the variance in the simulator sickness data could be explained by VOR gain changes. This low correlation

confirms some previous research (Bouyer & Watt, 1996; Watt, 1987) but it refutes the findings of a relationship between VOR adaptation and sickness found by others (Gordon et al., 1996). It would seem that Gordon's work may be a special case; it involves real motion (on seafaring ships) and vestibular habituation vs. VOR adaptation per se. This issue is explored further in the next experiment.

CHAPTER 6: TIME DELAY EXPERIMENT

6.1 OBJECTIVES AND HYPOTHESES

System time delays have been implicated as a significant source of visual-vestibular stimulus rearrangements in virtual interfaces. These time delays may drive phase adaptations of the VOR, though fixed time delays result in a variable phase adaptation demand dependent on head movement frequency (see Section 3.1.3.1). If head movements are restricted to a narrow range of frequencies, however, time latencies may appear as a relatively fixed phase demand that could theoretically be adapted to. Time delays are also often implicated in the literature as a provocative stimulus for simulator sickness. The following experiment explored the potential effects of time delays on simulator sickness and VOR adaptation and collected preliminary data on re-adaptation time-course.

As in the Image Scale Experiment, this experiment did not employ a traditional VOR adaptation protocol. Traditional VOR adaptation research often involves passive motion of the head/body at a specific frequency of oscillation, a head position that is fixed with respect to the rotating chair, meaningless visual stimuli (often random dots or vertical stripes) and no meaningful tasks to perform during the exposure period. The current experiment focused on exposure situations that are more likely to occur in actual VE applications. It incorporated active, unrestricted, head movements (across several frequencies) while the subject performed a meaningful task in a virtual environment. In other words, the question this dissertation asked was whether system time delays caused adaptations to occur as a result of exposure to realistic VE user scenarios. The answer to this question would serve to extend the knowledge base regarding VOR adaptive capabilities while also providing potential design guidelines for virtual interface design.

This experiment addressed main objectives 1, 2, 3, and 5 (see Section 1.8). The specific goals of this experiment were to ascertain if: 1) VOR adaptation could occur in VEs that involve natural head-coupled interaction with meaningful visual scenes over a short exposure period, 2) this adaptation magnitude could be modulated by varying system time delays, 3) any occurring VOR adaptation was frequency specific or if it generalized across the tested frequencies, 4) simulator sickness reports were affected by changes in system time delays, and 5) any occurring VOR adaptation covaried with sickness reports. In addition, data were collected on post-exposure re-adaptation time course for any occurring VOR gain/phase adaptation and sickness incidence.

It was hypothesized that increasing time delays would: 1) increase phase lag adaptation, 2) not effect VOR gain, and 3) increase reports of simulator sickness. Previous research suggests that VOR phase adaptation processes may be less sensitive than gain adaptation processes. Therefore, it was expected that any phase adaptation achieved would be far from fully compensatory for phase demand. This was especially likely given that head movement frequencies were not constrained in this experiment, reducing the amount of time exposed to each specific phase demand.

Given that no default threshold for VOR phase change 'meaningfulness' exists (i.e., when a VOR phase change becomes operationally important), an initial conjecture regarding this threshold was attempted. Since the fovea subtends only 2 deg of visual angle in the central visual field, any change in phase over 2 deg is hypothesized to be a potentially meaningful change, possibly resulting in retinal image slip. Since all phase demands in this experiment were greater than 3 deg for head movements greater than 0.05 to 0.1 Hz, any occurring phase adaptation above 2 deg was speculated to be 'meaningful'.

The equations of Chapter 3 do not predict a VOR gain change as a result of time delay, thus none was hypothesized. Lastly, though many researchers have implicated

system time delays as being a major factor effecting simulator sickness, two previous studies which addressed the issue found no statistically significant effect of increasing delay (Frank, et al., 1988; Uliano, et al., 1986). Nonetheless, sensory rearrangement theory predicts an effect of time delay on simulator sickness.

In addition, it was hypothesized that VOR gain and phase adaptation would be more robust at the lower frequencies, as several experiments on VOR adaptation have shown frequency specificity and subjects tend to reduce head movements while immersed in VEs. However, this hypothesis was tenuous given that the opposite result was found in the Image Scale Experiment. Lastly, it was hypothesized that there would be a moderate correlation between sickness reports and VOR adaptation.

6.2 SUBJECTS

A total of 11 adult subjects (7 males/4 females, mean age 27.4, age range 23 to 36) volunteered to participate in this experiment. None of these subjects participated in any other experiment in this dissertation. All subjects reported to be in good health with no history of epilepsy or vestibular medical problems (with the exception of one subject, who had been treated several times for migraine headaches)[70]. In addition, subjects were tested for normal or corrected visual acuity of 40/20 or better. Seven subjects wore glasses or contacts for corrected visual acuity and five of these reported some level of diagnosed astigmatism. Only contacts were acceptable for vision correction during the experiment due to incompatibilities between the eye tracking system and spectacles. Nine subjects reported that they had experienced motion sickness in the past and two of these reported experiencing simulator sickness in the past. Two subjects claimed to be nonsusceptable to motion sickness, six subjects reported slight susceptibility, one reported moderate susceptibility and two subjects reported high susceptibility. At the

[70] This subject was later dropped from the analysis as discussed in Section 6.7.

onset of testing, all subjects were new to the particular virtual environment involved and had not experienced a head-coupled virtual interface in the previous 30 days (four subjects had never before been exposed to head-coupled virtual interfaces).

6.3 EXPERIMENTAL DESIGN

The overall experiment was conceptualized as a 2 x 2 x 3 within subjects design. Two levels of Time Delay (DELAY: 125 ms, 250 ms) were crossed with two levels of testing time (TESTTIME: PRE-exposure, POST-exposure) and three levels of test frequency (FREQ: 0.2, 0.4, 0.8 Hz). Additionally, VOR measurements were collected 10 minutes post-exposure on most subjects. These data, obtained on less than the full set of subjects, were analyzed separately. Each subject participated in a total of two, 1.5 hour sessions (a session per DELAY condition). Each session was separated by a minimum of 7 days to minimize carryover effects. Sessions were counterbalanced across DELAY and SUBJECT.

The dependent variables for VOR gain analysis included averaged VOR gain estimates and percent gain change. Percent gain change is a derived variable of adaptation from the formula: ((POST-PRE)/PRE)*100. VOR phase estimates were also collected and averaged.

Sickness reports were collected before, during, and after VE exposure. These dependent variables included: 1) oral reports of sickness and 2) SSQ scores. The oral reports were collected at specified times during the VE exposure (10, 20, and 30 minutes). Each report was a number from the scale of 0 to 3, as discussed in Section 5.3. SSQ data were collected pre-exposure, immediately post-exposure, and 20 minutes post-exposure.

In addition, a metric of postural stability was used to assess balance pre- and post-exposure as a safety check prior to releasing the subject. This experiment employed the Chattecx Balance Platform to obtain this metric because it provided a more sensitive measure of postural stability than that employed in the Image Scale Experiment.

6.4 APPARATUS AND EXPERIMENTAL SET-UP

The experiment was conducted in the VR Effects Laboratory, a component of the HITL on the University of Washington campus. The apparatus and experimental set-up was as described in Section 4.3. Time delays were programmed into the WARP TV delay buffer via a software setting. Each DELAY condition employed a 1.0X image scale (i.e., GFOV = DFOV, as in the NEU condition of the Image Scale Experiment). The Chattecx Balance Platform was used for postural stability measurements.

6.5 PROCEDURE

The procedure was similar to that used in the Image Scale Experiment (Section 5.5) except for the following: 1) DELAY was manipulated while image scale was fixed, 2) for every VOR and VVOR test, three trials were collected and averaged instead of two, 3) balance assessments (PRE and POST) were made using the Chattecx Balance Platform, and 4) post-exposure eye calibrations were completed during the ten minute re-adaptation window (i.e., before the ten minute post-exposure VOR test). Only these differences in procedure are discussed below.

Since system time delays were the focus in this experiment, only this variable was manipulated. Image scale was fixed to 1.0X magnification. Image resolution (320 x 240), image size, and other display factors were the same as they were in the Image Scale Experiment.

Given that VOR phase adaptation processes may be relatively sluggish, a third trial was added to each VVOR and VOR test (i.e., at each FREQ). It was expected that this third trial would in essence 'break ties' that would sometimes arise between two competing trials and would therefore provide a more robust estimate of phase and gain. However, to minimize overall testing time, the number of sinusoidal cycles per trial at 0.4 Hz was reduced from 8 to 6.

The pre- and post-exposure postural stability test utilized the Chattecx Balance System. Subjects stood in a Sharpened Romberg position (eyes open) while on this apparatus, which measures changes in CG through the use of force-sensing plates (see Section 4.3). The subject performed two trials pre-exposure and two trials post-exposure. Each trial was 10 seconds long and the average total dispersion (computed automatically) across both trials was recorded as the postural stability metric.

To potentially increase the accuracy of estimating post-exposure VOR gain values, the post-test eye calibrations were performed immediately after the initial post-exposure VOR test was completed. In the Image Scale experiment, this eye calibration was completed after the 'ten minute after' VOR tests in most cases.

All other procedures were as described in Section 5.5.

6.6 STATISTICAL ANALYSIS

The statistical analysis was similar to that used in the Image Scale Experiment, with the following exceptions.

6.6.1 VVOR AND VOR DATA

Since MacEyeball calculated VOR gain and phase estimates over four cycles (0.2 Hz), six cycles (0.4 Hz) or eight cycles (0.8 Hz) of sinusoidal oscillation using two

different methods (Fourier and Varant), the final gain and phase value recorded per trial was obtained by averaging these two estimates. These two estimates were in most cases within 1 to 2 percent of each other for gain estimates and for phase estimates were within a degree of each other. Three trials were collected at each frequency (i.e., a cell). For cells with three valid trials (107 of 117; 91%) the three estimates were averaged to get the final VOR gain and phase values for that cell. A few cells had trial values that were deemed unacceptable due to excessive noise/distortion in the eye position data (e.g., due to lack of coherence), incomplete data collection, unwanted head movements by the subject, spurious head tracker output, etc. For cells that had only two acceptable trials (10 of 117; 9%) I averaged the two acceptable trials for the final value. There was never a case where two or all three values were invalid within a cell.

In most cases (14/20), the post-exposure eye calibration value was within two percent of the pre-exposure value. However, if the post-exposure value deviated by three percent or more, the post-exposure and 10 minute post-exposure VOR gain values were adjusted appropriately. The highest deviation between pre- and post-exposure calibration values was five percent (one case).

Since the dependent variables used (VOR gain estimates, percent gain changes, phase estimates) were found to be normally distributed with equal variances, parametric statistics were used. However when these assumptions were not met, appropriate non-parametric statistics were employed.

6.6.2 Simulator Sickness Data

Almost all SS data are positively skewed (i.e., not normally distributed). This was true with the data collected in this experiment as well. In addition, the sickness reports during exposure were ordinal, not interval data. Therefore, only non-parametric tests

were used in the analyses, which in turn obviated the need for homogeneity of variance and normality testing.

As stated above, 8 of the 10 subjects participated in both sessions. The ninth subject position was filled by 2 different subjects, one for each DELAY. Because of this design change, no 'related' or 'paired' statistics were used on the sickness data.

6.6.3 Head Position Data Analysis

Head position epochs (80 s each, yaw angle only) were collected at the beginning, middle, and end of each VE exposure session. These epochs were analyzed as described in the Image Scale Experiment.

6.7 RESULTS

Eight subjects successfully completed the two 1.5 hour sessions. One female subject withdrew after the first session (125 ms delay) because she had to depart on an extended trip before the second session could be scheduled. Rather than discard her data, another subject (a female, same age) was gathered and run in the 250 ms delay condition.

One subject was removed from the analysis after it was determined that he had a history of chronic migraine which violated the subject requirement of no such medical history. In addition, this subject had an average pre-exposure VOR gain of 0.24 which was over twice as low as the next lowest subject average. Finally, this subject was hyper-susceptible to motion sickness of all types, which would misleadingly skew the results across both delay conditions. The relationship between migraine and motion sickness is well established (Harker, 1993).

The results section will, in order, address VVOR data, adaptive changes in VOR phase and gain response, changes in sickness reports, head position analyses, the

relationship between VOR changes and sickness reports, and re-adaptation data. Lastly, the balance data collected pre- and post-exposure are presented.

6.7.1 VVOR DATA

To verify that the stimuli were appropriate to stimulate VOR adaptation, average real-world VVOR was compared to average virtual VVOR. These data are shown in Table 12 and graphically in Figure 59 and Figure 60.

Table 12: VVOR Gain and Phase Summary Data
(Mean and SD) (Time Delay Exp)

Condition	Gain (SD)	Phase (SD)
Real World	*0.99 (0.07)*	*-1.1 (3.0)*
125 ms (VR)	*0.89 (0.09)*	*-34.4 (7.8)*
250 ms (VR)	*0.74 (0.10)*	*-73.6 (5.5)*

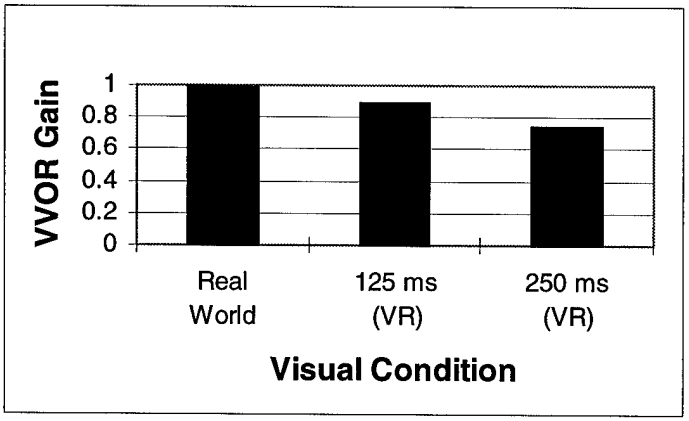

Figure 59: Mean VVOR Gain by Condition
(Time Delay Exp)

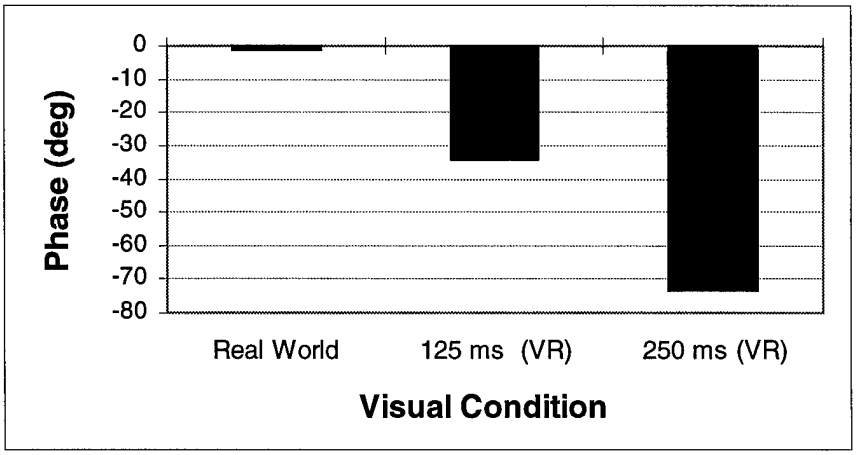

Figure 60: Mean VVOR Phase Lag by Condition
(Time Delay Exp)

VVOR data suggests that each condition provided the expected phase adaptation stimulus at 0.8 Hz. With increasing time delays, there was an increase in VOR phase lag (lag is represented by negative values). However, the data also indicated that VVOR gain dropped with increasing time delay, which was not predicted by the physics of the stimulus.

6.7.2 VOR Adaptation

A summary of VOR gain and phase change data across the two delays is shown in Table 13, Figure 61, and Figure 62. Each delay condition's statistical results is then individually presented.

Table 13: Summary Data (across DELAY)

Delay	N	Pre Gain	Post Gain	% Gain Change	# of subj w/ gain decrease	# of subj w/ no gain change	# of subj w/ gain increase	Pre-Phase (deg)	Post-Phase (deg)	Phase Change (deg)
125	9	0.633	0.585	**-7.4**	5	2	2	-0.6	-3.2	-2.6
250	9	0.605	0.546	**-9.8**	5	4	0	-2.6	-5.3	-2.7

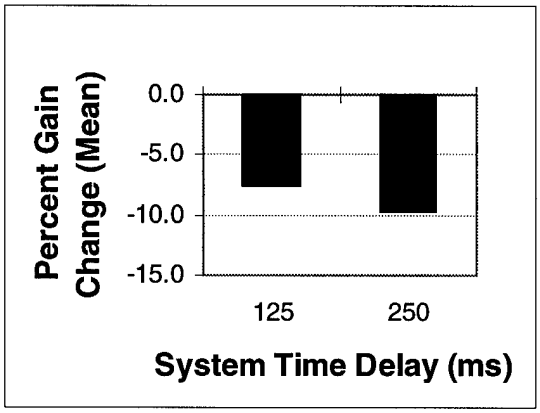

Figure 61: VOR Gain Change by DELAY

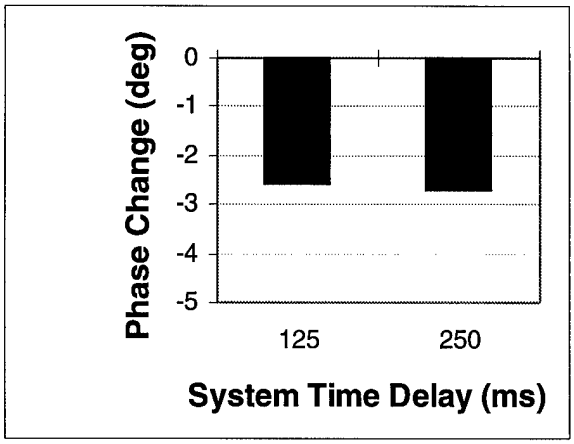

Figure 62: Mean Phase Change by DELAY

After verifying normality and homogeneity of variance, a one-way ANOVA of percent gain change by frequency (across DELAY) revealed no significant effect of frequency ($F(2, 51) = 0.41$; $p > 0.65$). Figure 63 depicts percent average VOR gain change across frequency. There was also no effect of frequency on phase change, when collapsed across DELAY (Figure 64). A one-way ANOVA was also not significant regarding the effect of SESSION on percent VOR gain adaptation, though a slight trend may exist ($F(1, 52) = 2.28$; $p < 0.15$), with percent gain change in the second session being half that obtained in the first session.

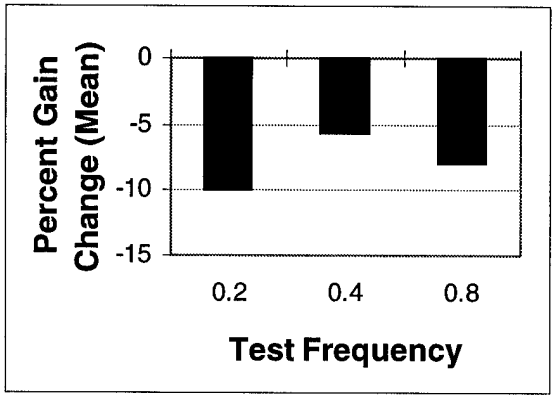

Figure 63: Percent Gain Change by Frequency (across DELAY)

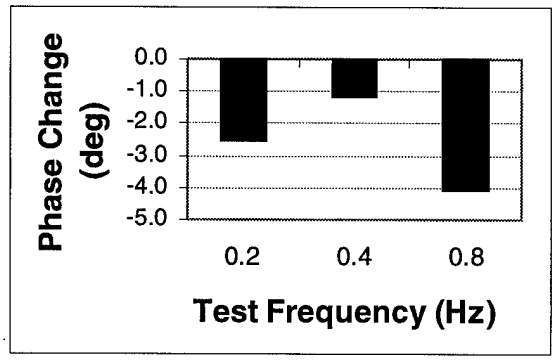

Figure 64: Mean Phase Change by Frequency (across DELAY)

6.7.2.1 125 ms Delay Condition

Summary data on VOR gain from the 125 ms delay condition are shown in Table 14. Both the PRE and POST gain data were normally distributed (Shapiro-Wilks) with equal variances (F-test) and the PRE-POST data were significantly correlated (r = 0.79;

$p > 0.0001$). A paired t-test (POST - PRE) revealed a significant reduction of gain in the POST tests ($t(26) = -2.63$; $p < 0.02$).

Table 14: VOR Gain Summary Data (125 ms Delay)

Test	N	Mean	Median	Variance	SD	SE	% Change
Pre	27	0.635	0.63	0.022	0.14	0.03	
Post	27	0.585	0.60	0.019	0.14	0.03	**-7.4**

Summary data on VOR phase from the 125 ms delay condition are shown in Table 15. Both the PRE and POST phase data were normally distributed (Shapiro-Wilks) and the PRE-POST data were significantly correlated ($r = +0.50$; $p > 0.009$). A paired t-test (POST - PRE) revealed a significant increase in phase lag in the POST tests ($t(26) = -2.50$; $p < 0.02$).

Table 15: VOR Phase Summary Data (in deg, 125 ms Delay)

Test	N	Mean	Variance	SD	SE	Phase Change
Pre	27	-0.6	13	3.6	0.71	
Post	27	-3.2	35	5.9	1.15	**-2.6**

6.7.2.2 250 ms Delay Condition

Summary data on VOR gain from the 250 ms delay condition are shown in Table 16. Both the PRE and POST gain data were normally distributed and the PRE-POST data were significantly correlated ($r = +0.77$; $p > 0.0001$). A paired t-test (POST - PRE) revealed a significant reduction of gain in the POST tests ($t(26) = -3.47$; $p < 0.005$).

Table 16: VOR Gain Summary Data (250 ms Delay)

Test	N	Mean	Median	Variance	SD	SE	% Change
Pre	27	0.605	0.59	0.017	0.13	0.03	
Post	27	0.546	0.53	0.021	0.13	0.03	-9.8

Summary data on VOR phase from the 250 ms delay condition are shown in Table 17. Only the PRE phase data were normally distributed but the difference data (POST-PRE) were normally distributed (satisfying the assumption for the paired t-test). The PRE-POST data were also significantly correlated ($r = +0.50$; $p > 0.009$). A paired t-test (POST - PRE) revealed a significant increase in phase lag in the POST tests ($t(26) = -3.12$; $p < 0.005$).

Table 17: VOR Phase Summary Data (in deg, 250 ms Delay)

Test	N	Mean	Variance	SD	SE	Phase Change
Pre	27	-2.6	17	4.1	0.79	
Post	27	-5.3	22	4.7	0.91	-2.7

6.7.3 Sickness Data

Mean and median SSQ total scores over time are shown in Figure 65. A Kruskal-Wallis test revealed that simulator sickness was induced as a result of VE exposure (collapsed across DELAY), ($X^2(2) = 7.76$; $p < 0.03$).

Nonparametric Scheffe post-hoc testing indicated that POST1 SSQ scores (those collected immediately after VE exposure) were significantly higher than either PRE SSQ scores or POST2 SSQ scores ($S_2 = 2.73$; $p < 0.025$). POST2 reports (collected 20 minutes post-exposure) were not significantly different than PRE sickness reports,

indicating that the accumulated sickness had returned to pre-exposure levels within 20 minutes after ending the VE exposure.

