A COMPARISON OF DIFFERENT EXERCISE PROGRAMS IN THE REHABILITATION OF PATIENTS WITH CHRONIC VESTIBULAR DYSFUNCTION

by MARY LESSING-TURNER

A THESIS

Presented to the Faculty of Graduate Studies
In Partial Fulfilment of the Requirements
for the Degree of
Master of Science

MASTER 0F SCIENCE

Faculty of Medicine
School of Medical Rehabilitation
Division of Physical Therapy
University of Manitoba

March 1995

ISBN 0-612-13293-5

Name _____

Dissertation Abstracts International is arranged by broad, general subject categories. Please select the one subject which most nearly describes the content of your dissertation. Enter the corresponding four-digit code in the spaces provided.

THE SCIENCES AND ENGINEERING
SUBJECT TERM

0382
SUBJECT CODE

U·M·I

Subject Categories

THE HUMANITIES AND SOCIAL SCIENCES

COMMUNICATIONS AND THE ARTS
Architecture 0729
Art History 0377
Cinema 0900
Dance 0378
Fine Arts 0357
Information Science 0723
Journalism 0391
Library Science 0399
Mass Communications 0708
Music 0413
Speech Communication 0459
Theater 0465

EDUCATION
General 0515
Administration 0514
Adult and Continuing 0516
Agricultural 0517
Art 0273
Bilingual and Multicultural 0282
Business 0688
Community College 0275
Curriculum and Instruction 0727
Early Childhood 0518
Elementary 0524
Finance 0277
Guidance and Counseling 0519
Health 0680
Higher 0745
History of 0520
Home Economics 0278
Industrial 0521
Language and Literature 0279
Mathematics 0280
Music 0522
Philosophy of 0998
Physical 0523
Psychology 0525
Reading 0535
Religious 0527
Sciences 0714
Secondary 0533
Social Sciences 0534
Sociology of 0340
Special 0529
Teacher Training 0530
Technology 0710
Tests and Measurements 0288
Vocational 0747

LANGUAGE, LITERATURE AND LINGUISTICS
Language
- General 0679
- Ancient 0289
- Linguistics 0290
- Modern 0291

Literature
- General 0401
- Classical 0294
- Comparative 0295
- Medieval 0297
- Modern 0298
- African 0316
- American 0591
- Asian 0305
- Canadian (English) 0352
- Canadian (French) 0355
- English 0593
- Germanic 0311
- Latin American 0312
- Middle Eastern 0315
- Romance 0313
- Slavic and East European 0314

PHILOSOPHY, RELIGION AND THEOLOGY
Philosophy 0422
Religion
- General 0318
- Biblical Studies 0321
- Clergy 0319
- History of 0320
- Philosophy of 0322

Theology 0469

SOCIAL SCIENCES
American Studies 0323
Anthropology
- Archaeology 0324
- Cultural 0326
- Physical 0327

Business Administration
- General 0310
- Accounting 0272
- Banking 0770
- Management 0454
- Marketing 0338

Canadian Studies 0385
Economics
- General 0501
- Agricultural 0503
- Commerce-Business 0505
- Finance 0508
- History 0509
- Labor 0510
- Theory 0511

Folklore 0358
Geography 0366
Gerontology 0351
History
- General 0578
- Ancient 0579
- Medieval 0581
- Modern 0582
- Black 0328
- African 0331
- Asia, Australia and Oceania 0332
- Canadian 0334
- European 0335
- Latin American 0336
- Middle Eastern 0333
- United States 0337

History of Science 0585
Law 0398
Political Science
- General 0615
- International Law and Relations 0616
- Public Administration 0617

Recreation 0814
Social Work 0452
Sociology
- General 0626
- Criminology and Penology 0627
- Demography 0938
- Ethnic and Racial Studies 0631
- Individual and Family Studies 0628
- Industrial and Labor Relations 0629
- Public and Social Welfare 0630
- Social Structure and Development 0700
- Theory and Methods 0344

Transportation 0709
Urban and Regional Planning 0999
Women's Studies 0453

THE SCIENCES AND ENGINEERING

BIOLOGICAL SCIENCES
Agriculture
- General 0473
- Agronomy 0285
- Animal Culture and Nutrition 0475
- Animal Pathology 0476
- Food Science and Technology 0359
- Forestry and Wildlife 0478
- Plant Culture 0479
- Plant Pathology 0480
- Plant Physiology 0817
- Range Management 0777
- Wood Technology 0746

Biology
- General 0306
- Anatomy 0287
- Biostatistics 0308
- Botany 0309
- Cell 0379
- Ecology 0329
- Entomology 0353
- Genetics 0369
- Limnology 0793
- Microbiology 0410
- Molecular 0307
- Neuroscience 0317
- Oceanography 0416
- Physiology 0433
- Radiation 0821
- Veterinary Science 0778
- Zoology 0472

Biophysics
- General 0786
- Medical 0760

EARTH SCIENCES
Biogeochemistry 0425
Geochemistry 0996
Geodesy 0370
Geology 0372
Geophysics 0373
Hydrology 0388
Mineralogy 0411
Paleobotany 0345
Paleoecology 0426
Paleontology 0418
Paleozoology 0985
Palynology 0427
Physical Geography 0368
Physical Oceanography 0415

HEALTH AND ENVIRONMENTAL SCIENCES
Environmental Sciences 0768
Health Sciences
- General 0566
- Audiology 0300
- Chemotherapy 0992
- Dentistry 0567
- Education 0350
- Hospital Management 0769
- Human Development 0758
- Immunology 0982
- Medicine and Surgery 0564
- Mental Health 0347
- Nursing 0569
- Nutrition 0570
- Obstetrics and Gynecology 0380
- Occupational Health and Therapy 0354
- Ophthalmology 0381
- Pathology 0571
- Pharmacology 0419
- Pharmacy 0572
- Physical Therapy 0382
- Public Health 0573
- Radiology 0574
- Recreation 0575
- Speech Pathology 0460
- Toxicology 0383

Home Economics 0386

PHYSICAL SCIENCES
Pure Sciences
Chemistry
- General 0485
- Agricultural 0749
- Analytical 0486
- Biochemistry 0487
- Inorganic 0488
- Nuclear 0738
- Organic 0490
- Pharmaceutical 0491
- Physical 0494
- Polymer 0495
- Radiation 0754

Mathematics 0405
Physics
- General 0605
- Acoustics 0986
- Astronomy and Astrophysics 0606
- Atmospheric Science 0608
- Atomic 0748
- Electronics and Electricity 0607
- Elementary Particles and High Energy 0798
- Fluid and Plasma 0759
- Molecular 0609
- Nuclear 0610
- Optics 0752
- Radiation 0756
- Solid State 0611

Statistics 0463

Applied Sciences
Applied Mechanics 0346
Computer Science 0984
Engineering
- General 0537
- Aerospace 0538
- Agricultural 0539
- Automotive 0540
- Biomedical 0541
- Chemical 0542
- Civil 0543
- Electronics and Electrical 0544
- Heat and Thermodynamics 0348
- Hydraulic 0545
- Industrial 0546
- Marine 0547
- Materials Science 0794
- Mechanical 0548
- Metallurgy 0743
- Mining 0551
- Nuclear 0552
- Packaging 0549
- Petroleum 0765
- Sanitary and Municipal 0554
- System Science 0790

Geotechnology 0428
Operations Research 0796
Plastics Technology 0795
Textile Technology 0994

PSYCHOLOGY
General 0621
Behavioral 0384
Clinical 0622
Developmental 0620
Experimental 0623
Industrial 0624
Personality 0625
Physiological 0989
Psychobiology 0349
Psychometrics 0632
Social 0451

A COMPARISON OF DIFFERENT EXERCISE PROGRAMS IN THE REHABILITATION OF PATIENTS WITH CHRONIC VESTIBULAR DYSFUNCTION

BY

MARY LESSING-TURNER

A Thesis submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

MASTER OF SCIENCE

TABLE OF CONTENTS

ACKNOWLEDGEMENTS

This work was carried out in 1990 - 1992 at the Division of Physical Therapy, School of Medical Rehabilitation, Faculty of Medicine, University of Manitoba, and the Health Sciences Centre, Winnipeg, Manitoba, Canada.

I am grateful for the financial assistance provided for this project by the Manitoba Medical Services Foundation and the Murphy Foundation of Winnipeg, Manitoba.

I wish to thank my advisor Dr. Tony Szturm for his assistance and guidance in developing and working through this project. His knowledge and insight in this field is very extensive and he guided me through the process of understanding the details necessary to complete this work. I would also like to express my appreciation to him for his patience during the really difficult periods.

I would also like to thank Dr. Dean Kriellars for his helpful suggestions and his enthusiasm. I appreciate that he accepted to be on my committee. I would like to thank him for his constructive criticism of this thesis.

Special thanks to Dr. Des Ireland who has provided information and guidance. His wish to start a rehabilitation program for patients with vestibular dysfunction at the Health Sciences Centre was the catalyst that projected this project into reality. I would also like to thank him for accepting to be on the committee, to review and evaluate this thesis.

A heart felt thank you to all the patients who were willing to participate in this study. I was impressed by there willingness to continue on even when we initially exasperated their symptoms. This study would not have been possible without their support and endurance.

I would also like to thank the staff of the Physiotherapy Department, Health Sciences Centre for their assistance in completing this project and in carrying the concepts of this research project into the clinical setting.

Acknowledgement is also extended to the Manitoba Medical Services Foundation and the Murphy Foundation of Winnipeg Manitoba for grants donated for this study.

A special thank you to Dr. Jell who stepped in at short notice to review this document and provide me with feedback.

Finally, I am grateful to my husband Andrew, for his encouragement, patience and understanding over the last five years and for making me believe I could finish this thesis.

LIST OF ABBREVIATIONS

TMC	Total body centre of mass
CNS	Central Nervous System
A-P	Anterior-posterior
CFP	Centre of foot pressure
EMG	Electromyography or Electromyogram
VOR	Vestibulo-ocular reflex
OKN	Optokinetic reflex system
RR	Recruitment ratio H reflex
SMR	Sensorimotor restriction
SN	Spontaneous nystagmus
SPEV	Slow-phase eye velocity
DC	Direct current
POC	Preoperative performance level
OKAN	Optokinetic after nystagmus
TC	Time constant
SPV	Slow-phase velocity
SPN	Horizontal spontaneous nystagmus
SP	Smooth pursuit
EHT	Eye-head tracking
AHR	Active head rotation
COR	Cervical-ocular reflex
UVH	Unilateral vestibular hypofunction
BPPV	Benign paroxysmal positioning vertigo
BPPN	Benign paroxysmal positional nystagmus
RT	Rotational test
PG	Posturography
PSPV	Peak slow-phase eye velocity
LA	Labyrinthine asymmetry
LW	Left warm
LC	Left cool
ES	Equilibrium score
RW	Right warm
RC	Right cool
ENG	Electronystagmography or electronystamogram
SO	Sensory organization test
EOG	Electroocular nystagmography
VHT	Vestibular habituation training
A	Dizziness
I	Intensity
T	Duration
NOR	Normal

VD	Vestibular dysfunction
VDP	Visual preference
VVD	Visual and vestibular dysfunction
VSD	Somatosensory and vestibular dysfunction
SEV	Severe sway
H	Home group
Reh	Rehabilitation group
OKS	Optokinetic stimulation
TD	Test day
Eqt	Equitest
CC	Clockwise
CW	Counterclockwise
STS	Strategy score
COVF	Centre of vertical force
OK	Optokinetic
ANOVA	Analysis of Variance
SEM	Standard Error of Mean
FSR	Force sensing resistors

LIST OF FIGURES

LISTS OF TABLES

LIST OF APPENDICES

CHAPTER 1- INTRODUCTION

The sense organs of the vestibular apparatus provide the central nervous system (CNS) with information about motion of the head and position of the head relative to the gravity vector. These sensory signals along with other sources of body orientation, motion cues from the visual and somatosensory receptor systems are required for postural control, coordination of eye-head movements and our awareness of spatial orientation.

Disruption to the vestibular apparatus or its pathways due to disease or trauma demonstrates the importance of the vestibular system. Damage or disease to the vestibular system will be manifested in a number of deleterious signs and symptoms. These may include all or a combination of: postural imbalance, problems with coordination of eye-head movements and gaze control causing blurred vision and spontaneous nystagmus, debilitating perceptual manifestations of vertigo, fatigue, nausea and lightheadedness.

An understanding of the complexity of the vestibular apparatus or labyrinth is essential to study the compensation process that allows a patient to recover function following a peripheral vestibular lesion.

The vestibular apparatus in each inner ear consists of three semicircular canals and two otolith end organs (utricle and saccule). One of the functions of the receptor region of each semicircular canal is the cupula which responds to angular acceleration of the head for reflex control of eye movements. The receptor region of the otolith is the macula which detects angular acceleration of the head, linearacceleration due to the position of the head relative to the gravity vector, and linear motion of the head.

The partial recovery of function which animals and humans frequently

demonstrate following the initial consequence of peripheral vestibular lesions has long been recognized, because of multisensory convergence lesions of the vestibular apparatus do not lead to complete deficits comparable to blindness or deafness (Cooksey 1946; Igarashi 1984; Kendel et al 1991; Lacour and Xerri 1981).

Research of vestibular compensation, defined as the ways patients recover from and learn to live with vestibular disorders, including substitution, perdiction and other cognitive strategies, has revealed that following peripheral vestibular lesions, the degree of functional recovery is dependent on adaptive CNS processes which require adequate visual and somatosensory feedback (Herdman 1994; Igarashi 1984; Kandel et al 1991; Lacour and Xerri 1984, Shepard et al 1990; Zee 1994).

Animal research has emphasized the importance of early intervention of goal directed activities post lesion that make use of visual and somatosensory feedback to facilitate the compensation process. Unfortunately it is common for patients with peripheral vestibular lesions to voluntarily decrease and/or stop most activities as these activities usually induce undesirable symptoms of dizziness, nausea and/or loss of balance. As a result these patients often experience persistent signs and symptoms for years following the onset of the disorder (Telian et al 1990).

Animal studies have shown that following unilateral labyrinthectomy or neurotomy, vestibulo-ocular reflex (VOR) compensation (gain symmetry and decreased spontaneous nystagmus) and recovery of postural reflexes are delayed and substantially reduced when the lesioned animals are deprived of vision and restrictions in somatosensory input (Igarashi 1984; Lacour and Xerri 1981). It was noted on the other hand that these animals with vestibular lesions

showed post-operative recovery of VOR gain symmetry and balance control when exposed to goal directed exercises such as running on a rail to particular targets.

In human studies vestibular exercise has been used to reduce and/or eliminate symptoms of vertigo and dizziness (Norre and Beckers 1987; Norre and Beckers 1988; Norre and DeWeerdt 1980; Shepard et al 1990). They based the value of their vestibular exercises on the theory of habituation, ie. repeated exposure to provocative stimuli will result in a reduction of the symptoms of vertigo and dizziness, in turn improving function.

Although extensive research has been done to examine the mechanisms underlying VOR compensation, and although a number of human studies have evaluated the effects of exercise in the management of vertigo, little work has been done to assess the adaptive capabilities of the nervous system in the regulation of postural stability.

The aim of this study is to evaluate whether a rehabilitation training program designed to benefit functional balance capabilities in patients with chronic peripheral vestibular dysfunction is effective in improving standing balance in this patient population.

CHAPTER 2 - LITERATURE REVIEW

The ability of the body to compensate (defined as substitute, perdict and use other cognitive strategies) and adapt (defined as adjustments in the basic VOR and VSR) following vestibular disorders has long been recognised (Cooksey 1946; Igarashi 1984; Kandel et al 1991; Lacour and Xerri 1981). Sensory information derived from visual, proprioceptive, exteroceptive and auditory sensors and cognitive knowledge of an impending movement all contribute to functional recovery (ie. compensation) following loss or reduction of vestibular inputs. It is this multisystem input to the CNS which is of particular importance when looking at a rehabilitation perspective on the treatment of peripheral vestibular lesions (Herdman 1994).

2.1. PERIPHERAL VESTIBULAR APPARATUS

The vestibular system plays an essential role in spatial orientation tasks, in particular: 1) control of postural stability or equilibrium; 2) eye-head co-ordination or stability of gaze; and 3) perception of body schema (body's internal representation or map) (Horak and Shupert 1994).

The vestibular system provides the most important input to the brainstem: 1) information about head motion; and 2) changes in head position with respect to gravity. For full details of vestibular anatomy, morphology and physiology, see Kandel et al (1991); and Herdman (1994). The following information will provide a brief overview of the peripheral vestibular apparatus.

The inner ear (or labyrinth) is a complex system of chambers and

passageways in the temporal bones of the skull. Within the bony labyrinth lie interconnected thin-walled sacs and tubes filled with a fluid called endolymph.

There is a hearing portion of the labyrinth: 1) the cochlea ; and 2) a motion detecting portion, the vestibular apparatus. This apparatus is critical in upright, bipedal locomotion.

The peripheral vestibular system is comprised of two distinct types of sense organs; the semicircular canals and the otolith end-organs.

The sensory transducers cells common to both semicircular canals and otolith end-organs are the hair cells. In the semicircular canals these hair cells are situated in the ampulla. In the otolith they are situated in the macula. These hair cells are mechanosensitive and are responsible for transduction and the neural coding of head motion and head position relative to the linear acceleration vector due to gravity. Gravity is defined as the pulling force of the earth and objects on the earth ($9.8 m/s^2$).

Projecting from the apical surface of the hair cells are bundles of hair hypertrophied microvilli: kinocilium and stereocilia. Bending of the hair bundles towards the kinocilium leads to depolarisation of the hair cell and increases the firing of the afferent fibres in the eighth nerve. Bending away from the kinocilium leads to hyperpolarisation and decreased firing in the vestibular fibres of the eighth nerve.

2.1.1. SEMICIRCULAR CANALS

The vestibular system in each inner ear consists of three semicircular canals (superior, posterior and horizontal) which lie at right angles to each other and are paired with functional counterparts on the opposite side of the head.

This permits detection of changes in angular or rotational acceleration of the head in all directions whether this movement is active (turning of the head to look from side to side) or passively imposed (sitting still on a rotating chair).

Each semicircular canal is partitioned off by a cupula containing the sensory receptor hair cells. The cupula is a gelatinous mass that stretches from the ampullary crest to the roof of the ampulla. When the head is rotated, the inertia of the fluid in the semicircular ducts causes the fluid to push against the cupula, bending the sensory hair cells. This bending of the hair cells leads to depolarisation/hyperpolarisation depending on hair cell movement direction. This receptor system of the vestibular apparatus is sensitive enough to respond to angular accelerations or decelerations as small as $0.5°/sec^2$. This high degree of sensitivity is due to the arrangement of the cupula, the diaphragm like partition across the ampullary lumen (Kandel et al 1991).

There is a mechanical damping of the cupola-endolymph system resulting in prolonged activation in the vestibular nerve.

The semicircular canals (horizontal canals) operate in pairs in a push-pull fashion. The semicircular canal of one side is coplanar to the semicircular canal on the opposite side. With angular head motion; the endolymph of the coplanar pair is displaced in opposite directions, as a result, neural firing increases in one vestibular nerve and decreases on the opposite side. There are three advantages to the coplanar pairings of the canals: 1) pairing provides sensory redundancy; 2) pairing may allow the brain to ignore changes in the neural firing that occurs on both sides simultaneously (common mode rejection); 3) the push-pull configuration assists in the compensation for sensory overload; and 4) enhances the sensitivity of the system (Baloh 1984; Herdman 1994; Kandel et al 1991).

2.1.2. OTOLITH ORGANS

The otolith end-organs are responsive to linear acceleration and position of the head relative to the gravity vector.

There are two otolith end-organs in each inner ear, the macula of the utricle and the macula of the saccule. Similar to the ampullae in the semicircular canals, a portion of the floor of the utricle is thickened and contains hair cells along with the terminations of the vestibular ganglion cells. The macula of the utricle lies roughly in the horizontal plane when the head is held horizontally.

The macula is covered with a gelatinous substance embedded with crystals of calcium carbonate, called the otoconia. If the head is tilted or undergoes linear acceleration, the otoconia deforms the gelatinous mass and bends the hairs of the receptor cells. There is also a receptor-rich macula in the saccule. This macula is orientated vertically when the head is in its normal upright position. The macula of the saccule responds selectively to vertically directed linear acceleration.

The hair cells in the utricular macule are not polarized in a uniform pattern therefore, they can respond to tilt or linear acceleration in any of several directions in the plane. Gravitational acceleration is directed downwards upon the hair bundles. When the head is tilted, the hair bundle is displaced along the axis of polarisation, causing it to depolarise and excite its afferent fibres. A tilt in the opposite direction has the opposite effect. The axes of the hair cells of the macula all point toward a single curving landmark, the striola. A tilt of the head in any direction depolarizes other macular hair cells. It is felt that this dual signal aids in providing the brain with an accurate indicator of head position (Kandel et al 1991).

2.2. POSTURAL CONTROL

In order to function in standing, the nervous system must maintain control over the position and motion of the total body centre of mass relative to the base of body support, the feet. This aspect of the motor behaviour is referred to, in general, as postural control or postural stability. Research has identified three neural subsystems or processes necessary for the normal regulation of postural stability in standing (Kandel et al 1991; Nashner 1976). These include: sensory receptor systems - vestibular, visual, and somatosensory - which provide information regarding body orientation/motion and external reference points, such as vertical or the ground, those components of the central nervous system responsible for the organization and integration of the different types of orientation and motion clues provided by vestibular, visual and somatosensory systems, and the motor centres responsible for the selection and execution of task-specific reactions or adjustments.

2.2.1. SENSORY SYSTEMS

Sensing the state of equilibrium defined here as the intended final limb position which maintains standing balance, in particular providing information on the position and motion of the total body centre of mass (TCM) with respect to the base of support is a function of the central organization and integration of multiple sources of sensory inputs. No one sense organ of the body detects the position of the TCM relative to the base of support or other external reference points. For example, knowledge of how body segments are orientated relative to each other is not sufficient to ensure postural stability, as an external spatial

reference is required (Horak and Shupert 1994). Besides vestibular inputs, visual inputs or cutaneous inputs from the feet (support surface inputs) can provide an external spatial reference for body orientation and balance.

Each of these sensory inputs has its own unique frame of reference and provide different types of orientation and motion information. The otolith end-organs have the only fixed frame of reference in detecting head position relative to gravity vector. Visual, cutaneous and semicircular canal information has limitations. As stated earlier the semicircular canals provide sensory input proportional to a change in head velocity but not absolute position. Vision is unavailable in the dark. In addition, vision motion cues can be misleading as they can only provide relative information about motion between oneself and the environment. Therefore, the CNS can interpret motion of the visual surround as if it results from movement of the self and vice versa, for example the sensation of self movement one experiences when sitting on a stationary train when a moving train passes by. The somatosensory system uses proprioceptive components (muscle spindles, Golgi tendon organs and joint afferents) to provide information about relative position of body segments (from muscle force, muscle length and muscle velocity) to one another. The exteroceptive components of the somatosensory system (touch and pressure mechanoreceptors) can provide information about the location of the support surface and distribution of foot pressure but this information can vary dependent on the configuration and characteristics of the surface one is standing on.

Researchers have looked at the relative role of each of the three sources of orientation information in regulating standing balance. Nashner (1976) and Black et al (1983, 1988) devised methods to manipulate sensory input while examining standing balance performance of healthy subjects and patients with

impaired vestibular function. In these studies, subjects were instructed to maintain a quiet, erect standing position while being exposed to altered somatosensory and visual conditions.

The "sway stabilized" support surface condition was achieved by rotating the support base in direct proportion to the amount of the estimated anterior-posterior (A-P) displacement of the TMC (Nashner 1985; Nashner et al 1982). Thus, as the subject swayed and the TMC was displaced, this procedure minimized the rotation that would occur about the ankle joints. The consequence of this perturbance is that misleading or inappropriate sensory information from the proprioceptive component of the somatosensory system is supplied to the CNS. Nashner has termed this a sensory conflict situation. Sway is defined as anterior/posterior movement of the body about the base of support.

In a similar fashion, a sway stabilized visual condition was achieved by rotating the visual surround in direct proportion to the amount of the estimated A-P displacement of the TMC. Unlike having eyes closed where no visual information is available, this sway stabilized visual condition results in no relative motion between the body and visual surround. Therefore, the visual information to the CNS is consistent with a body that is not moving.

To estimate A-P TMC displacement, two methods have been used. In one method usinf posturography, recordings of A-P centre of foot pressure (CFP) position and patient height were used to estimate A-P centre of mass sway reference. The second method involved recording hip horizontal displacement with respect to the ankles using a platform - mounted potentiometer at the ankle level and attached via a light rod about the hips.

The main findings from these studies were: 1) patients with vestibular deficits were able to maintain standing balance with eyes open or eyes closed;

2) performance measures (TMC displacement) decreased during visual and somatosensory sway stabilized conditions; but patients were still able to maintain standing balance; 3) the CNS can function to maintain balance with peripheral somatosensory input (muscle spindles, tendon organs, tactile, pressure, and/or joint receptors); 4) normal subjects could maintain their balance during eyes closed and stabilized somatosensory conditions or during sway stabilized visual and support surface conditions; whereas 5) patients with vestibular dysfunction had noticeably increased A-P body sway and frequently fell; 6) the degree of body sway during perturbations was roughly proportional to the degree of vestibular problem.

The researchers concluded that loss of an absolute spatial reference (vestibular input), interfered with the resolution of conflicting sensory information.

A number of studies (Diener et al 1988a; Held et al 1975; Lestienne et al 1977; Soechting and Berthoz 1979; Talbott 1980) were conducted to examine the impact of visually derived orientation and motion cues on balance control. These studies have demonstrated that significant body motion or oscillations can be induced by these visual perturbations. Subjects were exposed to various visual scenes to induce a visually evoked disturbance of posture and self motion perceptions. These researchers used stroboscopic illumination, stabilization of the visual surround with respect to head movements, appearing as apparent body movement in pitch using stripe patterns which moved continuously up or down in front of the subject, and eye closure.

It was observed that the extent of body sway is dependent on the field size of the moving scenes, the velocity of the moving scenes, and the spatial frequency of the image. It was also observed that the visual effect on body sway

was even greater with a combination of body and visual motion (Diener et al 1986; Forssberg and Nashner (1982).

A dynamic contribution of visual information has been shown in investigations which have eliminated visual cues using stroboscopic illumination. Amblard and Cremieux (1985) demonstrated that destabilization of standing balance could be induced by using stroboscopic illumination at low frequencies. This result was interpreted to mean that visual cues when available will dominate over vestibular or somatosensory cues. Visual information is essential to stabilize posture and, since stroboscopic lights at low frequency only provide position cues, the subjects would have only a poor spatial reference from visual information. They also studied the effects of vision on standing balance by testing subjects standing on a thick foam rubber support, which effectively removes ankle proprioceptive input with enhancement of visual input. The enhanced visual input consisted of a pattern of vertical grating and stroboscopic illumination. They measured mean power from the power spectrum of the component frequencies of the acceleration signal at each anatomical level (head, hips and ankles) using accelerometers. They found that the role of vision in control of lateral body sway mainly depended on dynamic visual cues, especially those involving peripheral vision. They also confirmed the findings of other research that a minor but significant contribution is made by static visual cues (Dichgans et al 1973a; Diener et al 1988).

Dichgans et al (1973a)studied monkeys with bilateral labyrinthectomy to examine the mechanisms underlying the recovery of eye-head coordination. They investigated the relative contribution of the vestibular and neck afferents to compensatory eye movements made during active and passive head turning in monkeys. They also looked at the mechanisms underlying recovery of

compensatory eye movements following the removal of either the vestibular or neck loop or both. They concluded from their study that recovery of ocular stability is a complex process which require the potentiation of a neck-to-eye-loop, preprogramming of compensatory eye movements, and recalibration of functional relationships between retinal error signal and subsequent saccades.