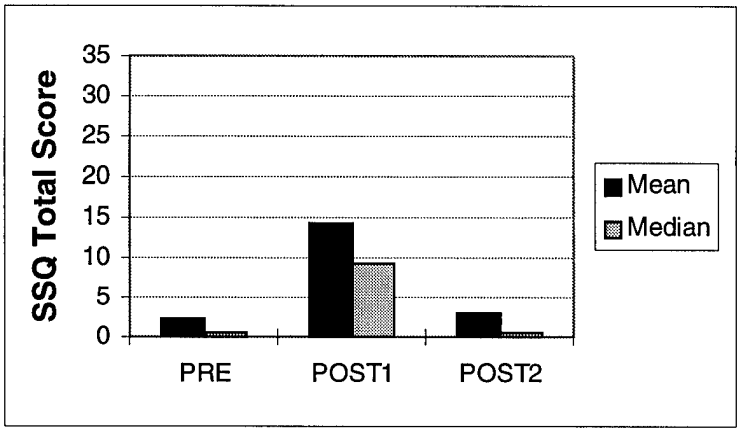

Figure 65: SSQ Total Score by Time
(Time Delay Exp)

Mean and median POST1 SSQ total scores by DELAY are presented in Figure 66. A Mann-Whitney nonparametric test indicated no effect of DELAY on POST1 SSQ scores ($U = 34$; $p > 0.60$). Mann-Whitney tests were also indicated no effect of DELAY on POST2 SSQ scores ($U = 38$; $p > 0.85$) (Figure 67) and on the final oral sickness report during exposure ($U = 33$; $p > 0.54$) (Figure 68).

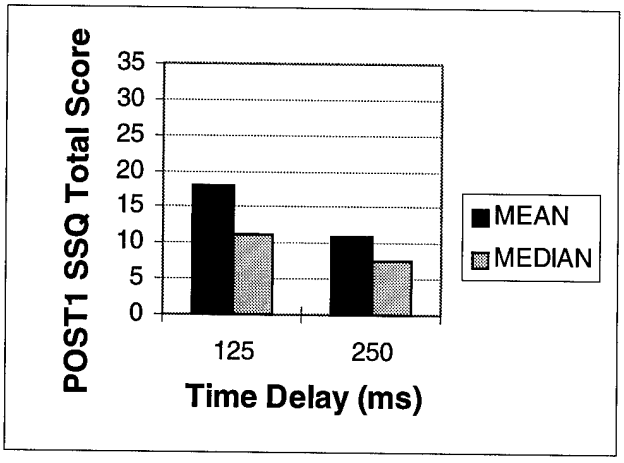

Figure 66: POST1 SSQ Total Score by DELAY

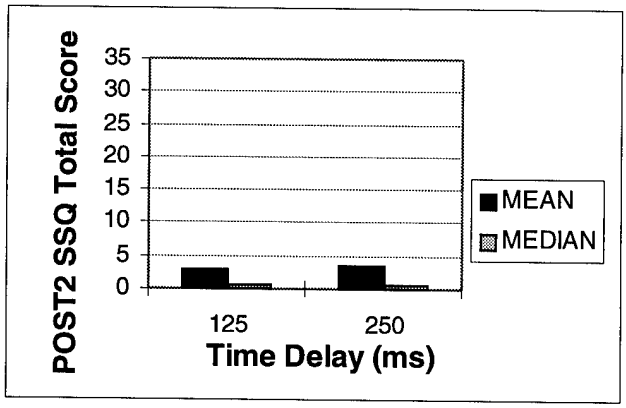

Figure 67: POST2 SSQ Total Score by DELAY

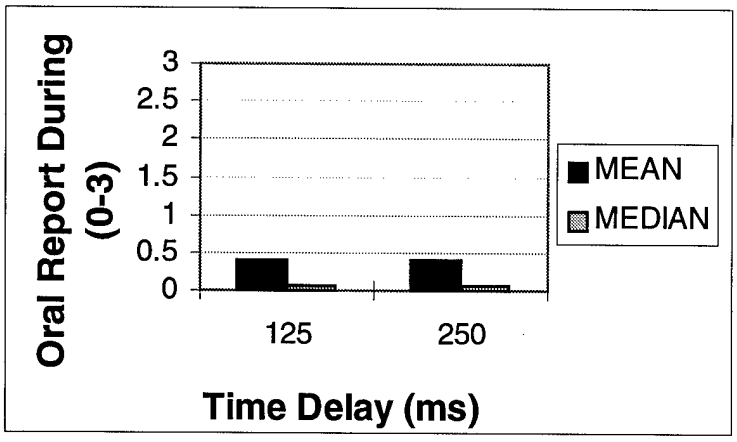

Figure 68: Sickness Report During by DELAY

Figure 69 presents POST1 SSQ data across sessions. A Mann-Whitney was not significant ($U = 37$; $p > 0.79$), indicating that subject's reports of sickness were not effected by prior exposures. POST2 SSQ data and sickness reports during also failed to show any obvious difference across sessions.

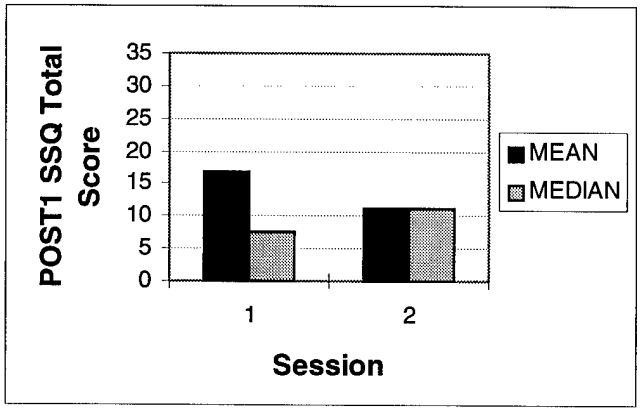

Figure 69: POST1 SSQ Total Score by SESSION
(Time Delay Exp)

A Friedman test indicated a borderline significant finding of sickness increasing with exposure time ($X^2(2) = 6.0$; $p < 0.06$) (Figure 70) when collapsed across DELAY. Sickness was significantly greater at 20 and 30 minutes than it was at 10 minutes.

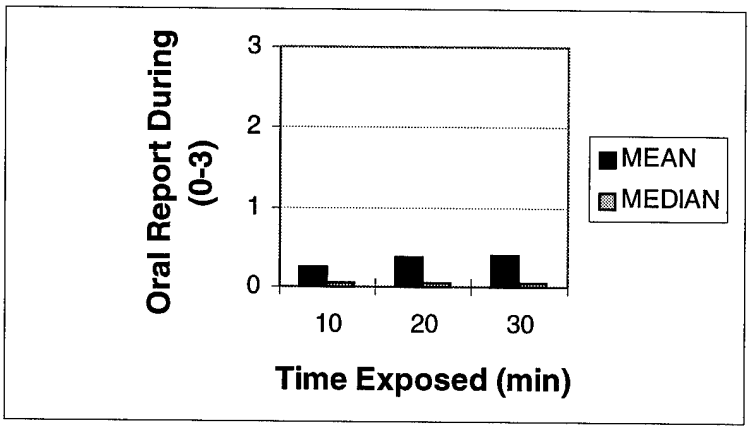

Figure 70: Sickness During Rating by Time Exposed (Across DELAY)

Results of Gender on sickness scores were mixed. There was a trend for females to report higher POST1 SSQ scores than males ($U = 16.5$; $p < 0.07$), but there was no effect of Gender on POST2 SSQ scores ($U = 28$; $p > 0.49$) or sickness ratings during exposure ($U = 20$; $p > 0.15$).

6.7.4 Head Position Analyses

A total of 51 of 54 potential head position epochs (80 seconds each) were collected during the experiment (nine subjects, two sessions each, with three epochs per session). Each head position epoch was analyzed using Labview's (National Instruments) auto-

power amplitude spectral analysis program to obtain a range of frequencies over which most head movements occurred and the associated power (deg RMS) at those frequencies. A typical example of an epoch's autopower spectrum is shown in Figure 71.

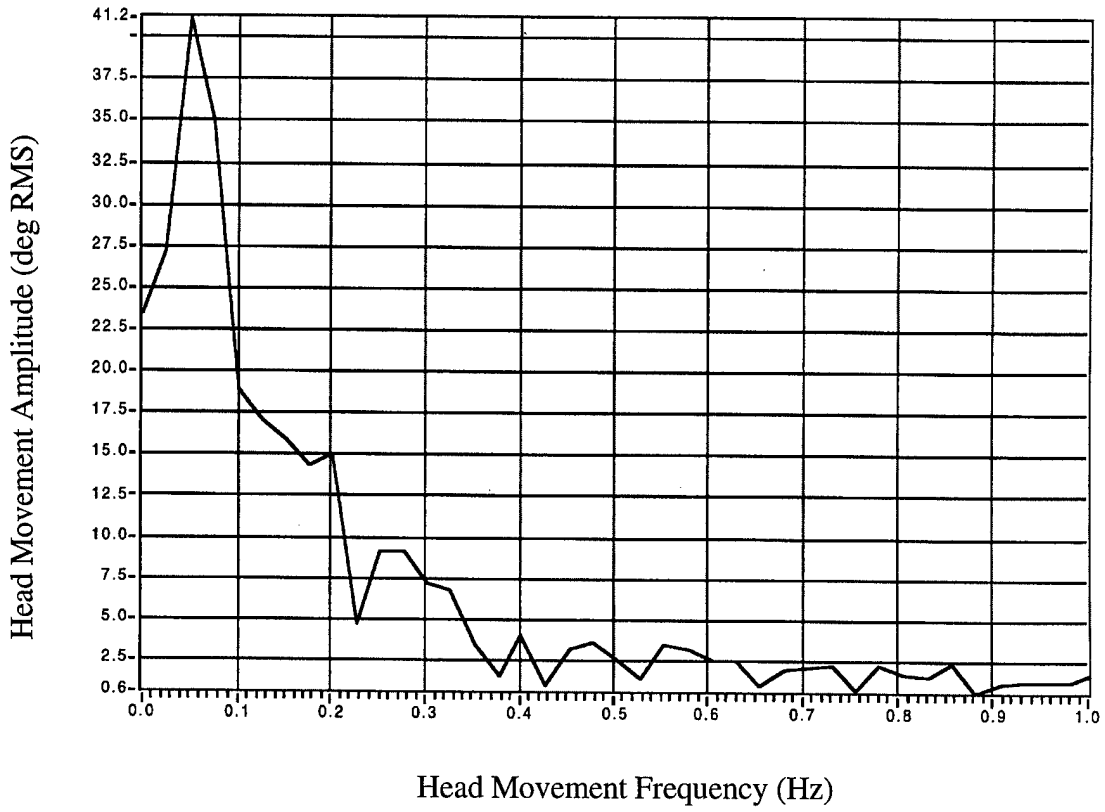

Figure 71: Typical Autopower Spectral Data:
Head Position (Time Delay Exp)

Four metrics were gleaned from each epoch: peak amplitude, frequency at which the peak amplitude occurred, frequency at which the power dropped below 10 deg RMS, and frequency at which the power dropped below 5 deg RMS. The last two metrics

were estimates of bandwidth. Table 18 presents summary data collapsed across conditions.

Table 18: Head Movement Summary Data
(Time Delay Exp)

	peak amplitude (deg RMS)	**peak freq** (Hz)	**BW** (10 deg RMS)	**BW** (5 deg RMS)
Mean	*36.09*	*0.06*	*0.21*	*0.38*
SD	*11.52*	*0.04*	*0.05*	*0.14*
Median	*34.70*	*0.06*	*0.21*	*0.35*

Table 19 compares the head movement data for each delay condition. There does not appear to be any meaningful difference in head movements across DELAY. Table 20 presents head movement data collapsed across DELAY but divided by collection time (beginning, middle, or end of a VE exposure). Rather than a decrease in head movements which would indicate fatigue or sickness, head movements seemed to increase slightly in both amplitude and bandwidth towards the end of each session.

Table 19: Head Movement Data by DELAY

	peak amplitude (deg RMS)	**peak frequency** (Hz)	**BW** (10 deg RMS)	**BW** (5 deg RMS)
125 ms				
Mean	*34.33*	*0.07*	*0.22*	*0.38*
SD	*11.22*	*0.05*	*0.06*	*0.12*
Median	*34.30*	*0.06*	*0.21*	*0.40*
250 ms				
Mean	*37.59*	*0.06*	*0.21*	*0.39*
SD	*11.77*	*0.03*	*0.05*	*0.16*
Median	*36.40*	*0.06*	*0.20*	*0.34*

Table 20: Head Movement Data by Exposure Time
(Time Delay Exp)

	peak amplitude (deg RMS)	peak frequency (Hz)	BW (10 deg RMS)	BW (5 deg RMS)
Beginning				
Mean	*30.86*	*0.07*	*0.21*	*0.36*
SD	*7.49*	*0.03*	*0.06*	*0.15*
Median	*33.50*	*0.06*	*0.21*	*0.34*
Middle				
Mean	*38.22*	*0.06*	*0.21*	*0.36*
SD	*11.19*	*0.03*	*0.05*	*0.13*
Median	*34.60*	*0.06*	*0.21*	*0.32*
End				
Mean	*39.96*	*0.07*	*0.22*	*0.43*
SD	*14.01*	*0.06*	*0.06*	*0.13*
Median	*41.00*	*0.06*	*0.21*	*0.40*

Individual subject head movements were not obviously related to either the amount of VOR adaptation achieved or the amount of simulator sickness reported. Also, head movement data were consistent across sessions.

6.7.5 VOR Adaptation / Sickness Relationship

Correlation tests were performed between percent VOR gain decrease and each of three variables: the final sickness-during rating (at 30 minutes), the POST1 SSQ total score, and the POST2 SSQ total score. A total of 18 pairs of data were compared (nine subjects, two sessions each) in each correlation. Due to the ordinal nature of the sickness data, Spearman's rho statistic was chosen over the Pearson statistic (which requires interval data). All correlations were examined using two-tailed tests.

The results of all correlation pairings indicate only a weak association between percent VOR gain decrease and sickness, with more sickness occurring as gain

decreases. With POST1 SSQ, $r = 0.243$ ($p < 0.24$), with POST2 SSQ, $r = 0.17$ ($p > 0.50$), and with the third sickness during rating, $r = 0.25$ ($p > 0.30$). Scatterplots did not reveal the existence of any nonlinear relationships.

Correlations between observed phase changes and the three sickness metrics were all less than 0.20.

6.7.6 VOR Gain and Phase Re-adaptation

Gain re-adaptation data are presented separately because not all cells contained re-adaptation information and because the question of re-adaptation time-course is relevant only after establishing the nature of any occurring adaptation. Two subjects were not tested ten minutes post-exposure due to the excessive amount of time taken to complete the session (mainly due to trouble with calibrations and HMD fit). Thus a total of 14 of 18 possible sessions remained in the analysis. Since the two DELAY conditions were not significantly different in gain or phase change obtained, re-adaptation effects were examined collapsed across DELAY.

Figure 72 presents all VOR gain data (PRE, POST, and AFT10: after 10 minutes) collapsed across DELAY. Gain re-adaptation was essentially complete after 10 minutes of re-exposure to the real world.

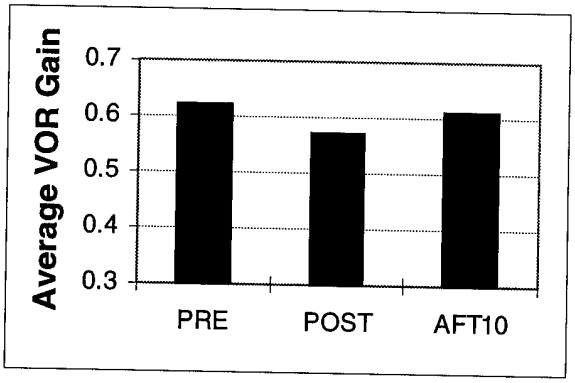

Figure 72: VOR Gain Re-adaptation
(across DELAY)

The phase re-adaptation data are presented in Figure 73. There appears to be little phase re-adaptation after 10 minutes, as the AFT10 value is nearly the same as the POST value (about -5.2 deg).

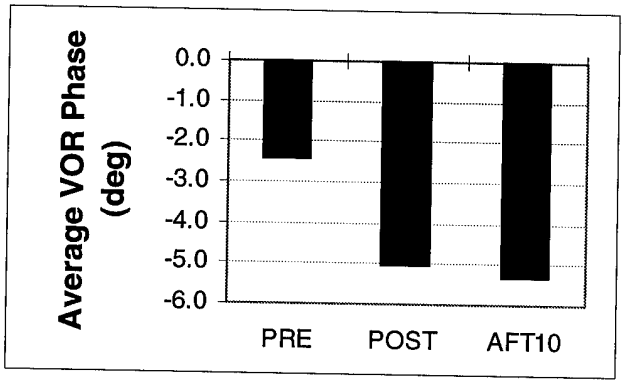

Figure 73: VOR Phase Re-adaptation (across DELAY)

6.7.7 BALANCE STABILITY DATA

Balance data were collected pre-exposure and approximately 15 minutes post-exposure. The data are shown in Table 21. There was no significant difference between PRE and POST data when collapsed across DELAY, nor was there any meaningful effect of DELAY on postural stability.

Table 21: Balance Platform Data (total dispersion score: Time Delay Exp)

Delay (ms)		PRE	POST
125	MEAN	*5.5*	*5.1*
	MEDIAN	*4.8*	*5.1*
250	MEAN	*4.9*	*5.7*
	MEDIAN	*4.7*	*4.6*

6.8 DISCUSSION

This section first considers findings regarding the VOR including VVOR, VOR adaptation, frequency specificity, and VOR re-adaptation. Simulator sickness is then discussed, followed by the head movement data. Lastly, the weak correlation between VOR gain adaptation and simulator sickness is addressed.

6.8.1 VVOR DATA

The VVOR phase data were as predicted, given the phase demands involved for each delay at 0.8 Hz (36 deg for 125 ms and 72 deg for 250 ms). However, the gain reductions were not expected given the stimulus. Both DELAY conditions had reduced gains compared to the gain of the real-world VVOR (0.99), with the gain at 250 ms being the lowest (0.74) and the gain at 125 ms being intermediate (0.89). The first course of action in explaining this result was to verify that the visual stimulus was not

the cause. It was re-confirmed that the virtual scene moved at the same amplitude as the head for each system time delay and that head movement amplitude data were not effected by changes in time delays to the visual stimulus. Therefore, the explanation for this gain reduction does not exist in hardware/software.

Another explanation exists which also accounts for the continual failure of the VVORs to fully compensate for large-magnitude gain change demands (see Section 5.8). The problem relates to tracking difficulty under conditions of visual-vestibular sensory rearrangement. In the virtual VVOR tests, subjects were required to fixate on a virtual target that was not space stable (Section 3.1.4). The motion of the virtual target in space is a sine wave with increasing amplitude from zero (when the phase lag is zero) to two times the amplitude of head motion (when the added phase lag due to time delay is 180 deg). Pursuit mechanisms are activated to correctly fixate on this moving target and perfect visual tracking would likely become progressively more difficult with increasing time delays[71]. This increased difficulty in tracking may require an increase in corrective saccades in the direction of eye motion when the eye falls behind the target and needs to 'catch up' (when the retinal position error becomes too large). These 'catch-up' saccades reduce the slow-phase eye movement amplitude required to each the target fixated. During the analysis, saccades were removed automatically, leaving only the reduced slow-phase amplitude eye motion which resulted in the reduction of VVOR.

To evaluate this hypothesis, I first looked at the standard deviations of VVOR gain. This hypothesis predicts that the data would be more variable with increasing time delay. This was what occurred, with the standard deviation rising from 0.07 (real world) to 0.09 (125 ms VR) to 0.10 (250 ms VR). It is interesting to note also that the

[71] For periodic motion this is only true until the increasing time delay produced a 360 deg phase demand which would result in an in-phase target one cycle removed.

variance in the NEU condition of the Image Scale Experiment was 0.06. Though the subject pool was different, the NEU condition was exactly the same condition as used in this experiment except for a reduced time delay (48 ms). Therefore, the data suggest that increasing time delay increased the variance in eye tracking performance.

VVOR head and eye raw position traces of a few subjects were inspected to determine if there were differences in the number/magnitude of corrective saccades occurring between the real world, 125 ms and 250 ms. Figure 74 indicates that for a 0.8 Hz sinusoidal head motion (30 deg/s peak velocity), visual tracking performance degraded as time delay increased. In the 125 ms condition, note what appears to be a few corrective saccades as described earlier. In the 250 ms condition, the number of corrective saccades increased and the eye movements lost smooth compensatory response.

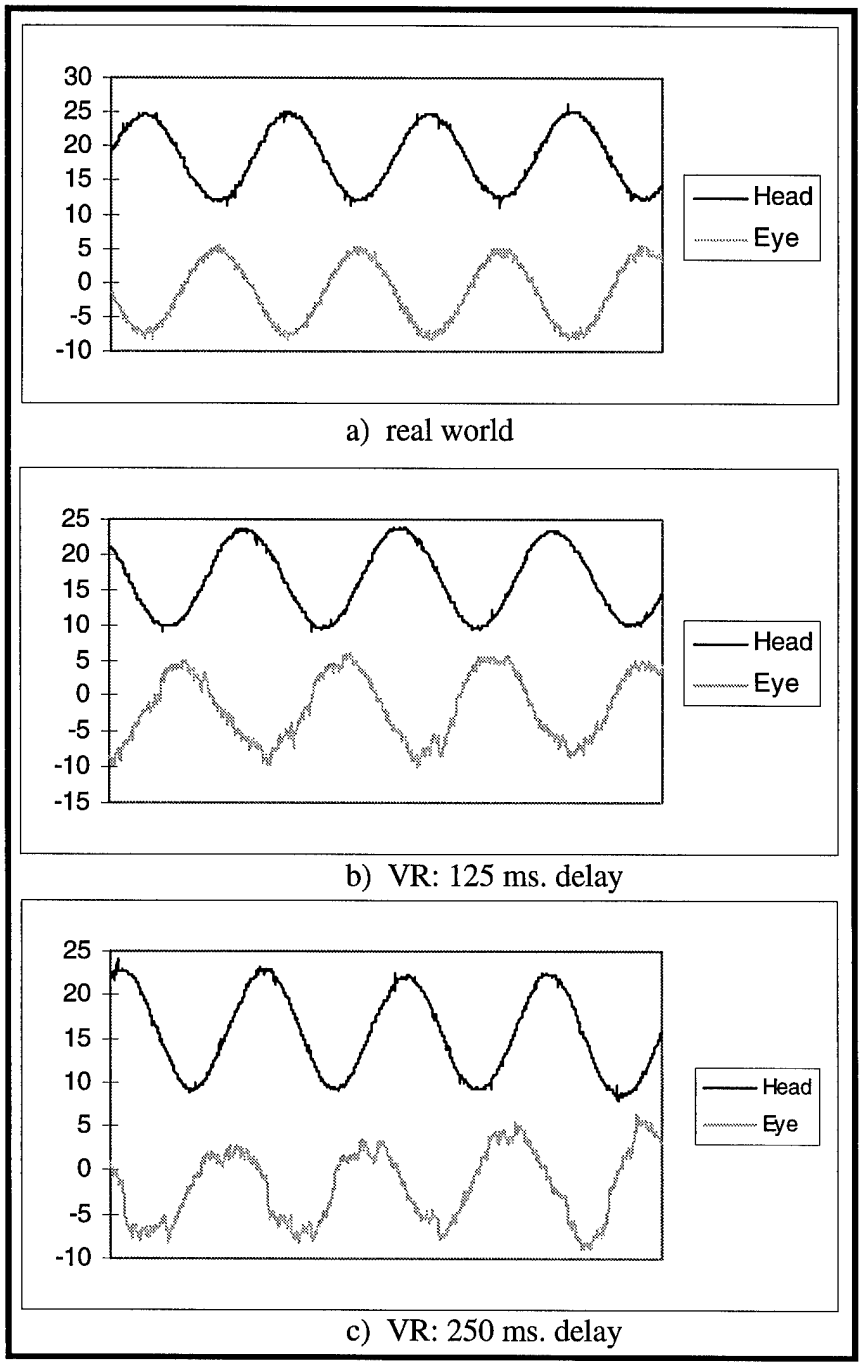

Figure 74: VVOR Data Traces by DELAY[72]

[72] Subject #1, Session #1, 0.8 Hz, Trial #2.