Dichgans et al (1973b) studied single unit activity recorded with tungsten micro-electrodes from the vestibular nuclei of gold fish. Two sensory inputs were tested by rotating the animal in the dark (exclusive vestibular stimulation) or rotating the visual surround with the animal stationary (visual stimulation). The visual surround was a projection of black and white stripes onto the inner wall of a cylinder that surrounded the turn-table. They also tested the interaction of the two sensory inputs when the animal was rotated in the light with a stationary visual environment. Their data indicated that for goldfish, the firing rate of a second order vestibular neuron gives accurate information about body motion only in the presence of a physically stationary visual scene. In the presence of a stationary visual surround as reference, the neuronal response to animal rotation showed activity was sustained during constant velocity rotation. They proposed their study indicates that vision improves the speedometer function of the vestibular system during constant velocity rotation.

Talbot and Brookhart (1980) reached similar conclusions by studying the postural response of the dog to oscillations of a table the dogs were standing on, during normal sighted conditions and during blindfold conditions. They demonstrated that the dogs with vision tended to minimize error with respect to a stationary surround as compared to the dogs which were blindfolded. They concluded that the visual component of sensory information may play a dominant role in balance.

Berthoz et al (1979) proposed that the visual effects demonstrated by other researchers may be of even greater magnitude during combined body and visual motion. They studied the pitch angle representing forward and backward sway by using potentiometric methods. Subjects stood on a platform attached to a mobile cart, enclosed on all sides. Subjects wore cardboard blinders that blocked lateral and inferior visual fields. The visual scene consisted of a black and white checkerboard pattern. They found that the influence of visual surround motion is very striking in the direction of the image motion. By measuring peak body pitch they found that the effect of visual surround is approximately twice as great when visual surround and body motion are combined.

2.2.2. SUMMARY

It is clear that postural stability is dependent on the interaction and integration of orientation and motion cues derived from multiple sources.

2.2.3. MOTOR CONTROL

In order to evaluate the motor control strategies utilized during balance reactions in response to unexpected postural disturbances or sudden loss of balance, investigators have used the "moving platform paradigm". Nashner 1977; Nashner et al 1982; Horak and Nashner 1986; Woollacott et al 1980; and Dietz et al 1984, used a procedure where they suddenly imposed translation to the platform upon which the subject was standing, (forward and backward). Keshner et al 1987; Nardone et al 1990, used a rotation of the platform toe-up and toe down about the axis of the ankle joint. With adequate displacement

during these procedures the individual is placed in an unstable position. To avoid loss of balance and falling, a compensatory motor response is required to re-establish balance or postural equilibrium. The body partially compensates for suddenly imposed displacement of the support surface in standing due to the body's inertia and the viscoelastic properties of muscle, tendons, and ligaments. However, when large (unspecified) imposed displacements occur compensatory reactions are required which involves activation of a number of muscles acting on several body segments.

Early studies by Nashner (1976, 1977), employing platform translations indicated that in most healthy adults, equilibrium was restored through fixed patterns of early muscle activity (100-120 ms latency). EMG activity began in the ankle muscles and then in sequence, the thigh and then the trunk muscles became active. This sway correcting response was called the "ankle strategy", to denote that the body behaved as a single inverted pendulum swaying only about the ankles with little motion about the knees or hips. One of the short comings in Nashner's study was the sparcity of information on what their kinematics showed.

In the study of Horak and Nashner (1986), different widths of support surface in relation to foot size, were used. When standing on a narrow beam during platform translations, most subjects displayed a proximal to distal sequence of muscle activation, the trunk muscles were activated before thigh muscles. Here the term "hip strategy" was used to describe this balance reaction; as the movement to regain balance was based on angular excursion about the hip joint. Subjects' abdominal and quadriceps muscles responded to forward sway perturbations, hamstrings and paraspinals responded to backward sway perturbations.

A mixed reaction (motion detected at the ankles and hips) was also observed during reduced support surface conditions. Four out of the ten subjects tested exerted both ankle torque and shear force against the surface while moving both the ankle and hip joints. This pattern included activation of gastrocnemius, rectus abdominis, and quadriceps, followed by hamstrings and paraspinals.

They concluded that these results supported the hypothesis that a continuum of balance responses can be generated by one or a combination of a limited set of muscle activation patterns. The body relies on different responses which have different muscle activation patterns, different body movements, and different joint forces called postural control synergies or strategies (Horak and Nashner 1986). Normal subjects use an "ankle" or "hip" strategy" to restore balance. The "ankle strategy" is typically used by most subjects recovering from body sway when standing on a firm, flat support surface. A "hip strategy" is used on narrow or compliant surfaces, or when centre of mass position must be corrected quickly (Horak and Nashner1986).

Studies have indicated that patients with bilateral peripheral vestibular deficit do not tend to use the normal motor synergies to restore balance in conflict situations.

Horak et al (1990) used three subject groups to evaluate the effects of somatosensory loss and vestibular loss on postural control. They compared results from normal control subjects with results from these same subjects following foot and ankle anesthesia due to hypoxic ischemia. They then compared the results of these normal subjects with the results of subjects with bilateral peripheral vestibular loss.

All subjects were exposed to brief anterior/posterior (A/P) horizontal

displacement of the support surface (13 cm/s, for 250 ms) at unexpected intervals. They also provided two conditions of support; a) normal support surface, both feet in contact with force plates and b) shortened support surface condition, standing on a narrow beam with only 9 cm. of the feet in contact with the surface. The authors did not specify as to whether the tests were performed in bare feet or whether the subjects wore shoes.

They measured the A/P torque and horizontal A/P shear force, and EMG signals were recorded from tibialis anterior, medial gastrocnemius, rectus femoris, hamstrings, rectus abdominis and lumbar paraspinal muscles. They also video taped and evaluated body motion about ankle, knee and hip angles.

Horak et al (1990), compared the subject groups on how well they used sensory information to control postural sway in quiet stance. They used the protocol described by Nashner et al (1982) and measured A/P body sway by a platform-mounted potentiometer at ankle position attached to a light rod fixed to the hips. Therefore their study had the same problem as Nashner, as previously stated, one of Nashner's short comings in his 1982 study was his lack of kinematic information.

They reported that subjects with somatosensory loss were able to maintain equilibrium in response to surface displacement but to do so they had to change their pattern of postural movements. They noted that in subjects with somatosensory inputs disrupted by the ischemia protocol, the changes in hip joint angles were about four times larger than those of the ankle. The subjects added early proximal muscle bursts in the abdominals and quadriceps at 100 +/- 10 ms and 125 +/- 16 ms respectively. All of these subjects demonstrated increased hip flexion in response to platform displacements.

When they compared the normal subjects to the bilateral vestibular loss

patients on the short surface tests, they noted the vestibular patients did not increase hip flexion and were therefore ineffective in moving the body towards equilibrium. The authors did not indicate to what degree the vestibular patients were unable to balance nor did they state whether these patients actually loss their balance and fell. They concluded vestibular information does not appear to be essential when using a normal ankle strategy, whereas, somatosensory information would appear to be critical for ankle strategy. On the other hand, vestibular information appears to be critical for hip strategy. It was suggested that execution of hip strategy requires a complete complement of sensory information as the somatosensory and vestibular systems provide different information both of which are required for accurate internal representation of body movement and relationship of the body to space.

They noted that during sensory conditions the vestibular patients were unable to balance with eyes closed or sway-referenced and that all four patients went into free-fall within seconds of the trial onset.

2.3. OCULOMOTOR CONTROL - EYE-HEAD COORDINATION

Gaze stabilization, the ability to maintain fixation of an object on the retina during head movements is an essential function for the normal processing of visual information. Inability to maintain gaze during head motion or the lack of eye-head coordination will not only result in blurred vision, but also may cause perceived sensations of self motion or of visual surround motion. These problems accompanied by the sensations of dizziness or vertigo can cause disturbance of balance (Black et al 1983; Nashner et al 1982; Soechting and Berthoz 1979). A number of neural processes contribute to gaze stabilization.

For an in-depth review of these processes see, Fuchs (1985); Fuchs et al (1989); Goldberg and Colby (1989).

There are five neuronal systems that keep the fovea on target. Two of these systems are primarily responsible for stabilizing the eye during head movements and three others keep the fovea on a visual target (Kandel et al 1991).

Of primary importance to eye-head coordination is the vestibulo-ocular reflex (VOR). The VOR operates to stabilize gaze if the position of the head is altered. The VOR is a disynaptic reflex arc. Angular motion of the head is detected by the semicircular channels. The afferent fibres associated with the semicircular channels synapse on cells within the vestibular nuclei. These vestibular neurons, in turn, synapse on alpha motorneurons of extraocular eye muscles. The VOR is responsible for the generation of eye movements which match head movements (eye movements which are equal in magnitude but opposite in direction to head movements). Since the main function of the VOR is to stabilize the visual world on the retina, the gain of the reflex must be close to unity, otherwise the image will slip. The VOR responds to brief or rapid head movements. During sustained or slow head movement the vestibular signal ultimately fails and gaze compensation is lost.

Other processes that are important to gaze stabilization and fixation of objects on the fovea are the optokinetic reflex system, smooth pursuit system, saccade, and vergence movements.

The optokinetic reflex system (OKN) can also generate eye movements in a similar fashion to vestibular nystagmus. Vestibular nystagmus is defined as a spontaneous horizontal nystagmus, usually occuring after vestibular lesion, with slow phase eye movements directed toward the lesioned side, coupled to quick

phases directed toward the intact side. The OKN compensates for the defects in the vestibular system by using visual motion of head movements to drive the eyes. The optokinetic system drives the eyes in the direction of full-field motion, which is opposite the head movement causing that motion. OKN works best at low field velocities, the vestibular system at higher head velocities. The OKN responds to slow visual motion induced by slow head movements.

The smooth pursuit system moves the eyes in space by calculating how fast the target is moving and then making sure the eyes move accordingly. The smooth pursuit system keeps a single target on the fovea. In comparison the OKN system stabilizes the eyes in space when the head movement would otherwise cause the entire retina image to move. The smooth pursuit is a voluntary movement which requires a moving stimulus to calculate the required eye velocity. This system is limited in that it has a maximum velocity of 100^0/sec.

The saccadic eye movement system is responsible for rapidly directing gaze to a target of interest in visual space. This system generates a saccade (conjugate, ballistic movement of the eye) that brings the image of the target on line with the fovea. The saccades are extremely fast at speeds of up to 900^0/sec. The saccade responds to visual stimulus, sounds, tactile stimuli, memories of locations in space, and to verbal commands. Saccadic velocity is determined by the distance of the target from the fovea. The amplitude and direction of saccades can be voluntarily changed but not the velocity of the movements.

The last system involved in keeping the fovea on a visual target is the vergence movement system which aligns the eyes to look at targets with different depths. The previous systems (VOR, OKN, smooth pursuit, and saccades) move both eyes the same amount in similar directions (conjugate). The eyes are also capable of moving in opposite directions when converging or

diverging to focus on objects at different distances. The vergence system generates these disconjugate movements. This system will keep the image of an object aligned on each fovea whether the object moves toward or away from the viewer.

All of the five systems work to maintain stability of gaze and to keep the fovea on a visual target: see Table 1.

Vestibular and neck reflexes provide information to help align the head and body with respect to gravity; the vestibular reflexes which are activated by changes in the position of the head, and neck reflexes which are triggered by tilting or turning the neck.

Vestibular reflexes act on the neck (vestibulocollic) and on limbs (vestibulospinal); both are evoked primarily by sensory input from the otolith organs. The vestibulocollic reflexes counteract head movements keeping the head stable. If the body is tilted forward without bending the head, the vestibulocollic reflexes contract the dorsal neck muscles to return the head to vertical (Goldberg et al 1991). One of the functions of the vestibulospinal reflex is to produce tone control of the limb muscles to prepare the body for landing, during a fall.

Other reflexes in the neck and limb muscles respond to bending and rotating the head relative to the body [cervicocollic (neck) and cervicospinal (limbs)]. Neck muscle spindles and joint receptors of the upper cervical vertebrae are the receptors involved in these actions. The cervicocollic reflexes act as synergists with the vestibulocollic reflexes produced by the same head movements. On the other hand, the actions of the cervicospinal reflexes on limb muscle oppose the actions of the vestibulospinal reflexes (Goldberg et al 1991).

Vestibulo-spinal and neck reflexes are not easily seen in the normal adult

TABLE 1. **Functional Eye Movements**

A. Movements that stabilize the eye when the head moves:

Eye Movement	**Function**
VOR	vestibular input to hold image stable during brief or rapid head rotation
OKN	uses visual input to hold images stable during sustained or slow head rotation

B. Movements that keep the fovea on a visual target:

Eye Movement	**Function**
Saccade	brings new objects onto the fovea
Smooth Pursuit	holds the image of moving target on the fovea
Vergence	aligns the eyes for different viewing distances in depth

but if the vestibular apparatus is damaged, reflexes of the neck, limb and eye muscles can become prominent (Goldberg et al 1991).

2.4. RECOVERY OF FUNCTION FOLLOWING PERIPHERAL VESTIBULAR DEFICITS

How does a patient regain function following a lesion or damage to the nervous system? Clinically we are aware that patients do recover motor and sensory function after injury to the nervous system (central and/or peripheral). This recovery has been called adaptation, compensation or plasticity (Igarashi 1984; Lacour and Xerri 1981; Shumway-Cook and Horak 1990). It has been stated by Igarashi (1984), that following an insult to the peripheral vestibular system, inputs from other sensory modalities must be mobilized and integrated at many levels before sufficient adjustments to the system can be achieved. Experimenters have frequently used the vestibular lesion model to gain insight into the adaptive mechanisms that underlie functional recovery (Igarashi 1984; Lacour and Xerri 1981; Precht and Dieringer 1981).

From a clinical perspective, however, it is common for patients with peripheral vestibular lesions to experience persistent signs and symptoms for years following the onset of the disorder. These signs and symptoms may include; postural imbalance, gaze instability during head movements, and vertigo (personal observations).

Two physiological mechanisms and a phenomena have been proposed to account for behavioral changes referred to as vestibular compensation. these include:

1) Sensory substitution (Igarashi 1984; Jenkins 1985; Pfaltz and Kamath 1983;

Shumway-Cook and Horak 1990). Other sensory modalities provide orientation and motion information, such as, visual and somatosensory input which can be used to substitute for loss of vestibular inputs. Sensory substitution would occur for static behaviours (spontaneous nystagmus, skew deviation of the eyes, and postural asymmetries) and dynamic behaviours (VOR gain during head movement).

2) A balancing of tonic neural activity between the left and right vestibular nuclei (Dix 1976; Dix 1984; Igarashi 1984; Kandel et al 1991; Pfaltz and Kamath 1983). It has been proposed that the compensation for unilateral peripheral vestibular deficits involves an initial inhibition of the resting discharge rate of neurons within the vestibular nuclei (neurons which receive inputs from the intact vestibular sensors). The cerebellum has been implicated as a source of inhibitory modulation of neurons in the vestibular nuclei (Dix 1976; Dix 1984; Monnier et al 1970). In addition, visual and somatosensory input to the vestibular nuclei may also contribute to re-balancing of tonic activity in the vestibular nuclei (Courjon et al 1977; Pukonnen et al 1977).

3) Habituation (Cooksey 1946; Norre and Beckers 1988; Shumway-Cook and Horak 1990); It has been proposed that habituation brought on by repeated exposure to a provocative stimulus or movement will result in a reduction in the intensity of vertigo.

The results of experimenters who have examined underlying mechanisms of vestibular compensation need to be reviewed in order to realize how these process contribute to recovery of function following peripheral vestibular deficits. The following sections will review the animal and human research into vestibular compensation.

2.5. ANIMAL STUDIES

In a series of experiments, Lacour and colleagues have examined vestibular compensation in different animal species: cats, baboons and monkeys.

In one study Lacour et al (1976) found that when the hindlimbs of baboons were restrained for four days after unilateral vestibular neurotomy there was a significant delay in their recovery rate from postural asymmetries and locomotor ataxia. Unilateral vestibular neurotomy was performed on 6 baboons. Soleus H-reflex was elicited and recorded bilaterally to assess excitability of soleus motoneuron pool ipsilateral and contralateral to the lesioned side. Two indicators were used: the H-reflex recruitment ratio (RR) and the H-reflex recovery cycle. Posture and sequences of usual motor activities, such as walking, running, climbing, and jumping were assessed from cinematographic recordings and from observation platforms. A record of the number of falls to the operative side per unit of time and of the degree of postural asymmetry were used as outcome measures.

The researchers found that following unilateral vestibular neurotomy, a severe imbalance appeared in the H-reflex responses. A substantial ipsilateral reflex hypoexcitability and contralateral reflex hyperexcitability was noted in all animals. Recovery to normal and symmetrical reflex responses occurred in three distinct periods: a critical stage (0-2 days postoperatively), an acute stage (2 days to 1 week postoperatively) and a compensatory stage (1-3 weeks postoperatively).

The critical stage was short lived and was characterized by ipsilateral reflex hypoexcitability and contralateral reflex hyperexcitability. The authors

report ipsilateral RR value averages of 30% of reference (pre operative level) 36 hours after surgery. On the contralateral side , the mean RR value represented 190% of reference. During the acute stage the baboons showed a progressive return to normal values of contralateral RR values as compared to an incomplete and less rapid recovery of the RR values on the ipsilateral side. The compensatory stage showed that 280 days postoperatively, the reflex responses had returned to normal levels on ipsilateral and contralateral sides. Lacour et al did not provide information to indicate any different results in H-reflex associated with the restrained baboons.

The researchers noted the recovery from postural and locomotor deficits also went through the same three stages in the unrestrained baboons: critical, acute, and compensatory. During the critical stage all animals exhibited: 1) asymmetrical distribution of muscle tone as measured by spontaneous EMG activity from the splenius muscle. 2) A head-tilt toward the operative side was observed; deviation of the ocular globes toward the operative side and a rotatory nystagmus. 3) leaning, staggering, and falling to the side of the lesion was also observed.

The acute stage showed attenuation of most of the disturbances noted in the critical stage. Posture was less asymmetrical although some degree of head tilt remained. Spontaneous nystagmus disappeared in all animals during this stage.

During the compensation stage the remaining defects were very slight. One month following surgery it was difficult to distinguish an operated from a non-operated baboon.

In the baboons restrained for four days postoperatively the time course of recovery showed serious alterations. Hindlimb restraint delayed the recovery

especially during the acute stage. This stage occurred later (began after one week) and extended for more than three postoperative weeks. Spontaneous nystagmus disappeared by postoperative day 2 or 3; a finding similar to the unrestrained baboons.

Leaning and falling continued for three weeks postoperatively in the restrained group, whereas in the unrestrained group there were no balance problems noted within 4-7 days.

In subsequent studies, Lacour and Xerri (1981) examined the effects of sensorimotor restriction on motor recovery following unilateral and bilateral vestibular neurectomy in cats. Postural symmetry in standing was assessed by calculating the ratio between the force exerted by each forelimb and each hindlimb. Locomotor function was assessed by the maximum performance of each cat corresponding to the highest speed of rotation of a beam which did not result in a fall, over four consecutive trials.

The neurectomized cats were divided into two groups. One group was allowed to remain free after surgery in their usual environment with normal vision. The second group underwent seven days of sensorimotor restrictions (SMR) but were allowed normal vision. The researchers did not provide information about the specifics of what form of restriction was used with the cats in this study. SMR was applied either in the first, third, or the eighth week postoperatively. They also submitted some of the cats who had a unilateral neurectomy to a second vestibular neurectomy one year after the first, to test the influence of the remaining labyrinth in the recovery process. The researchers reported that SMR applied in the first postoperative week delayed postural symmetry. They noted no difference in the postural asymmetry when comparing the cats two days post operatively and nine days following release of restrictions

in this group. It was not until after the ninth day that compensation of postural deficits is achieved. For the cats restricted in their third postoperative week, they noted that the recovery process was suspended, postural asymmetry values were identical before and after the restrictions but that they regained normal postural recovery with time. The restriction applied in the third week did not decompensate the cats. They noted no effect on postural recovery in the already compensated cats which were restricted after eight weeks. Their research identified that postural symmetry was eventually achieved whatever the time of the sensory restriction. On the other hand, recovery of locomotor equilibrium remained incomplete. They report that up to one year after unilateral vestibular nerve section, maximum performance was approximately 40% in the cats restricted in the first week postoperatively and 50% in the cats restricted in the third week. Cats restricted in the eighth week were not affected, as they had already compensated (symmetrical postural responses) prior to the restrictions being applied.

Based on the effects of sensorimotor restriction on motor recovery in the baboons and cats, it was concluded that compensation for loss of vestibular inputs requires active behaviours that would result in multi-sensory inputs, and that it is crucial to encourage activity early post lesion.

Support for the notion that activity which is guided and directed will promote recovery of postural stability following peripheral vestibular lesions comes from a series of studies by Igarashi and colleagues (Igarashi et al; 1975, 1979 and 1981).

Igarashi et al (1975) designed a study to determine the influence and effectiveness of physical exercise on balance during locomotion in monkeys following unilateral labyrinthectomy. Animals were trained pre-operatively on a

rail test for locomotor balance. This test consisted of two cubical compartments connected by an X-shaped rail. The rail could be rotated along its longitudinal axis. The squirrel monkeys were trained to traverse this rail while shuttling from one compartment to the other.

Each monkey's daily thresholds (maximum performance ability) were measured. They measured the rail performance level and the number of days it took to reach the pre-operative level. The pre-operative level consisted of the ability of the monkey to achieve more than four consecutive stable rail thresholds (traverse rail while shuttling from one compartment to the other avoiding a shock).

The monkeys were also tested for spontaneous nystagmus. Subdermal platinum electrodes were implanted bi-temporally, and the eyeball movement was recorded on a polygraph recorder. Post-operatively spontaneous nystagmus examination was performed on the first day, the third day, at one week and after that, once or twice a week.

The post-operative measures of locomotor performance were started on day two and were performed three times a week for six months.

The monkeys were divided into two groups. An exercise group who were given forced walking for 120 min. per day in a laboratory- built rotating cage. The other group had no exercise post-operatively. The authors did not indicate the level of activity allowed for the non exercise group postoperatively.

It was found that the exercise group had less spontaneous nystagmus, and when it existed, it disappeared faster than did spontaneous nystagmus in the non-exercise group but the difference was not significantly different between the two groups. Although the authors report that nystagmus disappeared before the monkeys had regained motor compensation they failed to provide

information on the length of days for the motor compensation.

Comparison of the locomotor performance levels did not show any statistically significant difference between the exercise and non-exercise groups. There was evidence of a tendency that in some of the animals the physical exercise might be helpful for locomotor compensation during the early postoperative stage. The researchers report that in the exercise group, the time when the monkeys regained their pre-operative level of locomotor function was found to be later than the time the spontaneous nystagmus disappeared (nystagmus disappeared after a mean of 10.5 +/- 7.632 days). This was not so for the non-exercise group (nystagmus disappeared after a mean of 35.66 +/- 27.982 days).

In a subsequent study, Igarashi et al (1979) studied the effects of two different exercise programs on locomotor recovery in squirrel monkeys following unilateral utricular nerve resection. Pre-operatively, squirrel monkeys were trained to run on a platform runway test. Once the subjects were able to attain the pre-operative criterion sequence, they were randomly assigned to one of three groups pre-operatively. The first group (N = 7) received an hour of physical exercise in a squirrel monkey rotating cage moving at 6 rpm. The second group (N = 7) was trained to do sixty running shuttles daily on the squirrel monkey rail rotating at 200 rpm. This exercise program was more impulsive and required maximum agility, as compared to the first group with their continuous and constant trotting in a rotating cage. The third group (N = 7) received no physical exercise.

They measured average total deviation counts between postoperative day one through day ten. This measure consisted of counting the number of times the animal activated the channel shock (animals received a shock whenever

they deviated from the set channel) while walking on the platform runway test. They measured the degree of deviation and days postoperatively (Igarashi 1974).

They found a slight reduction in the average total deviation counts in both exercise groups which was slightly faster than that of the control (non-exercise) group. The authors did not define the slight reduction in the average total deviation count in statistical terms. However, they did not find a difference in reduction rate between the two exercise groups. The mean postoperative days to regain pre-operative criteria were 17.3 days for the rotating cage exercise group, 19.4 days for the rail- traversing exercise group, and 23.3 days for the non-exercise control group. When comparing the number of days, they found that the rotating-cage group had a significant difference compared to the control group ($p<0.05$).

This experiment had a number of different aspects from their previous research Igarashi et al (1975). They used the monkey platform runway test (deviation measurement); previously they had used the squirrel monkey rail test. In this experiment they exercised the monkeys pre- and postoperatively whereas, in the previous study the animals were only exercised postoperatively. The other difference between these two studies was the type of vestibular lesion. In the first study they performed a unilateral labyrinthectomy and in the second study they used a unilateral utricular nerve resection, which is only a partial lesion leaving the semicircular canals and saccule end-organ intact. It was suggested that the effects of exercise which were more pronounced in the second study could have been due to these differences.

In subsequent research, Igarashi et al (1981) compared two groups of squirrel monkeys after unilateral labyrinthectomy. They used the squirrel monkey

platform runway test to measure the degree and direction of overground gait deviations in an ordinary trot. The monkeys were behaviorally trained to master running on a centre strip. Pre-operative performance criteria were measured. They compared pre- and post-operative daily gait deviation counts, and the number of calender days to reattain the pre-operative performance criterion between the two groups of monkeys.

The experimental group consisted of five squirrel monkeys assigned to the rotating cage exercise compared to another five monkeys for the control group with no exercise. The exercise consisted of two and one half hours of daily running on a rotating cage.

The animals in the rotating cage exercise group reattained the pre-operative performance criterion significantly faster ($p<0.05$) than the control non exercise group (about 20 days in the exercise group and about 35 days in the control group) .

They reported that a significant enhancement of locomotor balance performance was achieved post-operatively in the exercise group as compared to the non-exercise control group.

Igarashi et al (1988) conducted experiments on squirrel monkeys after two-stage bilateral vestibular lesions. They measured the extent and direction of gait deviations during a straight overground run, using the squirrel monkey platform test. They also measured the spontaneous nystagmus (SN) postoperatively in the dark through standard DC electronystagmography; the daily slow-phase eye velocity (SPEV) was manually calculated. The squirrels underwent unilateral labyrinthectomy and then were assigned to two groups. One group was assigned to exercise and the control group received no exercise. When an animal reached pre-operative performance levels they underwent

further operative procedure, contralateral labyrinthectomy.

After unilateral labyrinthectomy, it was found that the average number of days to reach POC-1 (first calender day the animal reached the pre-operative performance level) for the exercise group was 7.3 days, and 13.7 days for the control group. These differences were not statistically significant. There was a significant difference when comparing the two groups as to when they reached POC-2 (the calender day when the animal achieved eight uninterrupted trial days). The exercise group reached the second level in a shorter length of time ($p<0.01$). Total deviation counts also showed a significant difference ($p<0.05$) between the two groups.

On comparing the SPEVs of SN there was also a significant difference of ($p<0.01$) for the exercise group. The control group continued to have SN with high SPEV for later days in the study. The authors did not specify how many days later for the control group.

Following bilateral lesions all the monkeys showed great difficulty in regaining locomotor balance. However, there was a significant difference ($p<0.05$) between the two groups in total deviation counts. To reach POC-1 an average of 62 days was required for the exercise group as compared to 81 days for the control group (no significant difference). To reach POC-2 it took 118 days for the exercise group, and the control group took from 126 to 168 days, one animal did not reach POC-2 in 300 days post operatively. After bilateral lesion the average number of days to reach the 100^0/sec. level was 4.3 for the exercise group and 12.5 for the control group (no significant difference).

Igarashi et al (1987) demonstrated that physical exercise did make a significant difference in the total deviation count for animals after both unilateral and bilateral vestibular induced lesions. They also demonstrated that following

unilateral lesion the time to reach POC-2 for the exercise group was significantly shorter than the control group.

Taken together the results of the studies by Igarashi and colleagues support the view that physical exercise contributes to compensation. The authors make special note of the type of exercise that facilitates recovery of balance: those exercises that were goal orientated and involved agility (running on a rail to particular targets) were superior to continuous constant exercise that were not impulsive or taxing to the animal (natural trot on a normal support surface for 1 hour).

Mathog and Peppard (1982) evaluated the effects of exercise during recovery from unilateral labyrinthectomy in cats. In this study, four cats did not receive a labyrinthectomy, three cats had a unilateral labyrinthectomy and did not receive exercise, and three cats had unilateral labyrinthectomy and received exercise. Daily non goal directed exercise consisted of eight hours of roaming about the laboratory with fifteen minutes of play and jumping off a low stool.

Mathog and Peppard evaluated: 1) optokinetic nystagmus during constant velocity rotation at 24^0/sec. After one minute for equilibration, they recorded and measured induced eye movements (they used an optokinetic drum which rotated around the subject at 24^0/sec); 2) VOR in response to caloric stimulation; 3) horizontal VOR during sinusoidal oscillations at frequencies of 0.01 Hz., 0.05 Hz., and 0.10 Hz with peak velocities of 120°/sec and 300°/sec for each test frequency.

They measured the slow-phase velocity of nystagmus beat, and duration of slow-phase velocity for both the caloric and sinusoidal acceleration stimuli. They used a formula [((Vl- Vr) / (Vl+Vr)) X 100] where Vl and Vr represented the average maximum velocities for the slow phase components of nystagmus in the

left and right directions to calculate asymmetry.

Tests were performed on the four healthy cats for magnitude of nystagmus, symmetry of response, habituation, and differences in responses with changes in the frequencies and velocity of sinusoidal stimuli. They noted maximum slow-phase velocities were similar when comparing (L) and (R) ears during caloric testing and that repeated tests did not produce evidence of habituation. The sinusoidal rotation test also revealed a stable VOR, and the response was symmetrical with little or no tendency toward a reduced response with repetitive testing.

In the lesioned cats which did not exercise, they noted a directional asymmetry shift of 100% when evaluated by sinusoidal rotation tests, regardless of frequency and velocity of stimuli. The authors did not indicate the level activity the non exercise cats were permitted. There was characteristic complete asymmetry for 12-14 weeks, followed by gradual recovery. Although they report that the cats appeared to walk and carry on normally, at 19 weeks there was still a lasting asymmetry of approximately 20%.

In the three cats treated with exercise following labyrinthectomy the percentage of directional asymmetry was markedly altered; the period of complete asymmetry was shortened and recovery times were notably decreased. Their recoveries reached 20% directional asymmetry at 7-9 weeks and asymmetry approached zero regardless of the stimulus.Mathog and Peppard concluded that general types of exercise can reduce the recovery time for a cat that has undergone labyrinthectomy.

In a study by Fetter and Zee (1988), vestibulo-ocular and optokinetic responses in 6 Rhesus monkeys were examined before and after unilateral labyrinthectomy to quantify the extent and time course of compensation. Eye

movements were recorded using the magnetic-field search-coil technique.

Vestibulo-ocular responses were elicited by rotating about the earth-vertical axis, a frame that supported the magnetic-field coils and contained the primate chair. The VOR responses were measured pre and post-operatively, in both light and dark situations to sinusoidal oscillations and to constant velocity stimuli. For constant velocity stimuli the speed of rotation was varied from 30^0-300^0/s and they randomized the order in which the velocity steps were delivered. The peak head velocity was performed under three different visual conditions: darkness, viewing in the light (earth-fixed visual surround), and viewing in the light (chair-fixed visual surround).

Nystagmus in response to caloric simulation was also evaluated pre and post-operatively by infusing 20 ml of ice water into the external auditory canals.

Optokinetic nystagmus (OKN) was elicited using a full-field, rotating drum that contained a random pattern of black circles on a white background. Lights could be turned on and off during rotation. Constant-velocity rotations for 50 sec. were used at speeds of 30°-180°/sec., in clockwise and counter clockwise directions. Optokinetic after nystagmus (OKAN) was measured in four of the five monkeys.

Pre-operatively the researchers found:

1) symmetrical left-right VOR gain responses, average gain of 1.0 for sinusoidal rotations of 0.05 Hz. with peak velocities of 30° - 300° /sec. and slightly above 1.0 at 0.25 Hz. with peak velocities of 30° - 300° /sec.

2) VOR time constants (TC) varied with stimulus, being higher for the rotations from 30° - 120° /sec and lower for the higher velocities. The researchers reported that the TC was always the lowest at the highest speed of rotation (300° /sec) and the profile of decay of slow-phase velocity was not always

exponential.

3) Caloric stimulation produced a mean value for the maximum slow- phase velocity of 115°/sec. (range 40-250^0/sec).

4) Left-right OKN responses were symmetrical. the mean value of the initial component of OKN was 70% of the steady state response for 30° and 60°/sec. drum velocity, 60% for 120°/sec., and 50% for 180°/sec. The mean steady state OKN gains were 1.0 for 30 and 60 /sec., 0.9 for 120°/sec., and 0.8 for 180°/sec.

5) The time constant of OKAN decay varied considerably between animals. The overall mean time constant was less than 45 sec. at each velocity tested.

The researchers compared the pre-operative values to their findings postoperatively. Eighteen to twenty hours post- operatively, all the animals showed a tilt, a turn of the head towards the side of the lesion, and intermittent head nystagmus. The animals were unsteady and tended to circle toward the lesioned side. Postural stability improved within hours of being allowed to move around their cages in the light. In the light, all animals had either no or only small amounts of spontaneous nystagmus. Spontaneous nystagmus was present in the dark and decreased to a mean level of 180°/sec. at day ten.

When first measured the average VOR time constant had decreased from a pre-operative value of 35 sec. at 60°/sec. velocity to 7.4 seconds (range 6.0-9.1 sec.). There was a subsequent small but significant (p <0.005) increase (2-3 sec.) in three of the animals during the three month period following labyrinthectomy. They did not observe directional asymmetry. Fetter and Zee report a consistent finding in all of their animals of an immediate and lasting decrease in the time constant of the VOR. They presumed that these changes reflected a disabling of the velocity-storage mechanism. This is a central neural network that functions to prolong the VOR time constant above the value

predicted by cupula-endolymph mechanics alone. The reason for loss of velocity storage after labyrinthectomy is not clear but it is postulated that preservation of vestibular responses would be inappropriate when the VOR was malfunctioning due to an imbalance of peripheral input. The presence of spontaneous nystagmus might be because the CNS interprets the maintained input due to the imbalance as a persistent unidirectional rotation, and this could lead to a decrease in time constant of the VOR (habituation).

The initial value of the VOR gain in darkness was low in all six monkeys. The VOR gain during sinsuoidal rotations in the dark at a frequency of 0.25 Hz. with peak head velocities of 30°/sec-300°/sec. reduced from 1.0 pre-operatively to 0.5 when initially measured after the labyrinthectomy except for rotations to the lesioned side which had an even lower gain value. When exposed to the light, the VOR gain rapidly increased to 0.8. A similar behaviour was noted for rotations to the lesioned side at stimuli of 30-60 /sec. but at higher velocities the compensation was noted to progress more slowly with values that never reached the level of those for rotations toward the intact side (range of values to the intact side 0.77-1.03, values to the lesioned side ranged from 0.61-0.98). There was a significant ($p<0.05$) improvement in the ability to suppress and to enhance VOR (visual-following reflexes) during sinusoidal oscillations (0.25 Hz.) for the (chair-fixed visual surround) and (earth-fixed visual surround) viewing conditions. The values of VOR suppression gains increased from 0.57 pre-operatively to 0.71 at day 93 post lesion for sinusoidal rotations in the dark at a frequency of 0.25 Hz with peak head velocity of 30°/sec and from 0.49 to 0.73 at 120°/sec. stimulus. These figures represent an approximate increase of 25% in both the VOR enhancement and VOR suppression gains. They noted that most of the improvements in the ability to suppress the VOR were observed

after exposure to light. They note that these findings should be interpreted cautiously as the smooth pursuit system may be a major factor in providing the gaze stability. Nevertheless, they felt that the increase in these values could still be a useful adaptation in circumstances in which the pursuit system might not be immediately engaged (sudden trip or fall).

The initial values of slow-phase velocity of OKN was highly variable but average value showed characteristic changes with time that were similar for all stimulus velocities. Initially there was an increase in the value of slow-phase velocity for drum rotations toward the lesion side which started to decline at day 14 to values approximately 25% greater than pre-operative values. Values of slow-phase velocity for drum rotations to the intact side increased more slowly and gradually approached levels similar to those for drum rotations toward the lesioned side.

The researchers suggest that the net changes in the OKN reflect a combination of:

1) An inherent bias created by the labyrinthectomy which leads to better visual following when the optokinetic stimulus is in the same direction as the slow phase of the spontaneous nystagmus.

2) Adaptive need, which requires better visual following when optokinetic stimulus was in the direction opposite to the slow phase of the spontaneous nystagmus.

There was no response to caloric stimulus in the operative side of any monkey, but the response in the intact ear increased significantly from pre-operatively levels in 5 out of the 6 monkeys. The average value of the maximum response increased from 120°/sec. (range 50°-250°/sec.) to 195°/sec. (range 86°-312°/sec.). These values were significantly different ($p<0.05$).

Although Fetter and Zee report a significant difference in caloric response they do not indicate the time frame over which the results occurred.

In a companion study, Fetter et al (1988) examined the effects of unilateral labyrinthectomy and occipital lobectomies to investigate the influences of visual experience upon vestibular compensation in twelve Rhesus monkeys. Nine animals underwent a unilateral labyrinthectomy. Of these nine monkeys, three had a bilateral occipital lobectomy 6-10 months prior to the labyrinthectomy and three had their occipital lobectomy 4-6 months after the labyrinthectomy. The remaining three monkeys had only the labyrinthectomy. The last three monkeys (of the original twelve) had undergone the initial occipital lobectomy only.

The details of the methods and testing procedures have already been noted in the previous description of Fetter and Zee (1988) . Each of the nine monkeys was immediately placed in a dark room following the labyrinthectomy until after the first recording session (18-20 hours post operatively). The initial measurements of spontaneous nystagmus and the response to rotations were made in complete darkness. Of the nine monkeys without prior lobectomy (seeing monkeys) three were allowed normal visual experience (light-kept). Three (seeing monkeys) were kept in complete darkness for four days post operatively (dark-kept).They were then allowed normal visual experience and the remaining three were kept in darkness. The three animals which had undergone a prior occipital lobectomy only were treated with the same conditions as the light-kept monkeys.

The researchers reported pre-labyrinthectomy control data for VOR gain during sinusoidal rotation at 0.05 Hz with peak velocity of 30°/sec. was close to 1.0 in all monkeys. Mean time constant varied with the velocity, but it was the

highest for sinusoidal rotation at a frequency of 0.05 Hz. with peak velocities from 30°- 120°/sec. (ranging from 28-35 sec.) Pre labyrinthectomy there was no asymmetry of vestibular responses in any monkey either with constant-velocity or with sinusoidal stimuli. Optokinetic responses were symmetrical in all of the seeing monkeys pre-labyrinthectomy. In two of the three lobectomized monkeys there was considerable asymmetry of optokinetic responses.

They found that the labyrinthectomized monkeys who did not have an occipital-lobectomy recovered nearly normal posture, balance and locomotor behaviour within days. Whereas the monkeys with the lobectomy recovered more slowly. These monkeys showed larger head tilts and increased circling behaviour that persisted for weeks. The researchers did not distinguish between the light-kept and dark-kept non lobectomized monkeys in respect to general observations post labyrinthectomy.

All three monkeys who received a bilateral occipital lobectomy prior to the labyrinthectomy initially showed considerably larger amounts of spontaneous nystagmus in light than did the monkeys that were not lobectomized. After 20 hours the average slow-phase velocity was similar in the three groups with values ranging from 22° to 62°/sec. (occipital-lobectomized), 24° to 48°/sec (dark-kept), and 32° to 54°/sec (light-kept). The level of spontaneous nystagmus decreased in a similar fashion for all three groups.

The initial VOR time constant values taken 18-20 hours after the labyrinthectomy had dropped in all three groups to 7-8 seconds with a range from 6.2 to 9.7 sec. in the occipital-lobectomized monkeys, from 6.4 to 7.6 sec. in the dark-kept animals, and from 6.0 to 9.1 in the light-kept animals. The VOR time constant of the three seeing monkeys showed significant changes over the post operative observation period ($p<0.005$) increasing by 2-3 sec. whereas, the

time constant for the three blind monkeys (occipital-lobectomized) decreased from 7.3 sec. to 6.9 sec. (non significant) after three to four months post labyrinthectomy. The researchers do not supply information as to whether the values were symmetrical.

VOR gain in the dark showed an initial drop in the seeing monkeys immediately post labyrinthectomy at speeds up to 120°/sec. to approximately 50% of the pre-operative value. At higher speeds there was a 50% drop for rotations to the intact side but these values progressively declined with rotations toward the lesioned side. Within four days after exposure to light, slow-phase velocity increased to 80-90% for rotations to the intact side. The rate and extent of recovery of slow-phase velocity was much slower for rotations toward the lesioned side. For the animals with the lobectomies the slow-phase velocity decreased to 30-50% of the pre-operative values. Overall gain (right and left directions combined) stayed low except for some moderate improvement for low-velocity rotations. At 120°/sec. they noted a slight increase for rotations towards the intact side and a slight decrease in rotations toward the lesioned side.

When they compared the results of the light-kept and dark-kept animals they report that the light-kept monkeys had a prompt increase in gain of VOR within 48-72 hours after first exposure to light. This was true for rotations toward the intact side at all velocities and for rotations toward the lesioned side at low velocities. In the dark-kept animals the recovery of VOR gain was delayed until the animals were exposed to light. They showed similar recovery as the light-kept animals though slightly less complete at high velocities. For rotations toward the lesioned side, at velocities higher than 60/sec. the rate of recovery was much lower than that of the light-kept monkeys.

For the animals with the occipital-lobectomies there was an increase in VOR gain in the light above that of the VOR gain in the dark but only at low-velocity rotations. These animals were unable to use visual information to increase VOR gains at higher velocities, whereas the seeing monkeys could increase their VOR gain values at all velocities in the light.

Effects of occipital-lobectomy in animals that had undergone a prior labyrinthectomy were similar to those seen in animals without prior vestibular lesion.

The researchers concluded from their study:

1) The geniculostriate pathways play an important, but not exclusive, role in providing the necessary error signals to stimulate adaptive change in the gain of VOR;

2) Visual experience was not necessary for the compensation process that eliminated the spontaneous nystagmus created by the labyrinthectomy. The rate of disappearance of spontaneous nystagmus was not retarded in the dark-kept or occipital-lobectomized animals. They report that some rebalancing had already taken place before exposure to light.

3) Visual experience is critical for compensation of VOR gain following unilateral labyrinthectomy in monkeys. They report that the ultimate degree of recovery of the gain of the VOR may have been lower in the animals kept in darkness for the four days prior to exposure to light when compared to the animals with immediate light exposure.

4) They noted that occipital-lobectomy had little effect upon VOR in otherwise intact animals. There was a slight decrease in VOR gain for high velocities of rotation. When measured just prior to labyrinthectomy, the VOR time constant was lower in the animals with intact cerebral cortex. The authors felt, however,

that this difference could be due to the normal process of habituation, as the lobectomized animals had been tested more times prior to labyrinthectomy than the seeing monkeys.

Maeda (1988) examined glucose utilization in the brain stem, including vestibular nuclei, and cerebellum of: a) healthy control cats, and b) in cats at various times following unilateral labyrinthectomies.

Deoxyglucose [14C] was given as an intravenous bolus. The animals were sacrificed after 45 min. and the brain was dissected. Densitometric measurements of [14C] concentration from autoradiographs were performed.

For normal cats the highest value of [14C] was identified in the inferior colliculus. Two hours after right (R) hemilabyrinthectomy, when cats were uncompensated, they noted that distribution of activity within the brain stem and cerebellum was significantly different than that seen in the normal cat. The most noticeable change from the normal cat values (control values) was seen in the vestibular nuclei. Here, glucose utilization in the deafferented nucleus was well below (62% of the control value) that of the intact side (101% of the control value). There were similar values found in the cats sacrificed 24 hours after (R) hemilabyrinthectomy. From cats sacrificed four weeks after hemilabyrinthectomy (compensatory stage), the most important finding was the value for glucose utilization in the deafferented vestibular nucleus (85-95% of control values) increased so as to equalize that of the intact side (95% of control values). The results 10 months post hemilabyrinthectomy were similar to those reported at four weeks after the deafferentation.

Maeda suggests that this study supports the hypothesis that vestibular compensation results from the combined activity of many brain stem and cerebellar structures. Following acute labyrinthine deafferentation the level of

spontaneous activity in the vestibular nuclei is severely decreased and symmetry of function was lost. As the animal begins to compensate there is a proportionate increase in activity in the deafferented vestibular nucleus and in other structures. It was proposed that input from the intact labyrinth and information derived from other sensory systems is important for re-balancing of resting discharge rates in deafferentated vestibular nuclei, and consequently for vestibular compensation.

Schaefer et al (1979) used guinea pigs that were unilaterally labyrinthectomized to study the significance of somatosensory inputs on vestibular compensation. They were concerned with the influences exerted by neck somatosensory afferents on compensation of labyrinthectomy symptoms of postural asymmetry. To distinguish the effects of somatosensory afferents which directly feed into vestibular nuclei from those relayed in the cerebellum before reaching that system, they also studied vestibular compensation in animals with various cerebellar lesions.

The parameters used to measure the degree of compensation was head turning in the horizontal plane and ocular nystagmus (nystagmus beats per minute). In this study the authors did not describe the type or how they measured the nystagmus eye movements nor how the nystagmus was elicited.

The researchers used four groups of ten guinea pigs that were unilaterally labyrinthectomized on the right side. The first group served as the control group. The heads of the animals in the other three groups were restrained in three different positions for a period of five hours post operatively: 1) 60° to the right; 2) mid- position; and 3) 60° to the left.

They found that compensation was faster for the animals that had their heads restrained 60^0 toward the intact side, when compared to the control group

for head turning in the horizontal plane ($p < 0.01$). They noted no significant differences in rate of compensation between the control group and animals that had their head restrained toward the mid-line or towards the lesioned side.

Concerning compensation of ocular nystagmus following the labyrinthine lesion, there was no significant effect of restraining the head in the three positions. They also noted the effect on head turning about the longitudinal body axis when the labyrinthectomized animals were lifted off the ground. The head turning slowly increased after the head was free again but was still significantly lower than in the controls after eleven hours. The labyrinthectomized animals which were lifted off the ground returned to values identical to controls twelve hours later.

The same experiments were repeated with animals that had been cerebellectomized two weeks before the labyrinthectomy. It was found that head turning about the longitudinal body axis was unchanged by cerebellectomy. There was no difference in the time course or the strength of that symptom when the head had been fixed in the three positions, nor did the results differ from the controls. Further experiments revealed that ablation of the posterior vermis caused a marked retardation of vestibular compensation after labyrinthectomy, of head turning in the horizontal plane. It was concluded that compensation of the labyrinthectomy symptom of head turning in the horizontal plane is dependent on somatosensory afferents and that these influences appear to be mediated through the cerebellar vermis.

Paige (1983) studied horizontal VOR and OKN following and their interactions in eight squirrel monkeys before and after mechanical inactivation of the horizontal canal (plugging) on one side. The researchers evaluated VOR gain, VOR time constant, OKN, OKAN, and head and body posture. Data was

collected pre plugging, acutely, and one week and one month after horizontal plugging. Paige used sinusoidal rotations at 0.02 - 0.2 Hz with peak head velocity in range from 40°-360°/sec. and measured eye movement using attached eye coils.

Post operatively, two of the eight monkeys were returned to their cages and allowed unrestricted head movements under conditions of normal vision. The remaining six monkeys were placed in darkness with their heads fixed immediately post operatively. Of these six monkeys, two were allowed to move unrestricted with normal vision after 18-44 hours in darkness. The other four animals were restricted for one week following their surgery (one remained in darkness with his head fixed, two were maintained in a lighted room but with their heads fixed, and one subject was kept in the darkness but unrestrained).

Following the channel plugging, all of the monkeys displayed the same behavioural syndrome, head movement in one direction immediately followed by a head movement of smaller amplitude in the opposite direction. They noted that when the head was tilted back the monkeys would display head and trunk oscillations which persisted for several seconds or increased in amplitude until the monkey fell. For the animals allowed normal vision immediately post plugging, the syndrome disappeared over a period of days. A similar finding was also noted for the other animals but only after they had been returned to their home cages and allowed normal vision. Paige concluded that normal vision is necessary for recovery from post-plug head and body postural instability.

Immediately following the plugging, all of the animals displayed a spontaneous nystagmus in darkness. They noted that the slow-phase eye velocity (SPEV) was always directed to the plugged side. For animals that were allowed normal vision the spontaneous nystagmus was greatly reduced or

eliminated after one week averaging 3°/sec. The dark kept animals displayed peak values close to 25°/sec. immediately after the plugging and continued at that level for one week.

The authors reported that the animals showed low VOR gains in the acute phase post-plug. The two monkeys allowed normal vision displayed an increase in gain, which reached an average of 78% within week one and 82% by one month. On the other hand the monkeys deprived of normal vision for one week post-plug had a persistently low gain average of 52% +/- 5% at the one week tests. Three weeks post plug, these animals reached a gain average of 83% +/-11%. The authors concluded that normal vision is responsible for the VOR gain recovery post-plugging.

Paige reported no significant differences in the one week post-plug time constant between animals denied normal vision and those allowed normal vision during the first week post-plug.

In reporting their results of VOR response asymmetry, they report a bias velocity (SPEV's) at 0.2 Hz,120°/sec., is always directed toward the plugged side and increased with increases in head velocity reaching an average of 70°/sec at 120°/sec. peak head velocity. The response asymmetry was less pronounced at 0.02 Hz.(20°/sec. at 360°/sec.). The authors did not distinguish between the groups when they presented this data.

Paige did not distinguish between the groups for the following results but compared findings for the same animal pre (normal) and post-plug. For the OKN results to sinusoidal rotations, they report that one month post-plug the values are, on average, indistinguishable from normal values (values found pre-plugging in the same animal) except at higher drum velocities where the gains were greater than normal (at 120°/sec. drum velocity, average difference

of 11.4%, P<0.025). In the acute period, time constant of OKAN decay averaged 18.2 +/- 5.3 sec. not significantly different from normal (18.4 +/- 4.0 sec.) in the same animals. They concluded from their results that OKAN did not display secondary responses following horizontal channel plugging.

Putkonen et al (1977) used photographic techniques to study the evolution of lateral head-tilt following hemilabyrinthectomy in cats. The cats were divided into three groups post operatively: 1) one group was returned to normal laboratory cages. 2) the second group were immediately placed in a light-proof room in darkness for 10-31 days. 3) the third group was exposed to stroboscopic light. Video recordings and photographs were used to compare the three groups for compensation of gross postural and motor deficits observed post operatively.

They observed that the light kept animals displayed the classical postural syndrome following unilateral labyrinthectomy. In the critical stage (between 24 and 48 hours post lesion) the cats could not stand up and the head was tilted lesion side down. After two days the cats were able to stand but could only walk in inept circles to the unaffected side. After the third day the cats were able to walk in a straight line for several meters. After five to eight days, these cats displayed almost normal gait (occasional falls to lesioned side). The dark kept animals showed substantial postural asymmetry for up to thirty one days. These animals did not progress past the stage of being able to stand but only to walk in inept circling to the lesioned side. However, once these animals were re-exposed to light, within four days they were able to recover enough function to walk in a straight line.

The cats exposed to stroboscopic light were less impaired than the dark kept animals when first exposed to a normally lit environment (fifteen days post

operatively). These animals were able to walk in a straight line although they retained hypotonia of the limbs on the lesioned side.

Putkonen et al concluded from his study that vision is a necessary condition for postural compensation post hemilabyrinthectomy in cats.

Dichgans et al (1973a) investigated: 1) The relative contribution of the vestibular and neck afferents to compensatory eye movement made during active and passive head turning in monkeys. 2) The mechanisms underlying the recovery of compensatory eye movements following the removal of the vestibular and/or neck loop.

They performed bilateral labyrinthectomies and cervical rhizotomy (C1-C6). They used four adult monkeys who were trained to make a visual discrimination between horizontal and vertical light bars. The bars were thin black lines presented in a random sequence superimposed upon a one degree luminous spot. The targets were presented directly in front of the monkeys who were rewarded with a drop of water if they pressed a lever when the vertical bar appeared. During the tests horizontal eye and head movements were recorded.

A few days after bilateral labyrinthectomy the experimental sessions began with the animals placed in a primate chair with their trunks restrained. They assessed whether visual input was a factor in eye stabilization during spontaneous head turning by observing compensatory eye movements executed with and without a visible target light during head movements. The comparison of compensatory eye movements recorded under active head turning in darkness is completely adequate within the limits of electro- oculographic measurements. Therefore, they suggest that ocular stabilization during active head movement is entirely achieved by inputs from the semicircular canals and neck receptors.

To test ocular stabilization in passive versus active head movements they moved the head via a torque motor attached to the head holder at velocities within the range of active head movements. Their results indicated that compensatory eye movements matched the passive head displacement resulting in perfect ocular stabilization. There was a possibility of central modulation of vestibular afferent activity in the course of centrally-initiated eye-head movements, and that descending supraspinal control could modulate the inflow of afferent impulses from the neck proprioceptors during active head turning. They concluded that in the intact monkey there was no change in the overall gain of the vestibular and neck afferents during passive or active movements.

They looked at differentiating the roles of vestibular and neck inputs and found that when a monkey's body was rotated in the dark while his head was kept stationary in space (test of neck-ocular reflex), there was hardly any compensatory eye movements of measurable amplitude that could be detected. This indicated that in the monkey, ocular stabilization during active or passive head movements is essentially due to an unmodulated vestibulo-ocular reflex.

After bilateral labyrinthectomy, all the labyrinthectomized animals overshot visual targets when using combined eye and head movements and were unable to foveate the target within the usual interval after the target was presented.

Compensatory eye movements gradually increased in size (up to 25%) at the end of the first post operative week, but only during active eye-head movements. During the recovery period, compensation eye movements equivalent to about 50% of the amplitude of the head movement was evident by the 10th post operative day.

There was continuous improvement to 90% one month post

labyrinthectomy. The investigators felt the recovery of ocular stability was due to the combined results of several mechanisms. The most significant are the potentiation of the neck-to-eye reflex, the central programming of compensatory eye movements, and the "recalibration" of the saccadic eye and head motor systems.

Maioli and Precht (1985) studied unilaterally labyrinthectomized cats to assessed whether VOR plastic capabilities were affected by labyrinthectomy. They used forced oscillations and recorded specific VOR parameters to evaluate VOR compensation following unilateral labyrinthectomy in cats. The forced oscillations consisted of visual-vestibular stimulation performed by means of low frequency-small amplitude oscillations (0.1Hz+/-20°/sec.). The cats were oscillated for 3-5 hours daily, in front of a high contrast random dot pattern.

They used nine adult cats and measured horizontal eye movements recorded by DC electro-oculography. Forced oscillations and recordings were performed on animals whose head was immobilized. The researchers measured VOR amplitude in the dark before and at various times after the lesion, by means of sinusoidal rotations (0.1 - 0.5 Hz). Bilateral OKN responses to 40°/sec. constant velocity optokinetic stimuli were also measured.