6.8.2 VOR Phase Adaptation

A small but statistically significant amount of phase adaptation occurred in this experiment, though there was no difference in phase adaptation between the two delay conditions. Phase lag increased over 2.5 deg in both conditions. Since the visual angle subtended by the fovea is only 2 deg, this change in phase could *potentially* result in meaningful retinal slip after VE exposure if not compensated for by visual tracking systems.

The observed phase changes were far from compensatory, however, which was expected given the unrestricted, intermittent nature of the head motion during exposure. In fact, it could be argued that any obtained phase adaptation is significant given the nature of the interaction, the short exposure period, and the method of data collection (i.e., testing at only three frequencies). The phase change in this experiment was over 50% larger than that obtained in the Image Scale Experiment (where the minimum system time delay was used). However, it was surprising that phase changes were not effected by frequency given the differing phase demands involved. This result is likely due to insufficient exposure time at each frequency.

6.8.3 VOR Gain Adaptation

The gain decreased 7.4% in the 125 ms delay condition and 9.8% in the 250 ms condition, both of which were statistically significant. This was not predicted by the physics of the interaction (Section 3.1.4). Why then did this occur?

One possibility is fatigue. Subjects simply may have become tired and/or bored with the experiment which would be reflected as a decrease in post-exposure VOR gain. However, this is not likely for several reasons. First, subjects did not complain of

fatigue nor did the experimenters notice any overt signs of fatigue (i.e., yawning, lack of alertness) on the part of the subjects during the exposure. Second, head movement data indicated no appreciable decrease in amplitude or peak frequency of head motion over the course of the experiment which would have occurred had the subjects became fatigued. In fact, head motion seemed to increase slightly towards the end of the exposure, not decrease. Third, head movement data suggest that the movements were not strenuous or high-frequency in nature and so, therefore, physical fatigue due to excessive head movements seems unlikely. Fourth, VVOR data also indicated a gain decrease with increasing delay and this test was performed at the *beginning* of the session. Lastly, using the same protocol, subjects in the NEU condition of the previous experiment experienced only a 3 percent drop in VOR gain post-exposure, which itself was not significant. Therefore, fatigue effects are highly unlikely.

A second possibility is hardware problems or calibration errors that would have effected the results of both the VVOR data and VOR post-exposure data by misrepresenting the intended visual stimulus. However, as discussed in Section 6.8.1, the performance of the hardware and software was verified as accurately presenting the visual scene.

A third possibility is that the phase adaptation process is coupled to the gain adaptation process such that pure phase change demands result both in phase and gain changes. Other research has indicated a coupling between these two processes though the relationship is complex and ill-defined (Khater, et al., 1990; Kramer, et al., 1995). In addition, these models do not readily predict such a large gain decrease for a pure phase lag demand.

The most viable explanation again relates to the hypothesis presented in previous sections to account for the VVOR gain decreases. The premise is that the eye has difficulty tracking a virtual fixation target that is in fact moving with respect to space.

In explaining the VVOR data, I hypothesized the existence of 'catch-up' corrective saccades which return the eye on target when it slipped. That situation is not completely analogous to the search tasks experienced in the VE over the 30 minute exposure. However, there is a need for additional saccades and other eye movements (including smooth pursuit) in the performance of the search task to compensate for the visual-vestibular rearrangement. Research has demonstrated that catch-up saccades (saccades in the direction of the slow-phase VOR) do in fact occur during VOR testing after exposure to 2X magnifying spectacles, thus implicating a larger set of eye movements in the VOR adaptation process (Istl-Lenz, et al., 1985). These saccades, if existing in the current data as well, could result in an the VOR gain decreases found in this experiment (Istl-Lenz, et al., 1985). The data suggest that many of the subjects who demonstrated significant VOR gain adaptation also showed increases in the number and magnitude of saccades/quick phases post exposure versus pre-exposure (Figure 75).

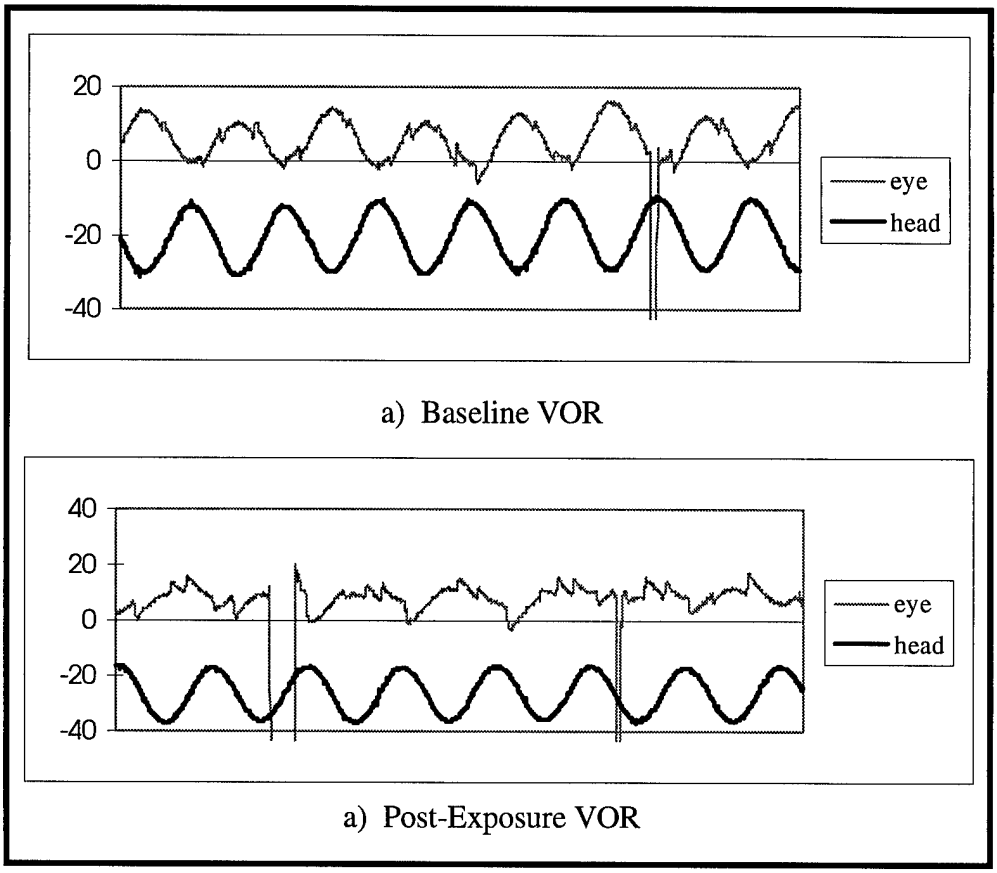

Figure 75: Head/Eye Position Traces During Pre- and Post-Exposure VOR Testing[73]

The issue may be more complex, however. Research has shown that adaptation to visual-vestibular rearrangements is an intricate, multi-factor process involving many oculomotor movements and central motor programs (Melvill Jones, Guitton, & Berthoz, 1988). Shelhamer, et al. (1994) cogently argued that the stimulus for VOR gain adaptation cannot be based upon image slip alone; compensatory eye movement information is likely utilized as well. Indeed, research has shown that VOR adaptation

[73] Subject #6, Session #2, 0.8 Hz, Trial #2, 7.5 of 8 cycles shown.

can occur with little to no existing retinal slip (Scudder & Fuchs, 1992; Shelhamer, et al., 1994). Shelhamer, et al. (1994) hypothesized that the true stimulus for VOR adaptation is based on a central estimate of 'error' which contains existing retinal slip along with the actions of compensatory eye movements generated to reduce retinal slip[74]. Therefore, corrective eye movements required during the exposure period may modulate the VOR gain in a complex manner, resulting in a gain decrease demand.

As stated earlier, time delays resulted in a gaze stimulus that was not space-stabilized during VVOR testing which likely stimulated saccades and smooth pursuit movements in addition to OKN and VOR. The requirement for increased smooth pursuit causes VOR suppression during the pursuit movement when the head is also moving. If smooth pursuit movements were increasingly required with increasing time delay and if these movements were repeated enough during the exposure process, this could result in increased VOR suppression during rotations in the dark (Barnes & Grealy, 1992).

Additionally, a time delay in the visual scene motion acts as a VOR suppression stimulus anytime the head moves from a stationary position. The duration of this VOR suppression stimulus equals the time delay of the system. During the VE exposure period, the head motions of each subject were a repeated series of starts and stops. This resulted in many instances of VOR suppression stimulation which could trigger a reduction of VOR gain over time.

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[74] The notion of "inferred head velocity" is also a factor in their model. Inferred head velocity is the internal estimate of head motion based upon a combination of labyrinthine, visual (OKN), and somato-sensory signals.

There is a counter-argument here, however. After the head completed a motion, the visual scene would continue to move in relation to the head for the length of the time delay. Theoretically, this motion could cause a VOR gain *increase* due to the retinal slip involved, which might in turn counteract the effects of the VOR suppression stimulus occurring at head movement initiation. Indeed, pure OKN stimulation has been demonstrated to drive VOR gain adaptation (Shelhamer, et al., 1994). Therefore, which is the provocative stimulus for VOR adaptation? Is it the visual and oculomotor stimulation occurring only *during* head movements or is the stimulation obtained during the period of OKN (i.e., after the head stopped moving) also potent? Assuming both are involved in some capacity, what are their relative weights?

These data suggest that the visual stimulus *during* head movements is more heavily weighted, which resulted in the observed gain decrease. This stimulus would predict that increasing time delays generate greater gain decreases. This was found in this experiment, though the difference was not statistically significant (likely due to that small sample size). In addition, the 3% decrease in the VOR in the NEU condition of the previous experiment (and the overall the gain change direction asymmetry found) could be partially explained by the short VOR suppression stimulus at the initiation of head movements (due to the minimum time delay of 48 ms).

Another factor supports the notion that the delay in onset of visual scene motion is more potent for VOR gain adaptation than the remaining OKN motion after the completion of head movement. At the onset of head movement, the VOR must directly be suppressed by an activated smooth pursuit system if the subject is looking at a target. However the converse is not true; the final OKN stimulus after the head stops is not as closely tied to VOR modulation (because the head is not moving and therefore there is no VOR).

If this explanation is correct, VOR adaptation obtained via OKN stimulation alone should be smaller in magnitude the VOR adaptation occurring as a result of head movements. VOR research is mixed on this issue (Shelhamer et al., 1994).

One potential way to determine temporal relationship between these stimuli may be to increase the exposure time to the visual stimulus. If long term exposure results in a larger or sustained VOR gain decrease, than the visual information during head motion is always the predominant stimulus for adaptation. If the gain begins to adapt back towards normal, it is possible that generalized adaptation processes occur which allows the visual motion information after head movements to have a greater effect over time. Data already exist that partially address this issue. There was a trend towards an effect of SESSION on VOR gain, resulting in approximately a halving of the gain reduction in the second session compared to the first. This suggests that adaptive mechanisms are at work which may include an increased effect of visual motion after head motion.

As a concluding note, it is important to restate the results of this experiment regarding VOR gain adaptation. Though there are competing explanations as to why it occurred, this research reliably demonstrated the existence of statistically significant VOR gain reduction as a result of exposure to a virtual interface with large time delays. Interface designers must recognize and account for this finding regardless of the underlying mechanisms involved.

6.8.4 VOR Adaptation and Frequency Specificity

As in the previous experiment, VOR gain demonstrated generalized adaptation across all three testing frequencies. This occurred despite finding that the majority of head movement power was below 0.2 Hz[75]. Gain data were even collapsed across

[75] Though the largest concentration of head movement power was below 0.2 Hz, subjects did occasionally move their heads at higher frequencies. These movements were simply less frequent than the slower, more deliberate head movements at or below 0.2 Hz.

DELAY in an effort to provide a more powerful statistical test for detecting any frequency specific effects that might exist, but none were found. In addition, an separate inspection of gain adaptation across frequency for each delay condition also showed the same generalized adaptation.

This result appears to contradict the notion of adaptation specificity but is congruent with other studies that have demonstrated generalized VOR adaptation across frequencies (Demer, et al., 1989). The explanation for these discrepant findings is likely related to differing interaction methods utilized during exposure as well as differing frequency ranges over which frequency specificity was examined.

Most VOR adaptation studies utilize passive exposure to a restricted frequency (or set of frequencies). In addition, they also require little to no tasking by the subject. Therefore, the adaptation achieved is artificial in that the visual-vestibular interaction was artificial. This stimulus/interaction method was used in the seminal experiments of frequency specificity (Lisberger, et al., 1983) and in studies that support this model (Powell, et al., 1991).

Generalizing frequency specificity from this artificially-induced adaptation to unrestricted, active human head motions presupposes that the brain utilizes no other inputs for adaptation response than that specifically called for in the motions of the head, visual scene, and eye. There is no latitude for the modulating influence of past experience, goals and intentions, information from motion commands (efference copy), and the existence of internal predictive models, all of which are hypothesized to play a role in human adaptation. Therefore, it is possible that VOR adaptation resulting from active, unrestricted head movements will not follow the frequency specificity model even if the majority of these head movements were centered around a small frequency range. The data obtained in these experiments support this premise.

An additional explanation for the contradictory results focuses on the differing ranges of frequencies over which VOR adaptation was tested. For instance, Lisberger, et al. (1983) demonstrated frequency specificity using a relatively large range of testing frequencies of 0 to 4 Hz. This experiment, however, had a range of only 0.6 Hz, and Demer et al. (1989) failed to find frequency specificity over a range of 1 Hz. Clearly, the range of test frequencies utilized is a meaningful factor to consider.

VOR phase adaptation is also generalized in magnitude across test frequencies, which is somewhat surprising given the increasing phase demands at higher frequencies. One could argue that if the metric employed was the percentage of phase adaptation achieved to that demanded at each test frequency, the results would favor adaptation specificity. However, examining frequency specificity using this metric after an exposure period restricted to a *high* frequency would likely result in "reverse" frequency specificity, with the demand at the *lowest* frequency tested being most easily met[76].

6.8.5 VOR Gain and Phase Re-adaptation

VOR gain and phase re-adaptation data were collapsed across DELAY to provide a more stable assessment of re-adaptation time-course. This was considered reasonable given that the gain and phase changes were similar between the two conditions.

VOR gain adaptation appeared to completely (or nearly so) return to baseline conditions after 10 minutes of re-exposure to the real world. This was expected given the magnitude of adaptation experienced in this study. However, it is not fully congruent with the results from the Image Scale Experiment. The differing stimuli for adaptation between the two experiments could have resulted in adaptations with different sustaining strength. Alternatively, this difference may simply reflect the

[76] This is only speculation at this point but the empirical test would be straightforward.

different subject populations, differences in attentiveness to the post-exposure controlled eye-head movement tasks, or other uncontrolled factor(s).

Phase re-adaptation did not occur after 10 minutes. This result was unexpected given the completeness of gain re-adaptation. Though there is a paucity of research that addresses time-course of VOR phase adaptation vs. gain adaptation, it is at least logical to assume that if a mechanism is slow to adapt, it may also be slow to re-adapt. Therefore, sluggishness of the VOR phase adaptation processes is one possible reason for the lack of phase re-adaptation after 10 minutes.

6.8.6 Simulator Sickness Data

Simulator sickness occurred as a result of exposure to the virtual interface. However, the magnitude was approximately half that found in the MIN and MAG conditions of the previous experiment.

Though some simulator sickness did occur, it was not differentially affected by the two time delays. This was true regardless of metric chosen: sickness report during, SSQ immediately post-exposure or SSQ 20 min post-exposure. This suggests that increasing time delays from 125 to 250 ms did not effect reports of simulator sickness.

Given that only between-subjects nonparametric statistics were used, perhaps these tests were not powerful enough to detect an existing effect of time delay. To answer this question, *pairwise* nonparametric statistics were subsequently employed on 8 of the 9 subject pairs (excluding the one subject pair that was between-subjects due to attrition). Using these pairwise tests, there was still no significance (or trend toward significance) for an effect of time delay on sickness.

Perhaps the range of time delays examined in this experiment was not large enough to detect an effect. However, the range could be expanded downward to 48 ms if the

results from the NEU condition of the Image Scale Experiment were considered. The NEU experimental condition was matched in every way to the two delay conditions used in this experiment save the time delay, which was set at 48 ms, and the different subject group.

Aware that the subject pool was different between the NEU condition of the Image Scale Experiment and the two larger delay conditions, the simulator sickness data were compared across these three different time delays. Figure 76, Figure 77, and Figure 78 display the results of this informal comparison. It is evident, with every sickness metric available, that sickness did not change appreciably over the three time delays, spanning 48 to 250 ms.

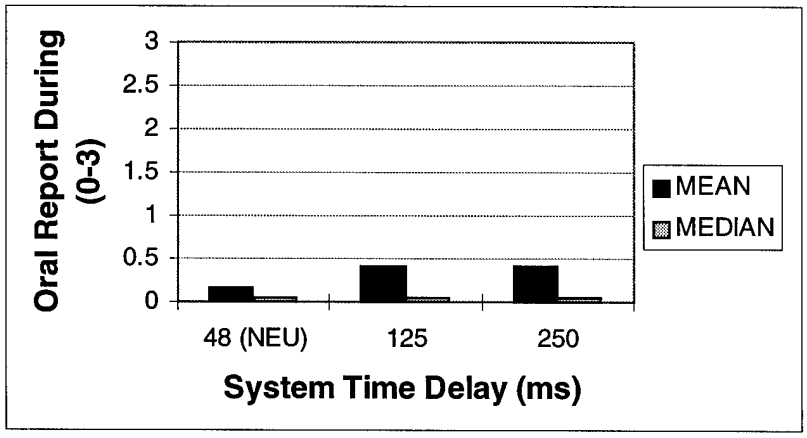

Figure 76: Sickness Reports During by Time Delay (across exps)

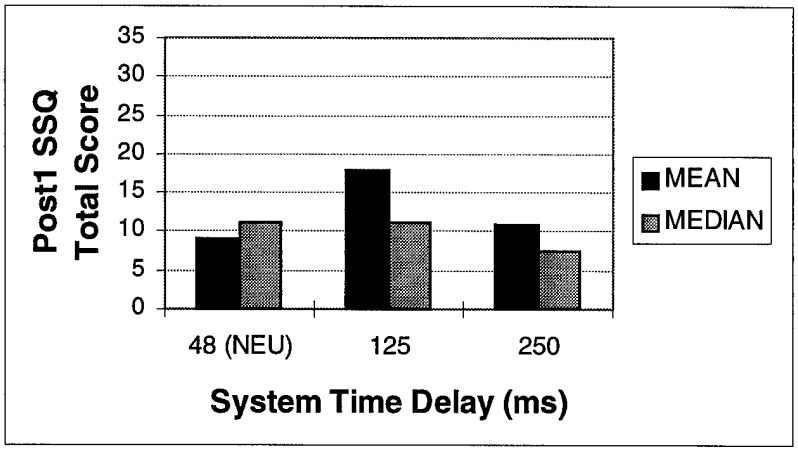

Figure 77: POST1 SSQ by Time Delay
(across exps)

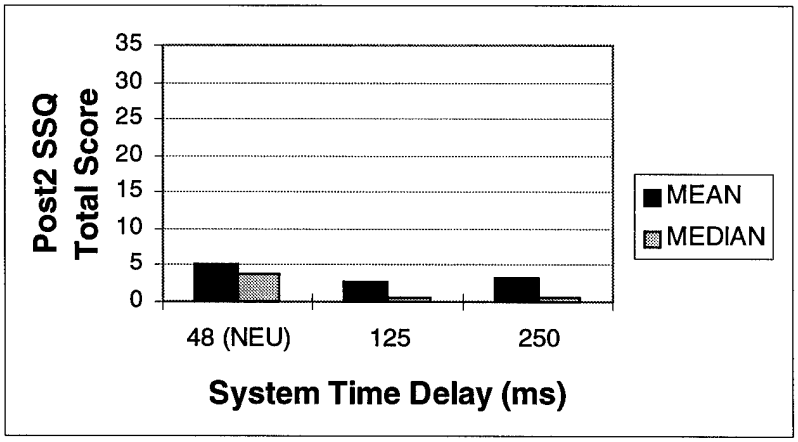

Figure 78: POST2 SSQ by Time Delay
(across exps)

These results are not readily explained by the sensory rearrangement theory, which would predict an increase in sickness with increasing time delay, at least to a point. If the theory is correct, then the data must be in error. There are a few potential arguments

for discounting the validity of these results. One is that the range of time delays involved was too restricted to detect an effect. This is perhaps the weakest of all arguments. First, there are very few virtual environments that have system time delays below 48 ms or above 250 ms (the former because of technology limits and the latter because participants would reject these VEs for performance and annoyance reasons). The range covered in these experiments does account for the majority of useable virtual interfaces. Second, percept varied with delay. The minimum time delay was not perceptible by the subjects whereas the 250 ms delay was very noticeable and annoying to many subjects. Third, these time delays must have resulted in a visual-vestibular sensory rearrangement given that both VOR phase and gain adaptation resulted.

A second argument might discount the comparison across experiments due to subject group differences. This is a valid concern. However, a review of the subject groups indicates much similarity in areas known to impact simulator sickness susceptibility. For example, females are normally found to be more susceptible to simulator sickness than males. Though significant differences regarding gender did not result form these studies, the groups were nearly equal with respect to the ratio of females to males (5/11 in the Image Scale Experiment versus 4/10 in the Time Delay Experiment). Subject groups were also nearly identical on motion sickness experience, simulator sickness experience, and ratings of susceptibility to motion sickness. The only difference noted was that the subjects in the Time Delay Experiment had slightly more previous experience with VR (8 subjects with 0 or 1 previous exposure to VR in the Image Scale Experiment vs. 6 subjects with 0 or 1 previous exposure in the Time Delay Experiment). Given the similarities in the subject groups, combined with the identical tasking/exposure protocol involved and the striking similarities in reported sickness between the two groups when restricted to the NEU condition, this comparison across experiments appears to be justified.

A third potential argument is that subjects may have been experienced with the virtual interface used. This experience would have resulted in a lesser sensory rearrangement for the given stimulus rearrangement because the internal set of expectations would have included sensory patterns stored from previous exposures to this interface (assuming the sensory rearrangement theory). This argument is not valid for a several reasons. First, upon beginning the first session, no subject in either the Image Scale or Time Delay Experiment had previously experienced the particular virtual interface employed in these experiments. Second, 50% of the subjects tested in these two experiments had never even experienced *any* head-coupled virtual interface prior to this experiment and 65% had one or less exposures. Lastly, there were no significant differences in simulator sickness across sessions in either experiment, which diminishes the overall argument of experience transfer.

Though other arguments may be raised, the data strongly indicate (both within and across experiments) that fixed time delays are not an overly provocative contributor to simulator sickness. In addition, two other studies that empirically addressed this issue revealed no effect of increasing time delay on simulator sickness (Frank, et al., 1988; Uliano, et al., 1986). Fixed time delays may be annoying and they likely decrease performance, but they do not make you overly sick[77].