The cats were divided into two groups: 1) group one was exposed to forced visual-vestibular stimulation which was initiated on the 3rd day post lesion; and 2) Group two was allowed to recover for 23 days before the forced oscillation program was started.

For the cats exposed to forced visual-vestibular stimulation, spontaneous nystagmus was reduced (2-10/sec. in the dark). For the other group there was no spontaneous VOR gain recovery noted after the 23 day period prior to the forced oscillation program. The researchers report a gain drop of 50% in the

response measured 2.5 days after the lesion compared to the first group (exposed to forced oscillations immediately post-lesion).

After the forced oscillation sessions were discontinued, there was an average VOR gain decrease of 18% which persisted at the last recording session for each cat. Although there was a decrease the authors state that these low values were above those measured in cats showing a poor spontaneous VOR recovery in their previous studies. They also found that the forced oscillations improved the VOR gain in the chronic post lesion stage (the animals exposed after 28 day recovery period post lesion). They noted that although forced oscillation induced some improvement in VOR gain, it had no effect on changing the response symmetry for either group of cats. The authors did not supply any data on the asymmetry scores for either group of cats.

OKN gain in response to rotations in both directions was larger at the end of the forced oscillation period then shortly after the lesion. However, the recovery they noted was not better than the ones reported in other research on spontaneously recovering animals by Precht et al (1981a).

From their personal observations, they report recovery from undefined motor deficits in cats that did not show VOR compensation and ones that did have some recovery VOR gain. From these personal observations it was concluded that a sustained amount of functional recovery can be accomplished without VOR compensation.

They concluded that their stimulation protocol did improve the VOR gain it did not induce a consistent improvement in VOR symmetry, nor a better compensation of optokinetic response, and therefore, forced oscillation is probably not the most effective way of improving responsiveness in the deafferented vestibular nucleus.

2.5.1. SUMMARY

In summary, the animal studies reviewed suggest that following unilateral labyrinthectomy or neurotomy, recovery of function is delayed and substantially reduced when lesioned animals were deprived of vision and somatosensory input. A number of the studies demonstrated that animals who were exposed to visual and/or somatosensory restrictions post lesion did not regain their pre lesion functional levels. Animals who were allowed normal vision and movement post lesion were able to regain most of their pre lesion levels of function.

In studies where exercise regimes and especially goal directed exercise regimes were employed, recovery of VOR gain symmetry and balance control were further enhanced.

These studies support the theory that early intervention with exposure to exercise, normal vision and somatosensory input can be beneficial in promoting compensation in animals with chronic vestibular lesions.

2.6. HUMAN STUDIES

Clinicians have experienced difficulty in the diagnosis and management of vestibular disorders partly due to the inability of patients to accurately describe their symptoms, lack of findings with a physical examination and the fact that the various components of the vestibular system cannot be directly tested. The main diagnostic tools have been the caloric test, rotational tests and more recently moving platform posturography testing.

The main focus of human research has been in developing tools to evaluate the degree of compensation following vestibular disease, and in

developing treatment for vertigo.

There have been human studies of long term VOR changes in patients with peripheral vestibular dysfunction which showed varying degrees of recovery of VOR gain and time constants (Fetter and Dichgans 1991;Jenkins 1985; Leigh et al 1987). Several studies (Black and Nashner 1984; Black et al 1988; Black et al 1983; Goebel and Paige 1989; Horak et al 1988; Voorhees 1989) have examined standing balance performance of patients with peripheral vestibular deficits under altered somatosensory and visual conditions. There has been little work done to assess the adaptive capabilities of the nervous system in the regulation of human postural stability. In human studies, the use of exercise to alleviate symptoms of vertigo and dizziness has been evaluated in different groups of patients with peripheral vestibular dysfunction (Cawthorne 1944; Cooksey 1946; Dix 1976;Hecker et al 1974;McCabe 1970; Norré and Beckers 1987; Norré and Beckers 1988; Shepard et al 1990; Telian et al 1990).

2.6.1. RECOVERY OF OCULOMOTOR FUNCTION

Human studies of long term VOR changes have been performed on patients with peripheral vestibular dysfunction which showed varying degrees of recovery of VOR gain and time constants.

Fetter and Dichgans (1991) investigated vestibular-ocular function in humans over a period of two months after an acute unilateral labyrinthine lesion. They studied nine subjects with acute unilateral vestibular lesions. Horizontal and vertical eye movements were recorded by DC electro-oculography in complete darkness. Vestibular hypofunction was evaluated by caloric testing in the supine position.

Static vestibulo-ocular disturbances were assessed by: 1) Average slow-phase velocity (SPV) of spontaneous nystagmus during a 1 min. period in the dark. 2) Maximum amount of SPV of nystagmus (average 10 sec. during peak elicitation) that could be induced by ice water calorics on the intact side. 3) Maximum SPV of nystagmus that could be elicited by high velocity rotation (180°/sec.) toward the lesioned side.

Dynamic vestibulo-ocular disturbances were quantified by measuring maximum SPV and calculating VOR gain, as well as VOR time constant of post rotary nystagmus after accelerations to 60° and 180°/sec. in clockwise and counterclockwise directions. To measure VOR imbalance between left-right sides, VOR gain and time constant asymmetry were calculated as a percentage (maximum eye velocity/maximum head velocity).

The patients were tested between day 3 and day 13, between day 7 and day 25, and finally between day 43 and day 68 after the onset of symptoms. On the first recording day, six of the nine subjects had no caloric response on the lesioned side and the other three had a marked reduction in SPV of nystagmus towards the intact side. All patients exhibited horizontal spontaneous nystagmus (SPN) in the dark on first measurements of 19.1 +/- 9.8^0/sec. By the second measurement (7-25 days after onset of symptoms) SPN was reduced in all subjects to 5.9 +/- 4.6^0/sec. By two months after lesion most of the subjects had further decreased SPN to almost normal levels of 2.6 +/- 1.6^0/sec.

Ten days after the lesion, 3 of the patients were able to produce a nystagmus in opposite direction to the SPN. Ten to twenty days following their lesion, 7 patients with incomplete lesions had increased their ability to produce caloric-induced nystagmus opposite to SPN. Two patients with complete lesions were slower regaining their ability to reverse SPN by ice water calorics on the

intact side. On day 43 and 68 even these patients had reached values comparable to patients who had regained bilateral labyrinthine function.

Initially a stop from high-velocity rotation (180°/sec.) toward the intact side elicited a nystagmus toward the lesioned side in all patients (average SPV 49.6+/-22.7/sec.). In the six patients with complete lesions the average SPV was 38.6+/-18.7/sec. as compared to the three incomplete with values of 71.6+/-10/sec. All patients recovered various degrees of ability to produce slow phases toward the intact side.

The VOR gain was initially low for both 60° and 180°/sec. stimulation with values of 25%+/-11% (lesioned) and 103%+/-16% (intact) sides at rotations of 60°/sec. The values at 180°/sec were 27%+/-13% (lesioned) and 83%+/-17% (intact)side. The VOR toward the lesioned side showed fast recovery (within 25 days) to values at 60°/sec. rotations of 69%+/-11% (lesioned) and 85%+/-22% (intact)side. For rotations at 180°/sec. values were 63%+/-17% (lesioned) side and 76%+/-16% (intact) side.

They noted an initial VOR asymmetry for rotations with 60°/sec. to be 63+/-12% and with 180°/sec. stimulus values of 51+/-16%. The dynamic imbalance remained incomplete at high velocity rotations. Within 25 days the asymmetry was reduced to less than 15% with 60°/sec. stimulus. At 180°/sec. stimulus only three patients improved to an average asymmetry of 14%+/-8%.

The average time constant on first measurements post lesion at 60°/sec. were 4.7+/-1.1 sec. (lesioned) and 6.6+/-2.2 sec. (intact) side and at 180°/sec. values were 6.2+/-2.3 sec. (lesioned) and 7.8+/-2.1 sec. (intact) side. These values were significantly different between the two directions only at 60°/sec. stimulus. On the 25th day post-lesion all of the patients showed a fast but incomplete recovery of time constant with final averages for rotations at 60°/sec.

of 9.3+/-2.1 sec.(lesioned) and 9.4+/-3.2 sec. (intact) side. At 180°/sec. stimulus the values were 9.8+/-2.0 sec.(lesioned) and 10.1+/-2.4 sec. (intact) side. There was no significant difference between the two directions of rotations for both of these stimulus velocities.

Fetter and Dichgans (1991) found that over the time of their study (68 days post lesion) that all of the patients increased their ability to reverse spontaneous nystagmus with ice-water calorics on the intact side. It was proposed that this indicated a restoration of tone in the vestibular nuclei on the lesioned side. They felt this finding supports the notion that restoration of tone on the lesioned side is partly responsible for the diminution of the static vestibular asymmetry.

The results of this study show that a VOR gain recovery is possible in humans, even in the case of complete unilateral vestibular lesion. The mechanisms for this increase are still unknown. However, only marginal increases in time constants were observed. It is unclear why recovery of the velocity storage mechanism seems to be prevented in cases of complete peripheral vestibular lesions.

Jenkins (1985) evaluated twenty six patients who had undergone translabyrinthine acoustic neuroma surgery. They also tested a group of 50 normal subjects who had no previous history of disequilibrium or of hearing loss.

Pre-and postoperative evaluation of vestibular reflexes included eye movement recordings by EOG techniques to measure responses to caloric stimulation, positional, smooth pursuit, optokinetic, and rotatory testing. Sinusoidal rotations were done in the dark at 0.0125, 0.05 and 0.2 Hz. at a peak velocity of 60°/sec. Jenkins did not provide any information on his positional testing procedures.

Post-lesion, Jenkins noted profound unilateral weakness in responses to caloric irrigation: greater than 70% difference in 12 patients, moderate weakness of 40-70% difference in 7 patients and slight weakness of 20-40% difference in 4 patients, with normal results in 2 patients. He noted positional nystagmus in 3 patients, 4 patients demonstrated decreased tracking ability, 2 had marked and another 2 had mild asymmetry of optokinetic nystagmus.

Changes in VOR gain postoperatively were most dramatically demonstrated at lower frequencies: at 0.0125 Hz 65% of the patients had responses lower than 2 SD below the mean for the group of normal subjects. The majority of the patients fell within the mean of the normal group at the other two frequencies tested (0.05 and 0.2 Hz). Four of the nine patients tested for a full year showed a return to within 1 SD of the normal group at 0.0125 Hz. The findings identified that gain increased with time and that the change was statistically significant at the 0.0125 Hz ($p<0.001$).

Jenkins reported that 95% of the patients showed abnormal phase responses at 0.0125 Hz., all at 0.05 Hz. and 57% abnormal responses at 0.2 Hz in the acute post operative condition. He notes that the phase decreased with time toward normal at 0.2 Hz at a significant difference level ($p<0.05$).

He also reported that in the initial post operative period, many of the patients showed a dramatic increase in VOR gain asymmetry. The asymmetry decreased with time toward normal at all frequencies (0.0125 Hz, $p<0.001$; 0.05 Hz, $p<0.01$; 0.2 Hz, $p<0.05$.)

Although Jenkins gives details on the level of significance with time he did not elaborate on the length of time in days, nor did he supply actual values of VOR gain and phase. He used the data from the 50 normal subjects as a normal baseline to compare the findings of the 26 patients post surgery.

Jenkins concluded that his findings showed that long-term changes in VOR gain, phase and gain asymmetry resulting from an ablative lesion of the peripheral end organ, follow a course that can be predicted.

Leigh et al (1987) investigated the effects of deficient labyrinthine function on smooth visual tracking in patients with bilateral peripheral vestibular disease. The researchers measured horizontal gaze (eye position in space) and head movements using a magnetic search coil system. Eye movements were recorded using infrared oculography.

They tested smooth eye and head movements under five conditions: 1) Smooth pursuit (SP) - the head was immobilized and subjects were encouraged to follow visual targets (laser-spot projected onto a target screen) moving in a sinusoidal fashion at 0.25 and 1.0 Hz. with amplitudes of +/-5 or +/-10 degrees. 2) Eye-head tracking (active EHT) - the subjects were asked to pursue the target with their heads. 3) Active head rotation (AHR) - the subjects were asked to actively rotate their heads in darkness while they imagined a stationary target. 4) Vestibulo-ocular reflex (VOR) - chair rotations in darkness (0.25 Hz and 1.0 Hz) while subjects attempted to fixate an imaginary earth-fixed target. 5) Passive eye-head tracking (passive EHT) - as subjects were rotated en bloc they were asked to view the laser target which moved in synchrony with the chair.

During SP, active and passive EHT (conditions 1,2, and 5) they measured tracking gain (peak gaze velocity/peak target velocity). During active head rotations (condition 3) and passive en bloc rotation (condition 4) they measured the compensatory response gain (peak eye velocity in orbit/peak head velocity).

They measured these parameters in ten healthy control subjects and in the ten patients with bilateral vestibular disease. They found no significant difference between tracking gain of SP and active EHT in normal subjects. In the

normal control subjects the compensatory gain response with active head rotation (in darkness) was significantly greater ($p<0.001$) than with passive rotation, with rotations at +/-10 deg. at 1.0 Hz but not with the other stimuli. When comparing SP with passive EHT, this group demonstrated a significantly higher tracking gain ($p<0.001$) during passive EHT at 1.0 Hz (+/-10 and +/-5 deg.) but not at 0.25 Hz. The tracking gain during passive EHT was not significantly greater than it was during active EHT.

The patients with deficient labyrinthine function showed significantly higher tracking gain during active EHT than during SP at 1.0 Hz. They observed no impairment of their ability to make smooth tracking movements of the head during EHT. Compensatory gain response during active head rotation in darkness (AHR) was much larger than during passive rotation (VOR gain). When comparing compensation gains for AHR the patients gain was significantly less than AHR or VOR in normal subjects. When passive EHT was tested it showed significantly higher tracking gains than during SP at 1.0 Hz. They reported that tracking gain during passive EHT was not significantly greater than during active EHT.

The major finding was that patients who had deficient labyrinthine function smoothly tracked a target better with active and combined movements of eyes and head than with smooth pursuit tracking alone. They stated that compensatory response during active head rotation in darkness was higher than during passive rotation, due to an enhancement of the cervical-ocular reflex (COR) or of smooth pursuit eye movements that occur during active head movements. They conclude that the data they collected provide direct support for the hypothesis that the VOR is cancelled by an internal smooth pursuit signal during active EHT.

2.6.2. RECOVERY OF POSTURAL CONTROL

Norré et al (1987a) evaluated balance reactions in two groups of vestibular patients, one group of seventy five patients diagnosed with Meniere's disease and a second group of one hundred and seventeen patients with unilateral vestibular hypofunction.

They studied the following parameters: 1) Caloric test results; they evaluated the nystagmus by measuring the maximum slow-phase velocity. Asymmetry was calculated by the formula R (right)-L(left)/R(right)+L(left) X 100 [((R-L) / (R+L)) X 100]; 2) Rotational tests; using both a sinusoidal stimulation and a stop-stimulus, they calculated the difference in VOR gain between rotations to intact and lesioned side and measured the maximum slow- phase of the nystagmus. Asymmetry was measured with the same formula mentioned above. 3) Posturography; they measured postural sway under two sensory conditions, eyes open and eyes closed. Postural sway was recorded by an x-y writer and analyzed by computer. They calculated the surface of the area and the total distance covered by the moving point (length). Degrees of posturography abnormalities were expressed by means and standard deviations.

They found that nearly the same proportion of abnormal posturographic results were observed in both patient groups. They then subdivided the groups into two categories with completely normal results on posturography (66%) and abnormal results (34%). No values were supplied regarding posturography scores other than a percentage value of 46.53% or 89 of 192 subjects demonstrated at least one abnormal posturography measurement. In all patients, recording with eyes closed showed more abnormal posturographic results than with eyes open.

The rotation test results showed that the patients with Meniere's disease had an asymmetrical nystagmus reaction which occurred less frequently than for the patients with unilateral vestibular hypofunction ($p<0.01$). They reported a higher frequency of rotary asymmetry scores in cases with typical "sudden loss" syndrome, which was significantly different ($p<0.01$) from the other cases. Of the patients tested they report 33.85% or 65 of 192 patients showed asymmetry in the rotation tests.

They concluded posturography can identify vestibulospinal disturbance in peripheral syndromes, whereas its occurrence was not predicted by other tests (caloric and rotational tests).They felt posturography adds functional information to the global evaluation and can assist in the evaluation of compensation as it applies to the vestibulospinal system.

Norré et al (1987b) studied vestibular compensation in one hundred and seventeen patients with unilateral vestibular hypofunction (UVH). The patients were divided into groups according to their complaint patterns. The listed grouping categories were as follows: 1). Category 0 (N=33) these patients had no complaints of vertigo, a number did have complaints of postural instabilities (N=17). 2). Category 1 (N=38) these patients presented with a history indicating a typical acute deficiency syndrome. 3). Category 2 (N=7) these patients had a typical paroxysmal positioning vertigo (BPPV). 4). Category 3 (N=21) these patients complained of atypical positioning vertigo. 5). Category 4 (N=18) these patients complained of vague dizziness and giddiness.

Norré et al (1987b) examined caloric test results, rotation test and posturography test results in the same manner as that described in the previous study documented above Norré et al (1987a). The aim of their study was to compare results from rotational tests and posturography as indicators of

compensation. They used gain symmetry of response for clockwise and counterclockwise stimulation as their indicator of VOR compensation and normal posturography as an indicator of decreased influence of the vestibulospinal reflex on the unilateral vestibular hypofunction. . The researchers found that 67 patients (57.26%) showed improvement in their VOR gain symmetry scores and 54 patients (46.15%) showed improvement in posturography scores.

They found that for the total group both examinations (rotation test [RT], and posturography [PG]) gave concordant results in only 60 patients (51.28%). There was discordance in 57 patients.

They concluded that in cases of UVH, it is necessary to evaluate both the VOR testing and posturography testing.

Goebel and Paige (1989) studied 159 patients who complained of dizziness, to evaluate the relationship between the symptoms of vertigo and findings to caloric stimulation and posturographic testing. They evaluated four groups of patients: 1) those who had experienced recent vertigo (within 1 month), (n=86); 2) past vertigo (more than 1 month before examination), (n=12); 3) equivocal (questionable sensations of vertigo), (n=12); and 4) absent vertigo (n=49).

They measured peak slow-phase eye velocity (PSPV) and calculated labyrinthine asymmetry (LA). The following formula was used: LA (%) = [(RW + RC) - (LW + LC)] X 100 with RW = right warm, RC = right cool, LW = left warm, and LC = left cool, using this formula they calculated the right-left differences in PSPV with caloric testing.

They used the Equitest device to evaluate balance performance under altered sensory conditions. They calculated an equilibrium score (ES) between 0 (fall) and 100 (perfectly stable defined as no falls) that reflected peak to peak

sway over a 20-second trial, scaled to the patient's calculated base of support. Of the six possible conditions they analyzed the results of two of the conditions (absent vision, sway referenced support, and sway referenced vision and support.

Goebel and Paige report that the LA scores for all groups were statistically indistinguishable, (LA scores, recent, 24%+/-25%; past, 22%+/-31%; equivocal, 19%+/-17%) except for the LA for the absent vertigo group (10%+/-10%, $p<0.01$). The caloric results of the recent and past vertigo groups were not statistically different. The equivocal and absent vertigo groups had fewer abnormal scores than either of the other groups. Therefore, a clear history of vertigo is associated with a positive caloric deficit in only about half the cases.

They report that the posturography results were statistically indistinguishable for all four groups (recent, 40%+/-30%; past, 47%+/-28%; equivocal, 36%+/-30%; and absent, 40%+/-30%).

When comparing relationship between caloric and posturography results they found that 26% of patients with recent vertigo had both abnormal calorics and posturography versus 25% for the past, 25% for equivocal and 8% for absent history groups. The only significant difference was between the recent and absent vertigo history groups. They also found that a greater percentage of patients with recent vertigo (70%) had at least one test abnormality as compared with 59% for the past, 58% for equivocal, and 47% for absent vertigo groups. The only significant difference was again; between the recent and absent vertigo groups ($p<0.01$).

They concluded that their results implied that approximately 50% of the patients with caloric test abnormalities were correlated with a clear history of vertigo (past and present), whereas platform abnormalities as reflected by

equilibrium scores or number of falls did not distinguish between patients who had and those who did not have vertigo. They note that the combination of calorics and posturography abnormalities did show a better correlation with a history of vertigo.

Voorhees (1989) studied one hundred and seventy five patients to assess the results of posturography testing using the Equitest device in a broad spectrum of patients with peripheral and central vestibular disorders, and non-vestibular disorders of the CNS.

All of the patients received neurotologic examinations and were tested using the following parameters: 1) Computerized ENG (electro-nystagmography) during caloric stimulation, symmetry response scores were calculated. 2) Sensory organization test scores using the Equitest device. They measured the degree of body sway which they classified as (0=fall and 100=perfect stability). 3) Motor coordination tests using the Equitest device were analyzed in terms of latency, amplitude and left-right symmetry of compensatory balance reaction to sudden platform translation and rotations.

Of the 70 patients with peripheral vestibular disorders, 44.6% had abnormal sensory organization scores and 27.7% had abnormal EOG findings. For the patients with central disorders (n = 53) 71.7% performed abnormally on their Equitest and of these patients 26 (49%) had abnormal sensory organization (SO) test scores only. Another 15 (22.7%) had abnormal motor coordination test scores. 8 patients had abnormal results in both sensory and motor tests scores for a total of 64% abnormal findings. EOG abnormalities were found in 22 (41.5%) of the patients in the central group.

Voorhees concluded that the sensitivity of posturography in peripheral deficits was 45%, the SO findings in central disorders detected 49% abnormal,

with elevated latencies providing an additional rate of 22.6%. He found that the combined detection rate for the peripheral and central groups was over 71% and felt this strongly supports the conclusion that posturography offers an effective means of analyzing individuals with balance and spatial orientation difficulties.

2.6.3. TREATMENT

The literature reports a number of studies which concentrated on the treatment of vertigoand related to the rehabilitation treatment of patients with peripheral vestibular disorders (Cawthorne 1944; Cooksey 1946; Dix 1976; Dix 1984; Hecker et al 1974; Norré and Beckers 1987, Norré and Beckers 1988; Pompeiano and Brodal 1957; Shepard et al 1990; Shumway-Cook and Woollacott 1985; Telian et al 1990) . The majority of these studies have examined the effects of specific training programs on symptoms of vertigo, whereas there is little information for VOR gaze compensation, oculomotor function or postural control.

As early as the 1940's Cooksey (1946) and Cawthorne (1944) developed a series of exercises to address their patients' complaints of vertigo and impaired balance. Their exercise program included eyes and head movements, total body movements, and simple balance tasks. They recommended that the exercises be performed first in sitting then standing and at various speeds of movement. They also asked their patients to perform the exercises with their eyes open and closed. Both Cawthorne (1944) and Cooksey (1946) felt that these exercise performed with eyes closed would decrease the reliance on visual information and possibly force more effective compensation by vestibular

and proprioceptive mechanisms. Cooksey stressed that patients be encouraged to perform activities in positions which provoke their symptoms, and that this could produce tolerance to the activity and position (habituation).

Hecker et al (1974) used Cooksey-Cawthorne exercises to treat a group of patients with vestibular disorders. It is not known how the exercises became known as the Cooksey-Cawthorne exercises.They studied eighty nine patients who were diagnosed with peripheral vestibular disorder. All patients were treated with the Cooksey-Cawthorne exercise routine.

They evaluated reports of decrease in episodes of dizziness, and nausea. Although the authors had indicated they had also measured vestibular nystagmus, they made no mention of these findings in their paper.

Hecker et al report that 84% of the patients showed a favourable response to the Cooksey-Cawthorne exercises. Although the authors report an 84% (75 of the 89 patients) improvement rate, they do not specify or quantify the favourable response .

Norré and collegues (Norré 1987; Norré and Beckers 1987,1988; Norré et al 1987a and b) have done a series of studies on treatment of patients for vertigo associated with vestibular disorders. Norré and DeWeerdt (1980a andb) proposed the use of vestibular habituation training (VHT) for the treatment of patients following unilateral peripheral vestibular dysfunction. Norré expanded on the work of Cawthorne (1944), Cooksey (1946) and Dix (1984) to develop a series of 19 manoeuvres that provoked symptoms of vertigo. He felt these 19 test manoeuvres produced an objective measure of his patients' functional condition. In their studies, they calculated the number of manoeuvres which provoked dizziness and asked the patient to rate the intensity, type (rotary or atypical), and duration of vertigo produced by each of the 19 test manoeuvres.

They used this information as their outcome measure. These 19 manoeuvres are also the treatment exercises which are referred to as the vestibular habituation training (VHT).

They used their test battery of movements and positioning to select the specific manoeuvres for each patient's exercise routine. They chose those manoeuvres which provoke symptoms of dizziness and used these as the individualized training program.

In the first study by Norré and DeWeerdt (1980) they examined the effects of vestibular habituation training (VHT) on vertigo in one hundred and thirty six patients. The patients were divided into 3 major categories: 1). Cases with the typical syndrome of acute vestibular failure (N=48). 2). Cases presenting with only a paroxysmal provoked vertigo (N=74). 3). Cases with recurrent attacks of spontaneous vertigo but also presenting with positional vertigo (N=14).

They used the number of manoeuvres which provoked dizziness for each patient, the reduction in their number from first to last test being computed as their outcome measure. They felt these measures would give an evaluation of individual improvement obtained after two months of treatment.

They reported a global reduction in positive manoeuvres of more than 50% in the first two weeks of training. They report that 63.97% had a very satisfactory result, 27.94% had some improvement and 8.08% had no improvement.

Norré and DeWeert felt that their treatment program based on the principles of habitation provided improvement to patients in a specific and genuine way. They also state that once treatment had started, however much time had elapsed from the onset of symptoms , the same satisfactory effect was observed.

In a subsequent study Norré (1986) evaluated the assumption that symptoms of vertigo could be cured by means other than drugs and surgery. He examined the use of vestibular habituation training as a treatment technique for vertigo. He studied six groups of patients: 1) Group A was comprised of cases with typical benign paroxysmal positional vertigo (BPPV), (N=82). 2) Group B was comprised of cases with unilateral vestibular hypofunction (UVH), (N=109). 3) Group C were patients with typical BPPV, treated by VHT, (N=20). 4) Group D were patients with typical BPPV, treated by sham exercises, (N=20). 5) Group E were a group of patients matched with groups A and B who did not have any exercise therapy, (N=28). 6) Group F were comparable to group A but were treated by suppressive drugs in the period before VHT was started.

A thorough history was taken on each patient to make a distinction between spontaneous and provoked vertigo. Diagnostic tests included audiometric evaluation, ENG recording of spontaneous and positional nystagmus (12 positions) and oculomotor tests (saccades, pursuit movements, and OKN). Positioning nystagmus was elicited by the Dix- Hallpike manoeuvres and observed under Frenzel glasses. Caloric testing (with ENG recording) was executed by bi-thermal stimulation (30°/44°C), they measured the slow-phase velocity of the nystagmus elicited and calculated the right left differences (asymmetry). Rotational tests were done using sinusoidal harmonic acceleration, 30 sec. acceleration at 30°/sec^2 and stop reaction after 90°/sec. All of these tests were used to identify patients in a steady state of peripheral vestibular dysfunction. Once this was established the patients were assigned to the groups listed above.

The outcome measures used for this study were similar to the previous study; a series of 19 manoeuvres was used to identify which ones provoked

vertigo. A description of the vertigo included data on whether it was typically rotatory or atypical dizziness, intensity,and duration of the vertigo were also estimated. Patients were reexamined at regular intervals of eight to ten days and percentage of reduction of the total score was calculated. End evaluations were completed after six weeks to two months after their treatment period.

Norré reports that when comparing results: 1) 80.5% of group A (atypical BPPV treated by VHT) had 100% score reduction, 12.2% had a greater than 75% reduction and 7.3% had less than a 75% reduction; 2) 65.1% of group B (unilateral hypofunction treated by VHT) had 100% score reduction, 16.1 % had greater than a 75% reduction and 18.3% <75% reduction; 3) 20% of group C (typical BPPV treated with VHT) had 100% score reduction, 50% had greater than a 75% reduction and 30% had 75-25% reduction and 0% had less than a 25% reduction; 4) 0% of group D (typical BPPV treated with sham exercises) had 100% score reduction, 5% had greater than a 75% reduction , 45% had 75-25% reduction and 50% had less than 25% reduction; 5) 5% of group E (BPPV and hypofunction with no exercises) had 100% score reduction, 15% had greater than a 75% reduction, 50% had 75-25% reduction and 70% had less than 25% reduction; and 6) when comparing group A and group F (treated with drugs), 79.5% of group A had 100% score reduction as compared to 36.7% of group F who had no complaints.

Norré concluded that these findings provide evidence for the favourable effects of vestibular habituation treatment when compared to the results of drug and sham treatments in similar cases.

In another study, Norré and Beckers (1987) studied the use of exercise as a method for treating benign paroxysmal positional vertigo (BPPV)in fifty one patients with unilateral vestibular dysfunction. Each patient had a thorough

history, a complete audiological examination, and a search for spontaneous and positional nystagmus (observation under Frenzel's glasses during the Dix-Halpike manoeuvre), and recordings during ENG with caloric, rotational and optokinetic tests.

Patients were submitted to Norré's 19-position tests to assess for the presence or absence of vertigo. They noted whether the vertigo was typical (rotatory) or atypical (dizziness), and asked patients to quantify the intensity and duration of their vertigo. The sum of the scores for the positive manoeuvres was calculated as the "total score" for each individual case.

The patients were treated with either vestibular habituation training (group B) or the brisk method (group A). The brisk method consisted of moving the patient from the sitting position towards the position eliciting vertigo. From this position the patient is moved rapidly to the mirror position on the opposite side. Finally, the patient is slowly returned to the sitting position and instructed to avoid the same positioning for the next two days. The brisk method is based on the hypothesis that there will be a dissolution of any dislodged debris on the cupula of the posterior canal.

They found that 52% of the patients in group A (brisk) obtained a zero score after one week. For group B (VHT) 32% had a zero score but the difference between the two groups was not significant. They found that eleven cases for the brisk group required complementary treatment with VHT. They noted a striking difference in the mean score for the two groups. The mean score for the group A (brisk) was 113 and remained unchanged for the cases still positive after one week. The mean for the group B (VHT) was 144 but was reduced to 66 for those cases with positive findings after one week. They concluded that either a complete result is obtained at once in the brisk method

or it has no affect on symptoms of vertigo. They found that after 1 week half of their patients treated by the brisk method were unchanged. They noted that all of the patients treated by VHT had a significant improvement. They suggest that the brisk method be used to treat typical unilateral cases of BPPV. If positional vertigo was still present after one week they felt treatment should be completed with VHT.

Norré, Forrez and Beckers (1987) studied eighty two patients presenting with BPPV to evaluate the effectiveness of vestibular habituation training (VHT). Norre et al submitted the patients to a series of 19 provoking manoeuvres and calculated a score using the formulae previously described. The sum of these scores, the number of positive manoeuvres, and posturography abnormalities were used as their outcomes measures. They recorded postural sway with eyes open, eyes closed and eyes closed with the head in retroflexion, as their conditions on posturography. Each examination lasted one minute and a statokinesimetric parameter L (length, the distance covered by the moving point of gravity) was calculated. The positive manoeuvres as assessed by the VHT test battery were the exercises used for training.

Their results showed that VHT has a positive influence on symptoms of vertigo in patients with BPPV (85.71% reached scores of zero). They report data on only 28 of the patients in respect to posturography results. Of those reported they indicate that 15 of the 28 had normalisation of posturography whereas 13 had an insufficient effect. They report 28 cases as pure BPPV cases. They state that there was an effect of VHT on the posturographic results but only in a restricted number of cases.

Norré and Beckers (1988) studied the effects of specific exercises in sixty patients who reported provoked positional vertigo.The VHT test battery of 19

manoeuvres was used to establish whether vertigo was elicited (M+ or M-) and whether the vertigo was typical rotary or atypical dizziness (A). They also measured intensity (I) and duration (T) of the vertigo reported. When nystagmus was observed it was also noted (Ny+). They found that the same manoeuvres could be positive with and without nystagmus (M + Ny+) or (M + Ny-). They computed how many times each manoeuvre was positive. They subdivided the patients accordingly to those with typical rotatory nystagmus in at least one of the manoeuvres (40 cases) and into a group with only vertigo and no nystagmus (20 cases).

All patients were treated according to the VHT protocol. Exercises were selected specific to each patient's need based on the manoeuvres which had positive results. They report that during the study they noted a common occurrence, whereby, manoeuvres that were positive with nystagmus (M + Ny +) became positive without nystagmus (M + Ny-) before becoming negative. There were 40 patients with M + Ny+ at onset and 33 one week after exercises commenced. There were 39 patients with M + Ny- at onset and 27 after one week of exercises. The authors report that after four weeks of VHT of 40 positive cases there were only 5 positives at the follow up, with 35 cases labelled cured. They do not report the level of significance of these findings. They concluded that their testing clearly distinguished two groups of patients, those with and those without nystagmus. They report that the VHT exercises worked effectively to reduce the number of positive cases with and without nystagmus.

Shepard et al (1990) performed a retrospective study to examine the effects of a rehabilitation training program on balance in ninety eight patients with peripheral vestibular disorders. They performed thorough oculomotor and visual-vestibular interaction testing, rotational chair testing, and dynamic

posturography using the Equitest device on all patients prior to their involvement in the program.

A customized therapy exercise program was designed for each individual based on the following findings: 1) functional deficits related to motion-provoked symptoms of vertigo, abnormal gait or postural control; 2) general muscle strength tests; 3) range of motion; and 4) dynamic posturography findings.

The individualized programs were developed to address two treatment aims: to reduce or eliminate motion-provoked and/or positional sensitivity (habituation therapy), and the correction of functional deficits of balance and gait (balance retraining). Shepard et al made two subjective measurements of overall therapy performance, a pre and post-therapy disability score and a post therapy symptom score of vertigo. The symptoms of vertigo included spontaneous episodic vertigo, motion or positioned provoked vertigo, type of motion or position provoked, and continuous vertigo. The disability scoring consisted of 0 - 5 grades: 0 was equal to no disability, negligible symptoms, to 5 was equal to long-term severe disability, unable to work for over 1 year or established permanent disability with compensation payments. The post therapy symptom score consisted of 0 - 4 grading: 0 was equal to no symptoms remaining at the end of therapy, to 4 was equal to symptoms worsened with therapy activities on a persistent basis relative to pre-therapy period.

They measured both scores as they felt an improvement in the disability rating would not necessarily indicate a reduction in symptoms but could indicate control over persistent, unchanged symptoms. In addition to the disability and symptoms scores they calculated a composite score of intensity and duration of vertigo elicited during the 19-position changes used by Norré and colleagues as described above. They also measured balance performance on 45% of the

patients (post-therapy). A composite score related to overall performance capabilities on the six sensory organization tests (Equitest) was used as an outcome to represent postural ability. The authors did not present details of what data they used for the composite balance score.

They found that 87% of the patients experienced at least some reduction of symptoms while on the treatment program with 31% reporting a complete absence of symptoms post-therapy. A significant shift toward lower disability scores was noted post-therapy (mean pre-therapy disability score was equal to 3.05, mean post-therapy disability score was equal to 1.3). The mean post-therapy score for Norré's 19-position test was significantly lower than the mean pre-therapy score for 45% of the patients who were evaluated post-therapy.

They identified six patterns associated with the sensory organization tests:

1. Normal (NOR) - test results normal for age group in all six conditions.
2. Vestibular dysfunction (VD) - Abnormal postural sway with vestibular input only.
3. Visual preference (VPD) - Abnormal postural sway when presented visually inaccurate information regardless of somatosensory cues.
4. Visual and vestibular dysfunction (VVD) - Abnormal postural sway when using vestibular system input even when accurate visual cues are available.
5. Somatosensory and vestibular dysfunction (VSD) - Abnormal postural sway when using vestibular input even with accurate support surface information.
6. Severe (SEV) - Abnormal sway on four or more test conditions that do not meet the definitions specified above.

They were able to categorize 94 patients using this system. Shepard et al

used mean post-therapy disability and symptoms scores for each of the six categories of patients identified by the sensory organization tests. They noted a significantly poorer performance of the patients with a pre-therapy visual and vestibular dysfunction pattern relative to all the other patterns. This was also significant for post-therapy disability scores at p values of 0.08 to less than 0.002, and post-symptoms scores at p values of 0.07 when compared with the NOR, VD, and VPD patterns. They concluded that their results indicate rehabilitation therapy for balance disorders can be of significant benefit for a wide range of patients with vestibular dysfunction.

Telian et al (1989) studied the effects of habituation therapy on vestibular symptoms (vertigo, postural abnormalities, positional nystagmus or rotational asymmetry) in patients with chronic vestibular dysfunction.They evaluated sixty five patients with symptomatology longer than six months (mean duration of symptoms was equal to 3.1 years). As in the previous study by Shepard et al (1990) a specific individualized exercise program was designed to address the functional problems identified through their testing procedure. The exercise program had three objectives: 1) to facilitate habituation of specific pathological responses; 2) to address underlying problems of postural control; and 3) generalized conditioning.

The patients' response to therapy was measured using a functional disability scale and a response level of persistent vestibular symptoms. The disability score consisted of 0 - 5, 0 was equal to no disability, negligible symptoms, to 5 was equal to established severe disability. The response to symptoms was categorized according to 5 separate headings A - E, A was equal to complete resolution of symptoms to E was equal to worse.

They report that post-therapy 82% of patients were assigned to a

response group which identified improvement in symptoms, 59% were placed in group A or B, and 23% in group C. The disability scores indicated that patients with lesser disability levels pre-therapy improved considerably after therapy. The patients with severe disability scores pre-therapy achieved dramatic recovery. The patients who scored 1 through 4 pre-therapy, achieved a mean disability score of 2, post-therapy. Telian et al (1990)noted that: 1) Patients' age and duration of symptoms did not adversely influence the outcome of therapy. 2) Patients with stable unilateral chronic vestibular lesions had an extremely favourable response to therapy. 3) Patients with severe disability only rarely showed improvement. 4) Patients with recent disabilities had a better prognosis for success with therapy. 5) Patients with objective test results suggesting poor compensation for peripheral vestibular lesions were not as likely to obtain remarkable recovery. However, their overall improvement rate was similar to the patients with evidence of satisfactory compensation. 6) Patients with mixed central and peripheral lesions did not respond as well as the average patient.

Takemori et al (1984) reported on the benefits of a generic non-customized exercise program for patients who had undergone labyrinthectomy, VIIIth nerve section, and unilateral ablation, as well as in patients with bilateral peripheral dysfunction. They studied 12 cases of unilateral labyrinthectomy, 3 cases of VIIIth nerve section, 22 cases of streptomycin infusion into the middle ear cavity and 8 cases of bilateral vestibular dysfunction. They compared the data from this group of patients with a group of normals ranging in age from 20 to 40.

Their generic goal directed exercise program consisted of : 1) standing upright with both feet close together eyes open or closed for 30-60 sec. times 10 repetitions; 2) stepping 50-100 times with their eyes closed and with both arms

held upward; 3) placing one foot in front of the other with eyes closed for 30-60 sec.; and 4) standing on one foot with eyes closed for 30-60 sec. These exercises were commenced as soon as possible post lesion and were performed 3 times a day on a daily basis.

Takimore et al (1985) measured movement of centre of foot pressure as an estimate of centre of gravity projection on to a base of support by stabilometer, while subjects stood quietly with eyes open and closed for 30 sec. intervals. The length or area of the centre of gravity displacement were calculated and used as outcome measures for balance performance during this task.

The authors report the length and area of centre of gravity displacements for 30 sec. were 281 +/- 83 mm, 2.6 +/-1.3 cm^2 with eyes open and 442 +/- 106 mm., 6.0 +/-4.0 cm^2 with eyes closed for the control group. The authors did not indicate whether the healthy controls were age-matched to the patients in the study.

After the unilateral labyrinthectomy or the VIIIth nerve sections, training was started as soon as the subjects could stand up. They noted that the movement of the centre of gravity with eyes open and eyes closed for these patients was larger than preoperative values. However the actual values were not presented by the authors.

Regarding the effects of treatment on outcome measures, they reported that the length and area of the centre of gravity displacement values gradually returned to normal over a period of 2-3 months. The exceptions to this were the cases with bilateral vestibular dysfunction who took from 3 to 6 months. It should be noted that although a 100% success rate was reported, the testing procedure only examined standing balance under benign conditions; they did not measure

dynamic postural control, gait control, or positional sensitivity .

Shumway-Cook and Horak (1990) used a case study to illustrate the applications and effectiveness of a rehabilitation approach to the assessment and treatment of vertigo and postural imbalance in a patient with peripheral vestibular pathology.

They evaluated balance performance using the sensory organization test protocol of the Equitest device. During the different test conditions they used a potentiometer device to record hip displacement as an estimate of body sway and consequently balance performance. To measure vertigo they used Norre's 19-positional test pre and post treatment. The patient received six weeks of therapy and performed her exercises twice daily at home. She also participated in a low impact aerobics program several times a week. Her exercises targeted her postural, gaze and motion perception disabilities.

Shumway-Cook and Horak report that the patient pre-therapy showed increased body sway when either vision or surface inputs were inaccurate for orientation, and that potentiometer measures showed excessive hip movement to control body sway. They note that the patient had normal body sway during all six sensory organization tests and potentiometer measures indicated that sway was occurring principally about the ankles, after six weeks of therapy. The authors only provided graphs to present the data for all six conditions over the four test periods.

The patient demonstrated positional vertigo in 14 positions pre- therapy with an average vertigo intensity score of 6 (scale 0-10). After the six weeks of therapy her complaints of vertigo had reduced to a score of 2 (scale 0-10) in 12 positions. The patient had no symptoms of vertigo in any of the positions after six months of therapy. The authors speculate that the patient's progress was due

to a rebalancing of tonic activity in the vestibular system because of an increased effectiveness of visual and somatosensory channels onto the vestibular nuclei.

The authors suggested that for a rehabilitation program to be effective in treating vestibular disorders it must use these physiological mechanisms to facilitate compensation and recovery. They feel there are four areas of function that need to be treated in patients with vestibular pathology: 1) postural control in sitting, standing and walking; 2) eye-head coordination and gaze stabilization; 3) motor perception; and 4) physical conditioning.

In a review of rehabilitation treatment programs for patients with vestibular disorders, Herdman (1989) presented two different exercise approaches used to manage patients with vestibular disorders. The exercises were designed to enhance the adaptation of the vestibular system and exercises that foster the substitution of alternate strategies to replace lost vestibular function.

She states the main goal of a vestibular exercise program is to: 1) Improve visual following when the head is stationary. 2) Improve gaze stabilization when the head is moving. 3) Improve visual-vestibular interactions during head movement. 4) Improve balance in stance and in ambulation.

Herdman noted that the exercises generally should mimic the conditions which produce the symptoms and should include both foveal and full-field visual stimuli. Exercises should incorporate vestibular, visual, and proprioceptive information to optimize recovery because there are major interactions between vestibular cues and both visual and somatosensory cues to stimulate compensatory eye movements and postural responses.

Herdman states that the best stimulus for inducing adaptation is the motion of an image across the retina combined with head movement. As

adaptation of the vestibular system is system specific it is important to perform head movement at different frequencies to improve VOR gain across many frequencies. Visual experience is an important factor in inducing adaptation of the dynamic vestibulo-ocular system (increased VOR gain).

The second exercise approach that Herdman discussed emphasized the substitution of visual and somatosensory information to stabilize the visual world and to maintain postural stability. She states that patients should be instructed to practice a combination of eye-head movements and body-on-neck rotation as each patient uses different set of strategies to compensate for loss of VOR. Patients should also be taught mechanisms to use vision and proprioceptive information to maintain postural stability.

She recommended exercises that enhance vestibular adaptation for patients who have some remaining vestibular function. For the patient with complete loss of vestibular function she recommended exercises that promote substitution of alternate strategies. To decide on the type of approach, Herdman noted that vestibular function tests such as caloric, rotational, and posturography should be used to assess the particular patient's vestibular functional levels.

2.6.4. SUMMARY

Although the literature reports that the severity of the lesion and loss of substitution systems may hinder the ability of the CNS to compensate there is evidence that patients with peripheral vestibular dysfunction can improve their function and decrease debilitating symptoms of vertigo, nausea, and loss of equilibrium through rehabilitative exercise programs.

Studies of the treatment of patients with vestibular dysfunction have

shown that there are therapeutic effects of exercise. These studies have suggested that to be effective the exercise program should: 1) provoke the symptoms of vertigo (Cooksey 1946); 2) be goal oriented (Igarashi et al 1981); and 3) include eye-head coordination exercises at different frequencies (Dix 1976).

Advances have been made in recent years in designing new rehabilitative approaches to the treatment of certain types of vestibular disorders but there has been sparse experimental work done to evaluate these approaches. Norré used a subjective description of dizziness, intensity and duration as his parameters to evaluate his habituation exercise program. Shepard used a retrospective study of his individualized treatment program and measured subjective measures of overall therapy performance and gives no indication of how his composite score was designed. Takemori used a limited measure of static balance to evaluate his generic non-customized exercise program. Shumway-Cook and Horak state the rehabilitation programs can relieve symptoms of body sway and vertigo but they did not use quantitative studies to compare their rehabilitation program to other modalities.

It would seem clear from this information that work still needs to be done to evaluate the effectiveness of rehabilitation exercise programs in improving the symptoms of patients with vestibular dysfunction.

CHAPTER 3 - PROPOSED STUDY

3.1 IDENTIFICATION OF THE PROBLEM

Although achievements have been made in the area of diagnosis of vestibular dysfunction, little has been achieved in the development of treatment programs for the varied population of patients with vestibular disease or damage. In a study by Nashner and Wolfson 1974) different programs of intervention were evaluated; two exercise routines, brisk and VHT and drug therapy. The researchers concluded from their study that drug therapy did not have as much of a benifical effect on relief of vertigo as VHT, and infact that drug therapy has had minimal success with this patient population. Other researcher (Tracois et al 1990; Wilson and Jones 1979; Zee 1985) have looked at the benefits of medication for symptoms of vestibular disorders and note that use of medication in recurring episodes of vertigo is dependent on the specific disorder affecting the vestibular apparatus. Only a limited number of studies have compared the benefits of exercise therapy versus drug therapy for alleviating symptoms of vertigo. As mentioned above, Norré (1987), noted a 36.7% relief of symptoms of vertigo for patients on drug therapy as compared to 70%-80% relief for patients treated with vestibular habituation exercises.

A number of medications such as baclofen, acetazolamide, clonazepam and isoniazid have been reported to having some effect on nystagmus (Currie and Matsuo 1986; Tracois et al 1990; Zee 1985).

Rehabilitation programs for treatment of patients with certain vestibular deficits date back to 1945, when Cawthorne (1944) and Cooksey (1946)

designed a set of eye-head exercises for patients with symptoms of dizziness. Since this time, most approaches to rehabilitation therapy for patients with peripheral vestibular disorders have been geared to treating the symptoms of dizziness or vertigo. Very little work has been done to develop and evaluate rehabilitation programs to address the issue of impaired balance.

3.2 AIM OF STUDY

The main aim of this study is to further develop andevaluate clinical treatment programs for patients with vestibular disorders to improve their functional capabilities. This treatment program should be based on a body of scientific evidence to support the procedure as a valid method of improving function. The treatment program should also be easily transferable to a clinical setting and should provide the clinician with valid tools for outcome measures.

3.3 PURPOSE OF STUDY

The research to date would support the idea that to facilitate and achieve optimal functional recovery following peripheral vestibular disorder or dysfunction, goal-directed activities under combinations of varied visual and somatosensory conditions should be encouraged, even though these activities may aggravate accompanying symptoms of vertigo and dizziness. Optimal function defined as a reduction in symptoms of vertigo and postural imbalance.

The purpose of this study was to evaluate the effects of two exercise programs on balance performance in patients with chronic peripheral vestibular dysfunction, and to assess whether these exercise programs do induce adaptive

modifications of the VOR gain and time constants and OKN gain and time constants..

The two exercise programs consisted of a home program of exercises based on the Cooksey - Cawthorne exercises and a new rehabilitation treatment program devised for this study based on goal-directed activities under a combination of varied visual and somatosensory conditions.

The exercise program developed for this research study consisted of balance retraining and goal-directed eye-head exercises, under a variety of visual and somatosensory sensory conditions. The somatosensory conditions included balancing on various thicknesses of sponges and balancing on a narrow beam. The visual conditions included varying degrees of optokinetic stimulation which consisted of: full view striped wall, dim lights and moving light stripes.

3.4 HYPOTHESIS

The hypothesis of the study was that the benifits of the comprehensive exercise program will be greater than those observed with the Cooksey-Cawthorne exercises in patients with chronic peripheral vestibular dysfunction. The benefits will manifest themselves in the form of improved standing balance performance and a reduction in left-right differences in VOR gain and time constants, and OKN gain and time constants.

3.5 CLINICAL RELEVANCE OF STUDY

The incidence of vestibular related disorders in Canada is approximately 0.1% (based on Manitoba statistics) which means that approximately 25,000 new patients would be examined each year in Canada. These patients experience a number of disturbing signs and symptoms which negatively affect the quality of their lives. The signs and symptoms interfere with their ability to work, enjoy leisure activities and in some cases even the ability to sleep well. The only treatment available to patients was medication, which has limited effect on vertigo (Nashner and Wolfson 1974; Tracois 1990; Zee 1985), and the Cooksey-Cawthorne eye-head exercises which again mainly addressed the problems of vertigo associated with vestibular dysfunction.

CHAPTER 4 - METHODS

4.1. SUBJECTS

Experiments were performed on twenty three (n=23) patients who met the criteria described below and who volunteered to participate in the study.

Patients were recruited between September 1990 and September 1992. Each participant was required to sign a consent form that fully described the study and gave a clear description of what was expected of them (see Appendix 1).

The study was approved by the Faculty of Medicine Committee on the use of Human Study Research, University of Manitoba (see Apendix 3).

4.2. INCLUSION CRITERIA

Inclusion criteria consisted of the following:

1) Clinical diagnosis of peripheral vestibular dysfunction and no other neurological deficits.

2) Had reported signs/symptoms for one year or more.

3) Were not currently taking medication for their vestibular problems.

4) Had an abnormal test score of balance performance in the sensory organization tests using the Equitest apparatus (Neurocom Inc., Clackamas, OR).

4.3. EXCLUSION CRITERIA

Exclusion criteria consisted of the following:

1) Central Neurological deficits.

2) Patients who could not understand English.

3) Patients with balance disorders for reasons other than Vestibular disorders such as musculoskeletal problems.

4.4. SUBJECT GROUPINGS

The subjects were randomly assigned by a toss of a coin to either the home exercise group (H), or the rehabilitation group (Reh). A third grouping consisted of patients who first received the home exercise program (A group) and then later received the rehabilitation program (B group).

The subjects are described according to age, sex, clinical diagnosis and duration of symptoms in Table 2.

4.4.1. GROUP H

Group H consisted of twelve subjects, six males and six females ranging in age from 29 to 66 years old with a mean age of 48.08 years. The duration of symptoms in group H, ranged from one year to twenty years with a mean duration of 4 years.

Table 2. **Patient Information and Clinical Findings**

Subj	Age	Sex	Clinical Diagnosis	History (years)	Caloric	VOR Gain CW	VOR Gain CC	VOR TC CW	VOR TC CC	OKN Gain CW	OKN Gain CC
R-NB	46	M	Neuritis	2	+L	0.85	0.55	15	22	1.1	0.95
R-NC	50	F	Neuritis	2	+L	1.0	0.7	21	13	1.0	1.1
R-SC	61	F	Etiology ?	8	+B	0.6	0.77	30	22	1.2	1.3
R-RD	47	M	Menier's	12	+L	0.8	0.45	12	6	1.1	0.96
R-LF	44	F	Etiology ?	9	N	0.75	1.0	19	12	1.05	0.98
R-AJ	42	M	Neuritis	13	+L	0.8	0.6	13	6	1.05	1.1
R-AK	56	F	Neuritis	4	+L	0.9	0.7	13	8	1.1	1.2
R-WP	63	M	NEURITIS	2	+L	0.95	1.1	15	9	1.2	1.15
R-VR	44	M	Neuritis	8	+L	1.4	1.0	30	21	1.2	1.1
R-SS	49	M	Neuritis	2	+L	0.43	0.23	5	3	0.88	0.8
R-JW	51	F	Etiology ?	2	+B	0.85	0.6	30	22	1.2	1.1
H-JB	29	M	Trauma	8	+L	0.85	0.5	25	12	1.1	1.0
H-WF	47	M	BPPV	3	N	0.9	0.7	27	14	1.07	0.99
H-TJ	47	F	BPPV	2	N	1.2	0.95	30	24	1.2	1.1
H-PM*	45	F	Trauma	2	+L	0.83	0.7	20	16	1.1	1.25
H-EM	46	M	Trauma	1	+R	0.6	0.9	22	32	1.2	1.2
H-JP*	43	F	Neuritis	1	+R	0.8	0.95	8	12	1.3	1.2
H-RP	53	M	Neuritis	3	+L	0.8	0.67	21	13	1.0	0.92
H-JR*	59	M	Neuritis	20	+R	0.6	0.9	20	27	1.0	0.85
H-AS*	66	F	Otoxicity	2	+B	0	0	0	0	0.9	0.85
H-IS*	60	F	Etiology ?	1	+R	0.7	1.15	28	30	1.0	1.1
H-FW*	31	F	Neuritis	5	+L	1.0	0.7	30	17	0.95	1.1
H-NW	51	M	Trauma	2	N	0.6	0.86	28	21	0.6	0.83

R is the Reh group, H is the Home group
* Subjects who entered the Reh training program (A-B group)

CALORIC
- + L/R is a positive asymmetry score (difference greater than 25%) using combined responses to 30° C and 40° C water, left/right;
- + B is reduced response, bilaterally (mean SPV less than 5°/sec.);
- N is response within normal limits

4.4.2. GROUP REH

Group Reh consisted of eleven subjects, six males and five females ranging in age from 42 to 63 years of age with a mean age of 50.27 years. The duration of symptoms in group Reh, ranged from two to thirteen years with a mean duration of 5.8 years.

4.4.3. DIAGNOSIS

The subjects in the study had a number of different diagnosis which included: Neuritis, Meniere's Disease, trauma, otoxicity, BPPV and unknown etiology .

4.5. TREATMENT PROGRAMS

4.5.1. HOME EXERCISE GROUP

The participants in this group were instructed to perform a modified version of the Cooksey-Cawthorne exercises; at home, on a daily basis. Home exercise subjects were asked to perform the exercises at least three times daily within their level of tolerance. Each subject was asked to perform the exercises for ten to twelve weeks. Compliance was monitored by regular telephone communication.