A few other notes about the simulator sickness data are in order. This experiment, like the Image Scale Experiment, found no significant effect of gender on sickness magnitude. This experiment also found a trend towards increasing sickness with exposure time though the increase was far less noticeable than in the previous study (probably due to a floor effect regarding sickness reports).

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[77] The time delays discussed here are relatively fixed at specific values. The potential for variable time delays to cause sickness is addressed in Chapter 10.

6.8.7 Head Movements

The head movement data were striking in that the power was concentrated at very low frequencies (less than 0.2 Hz) as in the earlier experiment. This was likely due in part to the rather large percentage of time that the subjects did not move at all (i.e., when waiting for confirmation that the target was correctly acquired or when waiting for the next target to be issued) which would result in the highest peak power frequency tending towards 0 Hz. Other factors potentially contributing to these low peak frequencies of head movement power include the encumbrance of the virtual interface, the novelty of the virtual environments, and the lack of a time constraint for acquiring each target. Though some high-frequency head movements did occur, the power associated with these movements was rather small. Head movements did not vary with delay condition or session.

The slight increase in amplitude and bandwidth of head movements at the end of the exposure as compared to the beginning is interesting. This was likely due to either the subject adapting to the motion effects of the time delay or increased motivation given that the end of the experiment was near.

Individual variability in the head movement data did not predict either simulator sickness or VOR gain/phase adaptation. This on the surface seems surprising. However, given the consistent nature of the head movement data combined with an apparent floor effect due to task requirements, the result is at least understandable.

6.8.8 Relationship between VOR Adaptation and Simulator Sickness

All correlations relating VOR gain and phase changes with the three sickness metrics were 0.25 or less. The relationship that existed indicated that as VOR gain decreased, simulator sickness increased. The weak correlations could be due to the relatively low levels of sickness experienced by the subjects. Considering the weak

correlations found in the Image Scale Experiment, however, it appears that VOR gain adaptation and simulator sickness are not strongly related, at least using final gain and phase change values as the adaptability metric. Nonetheless, another VOR metric may exist that more closely specifies the general adaptability of a person. This proposed metric is described in Chapter 10.

6.8.9 Postural Stability Data

These data were collected mainly to ascertain, for safety's sake, whether the subject had become measurably unstable as a result of being exposed to the virtual interface. If a subject scored poorly on the post-exposure trials, he/she was required to wait an extra five minutes before being re-tested. Only two subjects had to be re-tested due to balance instability on the first test, but both easily scored well enough on the second test to be released. The data seemed to indicate that there was a slight increase in postural sway after the 250 ms condition, but this difference was within the noise level of the test.

CHAPTER 7: LONGITUDINAL VOR ADAPTATION EXPERIMENT

7.1 OBJECTIVES AND HYPOTHESES

The purpose of this experiment was to gather initial data on VOR adaptation time course. Although re-adaptation data had already been collected in the previous two experiments to determine how long subjects must wait before they are considered fully re-adapted, there were no data on when meaningful adaptation responses begin to appear when a subject uses a virtual interface. This information could potentially support the formation of optimum exposure time guidelines to maximize time spent in the VE while minimizing any negative effects of that exposure (i.e., undesired physiological adaptations and simulator sickness). This experiment was conducted to ascertain when VOR adaptation likely appeared in the previous two experiments by testing VOR gain at 10, 20 and 30 minutes of a 30 minute VE exposure. As such, it addressed objectives 1 and 5 (see Chapter 1).

The hypothesis was that VOR adaptation to the virtual interface would follow a decaying exponential function, with initial adaptation occurring rapidly. Given the saliency of the specific interface used, a 5% change in VOR gain was expected to be noticed within 10 minutes of exposure, with an additional 3 to 10% change occurring between 10 and 30 minutes[78].

In addition, given the incomplete gain re-adaptation observed after 10 minutes in the Image Scale Experiment, this experiment examined the level of recovery after 20 minutes. However, to reduce potential contextual effects of the chair and HMD, subjects were released from the chair and allowed to move about the building naturally

[78] Values based upon previous findings.

for the re-adaptation period. It was hypothesized that the additional 10 minutes of recovery along with natural movements would return the VOR gain completely to pre-exposure levels.

Lastly, this experiment provided another opportunity to investigate the issue of frequency specificity vs. generalized adaptation across frequencies. The literature generally favors the notion of frequency specificity but the results of the previous two experiments supported the notion of generalized adaptation across frequencies. It was hypothesized that generalized adaptation across frequencies would again occur, given that the same exposure protocol was used.

7.2 SUBJECTS

Two adult subjects (both male, ages 27 and 35) were chosen to participate in this experiment based upon their demonstrated ability to adapt their VOR gain in the past (each had participated in the Image Scale Experiment and showed near average gain adaptation in each condition). Both subjects were in good health with no history of visual or vestibular medical problems. One subject wore corrective lens and had slight astigmatism. Neither subject had experienced the virtual interface for at least two weeks.

7.3 EXPERIMENTAL DESIGN

A two factor, within-subjects design was used, with five levels of TESTTIME (Pre, 10 min, 20 min, 30 min, and 20 min post) and three levels of test frequency (FREQ: 0.2 Hz, 0.4 Hz, 0.8 Hz). The dependent variables were VOR gain and phase estimates at each TESTTIME and FREQ combination, averaged over two trials.

7.4 EXPERIMENTAL SET-UP AND APPARATUS

Experimental set-up was as described in the Image Scale Experiment. To stimulate a significant VOR gain change, the virtual interface was configured with an image scale of 0.5X (minification) and a system time delay of 125 ms for the entire exposure. This configuration presented a significant VOR gain decrease demand.

7.5 PROCEDURE

The procedure was the same as in the Image Scale Experiment, except for the following. In addition to the pre- and post-exposure tests (at 0 and 30 minutes, respectively), VOR data were also collected at 10 and 20 minutes into VE exposure. No sickness data or posture data were collected, though oral sickness reports were recorded during the exposure for health and safety reasons only. Subjects removed the HMD after the 30 min test and were released from the chair so that they could naturally move within the building for 20 min prior to the final VOR test. Eye calibrations were completed: 1) prior to collecting pre-exposure data, 2) immediately after collecting the 30 min data, and 3) prior to the 20 min post testing.

7.6 STATISTICAL ANALYSIS

Given that two subjects were run, only descriptive statistics are presented.

7.7 RESULTS

Table 22 presents the VOR gain change over time for each subject, along with the average gain values and percent gain changes across subjects. Gain change over time per subject is presented graphically in Figure 79. The average percent gain change across subjects over the 30 minute exposure was 11.2%. Most of this change (7.8%)

occurred within the first ten minutes. There did not appear to be any additional change in gain between 20 to 30 minutes. After 20 minutes of re-adapting to the real-world through natural movements, the VOR gain had returned to within one percent of its pre-exposure value.

Table 22: VOR Gain Change with Exposure Time[79]
(Longitudinal Exp)

	PRE	10 min	20 min	30 min	Post 20 min
Subject 1	*0.51*	*0.48*	*0.45*	*0.47*	*0.54*
Subject 2	*0.65*	*0.59*	*0.56*	*0.57*	*0.62*
Mean	*0.58*	*0.54*	*0.51*	*0.52*	*0.58*
% Change		***-7.79***	***-12.99***	***-11.13***	***-0.99***

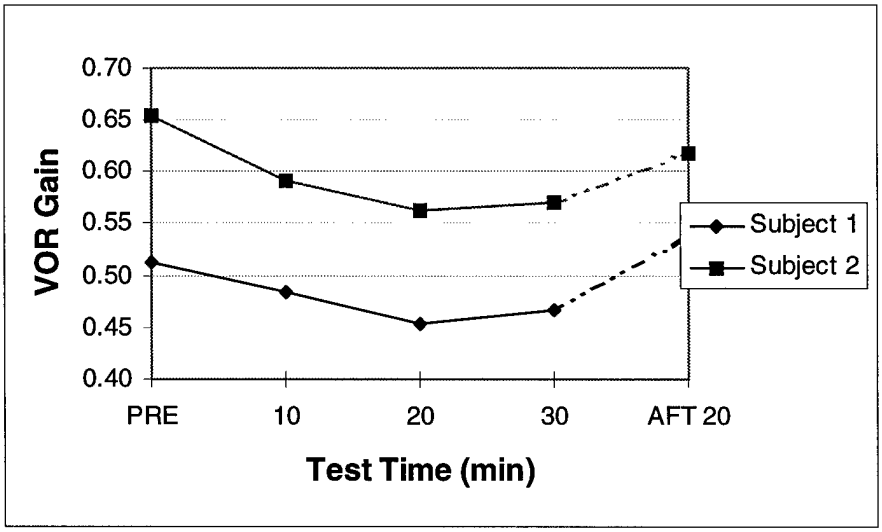

Figure 79: VOR Gain Change over Time by Subject[80]
(Longitudinal Exp)

[79] Gain values are rounded.

VOR gain change at each test frequency over time (collapsed across subjects) is shown in Figure 80. All frequencies resulted in similar percent gain changes over the 30 minute exposure (between 10.5 to 13% gain decrease). The individual curves varied a bit, as the 0.2 Hz curve levels off after 10 minutes, the 0.4 Hz curve levels off at 20 minutes, and the 0.8 Hz curve actually suggests a slight gain increase between 20 and 30 minutes. However, the small sample size prohibits detailed speculations on curve peculiarities.

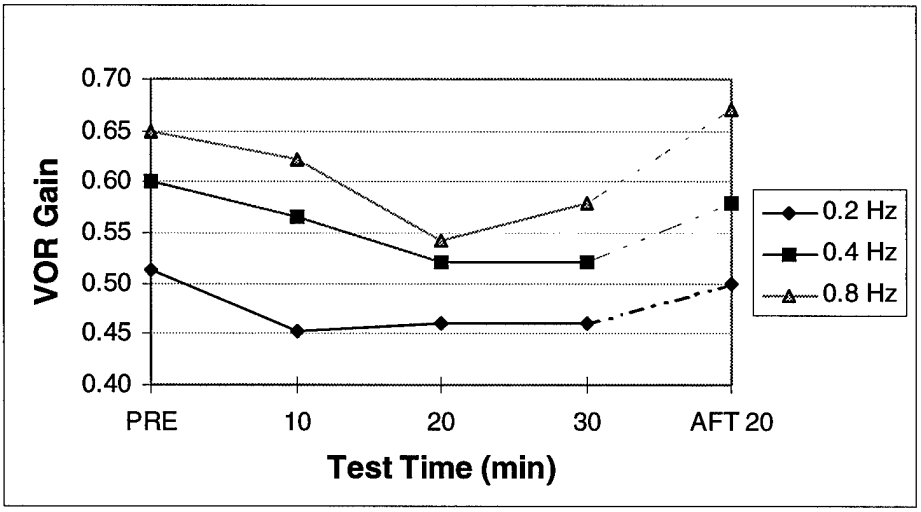

Figure 80: Longitudinal Gain Changes by Test Frequency[81]

[80] Dashed lines indicate the 20 min post-exposure period.

[81] Dashed lines indicate the 20 min post-exposure period.

7.8 DISCUSSION

Both subjects responded similarly and in accordance with the literature on adaptation time-course (Collewijn, et al., 1983; Welch, 1986). Subjects showed strong initial gain decreases within the first ten minutes, followed by less substantial changes thereafter. Using these data, a gain adaptation time constant can be roughly estimated to be 10 to 12 minutes. Note that gain adaptation would likely continue to occur beyond 30 minutes at a reduced rate.

However, it was interesting to note that neither subject showed any further gain decrease after 20 minutes. The reason for this is not entirely clear. The most likely explanation is that adaptation rate lessened after approximately 20 minutes and this decreased rate of adaptation was not picked up by the ISCAN system. Another possibility is that the images presented during the final 10 minutes were for some reason less conducive to VOR gain adaptation. This is less likely, as these images were successfully used in earlier experiments. Finally, some other uncontrolled factor may have been involved.

Implications of these data regarding the formation of optimal exposure time guidelines are quite interesting. Given that most of the gain change occurred early in the exposure period, it seems somewhat pointless to attempt to optimize exposure time to 'avoid the onset' of adaptation. However, another alternative for avoiding long re-adaptation effects exists given the results of this experiment. If a subject is repeatedly exposed to the same virtual interface for short periods of time, his/her adaptive mechanisms would be forced to adapt to the VE and then re-adapt to the real world several times in succession. This process could serve to speed up both adapting and re-adapting processes through the formation of adaptation sets (Parker, personal

communication, 1997; Shelhamer, et al., 1992). Re-adaptation time-course would thus be shortened, which lowers the risk of ill-effects due to the interface. Therefore, the subject's ability to quickly adapt could be employed to facilitate re-adaptation and lesson post-exposure safety risks. This is not a novel concept; only the application of an existing technique to virtual interfaces.

Once again, these data support the notion of generalized adaptation across frequencies. The three frequencies resulted in nearly the same gain change (10.5 to 13.3% decrease) over 30 minutes. However, while most gain change occurred early at 0.2 Hz, adaptation appeared to be more gradual at 0.4 Hz and 0.8 Hz. Perhaps the majority of early adaptation occurs at the dominant frequencies of head movements while adaptation at lesser encountered frequencies requires more time. Clearly more research is required to test this assertion.

CHAPTER 8: INCREMENTAL ADAPTATION EXPERIMENT

8.1 OBJECTIVES AND HYPOTHESES

The purpose of this experiment was to evaluate a fundamental concept underlying a potential rehabilitation technique for use with patients suffering from chronically low VOR gain. The concept, suggested by Viirre (1996), attempts to 'coax' VOR gain upwards using several small incremental increases in gain demand (e.g., 5 to 15%) instead of one large increase (50 to 100%). VOR gain mechanisms can work quickly and efficiently to compensate for small gain-change demands but are not able to ever completely compensate for large demands (Collewijn, et al., 1983). Patients with chronically low VOR gains face the latter problem in everyday life; there is too great a difference between their current gain level and normal levels which continually present a single, large, unachievable demand. If virtual interfaces are able to modulate VOR gain levels, perhaps this one large demand could be replaced by several smaller, more manageable demands experienced virtually.

The previous three experiments demonstrated that virtual interfaces can indeed modulate VOR gain levels and the first experiment demonstrated a link between image scale and change in VOR gain direction/magnitude. Therefore, small increases in VOR gain demand could be provided by slightly adjusting GFOV from the neutral condition (where GFOV = DFOV).

This experiment investigated an underlying premise supporting this proposed rehabilitation technique; i.e., that a series of small gain-change demands could result in higher overall VOR gain adaptation than one large gain-change demand over the same

period of time. This experiment addressed main objective 4 of this dissertation (See Chapter 1).

There are many issues to consider even with this one underlying premise. First, there is the question of how many increments to use and how big a gain change demand to provide with each increment. Should these demands be fixed in magnitude across increments or variable? Also, what are the criteria for switching from one step to the next? Should it be after a set period of time or should the subject's level of compensation dictate matters? These are all valid questions for which there are no solid answers. As a result, the configuration of this experiment represented initial conjectures. Subjects were provided gain change demands that varied slightly in magnitude over a series of 5 fixed intervals during a 30 minute exposure.

This experiment explored VOR gain increases instead of gain decreases for two reasons. First, gain increases are in the appropriate direction to aid those with chronically low VOR gains. Second, it was anticipated if the incremental method was effective, it would be more readily noticed in this condition (due to the lower magnitude gain changes observed in this direction)[82].

It was hypothesized that the incremental step method would result in a larger VOR gain increase than a single, large gain-change demand. The hypothesis is unavoidably qualitative at this point given the exploratory nature of this experiment.

8.2 SUBJECTS

Five subjects (3 male and 2 female, age range: 23 to 39) volunteered to participate in this experiment. All reported to be in good health with no history of visual-vestibular medical problems. Four subjects also participated in the Image Scale Experiment, so

[82] Reference the results of the Image Scale Experiment.

although they were not new to the virtual interface used, they had not experienced it in at least the previous 10 days. The fifth subject had not previously been tested.

8.3 EXPERIMENTAL DESIGN

A two factor within-subjects design was employed, with two levels of DEMAND (single, step) and two levels of TESTTIME (pre, post). Given that four subjects previously participated in the Image Scale Experiment and had already received the equivalent of a single gain increase demand stimulus (the MAG condition), they only needed to experience the incremental method of gain adaptation. DEMAND was thus confounded with session order, but this appears to be a minor issue given that past studies did not demonstrate any statistical effect of session on percent gain changes. The fifth subject was run only in the incremental method. Though the same three testing frequencies were used (0.2, 0.4, and 0.8 Hz), these were not separated out in the analyses (generalized adaptation had already been demonstrated in the three previous experiments).

The dependent variables were VOR gain and phase estimates, averaged across two trials per cell as before.

8.4 EXPERIMENTAL SET-UP AND APPARATUS

The set-up was as in the Image Scale Experiment (Section 5.4) except for the following changes. Subjects in the step condition received 5 virtual images as before (approximately 6 minutes per VE), but the image scale was increased for each successive VE presentation. Image scale began at 1.09X and progressed through 1.26X, 1.5X, 1.71X, and finally to 2.0X for the final image. This contrasts with the single demand condition, when subjects received an image scale of 2.0X for the entire 30

minutes. Image scale changes were driven by changing the GFOV in the WARP TV software. The minimum system time delay (48 ms) was employed throughout.

8.5 PROCEDURE

The procedure was as in the Image Scale Experiment (see Section 5.5) except for the following changes. No re-adaptation testing was conducted and no sickness testing was recorded (other than oral reports during, which were recorded as part of the safety protocol).

8.6 STATISTICAL ANALYSIS

VOR data were analyzed as in the Image Scale Experiment.

8.7 RESULTS

VVOR data are shown in Table 23. The virtual world VVOR data were collected with a image scale of 1.09X. VVOR phase showed an increase in lag in the VR condition.

Table 23: Mean VVOR Data (STEP Exp)

Viewing Condition	VVOR gain	VVOR Phase (deg)
Real World	*0.93*	*-3.1*
VR (1.09X)	*0.98*	*-18.4*

VOR gain and phase data by DEMAND are presented in Table 24. The subject who only participated in the STEP experiment did not show any gain change as a result of exposure. For ease of comparison, the STEP condition is shown both with and without

his data. Only the STEP condition with "N = 4" was used for statistical comparison with the corresponding SINGLE condition.

A one-tailed paired t-test indicated a strong trend toward an increase in gain within the STEP condition ($t(4) = 2.06$; $p < 0.06$). After verifying homogeneity of variance ($F = 0.24$; $p > 0.11$), a two sample t-test was performed comparing the two conditions with no statistical differences found ($t(3) = 1.94$; $p > 0.60$). The entire data set from the MAG condition is also included in Table 24 for comparison purposes.

Table 24: VOR Adaptation Data (STEP Exp)

Condition	N	Pre Gain	Post Gain	% Gain Change	Pre Phase (deg)	Post Phase (deg)	Phase Difference (deg)
STEP	*5*	*0.652 (0.10)*	*0.680 (0.10)*	***+4.6***	*0.0*	*-1.6*	*-1.6*
STEP (4 subjects)	*4*	*0.675 (0.09)*	*0.710 (0.09)*	***+5.0***	*0.1*	*-0.8*	*-0.9*
SINGLE (4 subjects)	*4*	*0.625 (0.09)*	*0.675 (0.11)*	***+8.0***	*-1.2*	*0.0*	*1.2*
SINGLE (total from MAG condition)	*9*	*0.608 (0.09)*	*0.644 (0.09)*	***+5.9***	*-0.6*	*-1.1*	*-0.5*

An informal comparison of DEMAND by subject found that two subjects had a greater gain increase in the STEP condition and the other two subjects had a higher gain increase in the SINGLE condition.

8.8 DISCUSSION

This experiment served as an initial investigation into the nature and value of the incremental step method. Due to the sample size used, only very large differences between the two methods would be capable of reaching statistical significance. However, the goal of this experiment was to search for trends favoring one technique

over another and also to gather knowledge that may help optimize the STEP technique for adaptation.

The VVOR gain data indicated once again that varying GFOV can create a gain change demand on the VOR adaptive system. In this case, the commanded gain change for the VR VVOR was small (approximately a 9% increase) and the VVOR gain response was more nearly compensatory (6% increase). The phase lag became greater in the VR VVOR, which has been previously hypothesized to be due to the effects of the minimum time delay along with the difficulty of tracking a virtual target that is stationary in virtual space but moving relative to real space.

The version of the STEP method used in this experiment did cause a VOR gain increase to occur that nearly reached statistical significance (at the 0.05 alpha level) after testing only 5 subjects. This indicates that it is at least a viable alternative technique for increasing VOR gain.

The comparison between the STEP and SINGLE methods were inconclusive. The failure of the difference between the methods to reach statistical significance is of minor importance given the small sample size. However, there appeared to be no trends favoring one method over the other. Looking only at the four subjects that used both methods, more adaptation occurred in the SINGLE method (8% increase versus a 5% increase). Expanding the sample sizes to include all subjects who used either method, the results still favor the SINGLE method but the difference between the two methods is reduced (5.9% versus 4.6%).

However, this emerging trend falters upon more investigation. Of the four subjects who used both methods, half performed better with the STEP technique. Additionally, one of the four data points (a data point being the average overall percent gain change per subject) in the SINGLE condition was twice as large as any other data point in the

STEP or SINGLE condition. This value was 23% gain increase and was not considered an outlier when compared to all the subjects who participated in the MAG condition. Therefore, it was not excluded from the analysis. However, this value disproportionally influenced the average gain increase in the SINGLE condition. Without it, the average gain increase in the SINGLE condition is 3.65, which is lower than the STEP average.

Lastly, the baseline gain of the VOR varied (within the same group of four subjects) between the two methods by close to 10 %. This resulted in a significantly higher pre-exposure gain in the STEP condition which could potentially have effected the results (e.g., if the subjects were overly alert during pre-gain testing in the STEP condition, larger increases in gain could be masked by the elevated pre-gain estimates). However, an individual's baseline VOR gain estimates are known to be variable over time, so the most accurate procedure for assessing VOR adaptation is to utilize the baseline gain measured just before the exposure period begins (versus some averaged measure over a longer period of time). Larger sample sizes would likely reduce this baseline gain discrepancy.

The results of this study are therefore inconclusive as to the relative benefits of the two methods. However, these results have demonstrated that the STEP approach is a valid method for increasing the gain of the VOR. Furthermore, the specific configuration of the STEP method employed in this experiment is likewise supported. These results, combined with Viirre's work with clinical patients (Viirre, Draper, Gailey, Miller, & Furness, In Press) provide a base of support from which to further study this method. Future experiments should concentrate first on optimizing the STEP method and then comparing the optimized version to the single method.

A few words on the phase data are in order. The phase appeared to decrease (lag) more substantially in the STEP method (as compared to either the SINGLE method: 4 subjects or the entire sample from the MAG condition). Assuming that these results are

confirmed by further research, this presents an interesting question. Why would phase adaptation more readily occur in the STEP method when the only parameter varying between the two methods is visual scene amplitude? The only plausible explanation is that phase adaptation is somehow adversely influenced by increasing amplitude of visual scene motion. A discussion of this possibility is presented in the next chapter.

CHAPTER 9: GENERAL DISCUSSION

9.1 INTRODUCTION

This chapter integrates the results found in these four experiments with results found in the literature to address the five main objectives of this dissertation. For clarity, the objectives are restated below. Additionally, some overall thoughts are presented regarding the relevancy of continuing to consider the VOR as a separate, independent oculomotor system, given the results found here along with other recent findings.

The five main objectives, as stated in Chapter 1:

1. Test General Hypothesis 1, which argues that specific sensory rearrangements of virtual interfaces drive VOR adaptation. Characterize any resulting adaptation in terms of magnitude achieved and time-course of re-adaptation following exposure.

2. Determine whether these same sensory rearrangements modulate simulator sickness and which of several existing theories of simulator sickness best fit the data.

3. Test General Hypothesis 2, which asserts that magnitude of VOR gain adaptation correlates with simulator sickness magnitude.

4. Given that objective 1 is satisfied, assess the viability of a proposed rehabilitation technique for increasing VOR gain.

5. Use results from this research to aid in the development of guidelines for virtual interface design that will minimize unintended VOR adaptations and/or simulator sickness symptoms.

9.2 OBJECTIVE 1: EXAMINE GENERAL HYPOTHESIS 1

Both VOR gain and phase adaptation consistently resulted from exposure to the virtual interface. Therefore, this discussion has been separated into two sections: VOR gain adaptation and VOR phase adaptation.

9.2.1 VOR GAIN ADAPTATION

Issues discussed in this section include the overall finding of VOR gain adaptation to virtual interfaces, how these interfaces can modulate VOR adaptation magnitude and direction, the resulting time-courses of adaptation and re-adaptation, and the concept of frequency specificity.

First, a few words are in order regarding the baseline VOR gain values obtained in this research. Baseline gain in these experiments (0.61 in the Image Scale Experiment and 0.62 in the Time Delay Experiment) are lower than some baseline values reported in the literature. Two factors address this discrepancy: subject tasking and statistics. In these experiments, subjects performed mental arithmetic during VOR testing while other research often had subjects imagine a point in the distance. This latter method (i.e., EVOR) results in higher VOR gains due most likely to the influence of a separate fixation system. Second, this experiment did not normalize VOR gain values to perfect compensation, VVOR data, etc. as some other researchers have. Using mental arithmetic and without normalizing the data, Robinson (1981) reported the average baseline value of the VOR to be approximately 0.65, which compares favorably with the data obtained here.

9.2.1.1 Overall finding: VOR Gain Recalibrates to Virtual Interfaces

All four experiments in this dissertation recorded VOR gain adaptation to virtual environments. This is rather remarkable given the short exposure period, the active,

natural, unrestricted, and intermittent nature of head movements produced by the subject in order to perform the task, and the approach to finding any occurring gain adaptation (three testing frequencies determined apriori). Nevertheless, VOR adaptive mechanisms responded consistently within and across experiments. This speaks to the power of the virtual interface as a stimulus for adaptation. As a result of these experiments, it can be stated with certainty that head-coupled virtual interfaces can indeed alter VOR response in a relatively short period of time.

This finding has many potential consequences, both positive and negative. VOR adaptation processes are often accompanied by oscillopsia and occasionally symptoms of simulator sickness. In addition, adaptation requires a re-adaptation period upon termination of the exposure. Alternatively, proper application of virtual interfaces to systematically control oculomotor response holds promise as a therapeutic aid. These possibilities are discussed further below.

9.2.1.2 Gain Adaptation Varies with the Virtual Interface

Simply finding that VOR adaptation occurs in typical interactions with virtual interfaces is interesting but not very useful information[83]. As stated in the introduction, a detailed characterization of VOR adaptive response is required for each potentially salient design parameter. This research, upon first demonstrating VOR adaptation to interactive virtual interfaces, has taken the initial steps toward understanding what parameters modulate this effect. The effects of image scale and system time delays are discussed below.

The Image Scale Experiment demonstrated that VOR gain can be stimulated to increase, decrease, or remain stable depending on the setting of a single parameter,

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[83] This is akin to experimental findings proclaiming that "simulator sickness occurs in VR". It is nice to know but does not help designers avoid such effects.

GFOV. It is ironic that such a poorly understood parameter can have such robust effects on the oculomotor response of the subject. GFOV, in isolation, is essentially a meaningless angle; it defines the angular extent of the virtual scene that is imaged by the 'virtual camera'. However, when combined with a specific DFOV of a virtual interface, this variable defines the magnification factor of the scene, the corresponding optic flow rate for head movements, and any associated perspective distortion of the resulting virtual image. As a result of this research, GFOV is now known to also present a VOR gain change demand when it is not equal to DFOV.

Theoretically, the gain change demand equals the magnification level of the visual stimulus (determined by the ratio DFOV/GFOV). However, the VOR gain can never fully compensate for large increases in gain demand (Collewijn, 1989). VVOR gain also fails to completely compensate for large increases in magnification, which has been hypothesized to be due in part to the activation of other oculomotor processes including corrective saccades (Section 5.8.1). Actual VVOR gain data obtained in this experiment may represent more achievable limits of VOR gain recalibration.

An unexpected result occurred in the Time Delay Experiment. VVOR data indicated the existence of a significant gain decrease demand and the VOR gain did decrease approximately 8 to 10% after 30 minutes, even though this gain decrease was not predicted by the physics of the stimulus[84].

The VVOR results imply that the gain decrease may be a result of difficulties in visually tracking the virtual target. The hypothesis explaining the obtained VOR adaptation is likely more complex, though it is similar in that other oculomotor systems are implicated[85]. There is likely a complex, multi-factor adaptation process that

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[84] This lack of a physical gain decrease demand is readily verified from the equations of Section 3.1.4. However, an aggressive yet unsuccessful attempt was made to falsify them once it was discovered that they would not fit the obtained data!

[85] See Section 6.8 for a detailed explanations of these VVOR and VOR adaptation results.

incorporates many oculomotor movements and central processes to compensate for existing visual-vestibular rearrangements. The hypothesis, fully described in Section 6.8.3, is briefly restated below.

Visual movement delay in response to the onset of head motion likely resulted in the brief occurrence of a VOR gain suppression stimulus (the duration of which would equal the magnitude of the existing system time delay). Since the 30 min exposure period was a long series of repeated starts and stops, the effect of this VOR suppression stimulus may have been amplified over time, resulting in the observed VOR gain reductions.

This hypothesis assumes that the VOR adaptation system weights more strongly the image slip detected during head motion than the retinal slip detected after head movements stop. This is an open question, though it seems reasonable given the existence of efference copy in this experiment. Intentional head movements imply an expected visual scene motion in response. Efference copy information serves to strengthen expectations regarding this visual motion (Collewijn, 1989). Therefore, resulting retinal slip during head movements more directly implies a failure of the VOR and visual systems to compensate. When the head ceases to move, relative scene motion is still experienced but without concurrent vestibular information or efference copy from head movements. Therefore, resulting retinal slip in this case is more likely to be corrected for by a saccade or smooth pursuit (at least initially) rather than through modification of the VOR.

In summary, VOR gain can be modulated by varying image scale of the VE and by increasing system time delay beyond approximately 50 ms. In addition, the focus on a simplistic VOR adaptation process to account for visual stabilization seems to be losing support in the field as research continues to point towards a more integrated system of

oculomotor adaptation (Shelhamer, et al., 1994; Melvill Jones, et al., 1988; Collewijn, 1989; Istl-Lens, et al., 1985).

9.2.1.3 Gain Adaptation and Re-Adaptation Time-Course

Only one experiment investigated the time-course of VOR gain adaptation to the virtual interface. The results suggest that VOR gain adaptation follows a decaying exponential function, with much adaptation occurring within the first 10 to 20 minutes of exposure to the virtual interface. This conclusion is supported by other research on VOR gain adaptation time-course (Collewijn, et al., 1983).

Regarding re-adaptation, the first two experiments demonstrated that VOR gain levels did not always return to baseline levels within 10 minutes after exposure. This was somewhat surprising because of the moderate levels of gain adaptation achieved and the short exposure period. Perhaps the strength of adaptation is influenced by the task involved (active, unrestricted head movements and goal-directed behavior). Nonetheless, when subjects were allowed to get out of the chair and move freely around for 20 minutes before post-exposure testing, gain levels fully returned to normal. Whether the primary influence for the successful re-adaptation was the additional time or the freedom of movement is left to future research. In addition, contextual cues (i.e., the chair and HMD) may also influence re-adaptation time-course. However, these results provide information on re-adaptation time-course limits for the exposure period involved (30 min).

9.2.1.4 Frequency Specific versus Generalized Adaptation

An unexpected but consistent finding from these experiments refutes the notion that all VOR gain adaptation is frequency specific. These data support the alternative that

VOR adaptation is more generalized across naturally occurring head-movement frequencies, at least within the range of 0.2 to 0.8 Hz.

As discussed in Section 6.8.4, these results are consistent with previous research once differences in experimental protocol are considered. The two major protocol differences include interaction method and range of test frequencies, both of which are discussed below.

Lisberger's model of frequency specificity is based solely on exposure conditions that involved passive sinusoidal motion at one adapting frequency (Lisberger, et al., 1983). This is not the protocol used in this experiment. Lisberger himself found that active motions during exposure to the adapting stimulus resulted in no evidence of frequency selective adaptations and others have also shown that passive exposures at multi-frequency rotations result in generalized adaptation (Powell, et al., 1991). These exceptions to frequency specificity suggest multi-frequency exposure as the modulating variable, though active head motions may have contributed to Lisberger's findings.

However, an argument can be made that, in both of these cases, frequency specificity was simply masked by a broadband equal-amplitude train of sinusoids that formed the head motion. Lisberger did not attempt to quantify head motions during the exposure period of his active head movement experiment so it is difficult to precisely test the concept of frequency specificity in this case (Lisberger, et al., 1983). Powell, et al. (1991) used passive rotation, but the multi-frequency head movements were composed of several equal-amplitude single sinusoids.

In contrast, this dissertation did quantify head movements during adaptation and found generalized adaptation though the vast concentration of head movement power was below 0.2 Hz. This suggests the VOR adaptation system may actually extrapolate

adaptation across a large range of head movement frequencies if active, goal-driven head movements are involved.

The second difference involves the range of test frequencies over which frequency specificity was evaluated. Lisberger, et al. (1983) used a range of 0.1 to 4.0 Hz and often only found noticeable changes in gain for very widely separated frequencies. Powell et al. (1991) utilized a reduced frequency range of 0.05 to 1.0 Hz for testing, but also often found meaningful gain changes only when comparing the extremes of that range. Conversely, the experiments of this dissertation found generalized adaptation to a further reduced set of test frequencies (0.2 to 0.8 Hz). Demer, et al. (1989) also found generalized VOR gain adaptation when comparing across a reduced range of frequencies (0.01 to 1.0 Hz). His subjects were passively rotated at one of 4 frequencies prior to testing, which combined with the results of Powell, et al. (1991) suggests that active vs. passive movement in isolation may not be a large factor in defining frequency specificity.

It is likely that a combination of factors determines whether frequency specificity will occur in VOR adaptation studies: the number and associated magnitudes of head movement frequencies during exposure, the range of testing frequencies, the existence of active, unrestricted head movements (which provides efference copy and proprioceptive influences), and possibly the nature of the task during exposure. Therefore, frequency specificity may be a special case of a more general, complex adaptation system that utilizes multiple inputs to adapt the VOR gain across several relevant frequencies, even those seldom invoked.

9.2.2 VOR Phase Adaptation

This section describes VOR phase data in terms of overall findings, specific contributions of time delays and scale changes, frequency specificity, and re-adaptation time-course.

9.2.2.1 Overall Findings

Statistically significant VOR adaptation was demonstrated in both the Image Scale Experiment and the Time Delay Experiment. Given that there was a phase lag demand (created by the system time delay involved), the VOR was mildly compensatory in its response.

Statistical significance, however, does not necessarily imply that a meaningful change occurred. The implications of a 1.5 to 3.0 degree increase in phase lag are hard to ascertain. If the VOR system was working purely in isolation to stabilize images on the retina, any change in phase would effect vision as the fovea itself only subtends 2 deg of the central visual field. Meaningfulness could also be evaluated using the knowledge that retinal slip results in a degradation in visual acuity if the slip velocity exceeds 2.5 deg/s (Collewijn, 1989). However, the VOR does not work in isolation. It works in concert with other oculomotor responses (e.g., OKN and pursuit)[86] which makes interpretation of the practical implications surrounding such small VOR phase changes very difficult. More research is required to determine the effects of these phase changes on visual performance and the perception of oscillopsia.

9.2.2.1 Phase Adaptation as a Function of Time Delay and Scale Change

Small phase changes occurred but did not vary with increases in system time delay (125 to 250 ms) in the Time Delay Experiment. However, it appears that phase

[86] The increased propensity for saccades with increasing time delay may also be related to these phase changes.

adaptation magnitudes did differ between the minimum time delay used in the Image Scale Experiment (48 ms: resulted in a 1.6 deg increase in phase lag) and those used in the Time Delay Experiment (125 and 250 ms: both of which resulted in a 2.7 deg increase in phase lag). Note that the observed phase changes were not nearly compensatory for demand. One possible explanation for these results is that VOR phase adapts in a sluggish fashion in response to small phase change demands and intermittent head motion, but as demand increases (above 125 ms), the phase adaptation system saturates.

However, note that only in the NEU condition (i.e., 1.0X magnification) of the Image Scale Experiment did a large change in phase occur (-3.1 deg). Since the Time Delay Experiment also utilized 1.0X image scaling, it is perhaps more appropriate to only consider the phase changes in the NEU condition when comparing across experiments. Considering only conditions of neutral scale, it appears that increasing system time delay did not increase phase adaptation magnitude.

The above statement suggests two interpretations. First, VOR phase adaptation did universally occur to a small extent but was unaffected by increasing phase demands. This implies one or more of the following: 1) the time delays used were too large (with excessively large phase change demands) resulting in the saturation of the phase adapting system, 2) the nature of the exposure (intermittent head movements, start and stop motion) was insufficient to effectively stimulate phase adaptation with a consistent phase demand, 3) the duration of exposure was too short for phase adaptation to occur, and/or 4) the phase adapting system is simply not very sensitive.

The exposure time employed may indeed be too short for significant phase adaptation to appear, though there is a paucity of research that addresses this issue. Previous research does suggest that the phase demands used in this experiment were not excessive when the nature of stimulus exposure was confined to a specific frequency

(Kramer, et al., 1995), which implicates the intermittent, multi-frequency nature of exposure as attenuating adaptation. Therefore, all of the above factors may have contributed to these results and further research is required to decipher their relative influence.

A second conclusion of the revised finding is the conjecture that phase adaptation may be contingent to some extent on VOR gain change demand. The Image Scale Experiment resulted in substantial changes to the VOR phase only in the NEU condition (though this difference failed to reached statistical significance). Additional support for the notion that image scale factor may effect the performance of the phase adaptation system was gathered in the STEP experiment. The STEP protocol incrementally increased image scale from 1.09X to 2.0X over the 30 minute exposure. The largest phase change (-1.6 deg) occurred using the incremental step method, which involved a larger percentage of exposure time at scale factors closer to 1.0X than in the single step method (which had a fixed scale factor of 2.0X). In addition, the -1.6 deg phase adaptation occurring in the incremental step method did not approach the phase change magnitudes achieved when using the neutral image scale throughout the exposure (-3.1 deg, found in the NEU condition of the Image Scale Experiment). This suggests that, in order to recalibrate effectively, the phase adaptation system requires an expected visual flow rate in response to head movements.

The above thoughts are merely speculation at this point. Though the data indirectly support the notion that image scale effects phase adaptation, the observed differences failed to achieve statistical significance. Though other research has indicated a potential coupling of the phase and gain adaptation systems (Kramer, et al., 1995), data could not be uncovered that specifically addresses a modulating influence of gain change demand on resulting phase adaptation. Clearly, this issue requires a closer look in future research.

9.2.2.2 VOR Phase Adaptation and Frequency Specificity

A discussion of whether VOR phase adaptation data was frequency specific or generalized across frequencies is an awkward task because, given a fixed time delay, phase demand varies with frequency. Two hypothesized strategies for phase compensation across frequencies were presented in Chapter 3. Strategy 1 (phase adaptation increasing with increasing frequency) definitely was not supported. Strategy 2 (phase adaptation demonstrating frequencies specificity) could be neither supported nor refuted because of the low magnitudes of phase adaptation achieved. In the Image Scale Experiment, the greatest phase changes occurred at 0.2 Hz. However, the Time Delay Experiment found the greatest phase changes at 0.8 Hz. Given that both experiments involved similar peak head movement frequencies (which were below 0.1 Hz), these results are inconclusive as to frequency specificity.

However, a third strategy is possible. VOR phase may have adapted only to the peak frequency of head movement and this change remained consistent across all other frequencies. The phase demand at 0.1 Hz (approximate peak frequency of head movement power in each experiment) was 1.7 deg in the Image Scale Experiment and either 4.5 deg (125 ms delay) or 9 deg (250 ms delay). Using this strategy, actual phase adaptation was fully compensatory in the Image Scale Experiment and less compensatory as demand increased. Therefore, this strategy is not fully supported by the data, unless the decrease in compensation with increasing time delay could be accounted for through the nature of the interaction during exposure.

An alternate metric of phase adaptation that accounts for changing phase demand with frequency is percent of adaptation demand achieved at each frequency. This metric could obviously support the notion of frequency specificity in these experiments, given the doubling of phase demand at each increasing test frequency without concurrent doubling of actual phase adaptation. However, as discussed in Section 6.8.4, this

metric would likely fail to support the notion of frequency specificity if head movements were restricted to a single high frequency during exposure but tested across several frequencies.

9.2.2.3 VOR Phase Re-Adaptation Time-Course

VOR phase re-adaptation time-course is also difficult to decipher given the small magnitude of changes observed in these experiments. However, both the Image Scale and Time Delay Experiments failed to show complete re-adaptation of VOR phase after 10 minutes. Phase data were not analyzed in the Longitudinal Experiment because the sample size was too small for meaningful interpretation, given the increased variability of the phase estimates as compared to the gain estimates. Therefore, based solely on the results from these experiments, phase adaptation was not fully dissipated after 10 minutes of re-exposure to the real world.

9.3 OBJECTIVE 2: EXAMINE SIMULATOR SICKNESS EFFECTS

This section summarizes the effects of image scale and time delays on simulator sickness. Additionally, various theories of simulator sickness are critiqued using the results obtained in these experiments, and this is followed by a few additional comments.

9.3.1 EFFECT OF IMAGE SCALE

Image scale deviations from the neutral condition increased incidence of simulator sickness. In fact, simulator sickness reports doubled in the MIN and MAG conditions as compared to the NEU condition.

The provocative stimulus was not simply optic flow rate because this rate varied by a factor of four between the MIN and MAG conditions with no significant difference in

sickness between these two conditions. Therefore, the provocative variable appears to be the difference between sensory expectations and actual sensory input, per the sensory rearrangement theory.

Though the difference was not significant, future research in this area may discover that image minification is more provocative than image magnification. Both subjects who prematurely ended a session in the Image Scale Experiment did so in the MIN condition. Additionally, two other subjects informally remarked that they felt ill for an extended period of time following the MIN session only.

9.3.2 Effect of System Time Delay

Fixed time delays do not appear to be especially provocative. This assertion arose from the Time Delay Experiment, where no differences in sickness occurred despite increasing the time delay from 125 to 250 ms. It was further supported when the results of the NEU condition of the Image Scale Experiment were compared to those of the Time Delay Experiment, effectively increasing the range of time delays from 48 to 250 ms (see Section 6.8.6). Though different subject groups were used in the two experiments[87], the magnitude of each sickness metric was similar across all three time delay conditions. In addition, the overall magnitudes of sickness reported (by all three metrics) were approximately half those of either the MIN or MAG condition from the Image Scale Experiment. Thus, all the data obtained in these experiments indicate that time delays alone do not have a large influence on simulator sickness.

A few caveats are in order, however. First, the time delays used in these experiments were approximately fixed at set values. Variable time delays can also occur in virtual interfaces, especially those that use large-scale VEs along with real-time graphics rendering. It may be that these variable time delays are provocative.

[87] These groups were similar in many important respects, however. See Section 6.8.6.

Secondly, only head rotations were tracked in these experiments. Time delays associated with linear translation in virtual environments also need to be investigated, though it should be noted that Uliano, et al. (1986) found no effect of time delay on sickness using mainly translational motion in a simulator.

9.3.3 A Critique of Simulator Sickness Theories

This section reviews how well available theories of simulator sickness predicted the results of these experiments. Theories covered include the sensory rearrangement theory, the subjective vertical theory, the postural instability theory, and optic flow models (see Section 2.4.4). Griffin's reflex-response theory and Ebenholtz's extraocular afference theory are not defined well enough to be specifically critiqued by these data.

Sensory rearrangement theory is by far the most accepted theory of motion and simulator sickness. This theory correctly predicted the results obtained in the Image Scale Experiment, which is quite an achievement given that none of the other theories did so. However, this theory failed to predict the non-effect of increasing system time delay. It is obvious that system time delays cause visual-vestibular sensory rearrangements, given the VOR phase and gain adaptation that occurred. However, simulator sickness was not effected by these sensory rearrangements. Therefore, the theory failed to predict the obtained results.

Where does this leave the sensory rearrangement theory? Oddly enough, with the same status it had coming into these experiments. Sensory rearrangement theory explains a good portion of existing simulator sickness research but it offers little predictive power as to which sensory rearrangements are provocative and which are not. Given that most sensory rearrangements that arise are not provocative[88], this failure to

[88] If they were, we would all likely vomit in response to each new experience in our lives.

predict is a major drawback of this theory[89]. If addition, ambiguities surrounding the notion of the internal store and neural comparator make the theory somewhat untestable. However, it does explains many findings involving simulator sickness which no other theory has been able to do.

Given that the sensory rearrangement theory seems to closely but not perfectly model the motion/simulator sickness process, many variations of this theory have arisen. The subjective vertical theory of motion sickness states that only motions that involve otolith stimulation have the potential to be provocative; pure yaw head movements are not effective at inducing simulator sickness. The data collected in this experiment contradict this theory, as simulator sickness was induced though the vast majority of head rotations were in yaw. In addition, this theory does not readily explain the differences in sickness across conditions in the Image Scale Experiment. However, given that head tilts were occasionally performed by the subject, these data do not directly refute the subjective vertical theory.

The postural theory of motion sickness does not appear to explain these data. This theory argues that motion sickness and simulator sickness are caused by extended periods of postural instability (degraded control rather than complete loss of control) brought about by interaction in a human-environment system. Therefore, if postural control behavior is fully restricted, simulator sickness should not occur. These data were collected while the subject was firmly strapped to a chair using a five-point harness and foot straps. Therefore, for all practical purposes, the subject was immobilized except for head and arm movements. There was little to no need for overt postural control behavior, yet sickness still occurred.

[89] And any existing theory for that matter, as theories are judged according to their ability to predict.

Nevertheless, proponents of the postural stability theory would likely argue that postural control behavior was not *fully* constrained which allows for the possibility that differences in postural control requirements between the different conditions could be manifest as subtle alterations in posture. However, pilot studies were subsequently carried out on the Chattecx posture platform to determine the relative effects of image scale changes on postural control. Subjects performed oscillatory yaw head movements (at 1.0 Hz) for 15 seconds while viewing images in each of four conditions (i.e., the MIN, NEU, MAG and 250 ms conditions). These studies indicated no effect of image scale on postural control whether the subject fixated on a target in the VE (N = 2; Figure 81) or changed his/her gaze from a central virtual target to a virtual target 60 deg left and 60 deg right during the oscillations (N = 4; Figure 82). This lack of effect is noteworthy given that this platform was designed to detect subtle changes in postural control. Therefore, restrictions on subject postural control during the Image Scale Experiment combined with the results of these pilot studies appear to contradict the postural control theory of simulator sickness.