This Home program consisted of the following exercises:

1.Exercises performed in sitting:

(a) Oscillating eye movements (head stationary) at first slowly and then quickly:

(i) left-right

(ii) up-down

(iii) diagonal movements

(iv) focus on finger moving 3 feet to 1 foot away from face

(b) Oscillating head movements at first slow then quickly. First with eyes closed, then eyes open:

(i) bending forwards and backwards

(ii) rotating from side to side

(iii) tilting right ear then left ear to shoulder

(iv) diagonal movements (head tilted chin up to R and then down to L shoulder, followed by chin up to L and then down to R shoulder)

(c) Shoulder shrugs ten times

(d) Bending forward and picking up objects from ground ten times (small objects such as tennis balls, books etc.)

2. Repeat 1(a) to 1 (c) in standing position

3. Change position from lying down (left side and right side) to sitting to standing, slow at first and then quickly (five times to each side).

These exercises are routinely prescribed for patients with peripheral vestibular dysfunction at the Vestibular Clinic, Health Sciences Centre, Winnipeg, Manitoba. Each subject in the H group was given a training session of approximately 45 minutes by a licensed Physical Therapist on how to perform the exercises, and was provided with a written description of each exercise(as listed above). The Home group were informed that they may experience a flare up of their symptoms following their exerciese and to contact the therapist if this occurred. Each participant was contacted by telephone on a regular basis to

review their exercise program and to respond to any questions they might have regarding their exercises.

As described below in Protocol and Procedures, the subjects were scheduled for tests at week seven and week thirteen. At these times the exercise program was reviewed and any necessary changes were made to correct the method of exercising. Any other feedback was provided by telephone communication.

4.5.2. REHABILITATION EXERCISE GROUP (Reh)

The participants assigned to the rehabilitation exercise program (Reh) were assessed and received an individualized training program. A detailed description of this training program is presented below.

Subjects received fourty five minutes of training at each treatment session, three times a week for twelve weeks. The training sessions were conducted by a licensed Physical Therapist.

Prior to starting the rehabilitation program each patient was put through a thorough assessment. An outline of the assessment protocol is presented in Appendix 2. The following is a description of the assessment performed on each subject.

In sitting, passive and active head rotations in different planes which were assessed with eyes open and closed. During each manoeuvre the subject verbally indicated to the Physical Therapist any symptoms of dizziness or unusual sensations. These movements were performed to indicate which types of head movements elicited symptoms and to document the type of symptoms experienced.

Passive and active head rotations were then performed while the subjects were asked to fixate on a visual target on the wall in front of them. The eyes were then closed to test whether this had any effect on the subject's perceptions of symptoms. Visual fixation was used to indicate whether the subject was able to focus on a target while moving their head, to test the VOR reflex. It was important for the therapist to note if this manoeuver elicited nystagmus in light. Subjects were asked to comment on any difficulty they were experiencing and/or whether they experienced blurred vision.

The subject was then seated in front of a striped wall and asked to perform all of the same test movements to evaluate their reaction to optokinetic stimulation. These tests were done to note any spontaneous nystagmus caused by the stimulus and to identify if the patient could overcome the distraction and maintain focus. All symptoms were recorded for each movement.

After all of these tests were performed sitting, if the subject could tolerate further testing they were then run through the same tests standing, in the same sequence except for the passive movements. All of these movements were tested standing to evaluate their effects on balance. Any body sway was noted, the extent of the sway and which movements in particular produced the greatest degree of sway.

If tolerated the subject was then asked to try and perform these exercises on a sponge standing. The sponge used for the assessment was the firmest density sponge. For treatment, three sponges of various densities were used to progress the balance activities and were progress from firm density to less firm which required more balance ability.This test was designed to evaluate the effect of altered somatosensory input on the subject's ability to balance. Any sway was noted, the extent of the sway and which movements in particular produced the

greatest degree of sway and/or symptoms of dizziness.

If the subjects were unable to tolerate all of the tests at one time, the tests were performed at subsequent appointments.

Each subject was informed they could experience a flare-up of their symptoms following the test and treatment procedures, and that it was important that they informed the therapist if and when this occurred.

Once the assessment was completed the therapist was aware of:

1) which movements produced symptoms,

2) whether the subject had problems focusing while moving their head,

3) whether the subject had problems in responding to optokinetic stimulus, and

4) an evaluation of the subject's balance capabilities.

An individual treatment program was then started for each subject which included goal orientated exercises for eye-head coordination and balance specific to their needs, as assessed by the test battery (see appendix 2).

The Reh program consisted of the following exercises:

1. In standing on a normal support surface subjects were instructed to:

   (a) Make oscillating head movements, all planes (rotation R and L, flexion and extension, side flexion R and L, diagonal), in front of a blank white wall and then in front of a white wall with vertical one inch black stripes (top to bottom) placed 8 inches apart, while focusing on a stationary target and while reading different pages of text aloud. Progressively increase amplitude and speed.

This exercise was intended to enhance somatosensory input to assist with balance while using the VOR to focus gaze and suppress the effects of the optokinetic stimulus of the striped wall. Once balance is established the amplitude and speed of the head movement is increased to challenge the VOR

system.

(b) Track a moving visual target (hand held laser light that was moved in an unpredictable fashion by the therapist), at first with head fixed and then with combined eye-head movements. Repeat in front of the blank white wall and then in front of the striped wall.

This exercise is based on the assumption that full somatosensory input will assist balance. The moving visual target encourages the subject to use the smooth pursuit eye movement to direct the fovea to the target of interest (a dot of laser light).

(c) Sway body forward-backward and side to side, at first with eyes open then closed.

This manoeuver taught the subjects their safe limits of sway on a firm surface with full somatosensory input and visual input. Once the subject had safely learned their sway limits the task was progressed by asking the subject to close their eyes, thereby eliminating visual input. This exercise was intented to make the patient concentrate on where their CFP was for balance reactions in sway.

2. Standing on modified support surface:

(a) Repeat tasks 1(a)-1(c) and then again with eyes closed while standing on pads constructed of 16-inch by 20-inch pieces of sponge-like material (varying in thickness and compliance) on top of which was placed a 14-inch by 16-inch piece of plywood.

This exercise was designed to add a degree of difficulty to the balance task by reducing the somatosensory input at the same time as working on developing the VOR to stabilize gaze.

(b) Repeat tasks 1(a)-1(c) and then again with eyes closed while standing

on a balance beam (6 inches in width).

This exercise eliminated visual input to balance and changes the somatosensory input which challenges the subject to rely on their remaining vestibular input to maintain balance.

3. Exposure to large-field optokinetic stimulus (OKS), at first with a dim light in the background and then in the dark. For this purpose, a portable optokinetic stimulator (nystagmus optokinetic stimulator, ICS Medical) was used to project a moving pattern of light and dark stripes, at variable speeds (0 - 100 c/sec.), on the wall in view of the subject. With the stripes moving left to right, up-down, and in diagonals, subjects were instructed to:

(a) In sitting:

(i) follow a single moving stripe across the wall, (smooth pursuit)

(ii) focus on a stationary visual target, (VOR)

(iii) perform oscillating head movements, all planes, without focusing on any particular visual target, and then while focusing on a stationary visual target, (VOR)

(iv) track a moving visual target (laser light that was moved in an unpredictable fashion) at first with head fixed and then head moving.

(b) Repeat (a) while standing on a normal support surface.

(c) Repeat (a[i]) and (a[ii]) while swaying body forward-backward and side to side.

(d) Repeat (a) while standing on sponge pads and while standing on a narrow beam.

(e) Repeat (a[i]) and (a[ii]) while walking on the narrow beam; forwards and back-wards and laterally, side to side.

Treatment was always started in sitting for subject safety and to assist

the therapist in developing an understanding of the subject's endurance and symptom occurrence. The eye-head coordination exercises consisted of the subject performing five head movements: flexion, extension, side flexion, diagonal movements, and rotation; in front of a full view blank white wall, with and without visual targets for fixation. Subjects were asked to perform these exercises five times in each direction. The speed and amplitude of movement was progressed based on the subject's tolerance.

Once the patient was able to manage those activities relatively symptom free (dizziness, nausea, blurred vision), they were progressed to the same exercises performed in front of a striped wall to add optokinetic stimulation. By adding the optokinetic stimulus the patient must suppress the spontaneous nystagmus to be able to focus on a visual target. Progression of speed and amplitude was again based on the subject's tolerance level.

Subjects were also asked to read text which consisted of a number of lines of text taken from the local newspaper, enlarged and placed on a moveable chart. The text was held in front of the subject. They were then asked to read the text aloud, first with head stationary and while the text was moved then with the text held stationary and the subject moved their head. The text was moved by the therapist in various directions, at various speeds and amplitudes. Subjects were asked to move their heads slowly and in small amplitudes and then to increase their speed and amplitude of the five types of movements.

When the patient was progressed to standing activities, biofeedback of centre of foot pressure (CFP) was used during these training sessions to heighten the subjects awareness of the balance performance, and to demonstrate and teach the subject their safe limits of sway for balance control. For this purpose a portable force transducer device was constructed. The device

consisted of four force sensing resistors (FSR) (Interlink Electronics Inc. Carpinteria, CA. USA) 2 cm. square and 0.4 mm thick, which were connected to a full-bridge amplifier with offset gain and sensitivity settings. The force sensing resisters were secured at fixed anatomical positions, to each of two foot sole pads (varying sizes to accommodate the different foot sizes of the subjects), one at mid-forefoot and one at approximately mid-heel. The foot sole pads with FSRs were placed under each foot of the subjects. A LED display and/or a variable frequency audible tone provided the subjects with information regarding direction and relative amount of centre of foot pressure (CFP) displacement during the various tasks they were requested to perform.

The biofeedback was continued until each subject was able to maintain their CFP with eyes closed and while moving their head at varing speeds and amplitudes. Once the subject was able to master this task, they were progressed to standing activities on a modified support surface. The modified support surface consisted of sponges of various thickness and compliance as described above. The subjects were asked to perform progressively more difficult eye-head and balance activities. The more difficult eye-head activities included: following a moving light, visually acquiring a random light, moving to a target on verbal command, these exercises were started slowly and then more quickly. Balance exercises were progressed from standing on sponge to swaying forward-backward and from side to side, first with eyes open and then with eyes closed.

Balance exercises were then progressed to walking on a narrow balance beam performing various eye-head exercises. These eye-head exercises included the focusing on a stationary and moving target while moving the head at various speeds and amplitude. All balance exercises were progressed

according to subject tolerance and ability along a similar treatment path.

The visual conditions were varied from dim lights situations to strong optokinetic stimulus. The subject was asked to follow a beam of laser light which was moved slowly in various directions and then moved more quickly. Subjects were asked to follow a beam of light as it moved left-right and in various other directions. All visual conditions were started in the sitting position to accustom the subject and to observe their reaction in a safe position. When the subject could tolerate it, the visual situations were tried standing. The surrounding light was dimmed and eventually all visual situations were repeated in complete darkness.

Subjects were instructed to perform the exercises in Section 1 of Reh treatment program in sitting and to progress to standing activities when the therapist felt the patient was ready to safely practice standing balance activities described above, at home on a daily basis (minus the laser light tracking). For the reading exercise, subjects were instructed to read the text on television (i.e. an information channel with text, such as, a weather or news broadcast) aloud while oscillating their head. When the subjects were capable of safely performing the exercises in Section 2 Reh treatment program, they were instructed to perform them at home. Each subject was provided with a sponge pad for home use. Patients were not instructed to try the narrow beam activities at home for safety reasons.

4.5.3. A-B GROUPING

In addition to the H group and Reh group, all participants assigned to the home (H) exercise group were given the opportunity to enter the rehabilitation

program if they did not think that they had improved as a consequence of the Cooksey-Cawthorne home training program. Seven of the twelve participants in the Home (H) exercise group chose to enter the Reh program after their five month follow-up period (see Protocols and Procedures). Thus in this group, seven subjects first performed twelve weeks plus five months of the home exercise program, designated as A group, then entered the rehabilitation program of exercises group for twelve weeks plus a five month follow-up, designated as B group.

Each subject regardless of grouping, was encouraged to walk outside and to remain as active as possible with daily chores and leisure activities.

4.6. PROTOCOLS AND PROCEDURES

All subject were tested at fixed intervals, beginning the day before commencement of treatment. Two tests were performed, the first consisted of the sensory organization tests using the Equitest apparatus (Neurocom Inc., Clackamas, OR.). This was immediately followed by the second test, which consisted of a series of step chair rotations in the dark and then in the light to elicit the horizontal vestibulo-ocular reflex (VOR) and optokinetic reflex (OKN). The tests were repeated as follows (see Table 3):

1) Test Day 1 (TD1) performed one day prior to the start of the respective treatment programs (H group and Reh group).

2) Test Day 2 (TD2) performed at the beginning of week seven which was six weeks after the start of treatment.

3) Test Day 3 (TD3) performed at week thirteen which corresponded to the end of the twelve week treatment period.

TABLE 3. **Test and Treatment Time Frames**

A. Test Days and Treatment Program for Reh and Home Groups

Test	TD1	TD2	TD3	TD4
Week	1	7	13	
	Start Treatment		Stop Treatment	<--------->
	(----------)			(----------)
	3 Months			5 Months

B. Test Days and Treatment Program for The B Group

Test	TD4/TD1 (B)	TD2 (B)	TD3 (B)	TD4 (B)
Week	32	38	44	
	Start Treatment		Stop Treatment	<--------->
	(----------)			(----------)
	3 Months			5 Months

4) Test Day 4 (TD4) performed at eight months which corresponds to five months following the end of the treatment.

5) Test Day 4 (TD4) for the A group (Home exercise group) was used as test day one (TD1) for the B group.

6) Test Day 2 (TD2) for the B group was performed six weeks after TD1 (9.5 months).

7) Test Day 3 (TD3) for the B group was performed six weeks after TD2 (11 months).

8) Test Day 4 (TD4) for the B group was performed five months after the end of treatment (16 months).

4.6.1. EQUITEST

This is a test of standing balance performance under a variety of altered visual and support surface conditions. The different sensory test conditions are fully described in Black et al (1983, 1988) and Voorhees (1989), and the methods for quantifying balance performance are described in Goebel and Paige (1989), Paige (1983) and Voorhees (1989).

An illustration of the Equitest device is presented in Figure 1. this device consists of:

1) a moveable platform upon which the subject stands and which rotates about an axis approximately collinear to the axis of rotation of the ankle joint,

2) a surrounding screen enclosure which rotates about an axis approximately collinear to the axis of rotation of the ankle joint.

Two identical force plates are embedded in the platform, one for each foot. Each force plate contains a pair of strain gauge type force transducers

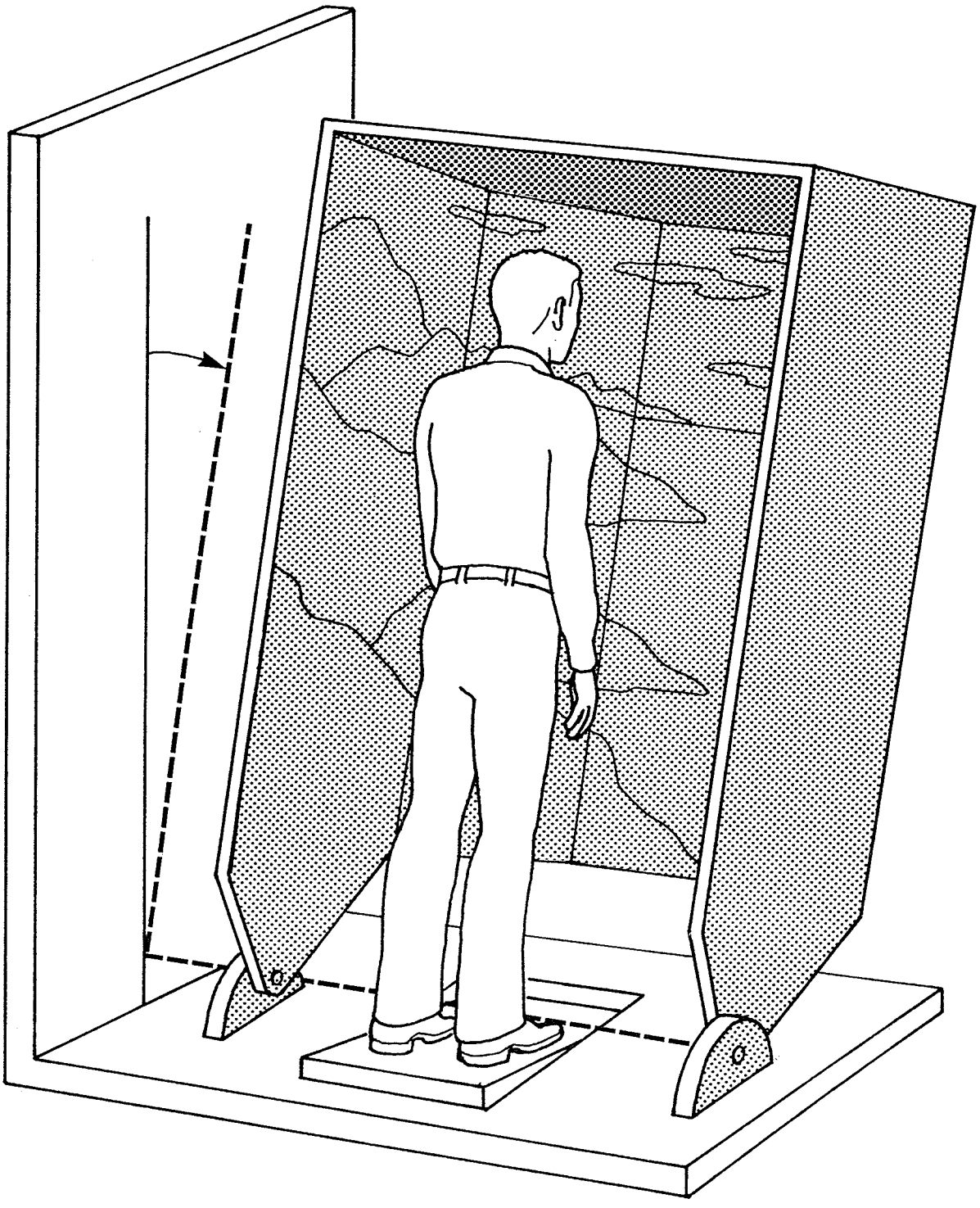

Figure 1. **Equitest device; a moveable platform and surrounding screen enclosure which rotates about an axis approximately collinear to the axis of rotation of the ankle joint.**

positioned at specific locations on the front and back of each force plate. From the vertical force signals the x axis (anterior-posterior) centre of vertical force position is calculated. In addition one force transducer is orientated to detect horizontal or shear reaction forces. Each subject was carefully positioned on the platform to align the lateral malleoli (ankle joint) with the axis of rotation of the platform/visual surround. During each test, subjects were instructed to stand still and erect for twenty seconds with arms resting by their sides.

The tests consisted of four different sensory organization tests (see Figure 2):

1) eyes open and sway stabilized visual surround (Eqt-1)

2) eyes open and sway stabilized support surface (Eqt-2)

3) eyes closed and sway stabilized support surface (Eqt-3)

4) eye open and sway stabilized visual surround and support surface (Eqt-4)

For a diagram to illustrate the Equitest conditions refer to Figure 2. Each test condition was repeated three times in succession. The anterior-posterior (A-P) centre of vertical force position and the patient's height was used to estimate A-P centre of mass sway angle. During the sway referenced conditions, the support surface and/or the visual surround were rotated by an amount equal to the estimated A-P centre of mass sway angle of the subject.

An estimated A-P sway angle (Equitest operator's manual) is geometrically calculated, using the following algorithm:

$$\text{Sway}(i) = \arcsin \frac{PXA(i-1) - PXA(i)}{0.55\,H}$$

Where:

- H is the subject's height in inches
- The 0.55 multiplier converts total height to centre of mass height based on the

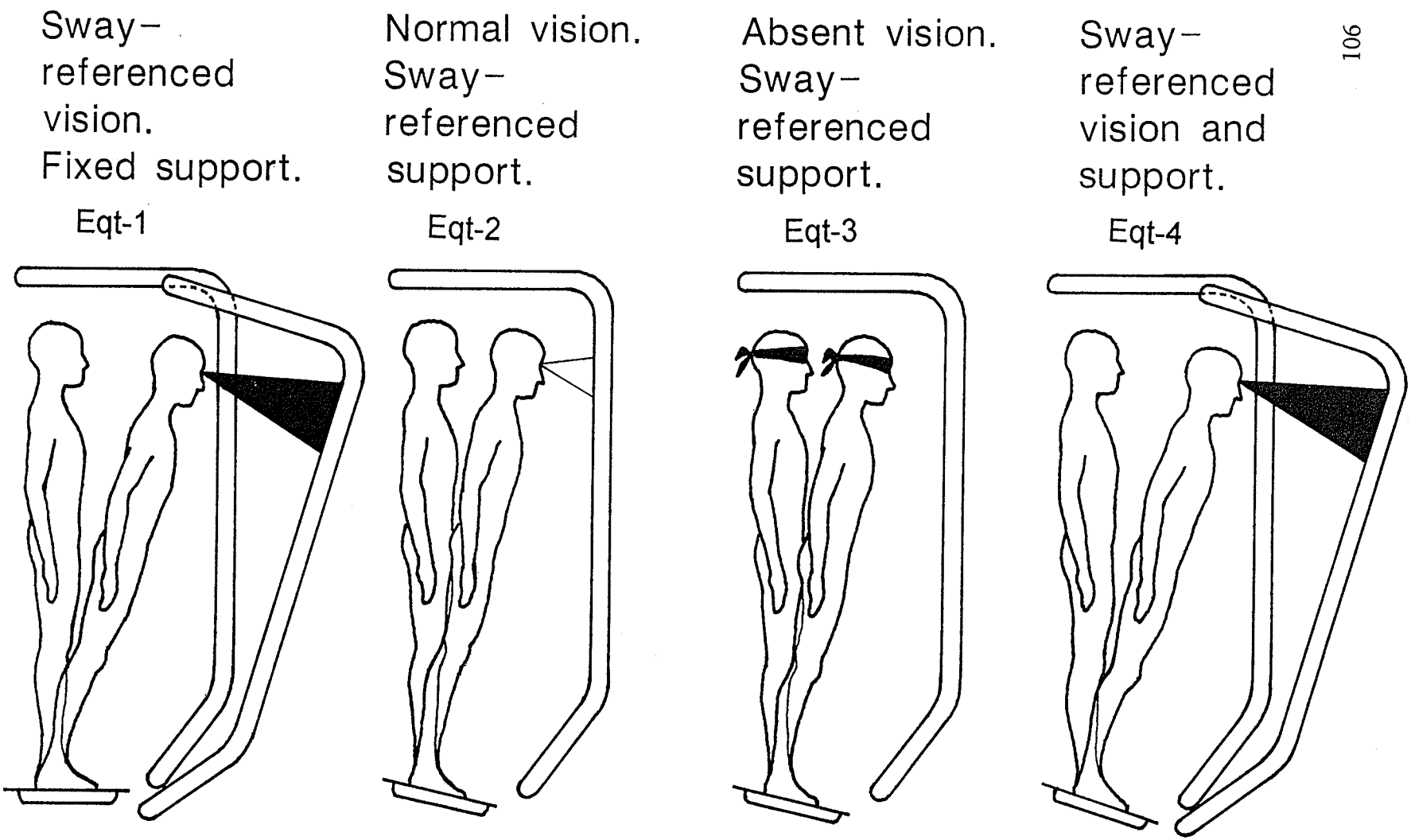

Figure 2. **The four sensory organization tests used to assess balance performance (Eqt-1, Eqt-2, Eqt-3 and Eqt-4).**

fact that over a wide range of adult heights and weights the centre of mass is approximately 55% of total body height.

- Sway (i) is the instantaneous sway angle at the time of the ith sample.

- PXA (i) is the weighted average position of the centre of total vertical force at the time of the ith sample.

The total angle os sway would be obtained by summing the Sway (i), SO = ($\sum$ $Sway_i$)

The value to the right of the arc sin is multiplied by a scale factor to produce the following relationship:

1 degree sway = 204.8 digital "counts".

Vertical and horizontal (shear) forces and calculated A-P centre of foot pressure were collected on an Amiga computer with a 12 bit analog to digital converter . The sample frequency was 200 Hz.

4.6.2. VOR AND OKN TESTING

Standard DC electronystagmography (EOG) methods were used according to the methods of Barber and Stockwell (1976). A surface electrode (silver/silverchloride, nonpolarising) was placed at the lateral canthus of each eye, and one (reference electrode) on the subject's forehead. These electrodes were used to record horizontal eye displacement during angular chair rotation and during optokinetic stimulation. An Amiga 2000 computer equipped with a 12-bit analog-to-digital converter was used to record and store eye movements obtained from the surface EOG electrodes, chair angular velocity, and a signal indicating timing of lights on and lights off. The sampling rate was 200 Hz.

Subjects were positioned and firmly secured in a rotating chair (see

Figure 3). The head was fixed in the midline position looking straight forward head tilted approximately 20^0 forward. They were subjected to the following two-step chair rotations in a clockwise (CW) and counterclockwise (CC) directions:

1a)Target angular velocity of 60°/sec. acceleration period to constant target velocity was four seconds (per-rotatory VOR) acceleration of $15^0/s^2$, and deceleration period to zero velocity was two seconds (postrotatory VOR) acceleration of $30°/s^2$;

1b) Target angular velocity of 120°/sec.-acceleration period to constant target velocity was four seconds (per-rotatory VOR), and deceleration period to zero was two seconds (postrotatory VOR).

2) The patients were also subjected to a 60-second period of optokinetic stimulation in the CW and CC directions. This test was integrated with the above described 60°/sec. chair rotation test to minimize total testing times. After the subject had been rotating in the dark at constant velocity for 60 seconds, the lights were turned on and the subject viewed a white circular surround containing full-field black vertical stripes at intervals of 18^0. The lights-on period of 60 seconds was followed by a 60 second period of constant velocity rotation in the dark before the chair was decelerated to a stop. Figure 4 shows the timing of the chair acceleration / deceleration, constant velocity period, and lights on/off.

An eye displacement calibration test was performed before each chair rotation.

Figure 3. **Illustrates the apparatus used in the rotatory chair test. This diagram also demonstrates the subjects positioning in the chair, the placement of electrodes and the striped drum wall used for optokinetic stimulation in testing the VOR gain, OKN gain and VOR time constant.**

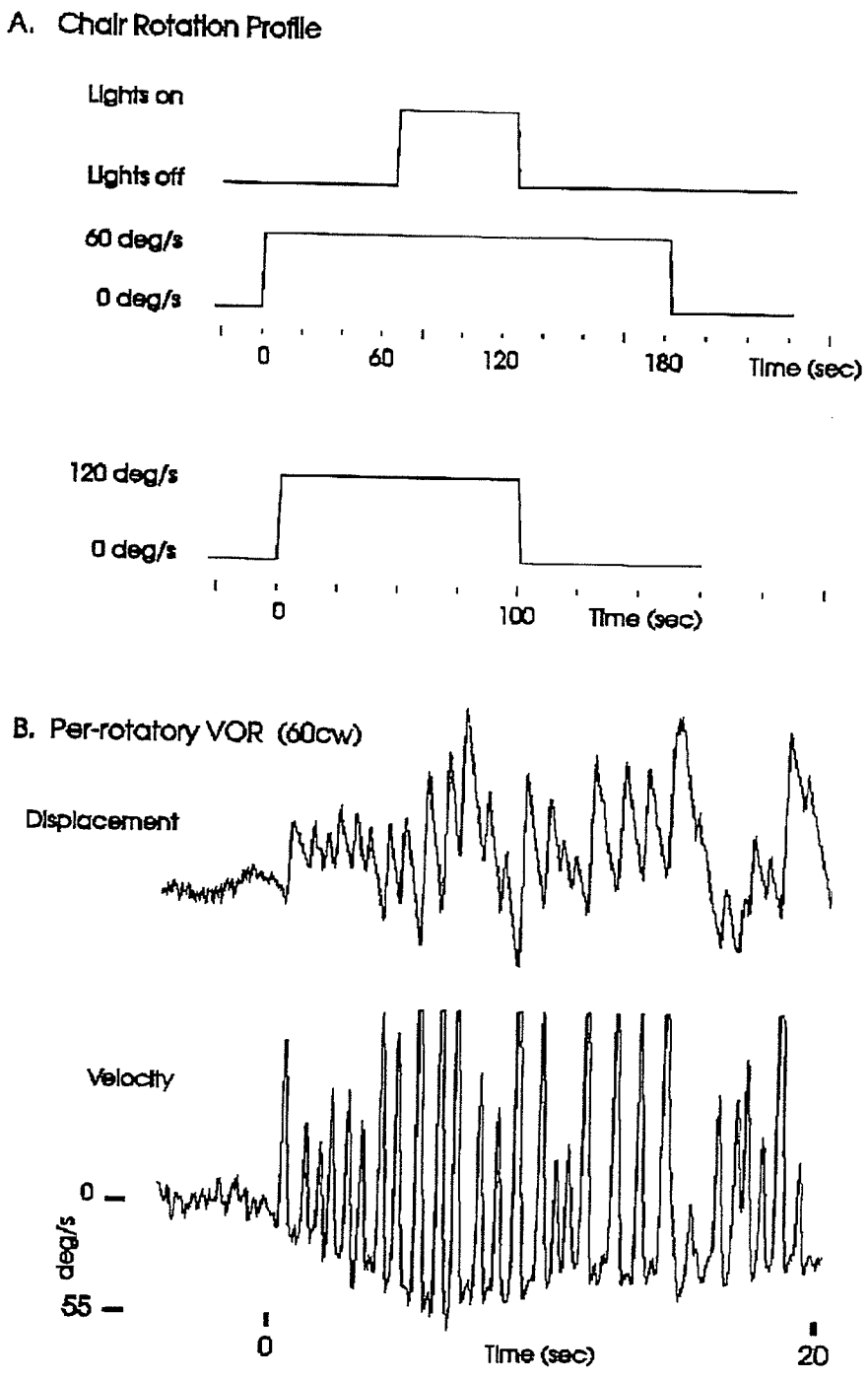

Figure 4. **(A) Timing of chair acceleration/deceleration, constant velocity period, and lights on/off for 60°/sec. and 120°/sec. step chair rotation tests. (B) EOG records of eye displacement (above), and respective trace of eye velocity (below) for one subject in response to 60°/sec. CW chair rotations.**

4.7. DATA ANALYSIS

4.7.1. EQUITEST

For each trial, two measures of balance performance were provided by the Equitest system:

1) an equilibrium score (ES) and

2) a strategy score (STS).

The ES represents the peak maximum to peak minimum displacement of A-P centre of vertical force (COVF) position over the twenty second test period. An ES score of 0% indicated a fall, and a score of 100% indicated no fall. The STS was calculated as the peak maximum to peak minimum shear force. In addition, the A-P COVF position and shear force (sampled at 200 Hz, low-pass filtered 20 Hz) were rectified and integrated over the 20-second test period to determine the total A-P COVF displacement and the total shear force. The average ES, average STS, average total COVF displacement, and the average total shear force of the three trials at each test condition were calculated. This procedure was repeated from TD 1 to TD 4.

4.7.2. FALLS

If the subject fell during any of the Equitest conditions this was scored as zero and counted for each of the Equitest 1 through 4. The total number of falls for each group over the four test days was tallied for each test condition Equitest 1, 2, 3, and 4.

4.7.3. VOR AND OKN

All EOG signals were analyzed on an IBM compatible computer. The raw EOG signals were low pass filtered at 8 Hz, using a fourth order digital zero-phase lag filter, and then differentiated with respect to time, to obtain eye velocity. Calibration was done immediately before each chair rotation.

For each identifiable nystagmus beat, the peak eye velocity of the slow phase component (PSPV) was determined. To avoid the relatively high velocities in the transitional periods between fast phase and slow phase, the first 40-80 ms and the last 40-80 ms of the slow-phase components of nystagmus were excluded in the determination of PSPV. Figure 4 presents typical raw EOG records of eye displacement, and below the respective trace of eye velocity for one subject in response to 60°/sec. chair rotation (per-rotatory VOR).

The eye velocity of the slow phase component of vestibular nystagmus reached its maximum value between five to eight seconds after onset of chair rotation or of chair deceleration, and then declined over time. At the point where slow- phase velocity reached its maximum value, the three greatest PSPV values were averaged and divided by the chair angular velocity (60° or 120°/sec.) to obtain the VOR gain. From the time of maximum PSPV, the VOR time constant was calculated using a single exponential regression model;

$Y = A \exp (- X/B)$ where Y is SPV,

X is the time of PSPV, and

1/B is the time constant in seconds.

The coefficient of determination (R^2) for each exponential regression was also calculated.

The OKN gain was determined in a similar fashion to VOR gain. After the

slow- phase velocity of OKN reached its maximum value (typically within five seconds after lights on), the three greatest PSPV values were averaged and divided by the chair angular velocity of 60°/sec. to obtain the OKN gain.

The symmetry scores for the VOR gain, VOR time constant and OKN gain were calculated using the following formula:

$$\frac{\text{(L response - R response)}}{\text{(L response + R response}} \times 100$$

4.8. STATISTICAL ANALYSIS

To determine whether there were significant changes that had occurred between the different groups (Home, Reh, A and B groups) over the four test periods (TD 1 to TD 4) in; a) balance performance, b) VOR gain and time constant symmetry scores, and c) OKN gain symmetry scores, a repeated measure ANOVA was performed on the following outcome measures:

1) ES, STS, total A-P COVF displacement, total shear force, and number of falls.

2) Per-rotatory and Post-rotatory VOR gain symmetry scores and VOR time constant symmetry scores.

3) OKN gain symmetry scores.

These statistical tests were done for each of the four groups (Home, Reh, A and B).

CHAPTER 5- RESULTS

5.1. OVERVIEW OF CHARACTERISTICS OF SUBJECT GROUPS

Table 2 (page 90) presents patient age, sex, clinical diagnosis, length of time in which subject experienced symptoms (history), caloric test results, per-rotatory VOR gains and time constants (TC) to 60°/ sec. rotations, and OKN gains.

The clinical diagnosis did vary somewhat between the Reh and the Home groups. The main diagnosis in the Reh group was predominantly Neuritis (seven out of the eleven subjects). The main diagnosis in the Home group was divided between Neuritis and Trauma, with four cases of Neuritis and four cases of Trauma out of the twelve subjects.

The distribution of males and females was quite even between the Reh and Home groups, (there were six males and five females in the Reh group, whereas there were six males and six females in the Home group)

The history for the Reh and Home groups was similar . There was a mean of 5.8 year duration of symptoms in the Reh group and 4 years in the Home group.

Caloric tests results were similar between the groups. In the Reh group eight subjects had positive asymmetry scores (difference greater than 25%), left/right, two subjects had reduced response bilaterally (mean SPV less than 5°/sec), and one subject had a negative caloric test. In the Home group nine subjects had positive asymmetry scores, one subject had reduced response bilaterally, and three subjects had a negative caloric test.

Low VOR gains were found in four subjects in the Reh group (NB, RD, AJ, and SS) and in three subjects if the Home group (JB, JP, and NW). One subject in the Home group (AS) had no VOR response to CW or CC chair rotations. The OKN gain values were within normal limits of unity gain for all subjects, except subject (NW) in the Home group.

Regarding outcome measures of: 1) balance performance - ES, STS, total A-P COVF displacement, and total shear force; 2) VOR gain and time constant symmetry scores; and (3) OKN gain symmetry scores, there were no significant differences between the Reh and Home groups on TD1 (*t* test).

5.2. GENERAL OBSERVATIONS

As to progression of patients in the Reh program, the first five to eight weeks were spent on the Reh program of exercises (Section 1 and 2) described in detail in the Methods section, and the last four to seven weeks focused on the Reh program of exercises (Section 3). The Reh subjects experienced moderate to strong sensations of dizziness, nausea, and/or disorientation at the beginning of the exercise program. These sensations subsided within two to five weeks of training. However, when exposed to optokinetic stimulation (OKS) used in Section 3, moderate to strong sensations of dizziness, nausea and/or disorientation returned. The subjects tended to sway in the direction of the moving light stripes and reported difficultly in righting themselves. It took another 4 to 6 training sessions for these sensations to subside.

Before training, with eyes closed or while viewing the OKS, the subjects were unable to maintain balance while standing on the sponge pads or narrow beam and were unable to walk on the narrow beam without falling . After

training, eight of the eleven subjects in the Reh and six of the seven patients in the B group progressed to a level where, with eyes closed or when viewing the OKS, they were able to maintain balance indefinitely while standing on the sponge pads of varying densities or narrow beam and were able to walk on the narrow beam. These patients could balance on the sponges and narrow beam while moving their heads at different speeds and amplitudes of movement. Three subjects (NC, SC, and JP), with eyes closed or while viewing the OKS, could only maintain balance for periods of one minute on the sponge pads and narrow beam without losing their balance. Subject AS (bilateral involvement) could only maintain balance on the sponge pads and narrow beam for 20-30 seconds with eyes closed or while viewing the OKS. Although this would appear to be minimal progress, AS was unable to maintain her balance on the firm floor surface with eyes closed prior to the training program.

5.3. BALANCE PERFORMANCE

5.3.1. FALLS

The numbesr of falls registered on Eqt-3 and Eqt-4 are presented in Figure 5, there were no falls recorded in Eqt-1 or Eqt-2 therefore no figures are shown for these tests.. From visual inspection there was a substantial decrease in the total number of falls over the source of training program for both the Reh and the B group.

While for the Home group and the A group (subjects in Home group who did not go on to the Reh training), there was little change in the total number of falls over the four test periods (TD 1 to TD 4).

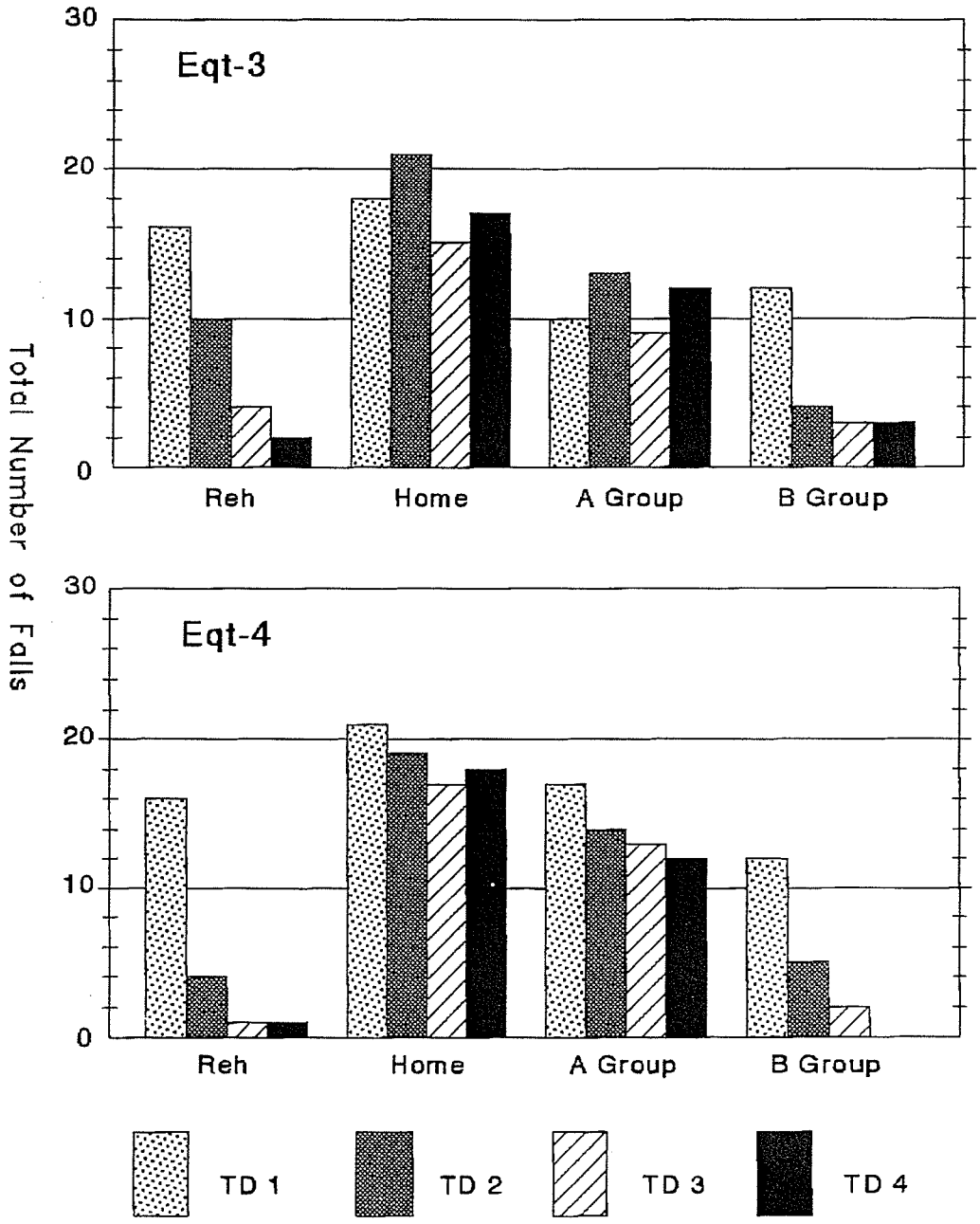

Figure 5. **Total number of falls during the Eqt-3 and Eqt-4 test conditions over the four test periods for the Reh group, the Home group, the A group and the B group.**

5.3.2. A-P CENTRE-OF-MASS DISPLACEMENT

Figure 6 presents records of A-P centre-of-mass displacement and shear force of one representative subject from the Reh group and one of the subjects who received the home program of exercises and then went on to receive the Reh exercise program (representing H group, A group and then B group) on the first test day (TD1) and final or follow up test day (TD4). These records demonstrate the typical changes observed in balance performance before and after the respective training programs. In Figure 6, the magnitude of the displacement curve in Eqt-3 and Eqt-4 for the Reh subject is noticeably reduced on TD4 as compared to TD1, indicating that the subject's ability to maintain balance under the same test conditions had improved. The same effect is noted for the subject in the B group, there is very little evidence of displacement in the waveform on TD4 as compared to TD1. When looking at the shear force readings in Figure 6 the same results are noted. The movements for the Reh subject are greatly decreased when comparing the results of TD4 to TD1. The same findings are noted in the waveform of the shear forces for the subject in the B group when comparing the results of TD4 and TD1.

The group means and standard errors of mean (SEM) for the ES, STS and A-P COVF displacement scores over the four test days are presented in the Figures 7A-9B. A significant improvement in all outcomes measures during the Eqt-3 and Eqt-4 test conditions was observed for the Reh and B groups.

Regarding the Eqt-1 and Eqt-2, statistically significant improvements were only observed for the ES during the Eqt-2 in the Reh group ($p < 0.05$) and for

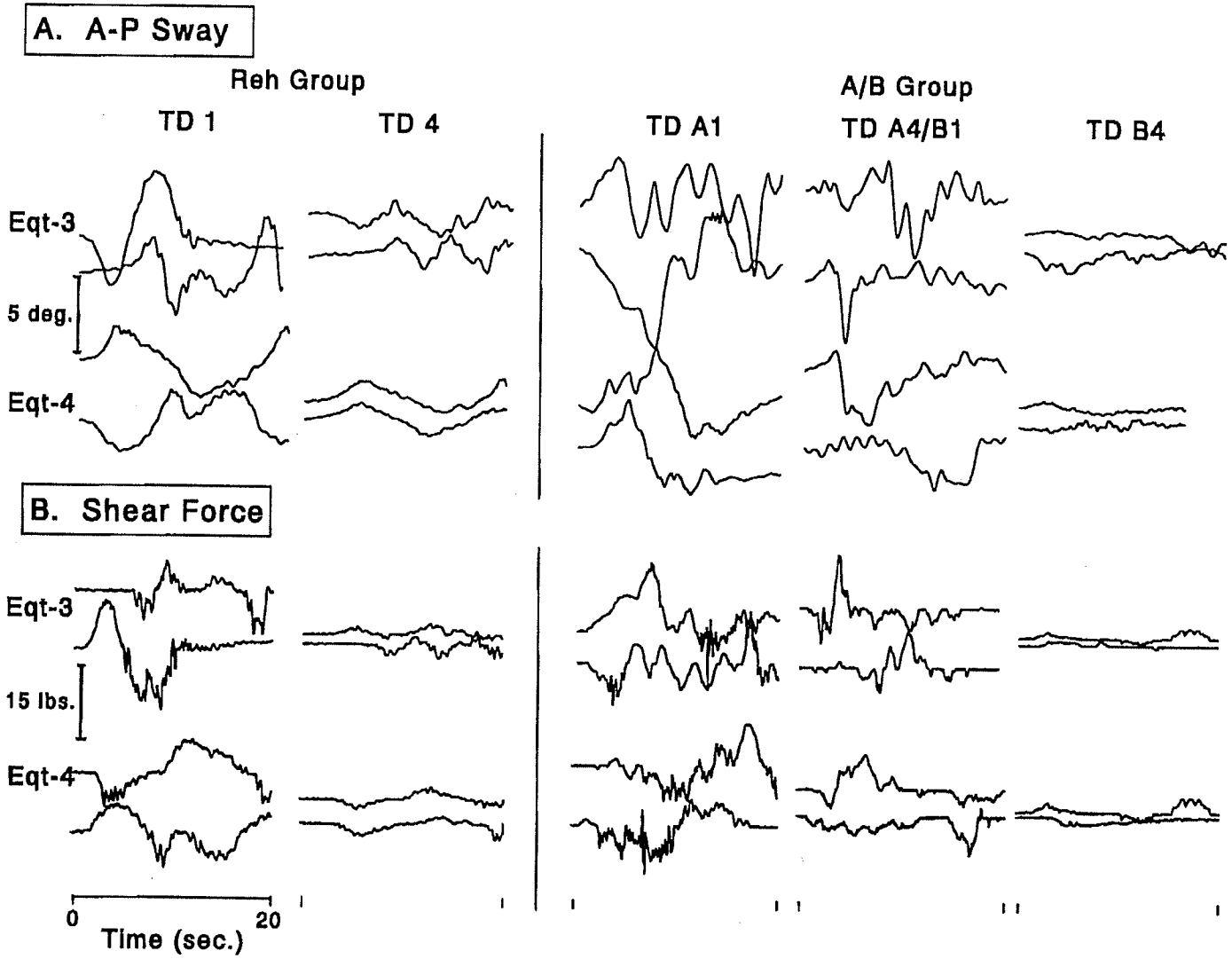

Figure 6. **Records of A-P centre-of-mass sway (A) and shear force (B) for one subject from the Reh group and one subject from the B group, during Eqt-3 and Egt-4 test conditions. Note that middle row of traces for subject FW, labeled "TD A4/B1," represents TD4 as A group and TD1 as B group. The first trial (top trace) and third trial (bottom trace) of each test are presented.**

STS during Eqt-2 in the B group ($p < 0.05$). However, as seen in Figures 7A-8B, consistent trends towards improvement in the ES and STS during Eqt-1 and Eqt-2 were evident for the other cases. In contrast, for the Home group and A group, the results showed no significant change in any of the outcome measures over the four test periods and no trend towards improvement in ES and STS scores over the four test periods (TD 1 to TD 4).

In general, the improvement in ES, STS and total displacement scores of the Reh group and the B group occurred mainly between TD 1 and TD 2, within the first 6 weeks of training (see figures 7A-9B). There was a slight improvement in the performance measures between TD 2 and TD 4, but the differences were not significant. However, it should be noted that the significant improvement in ES and STS scores achieved in the first six to twelve weeks of treatment was maintained at TD 4, five months after the end of the twelve week training period.

Normative data for ES are presented in Figure 7A and 7B as the crossed hatched bar outside and adjacent to each panel. The height of the cross-hatched bar represents the 5th percentile of the distribution of ES obtained from a sample of 110 healthy adult subjects aged 20-59 years old. This data was provided by Neurocom Inc. Equilibrium scores below the 5th percentile are considered as a positive sign of vestibular dysfunction (Lacour et al 1976; Lacour and Xerri 1981).

At TD 1, the mean ES for each group was below the 5th percentile level. By TD 2 or TD 3, the mean ES for the Reh group and the A-B group were above the 5th percentile level. The mean ES for the Home group and the A group were below the 5th percentile level at all four test periods and for two subjects (JW in the Reh group and AS in the A-B group). Although improvements in ES were

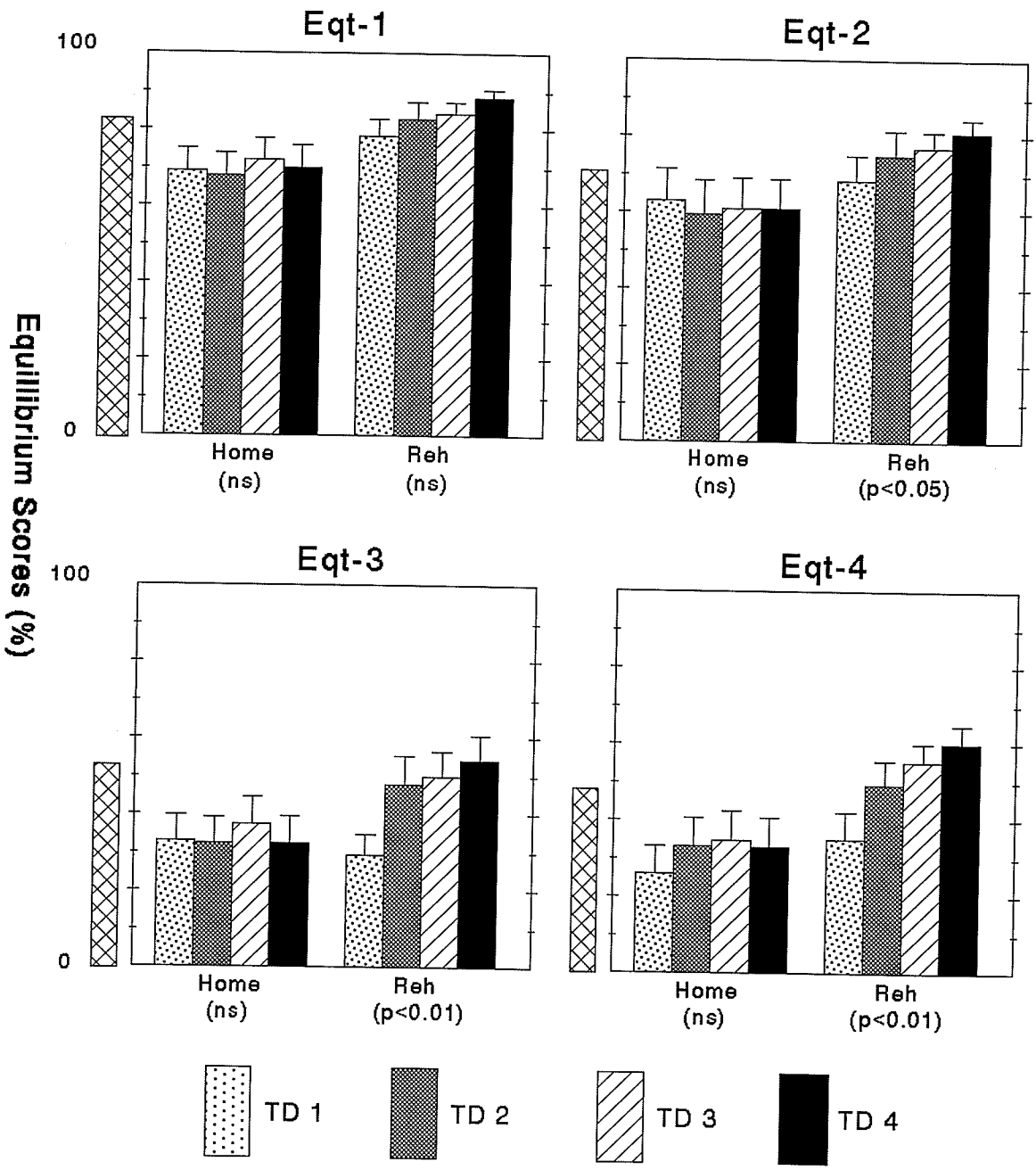

Figure 7A. **Group means and SEM of ES over the four test periodsfor Home and Reh groups. For each test condition (Eqt-1, Eqt-2, Eqt-3, Eqt-4) and group (Home and Reh), results of statistical analysis (*p* values) are presented, below, in parentheses (ns=no significance). The height of the hatched bars outside and adjacent to each panel represents the 5th percentile level of the distribution of ES obtained from a sample of healthy adult subjects (see results for details).**

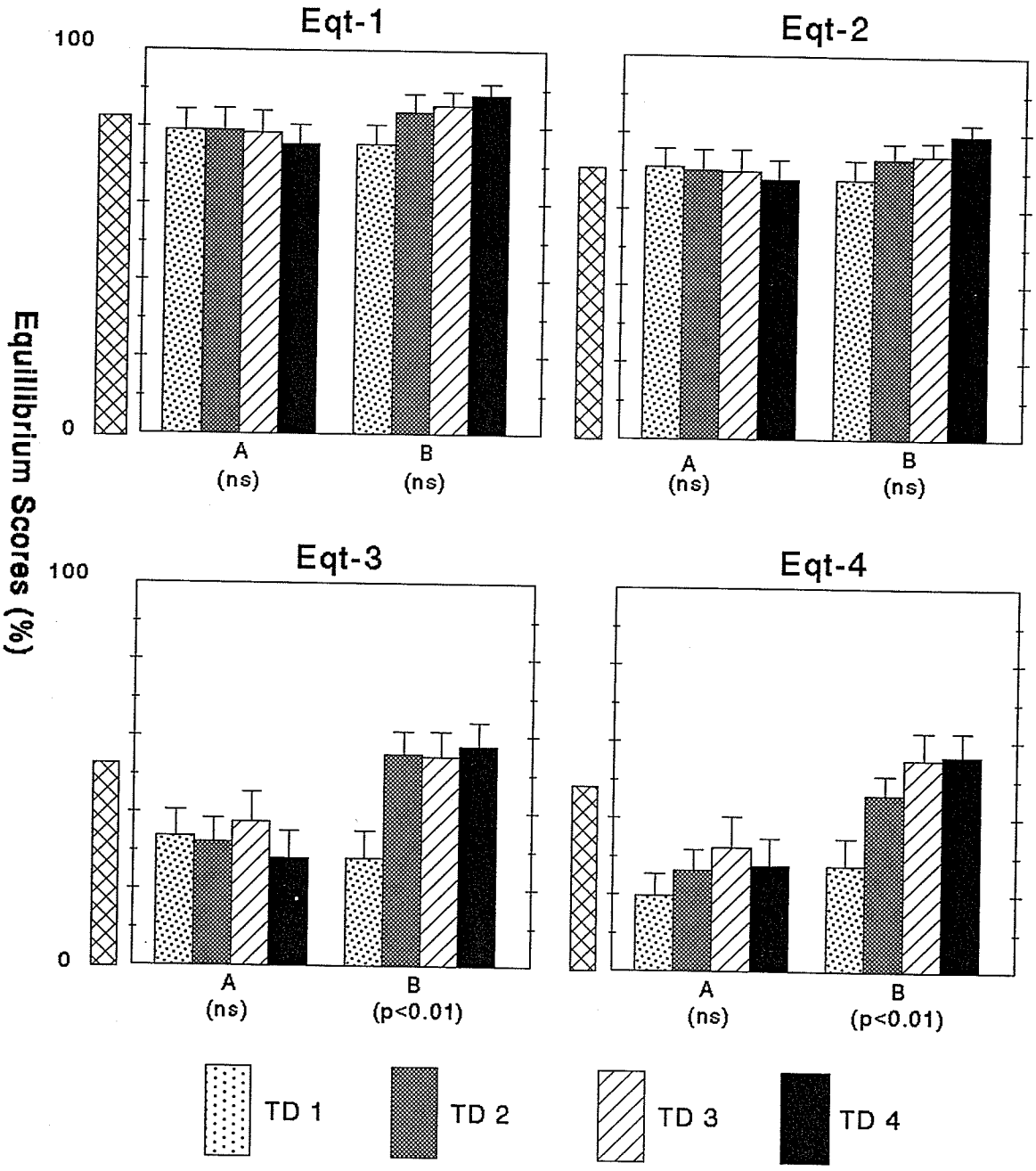

Figure 7B. **Group means and SEM of ES over the four test periods for groups A and B. For each test condition (Eqt-1, Eqt-2, Eqt-3,Eqt-4) and group (A and B), results of statistical analysis (*p* values) are presented, below, in parentheses (ns = no significance). The height of the hatched bars outside and adjacent to each panel represents the 5th percentile level of the distribution of ES obtained from a sample of healthy adult subjects (see Results for details).**