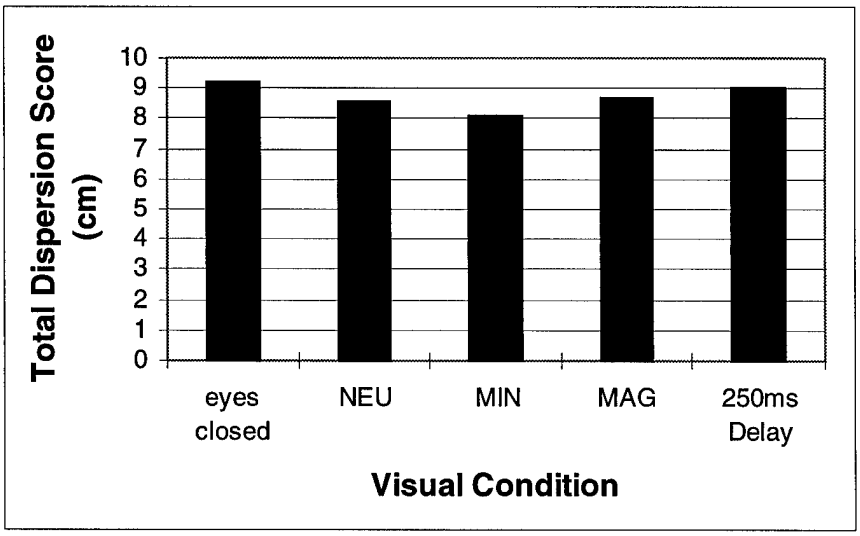

Figure 81: Postural Stability by Visual Condition (fixated on virtual target)

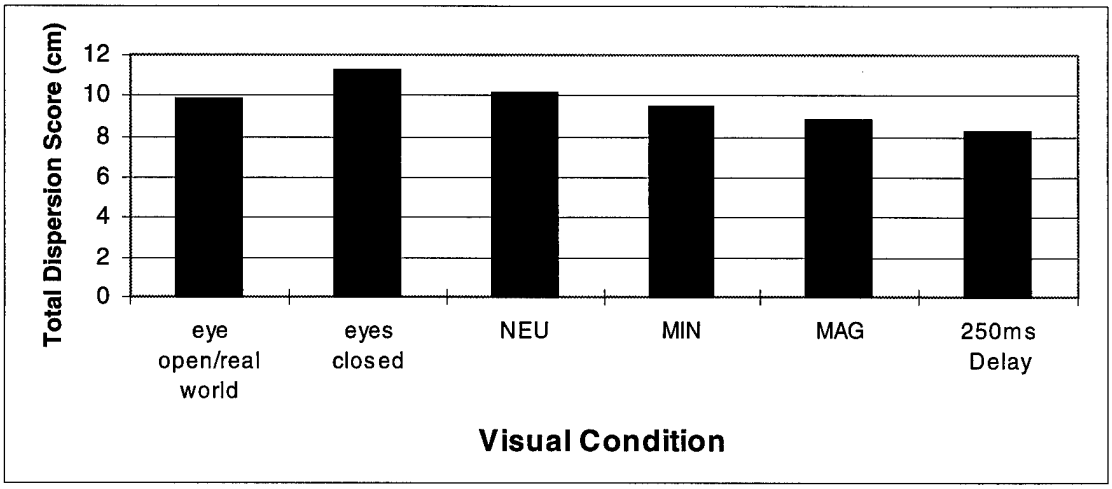

Figure 82: Postural Stability by Visual Condition
(gaze shifts within the VE)

Finally, there has been some interest in exploring the influence of optic flow on simulator sickness. Some researchers have attempted to characterize which parameters of optic flow correlates with sickness (Kennedy, Berbaum, & Smith, 1993) while others have advocated increased optic flow as being a significant contributor to sickness, based upon data from flight simulators (McCauley & Sharkey, 1992; Hettinger & Riccio, 1992). This research does not support a *direct* relationship between optic flow and simulator sickness. Subjects became sick regardless of whether the optic flow was reduced or increased from that normally expected. Thus it is concluded that although optic flow may indeed contribute to simulator sickness in flight simulators, it appears to be less influential in head-coupled virtual interfaces. This difference in sickness result between simulators and head-coupled virtual interfaces is addressed in a proposed classification system for simulator sickness research (Appendix G).

9.3.4 Other Issues Concerning Simulator Sickness

One issue that is often ignored in simulator sickness research is the contribution of hardware fit on simulator sickness reports. Simulator sickness is mainly considered to be motion sickness symptoms experienced in virtual environments. While this is indeed a significant part of the syndrome, there can be no denying that a virtual interface user can be made to feel uncomfortable strictly due to hardware issues such as HMD weight and fit, misadjustment (or lack of adjustment) of the optics and IPD, heat generated as a result of wearing the HMD, tethering constraints of head trackers, etc. These hardware issues are often ignored because there is no way to separate out these effects on the current SSQ. Below is a proposal[90] for classifying simulator sickness in terms of the underlying nature of the stimulus, followed by possibilities for modifying the SSQ in order to capture any HMD-related influences on simulator sickness.

Simulator sickness can be parsed into three components: 1) sickness that results from an accurate simulation of a provocative environment in VR, 2) sickness caused by simulation inaccuracies, and 3) sickness caused by hardware. The first component is essentially pure motion sickness. If the virtual simulation of a nauseogenic situation results in nausea, then this simulation accurately triggered the desired effect. As virtual interfaces progress and simulation inaccuracies/hardware problems decrease, this will become the major contributor to simulator sickness. However, since motion sickness is desired and expected in this condition, it is not the concern of this dissertation. The second component of simulator sickness occurs inadvertently/unavoidably due to inaccuracies in the simulation of a fundamentally benign environment. This sickness, more aptly termed 'interface sickness', is the focus of this dissertation and most other simulator sickness research. The goal is to eliminate those factors of the virtual simulation which cause a benign environment to become provocative. The last sickness

[90] developed through several helpful conversations with a fellow graduate student, Jerry Prothero.

component is appropriately termed 'hardware sickness'. Improvements in HMD ergonomics will largely eliminate this component.

A problem that exists with current metrics of simulator sickness is that one cannot determine if the sickness reported is due to motion-, interface-, or hardware sickness. The parsing out of the first component (true motion sickness) could simply require a matching of diagnostic profiles (perhaps using the same subscales of the existing SSQ and/or MSQ) and incidence rates between real environment and simulated environment. However, this is a non-issue for the vast majority of virtual interfaces which seek to simulate only benign environments. Thus the problem becomes one of separating out hardware sickness from interface sickness.

How does one isolate the negative effects of hardware from those due to the inaccuracies in the simulation? Two possibilities come to mind. One strategy is to collect a large pool of data purely on HMD comfort using the SSQ as the main metric with space for subjects to write additional comments. These HMDs would need to be specially modified so that subjects could view the outside world through existing optics. The resulting symptom profile could then be used as a hardware-specific template when investigating virtual interfaces that utilize HMDs. A second strategy is to consult an expert on ergonomics on how best to modify the SSQ to optimize diagnosis of hardware effects. Additionally, there may be existing questionnaires designed to detect hardware fit/comfort that could be combined with the SSQ to better diagnose the cause of reported symptoms

9.4 OBJECTIVE 3: TEST GENERAL HYPOTHESIS 2

This hypothesis was not supported by the data. Correlation values between percent VOR adaptation and simulator sickness never exceeded 0.30. This result is strengthened

by similar findings in the literature (Watt, 1987; Bouyer & Watt, 1996) though it contradicts other findings (Gordon, et al., 1996).

This result appears reasonable when one considers the relative influence of experience on these two processes. Successive exposures to sensory rearrangements often result in a decrease in reported simulator sickness but continued VOR adaptation. Therefore, the most likely conclusion is that VOR adaptation and simulator sickness are not strongly correlated.

However, given that Hypothesis 2 was based on the existence of a general adaptability trait within individuals, it is possible that the wrong VOR metric was used to assess a subject's adaptability. Percent VOR gain change from baseline values was used in these experiments. It is possible that a more pure measure of adaptability is actual *rate* of VOR adaptation. A proposed experiment to explore the relationship between VOR adaptation rate and simulator sickness is presented in Chapter 10.

9.5 OBJECTIVE 4: VIRTUAL INTERFACE AS A REHABILITATION TOOL

The STEP Experiment did not demonstrate an improvement in VOR adaptation efficiency with the incremental step method over the single step method. However, there was much to support a continued investigation of this technique. For instance, the STEP method did increase VOR gain over the 30 min exposure which makes it a valid technique for comparison. Second, there are many possible variations of the incremental method, only one of which was explored in this dissertation. Third, preliminary work suggests that the incremental technique can increase the gain in subjects with chronically low VOR gain levels (Viirre, Draper, Gailey, Miller, & Furness, In Press).

However, there are also some significant issues that need to be addressed before the STEP method can be given serious consideration as a rehabilitative tool. For instance, this technique is designed to incrementally drive a low VOR gain back towards normal. If real-world interaction provides too great a gain change demand, this technique will have to utilize virtual interfaces with gain demands below 1.0X (the NEU condition). Subjects with normal VOR gain will respond to a VOR gain demand below 1.0X by decreasing VOR gain. Since the goal is to raise the VOR gain in those with chronically low levels, will VOR gain demands that are below 1.0X stimulate a gain increase or gain decrease in these patients? Initial data suggests that the VOR gain will increase (Viirre, Draper, Gailey, Miller, & Furness, In Press).

A second concern involves the incremental procedure. Assuming a viable means of incrementing VOR gain upwards is possible, how can these values be maintained between rehabilitative sessions? A common characteristic of the VOR is to revert to baseline conditions after termination of the exposure to a sensory rearrangement. A means must be found to retain newly obtained levels over time. The concepts of adaptation sets and contextual adaptation might be part of the solution. Repeated exposure to the gain demand, combined with additional context cues that overlap from exposure period to real world with increasing durations, might help to extend the adaptive effects further into real world situations.

9.6 OBJECTIVE 5: PRELIMINARY VIRTUAL INTERFACE DESIGN GUIDELINES

The following list serves as preliminary design guidance for the development of virtual interfaces that minimize the influence of negative effects (simulator sickness and undesired re-adaptation processes). These guidelines were based primarily on the data obtained in this dissertation, though outside research was leveraged as appropriate to solidify or extend these recommendations.

- Avoid image scale deviations from 1.0X magnification to reduce potential VOR gain adaptations and simulator sickness. This can be accomplished by equating GFOV and DFOV in both the horizontal and vertical directions (assuming a spatially accurate model of the virtual environment exists).

- Verify that the gain of the head tracking system is accurately calibrated to minimize simulator sickness and VOR gain adaptation through unintended changes in optic flow rate during head rotations[91].

- Minimize system time delays to reduce VOR gain and phase adaptation. In addition, though time delays have not been shown to effect magnitude of simulator sickness, they can effect user performance and satisfaction levels. However if the objective is solely to reduce sickness levels, look elsewhere[92].

- Allow 20 minutes minimum after a 30 minute exposure to a virtual interface for VOR gain and phase levels to return to pre-exposure conditions. Allow the subject ample opportunity to interact with the real world during this time. Note that other adaptation processes, including simulator sickness, may not follow the same time-course. Also note that increasing exposure time will likely increase re-adaptation duration requirements.

- Limit VE exposures to under five minutes to minimize simulator sickness and undesired VOR adaptation effects. Exposure durations beyond 5 minutes may result in significant VOR gain adaptation, with adaptation likely following a decayed exponential function. Simulator sickness increases with exposure time.

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[91] Though actual empirical evidence for this recommendation does not yet exist, it is a logical extension of this research.

[92] This guidance is currently limited to fixed time delays and rotational motion only. The need for further research that examines variable time delays and linear motion is discussed in Chapter 10.

- For a specific virtual interface and exposure duration, large DFOVs will likely increase simulator sickness, VOR adaptation rate and magnitude, and other adaptation processes. However, small DFOVs can still cause these events to occur as well.

- Physiological adaptations and simulator sickness may be minimized by providing concurrent presentation of the real-world visual scene, either though the display or in the periphery[93].

9.7 THE VOR REVISITED

This dissertation embarked under the assumption that the VOR was generally an independent, separate entity that could effectively be investigated separately from the other oculomotor systems (with the exception of OKN). There is a preponderance of VOR research that also follow this assumption and as a result the information in Chapter 2 reflects this view. Models have been developed on VOR operation and data have been obtained which support these models. However, as my knowledge of the VOR has increased, it has become apparent that the performance of this system is highly influenced by other systems including proprioceptive input, visual factors, and cognitive strategies. As such, it has become more difficult to consider the VOR as a pure reflex.

The unanticipated results of the Time Delay Experiment contributed to a rethinking of VOR processes. The only hypotheses that could be developed to account for the observed gain reduction to a pure phase lag demand involved the influence of other oculomotor systems. Subsequently, I discovered other literature that has suggested a more intricate coupling of VOR to other eye movements. This strengthened my

[93] Reference the ataxia studies described in Chapter 4.

arguments in Section 6.8 and also caused me to pursue this issue further. I have since reviewed a paper by Collewijn (1989) that critically reviewed and dismissed the notion of a separate, independent VOR system as well as the other oculomotor systems (saccadic, smooth pursuit, vergence and OKN). In its place, Collewijn proposed a holistic concept of oculomotor control that functions to best orient the eye towards an object of interest. In his model, all eye movements work in concert, along with goals/expectations, retinal slip, efference copy, proprioception, and audition to achieve the goals of target identification and fixation.

This revisionist view of the VOR supports many of the findings and accompanying explanations in this dissertation such as the generalized adaptation of VOR gain, the VVOR data from the MAG condition and two time delay conditions, and the VOR gain reduction in the Time Delay Experiment. All were explained using processes in addition to the VOR.

This revisionist thinking, however, is not without flaws. The visual/oculomotor system may indeed be as fully integrated as Collewijn suggests, but this assertion creates a difficult challenge for experimental design. Under such a holistic scheme, how does a researcher control the numerous nuisance variables involved? Or conversely, how can a researcher interpret results with so many freely varying parameters? Lastly, does this revisionist thinking nullify a large percentage of previous data collected under the assumption of 'separate' oculomotor systems?

In addition, some of the arguments Collewijn employed seem a bit pessimistic. It implies no advantage to the separate study of oculomotor systems. As an example, Collewijn claims that VOR adaptation may be an awkward and indirect way to

investigate oculomotor/visual adaptation[94]. Regardless of the veracity of his statement, there is no denying that visual-vestibular rearrangements which cause VOR adaptation often entail negative perceptual effects during and/or after the exposure. If these aftereffects are the focus of the study, VOR adaptation research is warranted. Therefore, there remains validity in studying the VOR and the other oculomotor systems separately, depending upon the specific objectives involved.

Given Collewijn's arguments, the nature of the VOR, and the preponderance of existing research that indicates increased coupling between oculomotor systems, there seems to be substantial support for the existence of a tightly integrated, holistic visual system that functions to find and fixate targets of interest. However, rather than discounting the valuable knowledge obtained to date on the separate oculomotor systems, research should continue along current lines, albeit with a bent towards inclusion of other oculomotor systems either in the experimental design or in interpretations and generalizations. In this way, a holistic model will eventually be built through a process of induction while manageable experiments are conducted to solve problems and answer meaningful questions along the way.

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[94] He actually used the term 'spatial localization' adaptation which I modified to 'oculomotor/visual' in order to avoid an extended discussion of his thoughts on spatial localization. For a full discussion of his ideas, see Collewijn (1989).

CHAPTER 10: FUTURE RESEARCH OPPORTUNITIES AND CONCLUSION

10.1 FUTURE RESEARCH OPPORTUNITIES

Though the preceding experiments addressed the five main objectives of this dissertation, there remain several unresolved issues and interesting questions generated along the way. This section serves as a springboard for future research in the area of VOR adaptation to virtual environments, virtual interface parameters that modulate simulator sickness, and the relationship between oculomotor adaptation and simulator sickness. In addition, this dissertation advocates research on an overall oculomotor adaptation strategy to virtual interfaces that considers all types of eye movements as part of a singular, integrated visual-oculomotor system.

10.1.1 CONTINUED CHARACTERIZATION OF VIRTUAL INTERFACE PARAMETERS

This is the most obvious of research recommendations: to continue the process of characterizing the saliency of each visual-vestibular stimulus rearrangement found in virtual interfaces with regards to adaptations and simulator sickness. Adaptations should not be limited to the VOR, nor only oculomotor reflexes, but should cover other physiological and perceptual adaptations as well.

Some specific opportunities include the following. Different image scale magnitudes should be investigated, particularly those closer to 1.0X magnification. These changes are thought to be more efficiently adapted to and as a result may be more of a concern to designers. These images would also test the assertion that the closer the altered sensory patterns are to those normally expected, the greater the magnitude of the resulting

sensory rearrangement signal[95]. Inaccuracies in the rotational gain of head trackers should also be investigated to see if the same effects (i.e., VOR gain adaptation and simulator sickness) can be elicited by only changing optic flow rate. DFOV magnitude is also an unresolved issue with respect to VOR adaptation, though its effects on simulator sickness are well known. Other factors that should be explored include positional inaccuracies of the head tracker, display quality, resolution issues, and the effects of stimulus rearrangements associated with translational motion. VOR adaptation should also be characterized in three dimensions rather than solely the yaw dimension.

In addition, the area of augmented reality is receiving more emphasis in the VR field. The concept of wearable computers is driving potential applications and associated technology development. As a result, perhaps the unique issues of "see-through" virtual interfaces should be explored first.

10.1.2 Time Delay Experiment Findings: Replication and Advancement

It is important to replicate the findings regarding the effects of time delays on VOR gain and simulator sickness. Both of these results were striking for different reasons; VOR gain decreases were wholly unexpected and the lack of simulator sickness contradicts popular opinion and theory[96]. Therefore, replication is a necessity. If VOR gain decreases are replicated, the explanation put forth in Chapter 6 should also be tested. Since only fixed time delays were explored in this dissertation, the effects of variable delays must be examined as well.

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[95] This idea was borne from repeated observations in simulators that as simulation fidelity improved, simulator sickness worsened. It is one of the many dichotomies of the syndrome.

[96] Though the sparse data that exists are generally in agreement with these findings.

10.1.3 Investigation of VOR Re-Adaptation Time-Course and Safety Issues

This is a major issue since only preliminary data on adaptation and re-adaptation were collected in this research. Complete adaptation curves describing specific physiological and perceptual adaptation/re-adaptations to each salient sensory rearrangement is required to fully understand the effects of virtual interfaces.

A detailed characterization of any identified side-effects is also needed. While it is understood that oscillopsia, blurred vision, headaches, dizziness, and nausea have been associated with VOR adaptation and re-adaptation, what is the threshold for these effects to appear? How long do these effects last? What performance decrements are likely to result from these effects?

10.1.4 Expansion of Virtual Interface Design Guidelines

Future research in this area should be specifically tailored towards addressing virtual interface design issues. The results of such research could help refine and expand the initial guidelines presented here.

10.1.5 Miscellaneous VOR Research

There are numerous issues to explore in the area of fundamental VOR research, given the results of these experiments. For instance, the relative influence of active vs. passive head motions, meaningful vs. meaningless tasking, and expanded test frequency range on frequency-specificity of VOR adaptation is needed. Active vs. passive testing should use naturalistic patterns of head movement amplitudes and frequencies (i.e., the patterns generated by the head movement analyses) in order to determine the effective stimulus for the generalized adaptation found in this research.

The lack of a significant effect of time delay on phase adaptation is interesting and should also be explored further. Research could examine the reasons why and also

investigate the suggestion that phase adaptation seemed greater when the image scale was neutral vs. magnified or minified. Also, perhaps longer duration exposures at a fixed time delay would increase the magnitude of phase change.

10.1.6 Explore Adaptive Effects of the Entire Visual System

This is a difficult task but the results could be far-reaching. The idea would be to build upon the model presented by Collewijn (1989) of a single holistic visual system, so that the oculomotor effects of virtual interfaces can be more fully characterized. This would not be an easy undertaking and it would require use of a highly accurate (temporally and spatially) 3-D eye tracking system with sophisticated analysis techniques[97].

10.1.7 Further Develop the Incremental Step Technique

Dr. Viirre is continuing this research. After conceptual issues such as the maintenance of gain adaptation between therapy sessions are solved, research should concentrate on the optimization of the incremental step technique, both for gain increase and gain change maintenance. Comparison testing of adaptation protocols should then be conducted on normal subjects as well as those with chronically reduced gain.

10.1.8 A Final Attempt at Relating VOR Adaptation to Simulator Sickness

Hypothesis 2 should be reexamined. However the metric of adaptability should not remain as overall gain change achieved within 30 min. Since *rate* of VOR gain adaptation is considered a more pure estimate of a person's ability to adapt, this metric should be empirically tested. Below is an outline of an experiment designed to

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[97] Perhaps VR could be utilized as a data visualization tool to facilitate the presentation of eye-movement data collected while in the VE!

maximize the chance of finding a significant relationship between rate of VOR gain adaptation and simulator sickness.

This design is similar to that used by Gordon, et al. (1996) in research relating VOR metrics to seasickness incidence. Subjects would be classified into two groups of approximately 10 subjects each (high susceptibility, low susceptibility) on the basis of their responses to a motion sickness history questionnaire (and perhaps empirical validation of group membership using virtual interfaces).

The dependent variables would be the maximum slope of gain adaptation achieved after 50 minutes of forced rotation to an adaptation stimulus and the maximum slope of VOR gain adaptation achieved within the first 16 minutes only. A direction adaptation stimulus would be utilized per Khater, et al. (1990). This method of adaptation is preferred because it begins with a vertical VOR gain component of zero. This vertical VOR gain increases over time (from zero) as the subject adapts, making subtle changes in adaptation more noticeable (Rude, personal communication, 1997). VOR testing would occur every 4 minutes for the first 16 minutes and every 8 minutes for the remainder of the session. Sickness data could also be collected during exposure.

Differences in maximum rate of gain adaptation (either through the entire session or within the first 16 minutes) between groups would indicate that a relationship exists between VOR adaptation rate and simulator sickness susceptibility. Correlations could then be performed between adaptation rate and MSQ scores or sickness data collected during the experiment.

10.2 CONCLUSION

This dissertation set out to determine some of the effects virtual interfaces had on users. As stated in the preface, these experiments were designed to be useful to three

distinct and mostly independent audiences. It is sincerely hoped that the results surrounding VOR gain and phase adaptation, including the finding of generalized adaptation across frequencies, benefit the efforts of oculomotor physiologists. Likewise, experimental psychologists and others who are actively working to decipher the motion/simulator sickness syndrome may find the sickness data and accompanying discussions beneficial to their efforts. Most importantly however, I hope that these experiments and the resulting preliminary design guidelines will assist virtual interface designers in developing systems that are safe and comfortable to use, as this was the primary purpose of this effort. It is also hoped that these guidelines will be expanded and refined though future research.

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APPENDIX A: SYSTEM TIME DELAY CALIBRATION

This appendix describes the procedure used to determine the minimum system time delay of the virtual interface employed in these experiments. This calibration also determined the relationship between time delay value manually set in WARP TV and the actual system time delay that resulted. Figure 83 and Figure 84 illustrate the overall equipment configuration for the calibration task. This configuration is discussed first using the seven numbered arrows to direct the reader. A description of the procedure follows and lastly the generated regression line and equation is presented.

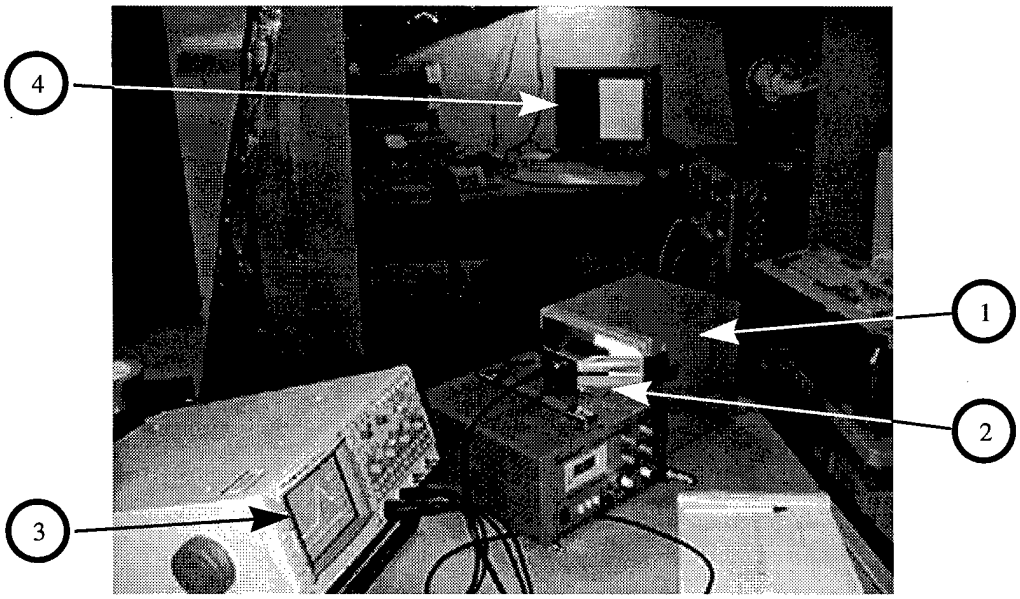

Figure 83: Overall System Time-Delay Calibration Set-up

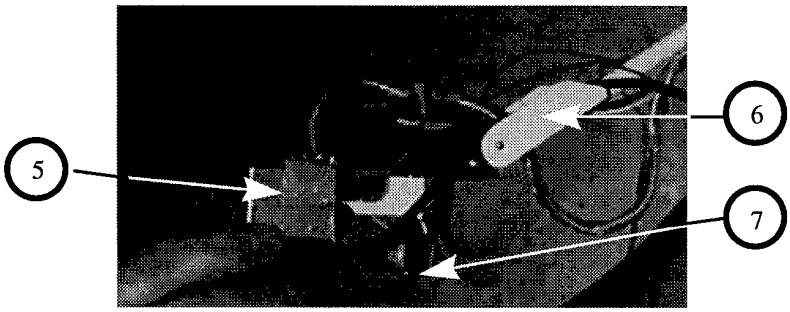

Figure 84: Head Tracker Coupling

The system was configured as follows. A New Focus Photo Detector/Amplifier unit[98] (arrow 2) was attached to a small CRT monitor (arrow 1) on which was displayed a half black/half white image generated by WARP TV. Figure 85 presents an isolated view of this arrangement. The photo detector identified changes in luminance when the image moved and the black/white boundary crossed the detector's active area. Photo detector output (analog) was sent directly to a Fluke four-channel, 200-MHz oscilloscope (Model 3394) for display (arrow 3). Arrow 4 on Figure 83 identifies the PC computer used to run the WARP software, with its monitor slaved to the same image being presented on the small CRT.

[98] This photo detector (Model 801) is of high quality with an onset time of a few ns.

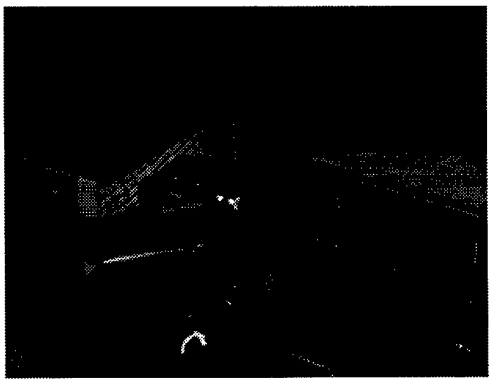

Figure 85: Close-up of Photo Detector
with CRT

Image-update commands to the WARP TV computer were generated by the InterSense tracker employed in these experiments (see Section 4.3). This sensor (arrow 5) was securely fastened to the ADL-1 mechanical position-sensing tracker (arrow 6) as shown in Figure 84. A high precision analog potentiometer of the ADL-1 was tapped (arrow 7) to provide tracker output directly to the oscilloscope. Therefore, the purpose of the ADL-1 was simply to restrict movement of the InterSense tracker to yaw rotations only and to transmit position changes to the oscilloscope with minimum latency (1 to 2 ms) via direct tapping of the activated potentiometer.

The procedure for each test trial was as follows. The experimentor preceded each test by alligning the visual image such that the white-black contrast line of the image was just to one side of the photodetector's maximum sensitivity area, with the white (high luminance) side being detected. The oscilloscope was then configured to trigger off a change in head-tracker position. Once the system was verified stable and in the correct allignment, the experimentor initiated an impulse yaw rotational movement to the InterSense sensor. This sensor swiveled along the vertical axis sensed by the tapped potentiometer of the ADL-1 and this potentiometer sent position data (analog) directly to

the oscilloscope for display. The InterSense tracker also detected this change in yaw angle and sent this information via RS-232 bus to the WARP TV computer. The computer generated an updated image signal for display in accordance with the tracker movement. This signal, upon passing through an VGA-to-NTSC converter, resulted in image motion on the small CRT monitor[99]. The photo detector detected the change in luminance as the contrast boundary swept across its active zone and this data was also sent to the oscillosope for display. The result was an oscilloscope screen displaying both changes in 'head' (via the ADL-1 potentiometer) and 'image' (via the photo detector) as shown in Figure 86. The system time delay was then determined by visual inspection of the time difference between when the 'head' trace indicated movement initiation and the resulting change in the image data.

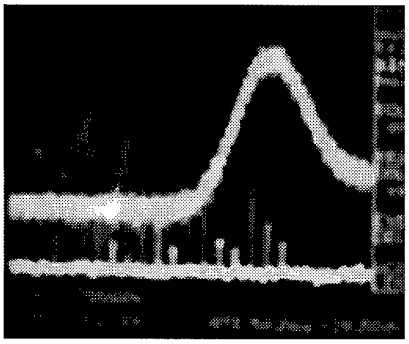

Figure 86: Close up of Oscilloscope Display

[99] The existence of the NTSC converter is vital because the HMD used in these experiments also required conversion from VGA to NTSC. Given that it is reasonable to suggest that this conversion effects overall delay, it needed to be included. That is the reason the WARP PC monitor was not used to trigger the photo detector; it only accepted VGA inputs.

The data on the oscilloscope require a bit more explanation, however. The trace of head position in Figure 86 is rather obvious as the data smoothly transitions from steady state. The image data trace appears as a train of spikes however, because the detector only signals when the CRT beam strikes its active area directly. This occurred once every 16.7 ms, due to the 60 Hz refresh rate. As a result, the change in image luminance was marked on the oscilloscope as an attenuation in spike amplitude. For standardization, the last large spike was defined to be the marker for when the system was updated.

This definition created a systematic timing error due to the fact that the photo detector only signaled every 16.7 ms. In order to get a handle on this bias, the limits of error must be understood. During some trials the CRT beam may be positioned just before the photodetector location and on other trials the beam may be positioned just beyond the photodetector's location. In the former case, the beam would reach the photodetector's location directly after the image change, resulting in no spike and with the last spike occurring approximately 16.7 ms earlier. This would result in the time delay estimation being 16.7 ms too small. In the latter case, the beam would have just caused a spike prior to the image movement which means that there would be no error in this case (since the position just after last spike is operationally defined as the point in which the image change occurred).

To correct for this bias, each trial was repeated 50 times. Assuming randomized beam locations at start time over the 50 trials, the eventual error would have a magnitude equal to the mean expected value, approximately 8.5 ms. Since the errors were unidirectional, adding 8.5 ms to the overall mean after 50 trials effectively eliminated the effects of this measurement bias.

This calibration was performed under the following conditions: a visual image resolution of 320 x 240 lines, an image scale factor of 1.0X magnification (NEU condition), the baud rate for RS-232 communication with the InterSense tracker set at

38400, and the InterSense tracker set to its default settings. All of these values corresponded with those used in the dissertation research.

A total of 50 trials were conducted at each of the following time delay settings on the WARP TV command line: 1, 25, 50, 75, 100, 100, and 200 ms. Later, 81 ms and 211 ms settings were tested to verify that they resulted in the desired system time delays utilized in the Time Delay Experiment (i.e., 125 and 250 ms).

The results of the calibration tests along with the calculated regression line are shown in Figure 87. As can be seen, the system is very linear with a very tight fit by the regression. In fact, the predicted and actual lines overlap. Actual system time delay (S_t) can be reliably predicted from WARP command line setting (W_t) by the following:

$$S_t = 46.9 + 0.97 * W_t$$

A few additional points are relevent. Though detailed tests were not completed at VGA resolution (640 x480), the doubling of resolution approximately doubled the system time delay. This was likely due to video card limitations. Also, slight changes in system time delay occurred with image scale changes, but these were relatively minor (approximately 4 to 9 ms between MIN and MAG conditions).

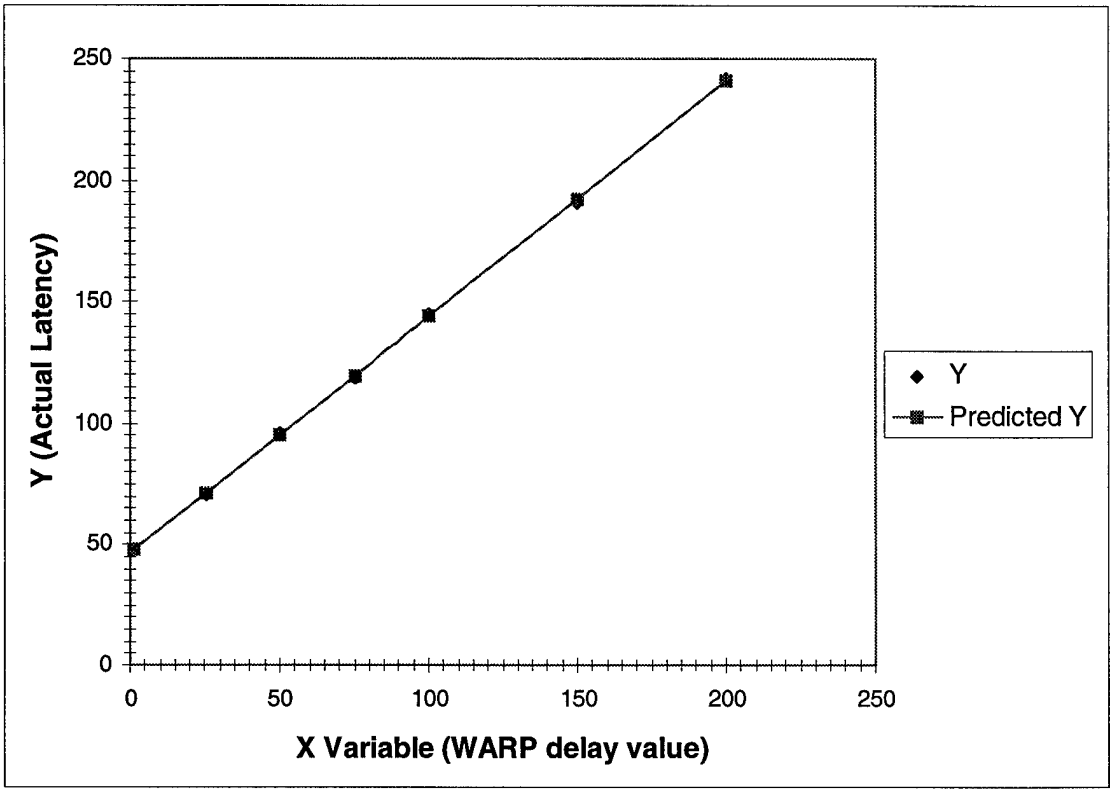

Figure 87: Regression Line For System Time Delays

APPENDIX B: EXPERIMENTAL PROTOCOLS

Below is the experimental protocol utilized in the Image Scale Experiment. Protocols for the other three experiments were similar except for changes noted in the procedure sections of each experiment.

IMAGE SCALE EXPERIMENT

Experimental Protocol (1 of 6)

Experiment Set-up/Prep

-verify all equip on and ready (see specific instruction sheets)
--ISCAN system (TEST)
--WARP computer
--SERVO system (inc. siggen)
--HMD (TEST then turn off)
--MacEyeball System
-enter correct data in MacEyeball (TEST)
-preset WARP to first condition/image/gfov (TEST)
-preset SERVO & siggen to first freq (TEST)
-verify room is light-tight
-turn down brightness of monitors
-verify shed is light-tight
-REVIEW SAFETY PROTOCOLS
-door open
-start fans
-verify refreshments
-organize all paperwork

Assistant **Experimenter**

Pre-Exposure (when subject arrives)

-hang 'experiment in progress' signs
-close door/seal out light

-verify all equip is on and ready

-verify MacEyeball data
-verify all is light-tight
-pre-briefing
-consent form
-visual acuity test
-pre-ssq

-balance test (2 trials in Sharpened Romberg)

Experimental Protocol (2 of 6)

Assistant	Experimenter
Baseline VOR Measurement	
-seat and strap subject in chair	-seat and strap subject in chair -prepare servo/sig gen -- (See SERVO Sheet)
-FIT EYE TRACKER (ISCAN sheet) --important that it is well fitting, snug, pupil in center of video image --have subject perform head movements while looking straight ahead --check ISCAN graph, verify OK	-Assist with fitting eye tracker
-center chair -adjust neck rest for support without obstructing HMD	
TELL SUBJ:NOT MOVE HEAD DURING	-EYE CALIBRATION: -perform eye calibration --(See ISCAN sheet) --subject only move eyes --RECORD CAL DATA!!
-turn off and cover video monitor #1 and ISCAN monitor	
[VVOR]	-VVOR TEST (0.8Hz/3V/BC30) --record data
-turn main lights off -cover test area with blinds	
	-close/seal shed
-test light off -remind subject to look straight ahead and remain still/hold grips -instruct subject to begin performing mental arithmetic -verify eye image & incoming data	VOR TESTS: -start rotator Siggen:13BC
-spotter: guard against excessive chair excursion/reset chair upon completion	-start test (0.2Hz) x 2 --switch freqs/sigen(24BC)/Mac -start test (0.4 Hz) x 2 --switch freqs/sigen(30BC)/Mac -start test (0.8Hz) x 2 -stop rotation
-STAY CLEAR IN CASE OF CHAIR FAILURE	
-lights on -loosen chair straps if nec. -center chair	-reset chair/servo in idle/on

Experimental Protocol (3 of 6)

Assistant	Experimenter
	Treatment
-Turn on HMD	
-explain objectives/procedures again -lights off	-check warp TV (settings correct?)
[VVOR in VR]	-VVOR in VE (0.8/3v/bc30) *"maintain fixation on target"*
	-set timer (10 min) for SS reports
Assist subject as needed	-series of images (5) --explore time (1 minutes) --search time (5 minutes) --record head epochs
	-subject **CLOSE EYES** and **NOT MOVE HEAD** between images
	-remind subject to call out sickness rating every 10 minutes
	-record sickness rating

SS Rating#1: collect head movement epoch

SS Rating#2: collect head movement epoch

SS Rating#3: collect head movement epoch

Experimental Protocol (4 of 6)

Assistant	Experimenter
	Post Exposure VOR Test
-TURN OFF HMD!	
	-prepare servo/sig gen --(See SERVO Sheet
	-verify eye image is still good
-help to adjust HMD if required --may need to turn on monitor #1here --HAVE SUBJECTS CLOSE EYES PRIOR TO THIS! -tighten chair straps	
	VOR TESTS:
-REMIND SUBJECT TO LOOK STRAIGHT AHEAD AND TO REMAIN STILL/HOLD GRIPS -instruct subject to begin performing mental arithmetic	-start rotator (13BC) -verify eye image & incoming data
-spotter: guard against excessive chair excursion/ -reset chair upon completion of each test --STAY CLEAR IN CASE OF CHAIR FAILURE	-start test (0.2Hz) x 2 --switch freq/siggen(24BC)/Mac -start test (0.4 Hz) x 2 --switch freq/siggen(30BC)/Mac -start test (0.8Hz) x 2 -stop rotation
-lights on -loosen straps if necessary -center chair	
	-reset chair/servo in idle/on

Experimental Protocol (5 of 6)

Assistant	Experimenter
Re-adaptation tests	
	-set timer (10 minutes)
-post SSQ #1	
-subject perform simple eye-head movement task	
<<<<<<<<<*AT 10 MIN*>>>>>>>>>	
-tighten chair straps if necessary -lights out	
	VOR TESTS:
-remind subject to LOOK STRAIGHT AHEAD AND REMAIN STILL/ HOLD GRIPS -instruct subject to begin performing mental arithmetic	-start rotator/siggen (13BC) -verify eye position & position data -start test (0.2Hz) x 2
-spotter: guard against excessive chair excursion -reset chair upon completion of each test --STAY CLEAR IN CASE OF CHAIR FAILURE	--switch freq/siggen(24BC)/Mac -start test (0.4 Hz) x 2 --switch freq/siggen(30BC)/Mac -start test (0.8 Hz) x 2 -stop rotator
-center chair	-reset chair/servo in idle
-lights on	
-post calibration of eye --blinds up --center chair --verify eye centered on monitor	-Post-test eye calibration --SUBJ ONLY MOVE EYES! --minimize blinks

Experimental Protocol (6 of 6)

Assistant	Experimenter
	Post-test
	-general post-questionnaire -power-down equipment
-posture test (twice, compare with first) -open door	
	-debriefing
[Subject cooling-off period (10 min.)]	
	-post SSQ #2
	-take down sign -BACK UP DATA!

Safety Protocol for Rotating Chair

1) (**Experimenter**) Verify all local settings (RATE and ACCEL) are set to '0' before turning the SERVO on.

2) (**Experimenter**) Finger on 'STOP' button when enabling, until told that chair is operating acceptably by the HOST.

3) (**Experimenter**) Press 'STOP' button at first indication of trouble.

4) (**Assistant**) Use hand to monitor chair motion. If continuous chair rotation is sensed, or if rapid acceleration is sensed, yell 'STOP!'. Turn flashlight on to assist subject.

5) (**Assistant**) Turn test light on after chair is stopped to further assist subject as necessary.

Sample Mental Alerting Tasks

1) Count back from ____ by __:
   301, 3
   650, 7
   450, 3
   etc.

2) Think of as many male/female names as you can that begins with the letter ___:
   B
   D
   S
   T
   M
   C
   etc.

3) Give me a male (female) name that corresponds to each letter of the alphabet.

4) Add together the numbers as you count from 1 to 25.

5) Name states in the union; countries in Europe, sports team names, etc.

APPENDIX C: SUBJECT CONSENT FORM

Human Interface Technology Laboratory
University of Washington
PARTICIPANT CONSENT FORM
"Optokinetic/Vestibular Research"

Purpose and Benefits

You are being asked to participate in a research study. The purpose of this study is to investigate how head-coupled visual images affect the way in which the eye moves. This research will be useful both for basic science and for the design of virtual interfaces.

Procedures

First we will measure how stable your balance is. You will stand heel-to-toe with your arms crossed for 10-30 seconds. We may repeat this test several times until you get used to it. We will have people guarding you so that you do not fall.

Next, we will measure your eye movements in the dark, using a rotating chair that operates at low speeds and a video camera to record the position of one of your eyes. Then you will view visual scenes by wearing a helmet mounted display. We will ask you to search for items that appear in these visual scenes. You will view these scenes for 30 minutes.

After the experiment, we will again measure your eye movements in the dark, once right after exposure, another time 10 minutes after exposure. Lastly, we will again measure your balance.

You will be asked to answer written questions about your experiences after the experiment. For some experiments, data collection may involve recording (e.g., videotaping, voice, photos) the experience and/or interviews with the participants. For these experiments, subjects will have a chance to review and delete any (or all) portions of the recordings. Sessions may take 1-2 hours. You may be asked to take part in more than one session.

Risks of Negative Effects

It is possible that you may suffer discomfort or mild nausea as you view the visual images. If you experience these or any other negative effects, please let us know, and we will try to correct the situation. If this is not possible, we will end the session early. For safety reasons, persons who are pregnant or who have back problems should not participate in this study.

Other Information

Your participation in this experiment is entirely voluntary. If at any time you decide to withdraw from the study, simply inform the experimenter and you will be excused without penalty. Your responses will be confidential. Your data will remain anonymous and will be kept for a period of 5 years by the co-investigators under conditions of restricted access. If you have any questions about this study or about your rights as a subject, please feel free to ask.

_____

Signature of Investigator Date

Participant's Statement:
The study described above has been explained to me. I voluntarily consent to participate in this activity. I have had the opportunity to ask questions, and I understand that future questions I may have about this research, or about my rights as a subject will be answered by one of the investigators listed above.

copies to: subject
experimenter's files

_____

Signature of Subject Date

APPENDIX D: SIMULATOR SICKNESS QUESTIONNAIRES

PRE-EXPERIENCE COMFORT QUESTIONNAIRE

Instructions: Please circle the severity of any symptoms that apply to you right now.

	(0)	(1)	(2)	(3)
1. General Discomfort	None	Slight	Moderate	Severe
2. Fatigue	None	Slight	Moderate	Severe
3. Headache	None	Slight	Moderate	Severe
4. Eye Strain	None	Slight	Moderate	Severe
5. Difficulty Focusing	None	Slight	Moderate	Severe
6. Increased Salivation	None	Slight	Moderate	Severe
7. Sweating	None	Slight	Moderate	Severe
8. Nausea	None	Slight	Moderate	Severe
9. Difficulty Concentrating	None	Slight	Moderate	Severe
10. Fullness of Head	None	Slight	Moderate	Severe
11. Blurred Vision	None	Slight	Moderate	Severe
12. Dizzy (Eyes Open)	None	Slight	Moderate	Severe
13. Dizzy (Eyes Closed)	None	Slight	Moderate	Severe
14. Vertigo*	None	Slight	Moderate	Severe

*Vertigo refers to a loss of orientation with respect to upright (i.e., you don't know "which way is up")

15. Stomach Awareness**	None	Slight	Moderate	Severe

**Stomach awareness is usually used to indicate a feeling of discomfort which is just short of nausea

16. Burping	None	Slight	Moderate	Severe

Are there any other symptoms that you are experiencing right now? If so, please describe the symptom(s) and rate their severity on the other side.

POST-EXPERIENCE COMFORT QUESTIONNAIRE

Instructions: Please circle the severity of any symptoms that apply to you right now; after experiencing the virtual environment.

	(0)	(1)	(2)	(3)
1. General Discomfort	None	Slight	Moderate	Severe
2. Fatigue	None	Slight	Moderate	Severe
3. Headache	None	Slight	Moderate	Severe
4. Eye Strain	None	Slight	Moderate	Severe
5. Difficulty Focusing	None	Slight	Moderate	Severe
6. Increased Salivation	None	Slight	Moderate	Severe
7. Sweating	None	Slight	Moderate	Severe
8. Nausea	None	Slight	Moderate	Severe
9. Difficulty Concentrating	None	Slight	Moderate	Severe
10. Fullness of Head	None	Slight	Moderate	Severe
11. Blurred Vision	None	Slight	Moderate	Severe
12. Dizzy (Eyes Open)	None	Slight	Moderate	Severe
13. Dizzy (Eyes Closed)	None	Slight	Moderate	Severe
14. Vertigo*	None	Slight	Moderate	Severe

*Vertigo refers to a loss of orientation with respect to upright (i.e., you don't know "which way is up")

15. Stomach Awareness**	None	Slight	Moderate	Severe

**Stomach awareness is usually used to indicate a feeling of discomfort which is just short of nausea

16. Burping	None	Slight	Moderate	Severe

Are there any other symptoms that you are experiencing right now? If so, please describe the symptom(s) and rate their severity on the other side.

APPENDIX E: VIRTUAL ENVIRONMENT IMAGES AND TARGET LIST

This appendix presents the 15 virtual images used in these experiments. Each image was a 360-degree cylindrical, full-color, QuickTime VR panaramic image (though the versions presented here are in a lower resolution JPEG format). The images can be accessed at the following internet site: http://www.vrseattle.com/vrsea.home.html. Following these images is the list of visual search targets for the Kingdome image (others omitted for brevity).

Figure 88: Pike Place Market 1

Figure 89: The Kingdome

Figure 90: Pioneer Square 1

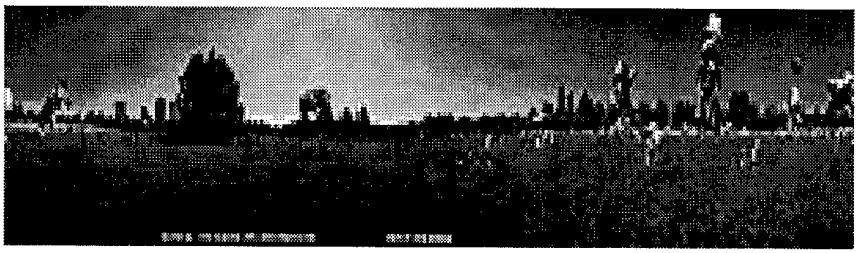

Figure 91: Bellevue Park

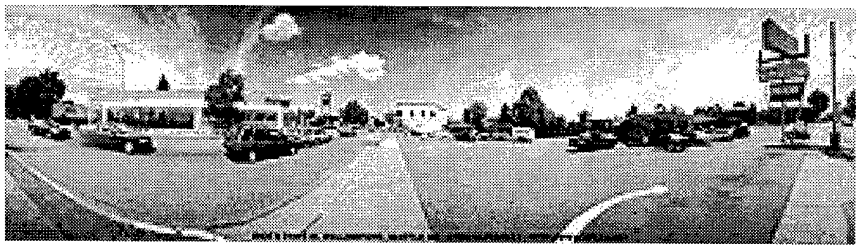

Figure 92: Dick's Drive-In

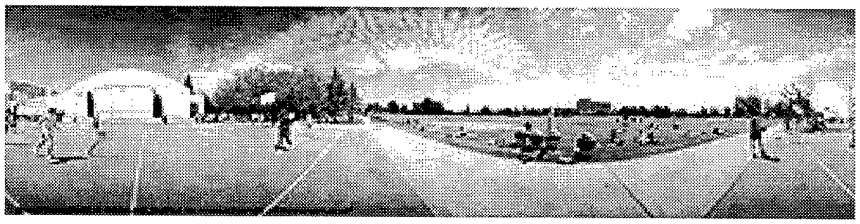

Figure 93: Greenlake

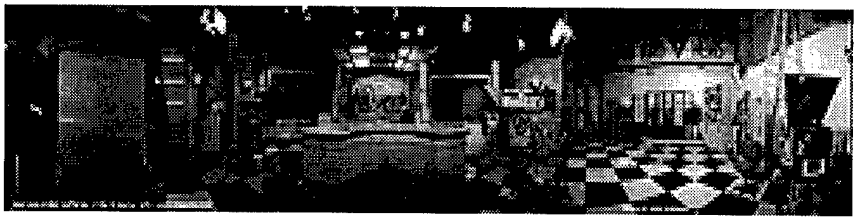

Figure 94: KIRO News Room

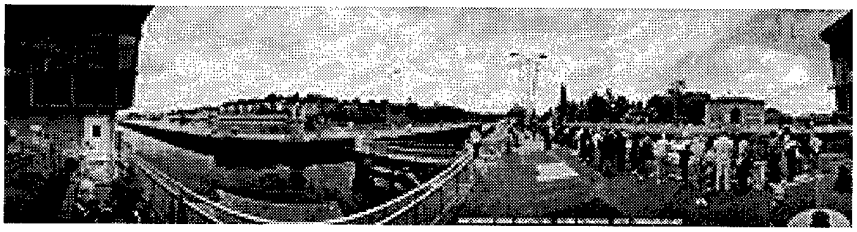

Figure 95: Ballard Locks

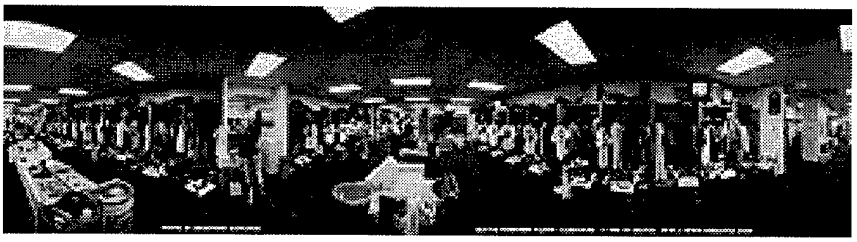

Figure 96: Mariners Locker Room

Figure 97: Seattle Center

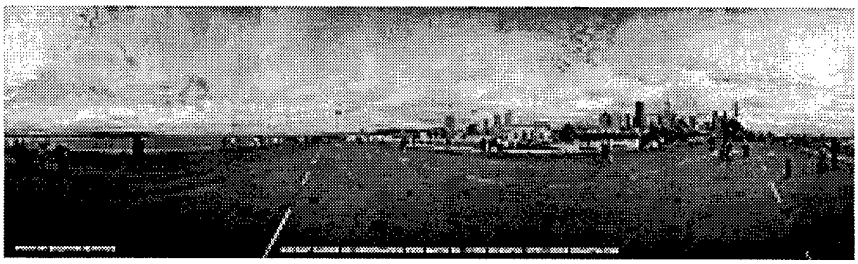

Figure 98: Aircraft Carrier 1

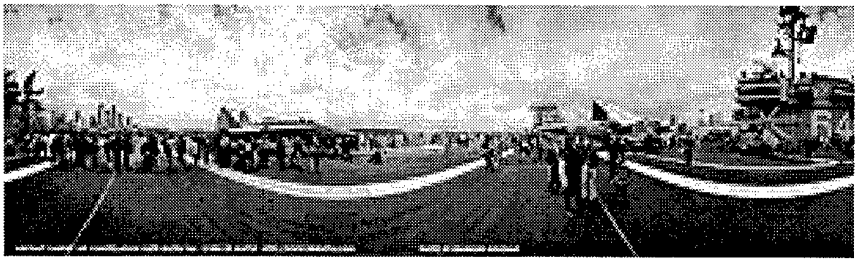

Figure 99: Aircraft Carrier 2

Figure 100: Waterfront

Figure 101: Pioneer Square 2

Figure 102: Pike Place
Market 2

Kingdome Target List (see Figure 89)

- left dugout
- right dugout
- orange-brown pennent
- man in shorts
- 2nd base area
- number of bats on ground
- Seattle mariner symbol on ground
- player bending over
- number of people around batting cage
- large banner hanging in distance
- man closest to view
- child in white shirt on field
- pitcher's mound
- man in white shirt w/ stripes
- banner behind batters box
- number of people in white shirts
- batter
- 3rd base area
- man on chair
- 1st base area
- both foul poles
- person squatting on field
- man in gray sports coat
- black box object on ground
- large TV screen

APPENDIX F: GENERAL POST EXPOSURE QUESTIONNAIRE

Subject # Session #
Date:

Age:
Gender: **M F**

1) Do you wear corrective lenses? **YES NO**
If yes:
type: **CONTACTS GLASSES BOTH**
relative lens strength: **MINOR MODERATE LARGE**

2) Do you have astigmatism? **YES NO**

3) Do you have a medical history of any visual and or vestibular problems (besides need for corrective lenses)? If so, please briefly elaborate.

4) How would you rate the movement of the virtual worlds that you saw?
NOT REALISTIC FAIRLY REALISTIC VERY REALISTIC

5) Did you get used to the virtual environment or did it bother you more as time progressed?
GOT USED TO IT BOTHERED ME MORE WITH TIME

6) Have you ever been in a VR world before? **YES NO**
If yes, how many times? **1 2-5 6-10 MORE THAN 10**

7) Do you play video games?
NO OCCASIONALLY OFTEN DAILY

8) Have you ever experienced motion sickness in the past? **YES NO**

9) Have you ever experienced simulator or cyber-sickness in the past? **YES** **NO**

10) How would you rate your susceptibility to motion sickness?
NOT SUSCEPTIBLE **SLIGHT** **MODERATE** **HIGH**

11) Did you develop a strategy for searching for targets? **YES** **NO**
If 'YES', please briefly describe this strategy:

12) Which image did you think was most realistic and why?

13) Was the resolution of the image enough to discern visual targets?
YES **KIND OF** **NO**

14) In general, how would you describe your ability to adapt to new perceptual environments?
LOW **MODERATE** **HIGH**

APPENDIX G: CLASSIFICATION SCHEME TO AID GENERALIZATION OF SIMULATOR SICKNESS RESULTS

No one doubts that sickness symptoms can occur in synthetic environments. However, a major drawback of simulator sickness research is the inability of empirical results derived from one environment to reliably generalize to another environment (Hash & Stanney, 1995; Pausch, et al., 1992). While simulator sickness is a complex phenomenon with many complicated interactions, this lack of generalization may be partially due to poor identification and classification of provocative environments.

The majority of researchers who study motion sickness and simulator sickness believe that a main source of sickness are visual-vestibular sensory rearrangements (i.e., the sensory rearrangement theory). Given this assumption, it would be advantageous to classify synthetic environments in terms of their specific visual-vestibular relationships.

This idea of classifying environments according to visual-vestibular coupling is not new. It essentially extends Reason and Brand's (1975) motion sickness environment classification scheme to synthetic environments. Reason and Brand argued that all motion sickness results from either visual-vestibular rearrangements or canal-otolith rearrangements, each with three possible permutations of the involved variables (see Section 2.4.4.1). Using this classification scheme, Benson (cited in Griffen, 1990) asserted that all simulator sickness is caused only by visual-vestibular rearrangements, especially those in which there are no vestibular motion signals to accompany existing visual motion inputs.

As a further example of classifications, Kennedy has long recognized the importance of classifying flight simulators into such dimensions as fixed-base/motion-base and demonstrating how sickness varies between these different simulators (Kennedy, Drexler,

& Berbaum, 1994; Baltzley, et al., 1989). Recently, he has also attempted to identify and quantify provocative visual stimuli at a much more detailed level than ever before, using an visual frame grabber to capture and calculate optic flow velocities and accelerations along several dimensions (Kennedy, Berbaum, Dunlap, & Hettinger, 1996). The argument is that the visual stimulus must be fully defined if there is to be any hope of determining true causes of simulator sickness.

Although this line of inquiry is perfectly acceptable within the domain of fixed-base flight simulators, it represents only one componant of the motion stimuli relevant in head-coupled VEs. To accurately define the motion stimuli in head-coupled VEs, both the visual and *vestibular* components must be considered, and quite possibly the most important aspect is the interaction between visual and vestibular signals (per the sensory rearrangement theory).

There is often more than one type of visual-vestibular coupling manifest in a single synthetic environment. Current simulator sickness literature frequently fails to recognize this fact. Papers discuss only the most obvious visual-vestibular interaction involved in a synthetic environment. For instance, consider a fixed-base flight simulator which has perfect visual-vestibular coupling during active eye-head gaze shifts but no such coupling during simulated vehicular movement. Therefore, an appropriate classification scheme needs to account for several potential couplings coexisting within a synthetic environment.

A review of the literature makes it clear that classification of synthetic environments in terms of specific visual-vestibular interactions has not yet occurred, which may have contributed to the complexity of the simulator sickness problem. Therefore, a classification scheme is proposed to more completely classify the visual-vestibular interactions of synthetic environments.

Visual-vestibular relationships should be separated into those arising as a result of '*head movements*' and those arising as a result of '*vehicular movements*', because it is through these two types of movements that all visual-vestibular interactions occur (Table 25). These two categories can be further separated into *rotational* and *translational* motion.

The 'head movement' category entails visual and vestibular motion stimuli in response to active, self-generated movements of the participant's head (either through neck rotations, torso movements, or natural locomotion). Head movements are assumed to be actively controlled by the subject and would, therefore, always have an appropriate vestibular signal. These motions also include efference copy signals in perceptual-motor control loops.

The 'vehicle movement' category applies in those situations where the participant experienced simulated 'vehicle' motion (such as in car and flight simulators), either actively controlled or passively experienced. In VR, this vehicular motion would include 'flying' through an environment by means of a hand controller button or other such device. Vehicular movement would also include the changing of rotational views by means of an artificial control device.

Table 25: Proposed Classification Scheme

	Head Movement	**Vehicle Movement**
Rotation	{active} synchronized; congruent, vest/no vis	{active or passive} synchronized; congruent, vis/no vest; vest/no vis
Translation	{active} synchronized; congruent, vest/no vis	{active or passive} synchronized; congruent, vis/no vest; vest/no vis

Each cell in Table 25 has one of the following possible visual-vestibular couplings: synchronized (both visual and vestibular motion signals are assumed to be perfectly coupled as in the real world), congruent (both systems signal congruent motion information but they may *not* be perfectly coupled due to latencies, tracking inaccuracies, scale factor deviations from 1.0X, etc.), and vest/no vis (vestibular inputs not accompanied by corresponding visual motion inputs). The vehicular movement category also includes a fourth potential coupling: vis/no vest (visual motion signals are not accompanied by corresponding vestibular signals). Furthermore, as control of vehicular motion has been shown to modulate the severity of sickness experienced, there is identification of whether vehicular control task is *active* or *passive* for translation as well as for rotational motion.

Figure 103 provides examples of how the visual-vestibular relationship can vary with category of movement (head movement vs. vehicular movement). The top figure pair indicates how head movements (in translation and rotation) are coupled to visual optic flow in a head-coupled environment. Note that head movements result in a visual image motion response. Whether that response is synchronized, coupled, or vest/no vis depends on the specific environment involved.

The remaining figure pairs in Figure 103 show examples of vehicular movement. The middle pair demonstrates vehicular control in a motion-based simulator, where small, transient, externally-applied inertial motion stimuli (represented by the small dot to the right of the head) are provided to the entire subject's body and to which the visual image is coupled. The visual-vestibular relationship is congruent in this example, as vestibular cues do not perfectly correspond with visual optic flow. The bottom pair represents vehicular movement in a fixed base simulator, where there is no vestibular coupling (internally or externally produced) with optic flow. This results in a vis/no vest relationship. These examples are intended simply to highlight the significant differences in visual-vestibular couplings that can occur in synthetic environments.

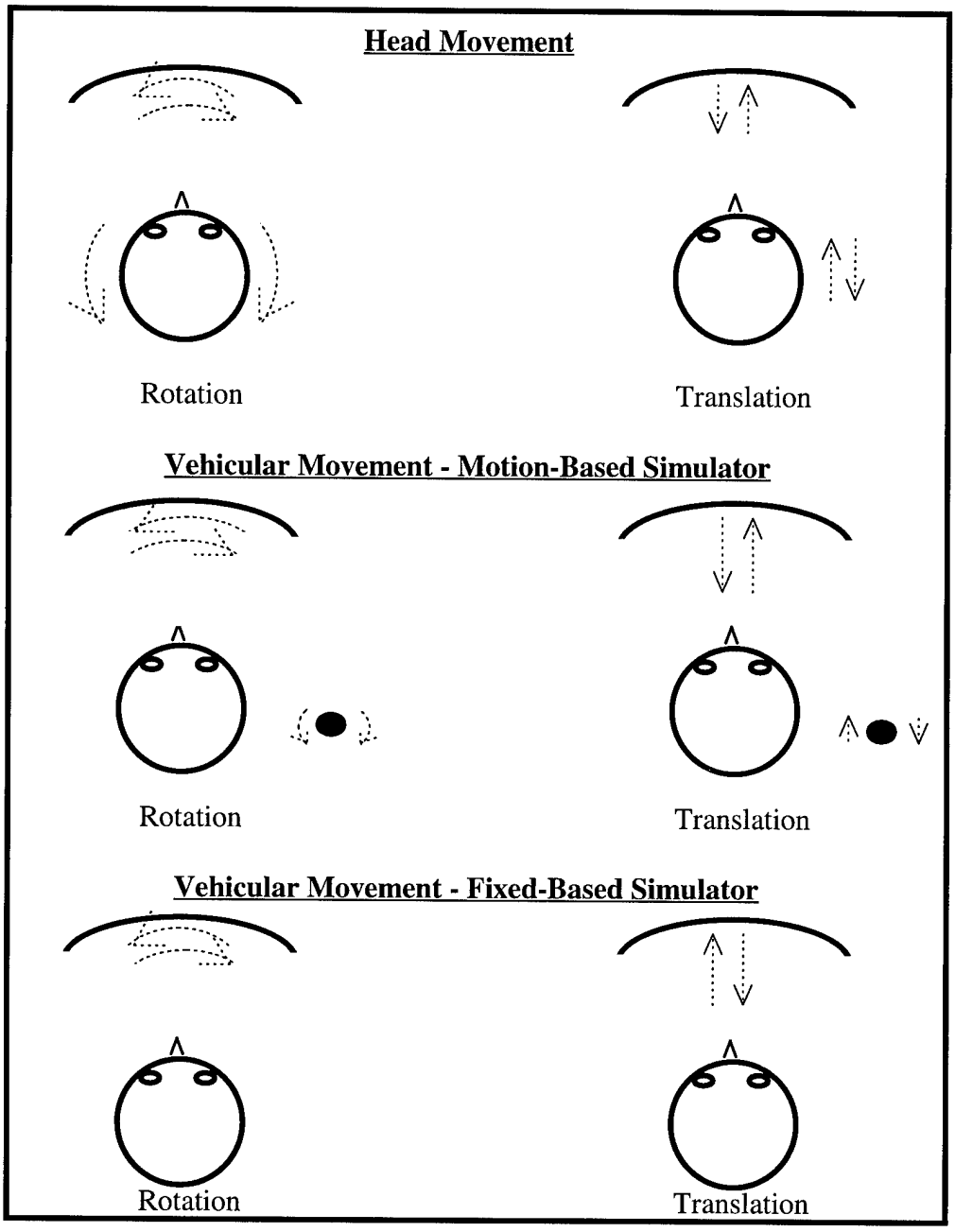

Figure 103: Various Visual-Vestibular Couplings

This proposed system specifies the visual-vestibular couplings of all synthetic environments according to the four quadrents of Table 25. Table 26 gives a few examples of how specific synthetic environments would be categorized. Note the differences in visual-vestibular couplings that exist between simulators, between VEs, and between simulators and VEs. This suggests that one should not speak generically of 'visual-vestibular conflict' without detailing its specific nature.

Also note how DFOV modulates the potential for head movements to occur in non-head coupled simulators. If the DFOV is small (like a computer display monitor), there will be little if any resulting head motion but if the DFOV is large (like a dome simulator), there likely will be much more head movement (as users look around their environment). So in essence, DFOV may act as a gain modulator for the 'head movement' visual-vestibular couplings in non-head-coupled displays, but this modulation may not carry over into head-coupled displays.

Table 26: Classification of Synthetic Environments[100]

Synthetic environment	Head Movement (A) Rotation	Translation	Vehicular Movement Rotation	Translation
1) Fixed-base domed flight sim (non-head coupled)	sync'd	sync'd	A:vis/no vest	A:vis/no vest
2) 6 DOF motion dome flight sim	sync'd	sync'd	A:congruent	A:congruent
3) fixed-base small FOV flt sim	N/A	N/A	A:vis/no vest	A:vis/no vest
4) 3 DOF head-cpld (rot. only) VR (gaze direction = translation dir.)	congruent	vest/no vis	N/A	A:vis/no vest
5) 3 DOF head-cpld (rot. only) VR (hand direction = translation dir.)	congruent	vest/no vis	A:vis/no vest	A:vis/no vest
6) 6 DOF head-cpld VR (gaze direction = translation dir.)	congruent	congruent	N/A	A:vis/no vest
8) 0 DOF small FOV passive sim flythrough	N/A	N/A	P:vis/no vest	P:vis/no vest
9) 3 DOF (rot only) VR passive flythrough (active gaze shifts allowed)	congruent	vest/no vis	P:vis/no vest	P:vis/no vest

In Table 26, fixed-base domed flight simulators involve active motion control and synchronized visual-vestibular couplings during head movements because the visual image always surrounds the pilot and it responds with 0 ms latency, 1.0 gain, and no phase shift to head rotations and translations (as in the real world). However, motion from vehicular control results in a significant conflict between optic flow and an uncorrelated vestibular signal (i.e., vis/no vest). Compare this to a commercially available head-coupled VR system that only responds to head rotations (3 DOF) and where the subject translates through virtual space in the direction he/she looks by virtue of a button press. In this case, although the subject actively controls the motion just like in the flight simulator condition, the underlying visual-vestibular interactions are different. Head movements result in 'congruent' but not 'synchronized' visual-vestibular interaction (due to latencies, scale factor deviations, etc.) and vehicular motion, only relevant in translation, involves visual motion cues only (i.e., vis/no vest). Also consider that

[100] 'A' = active control, 'P' = passive control; 'VR' assumes the use of an HMD, 'sim' assumes no HMD.

translations only in the direction of current gaze result in more rotational head movements and less asynchronous translational optic flow in the visual stimulus. Thus these two synthetic "simulations" (fixed-base flight simulator and 3 DOF virtual interface) result in very different visual-vestibular couplings, and it should be apparent that generalization of simulator sickness factors/incidence from one to the other may be limited at best. Indeed, researchers have begun to investigate whether sickness symptoms arising from flight simulators are different than symptoms resulting from virtual interfaces (Stanney, Kennedy, & Drexler, 1997).

The purpose of this proposed classification scheme is to provide a rational grounds for generalization of simulator sickness results to other synthetic environments based upon the relative similarity of visual-vestibular couplings involved. A secondary purpose to offer a framework in which to discuss: 1) conflicting results in the literature, and 2) the potential saliency of specific visual-vestibular rearrangements. Given that any complete description of simulator sickness needs to fully include the task, the stimuli, and the individual, this framework is necessarily incomplete. However, for a particular task and individual profile, this classification scheme offers a way to specify and categorize the visual-vestibular couplings in synthetic environments while also specifying the salient task variable of vehicular motion control (active versus passive).

This classification also offers the potential for predictions to be made regarding the relative "potency" of synthetic environments, according to their aggregate of visual-vestibular rearrangements across vehicular and head movements. This presupposes that knowledge is first gathered on the relative saliency of each potential visual-vestibular coupling per movement category. If the task and individuals were equated across two environments to be compared, however, this scheme might at least provide a "suggestion" as to which environment may be more provocative based on some relevant combination of the visual-vestibular rearrangements involved. Nevertheless, the main purpose for

developing this classification scheme was to guide the generalizing of simulator sickness results and to better frame discussions of existing research.