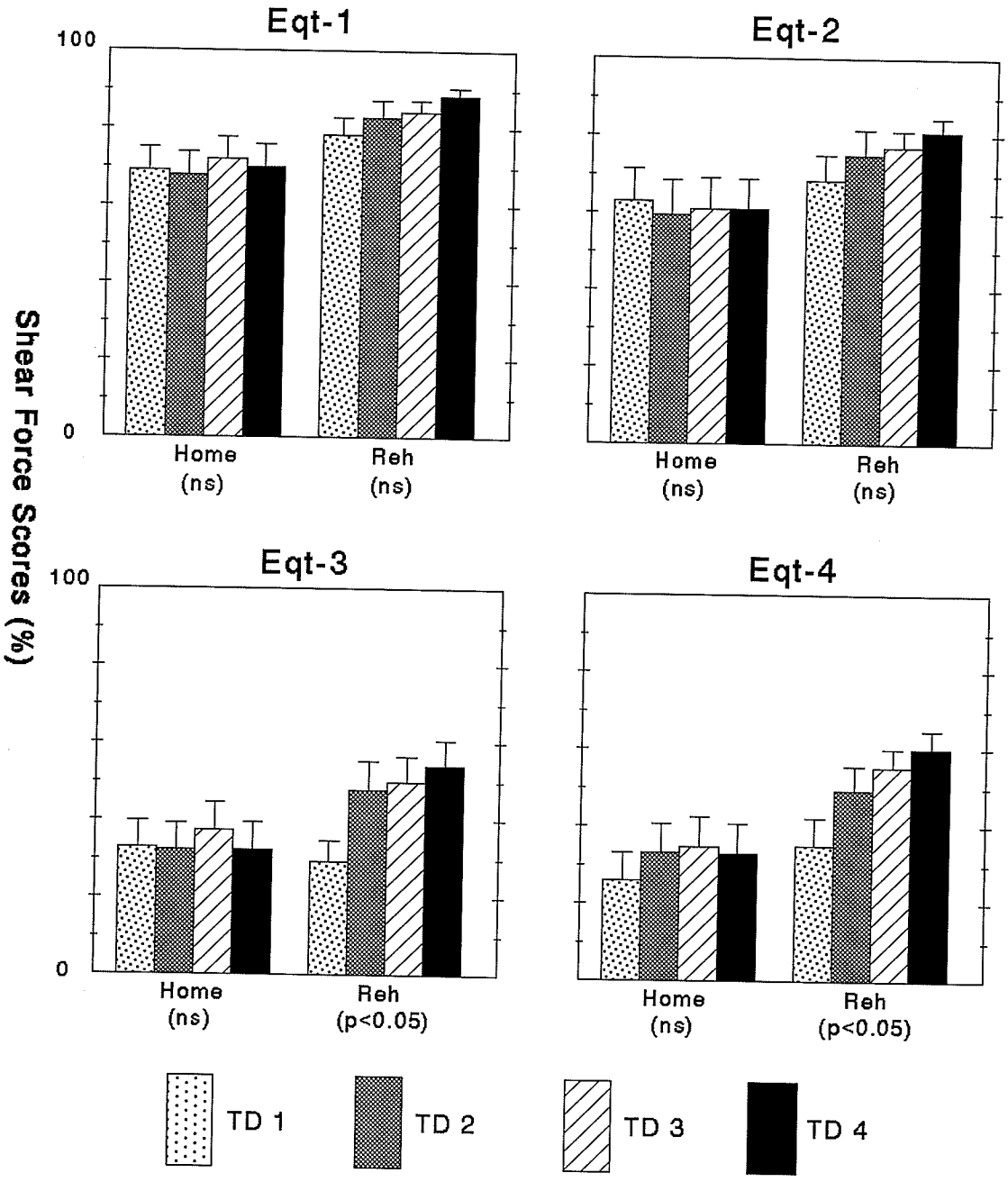

Figure 8 A. **Group means and SEM of STS over the four test periods for the Home and Reh groups. For each test condition (Eqt-1, Eqt-2, Eqt-3, Eqt-4) and group (Home and Reh), results of statistical analysis (*p* values) are presented, below, in parentheses (ns=no significance).**

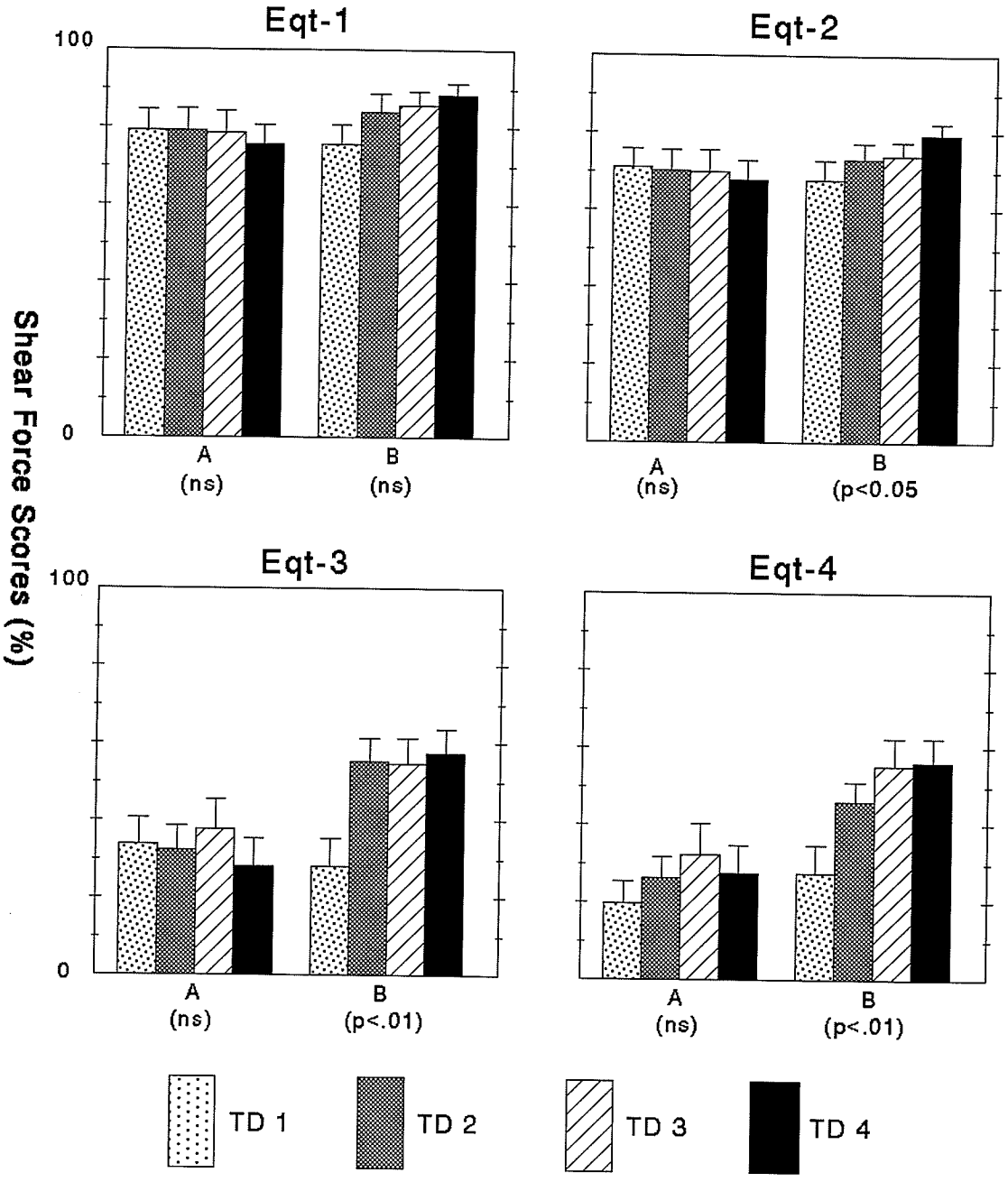

Figure 8 B. **Group means and SEM of STS over the four test periods for the A and B groups. For each test condition (Eqt-1, Eqt-2, Eqt-3, Eqt-4) and group (A and B), results of statistical analysis (*p* values) are presented, below, in parentheses (ns = no significance).**

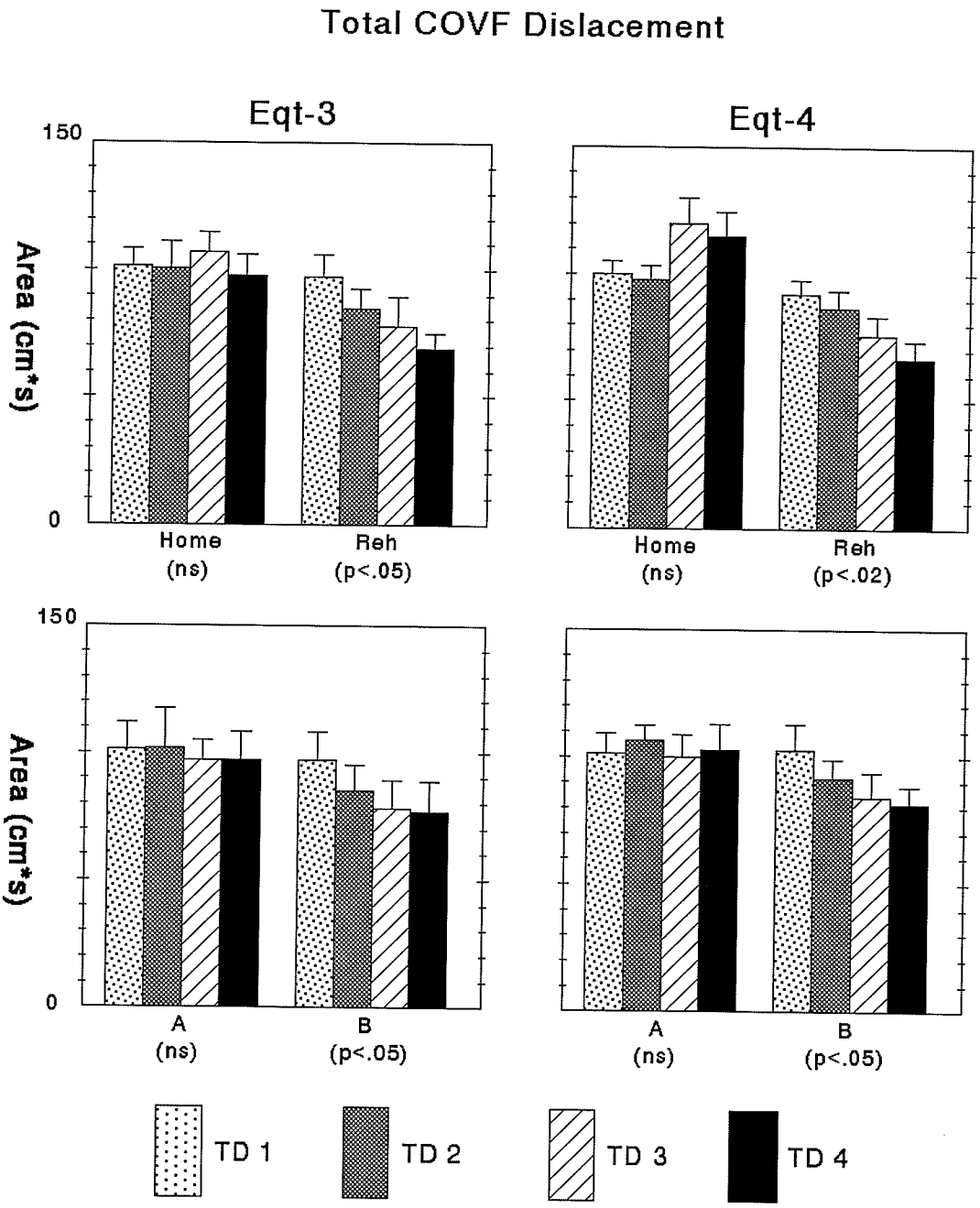

Figure 9 A. **Group means and SEM of total COVF displacement over the four test periods for all four groups. For each test condition (Eqt-3 and Eqt-4) and group (Home, Reh, A and B), results of statistical analysis (*p* values) are presented, below, in parentheses (ns = no significance). Y = axis scale for total COVF displacement are in cm*s.**

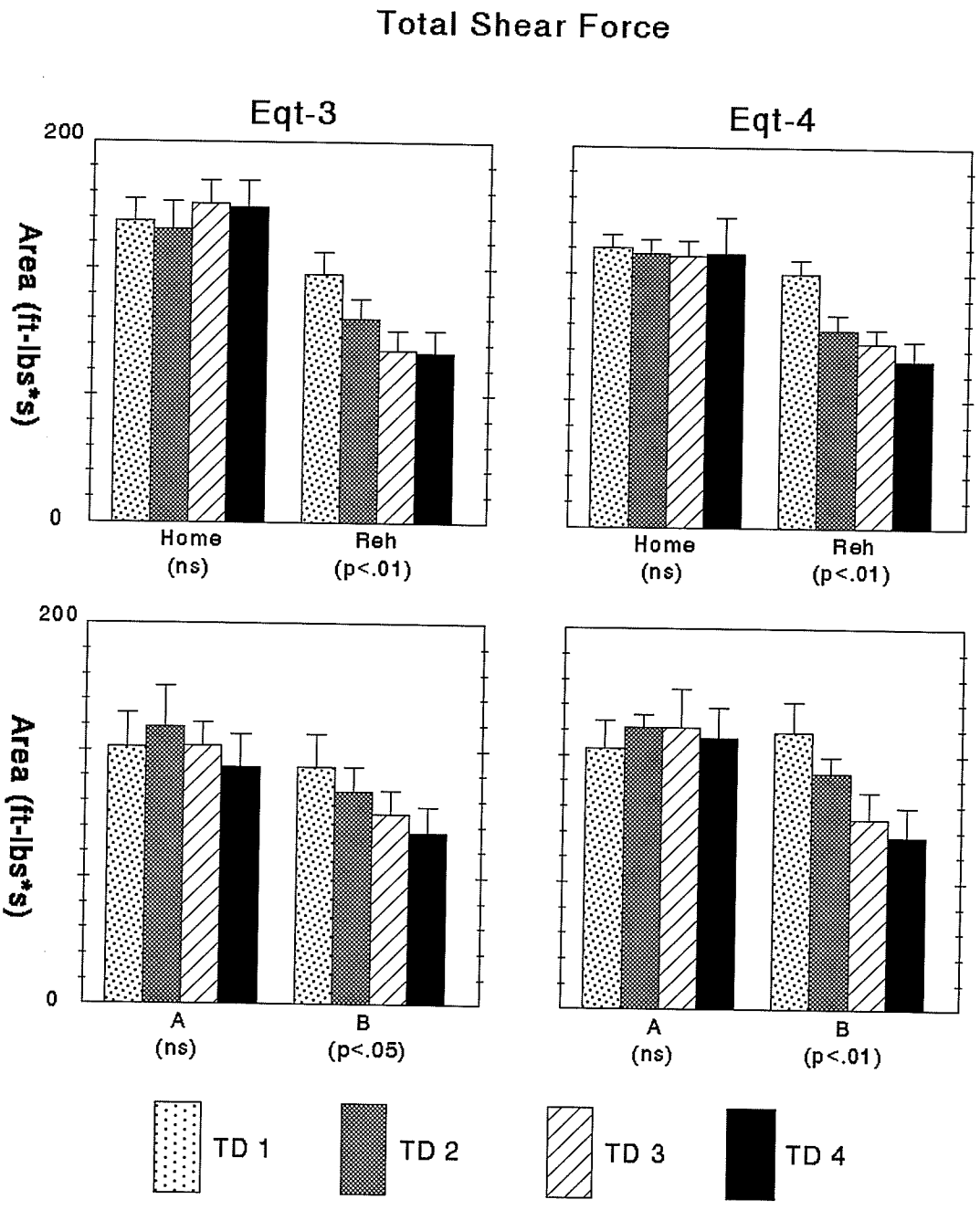

Figure 9 B. **Group means and SEM of the total shear force over the four test periods for all four groups. For each test condition (Eqt-3 and Eqt-4) and group (Home, Reh, A and B), results of statistical analysis (*p* values) are presented, below, in parentheses (ns = no significance). Y = axis scale for total shear force are in ft-lbs*s.**

observed with training (Reh program), ES during Eqt-3 and Eqt-4 were still below the normal range (5th percentile level).

5.4. VOR and OKN

Subjects with no VOR response (AS) at either 60°/sec. or 120°/sec. chair rotations, as well as the two subjects in the Home group with a clinical diagnosis of benign paroxysmal vertigo (BPPV) were not included in the VOR and OKN analysis.

5.4.1. VOR

Figure 10 presents scatter plots of SPV versus time and exponential regression curves of one representative subject from the Reh group and one of the subjects who received the Home program of exercises and then received the Reh program of exercises (A group and B group) on the first test day and final or follow up test day. These results demonstrate the typical changes observed in VOR before and after the respective training programs. The R^2 values obtained from the exponential regression of SPV versus time were in the order of 0.8. A comparison of the R^2 values between groups (*t* Test) revealed no significant difference. The average R^2 value (SEM) for the Reh group was 0.81 (0.08), and for the Home and A group 0.79 (0.11).

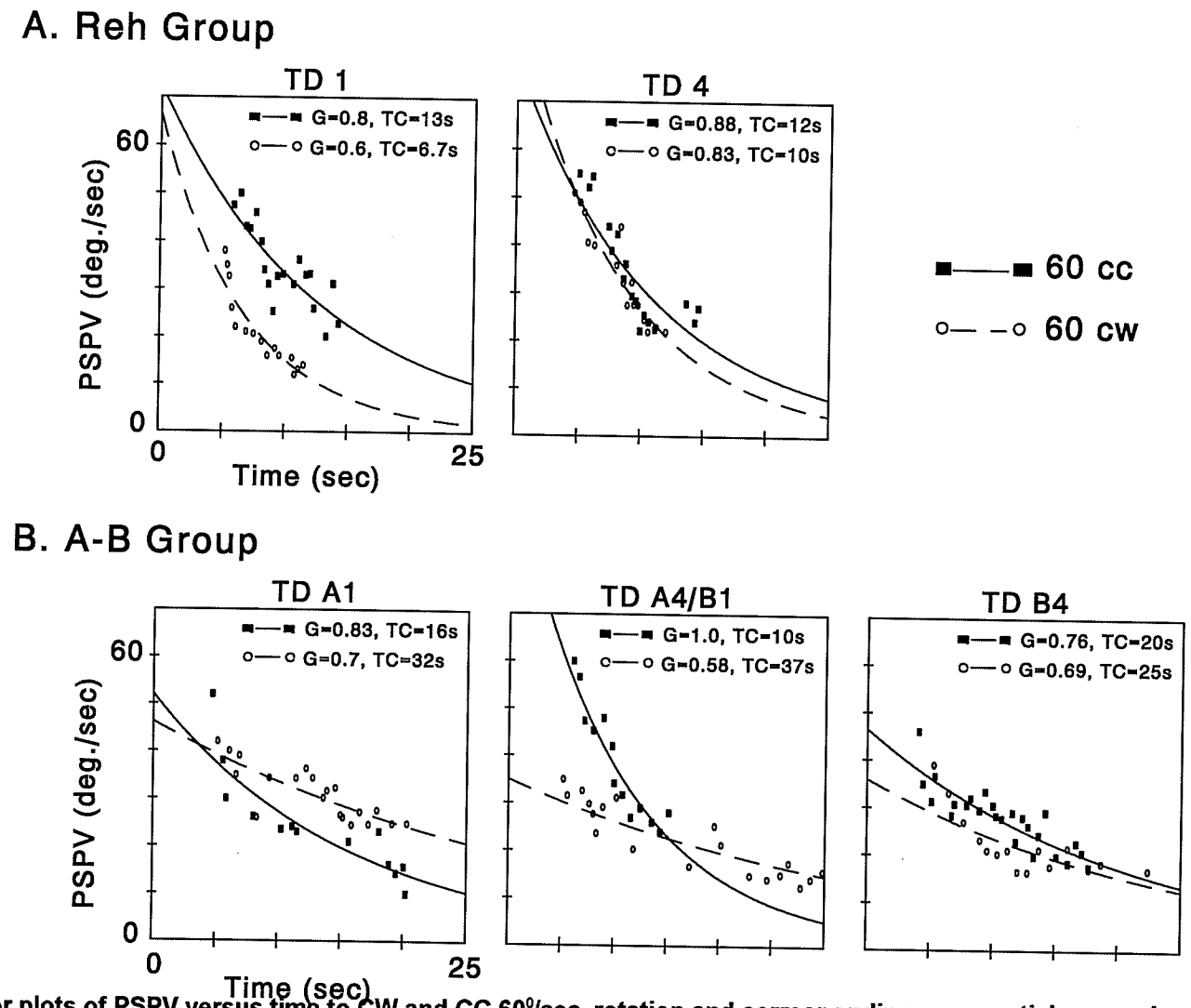

Figure 10. **Scatter plots of PSPV versus time to CW and CC 60°/sec. rotation and corresponding exponential regression curves for one subject from the Reh group, and one subject who completed the Home program (A group) and then the Reh program (B group). Note that middle panel for subject PM, labeled "TDA4/B1," represents TD 4 as A group and TD 1 as B group. VOR gain (G) and time constant (TC) are presented in each panel.**

5.4.2. VOR GAIN AT 60^0/SEC. CHAIR ROTATION

Group means of VOR gain symmetry scores and SEM, to 60°/sec. chair rotations for the four test days are presented in Figure 11A . The results of the repeated measures ANOVA revealed significant changes in the VOR gain symmetry over the four test periods in the Reh and B groups but not in the Home or A groups.

The probability values for the VOR gain symmetry scores are presented in Figure 11A and 11B for each group.

In the case of the Reh group and the B group, the magnitude of the VOR gain symmetry scores decreased over the 12-week training program, and the decrease was maintained at the five month follow-up test.

The mean per-rotatory VOR gain symmetry scores for: (a) the Reh group decreased from the initial value of 17% on TD 1 to 9.9% on TD 4, (b) the B group, 23% on TD 1 to 17 % on TD 4.

5.4.3. VOR TIME CONSTANT

The group mean and SEM for VOR time constant (TC) symmetry scores to 60°/sec. chair rotations are presented in Figure 11B.

The results of repeated measures ANOVA revealed no significant changes for any of the groups over the four test days. The probability values for the VOR time constant scores are presented in Figure 11B for each group.

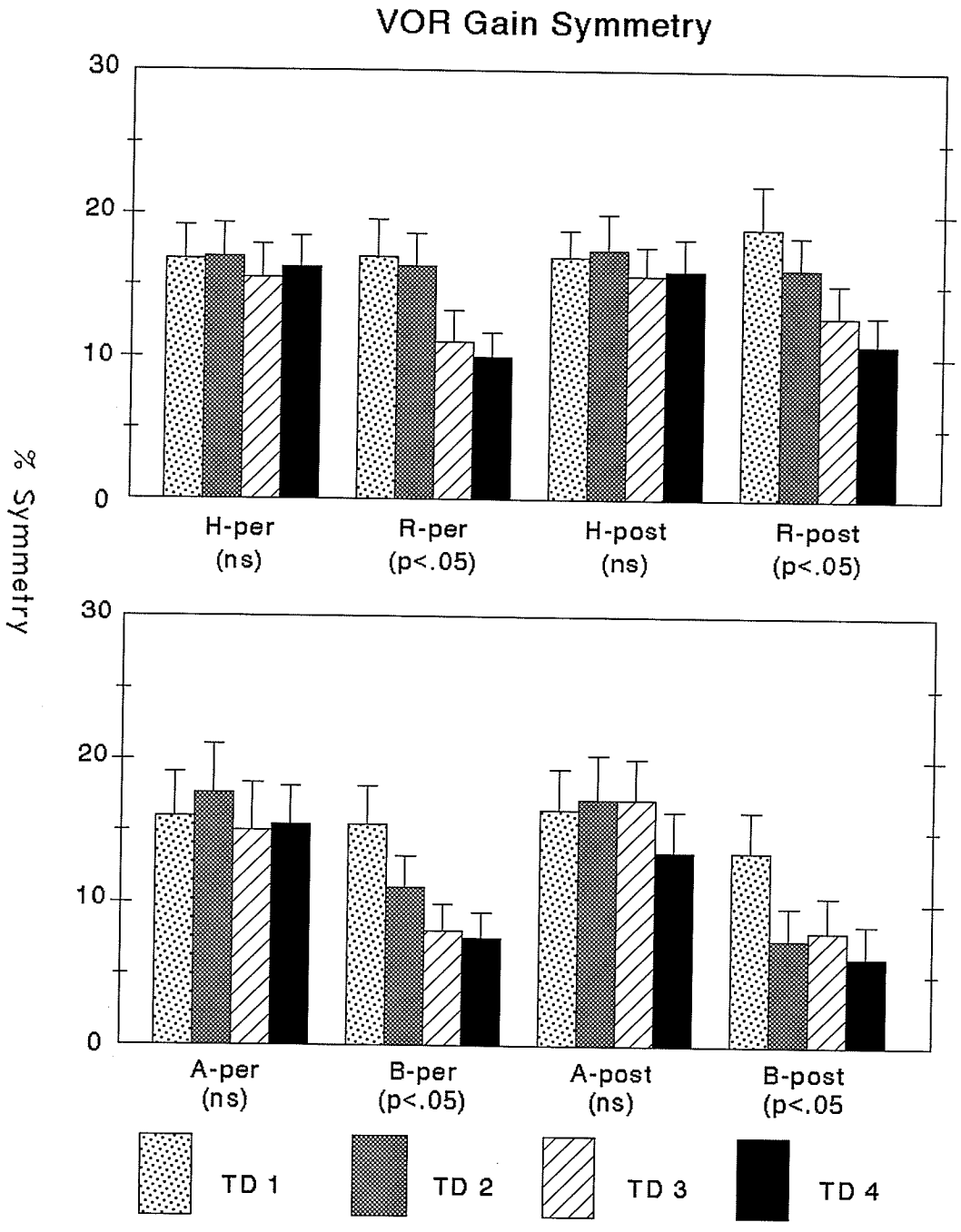

Figure 11 A. **Group means and SEM of VOR gain symmetry scores to 60^0/sec. chair rotation over the four test periods. For each group - Home group (H), Reh group (R), A group (A) and B group (B), and for per-rotatory (per) and postrotatory (post) responses - results of statistical analysis (*p* values) are presented, below, in parentheses (ns = no significance).**

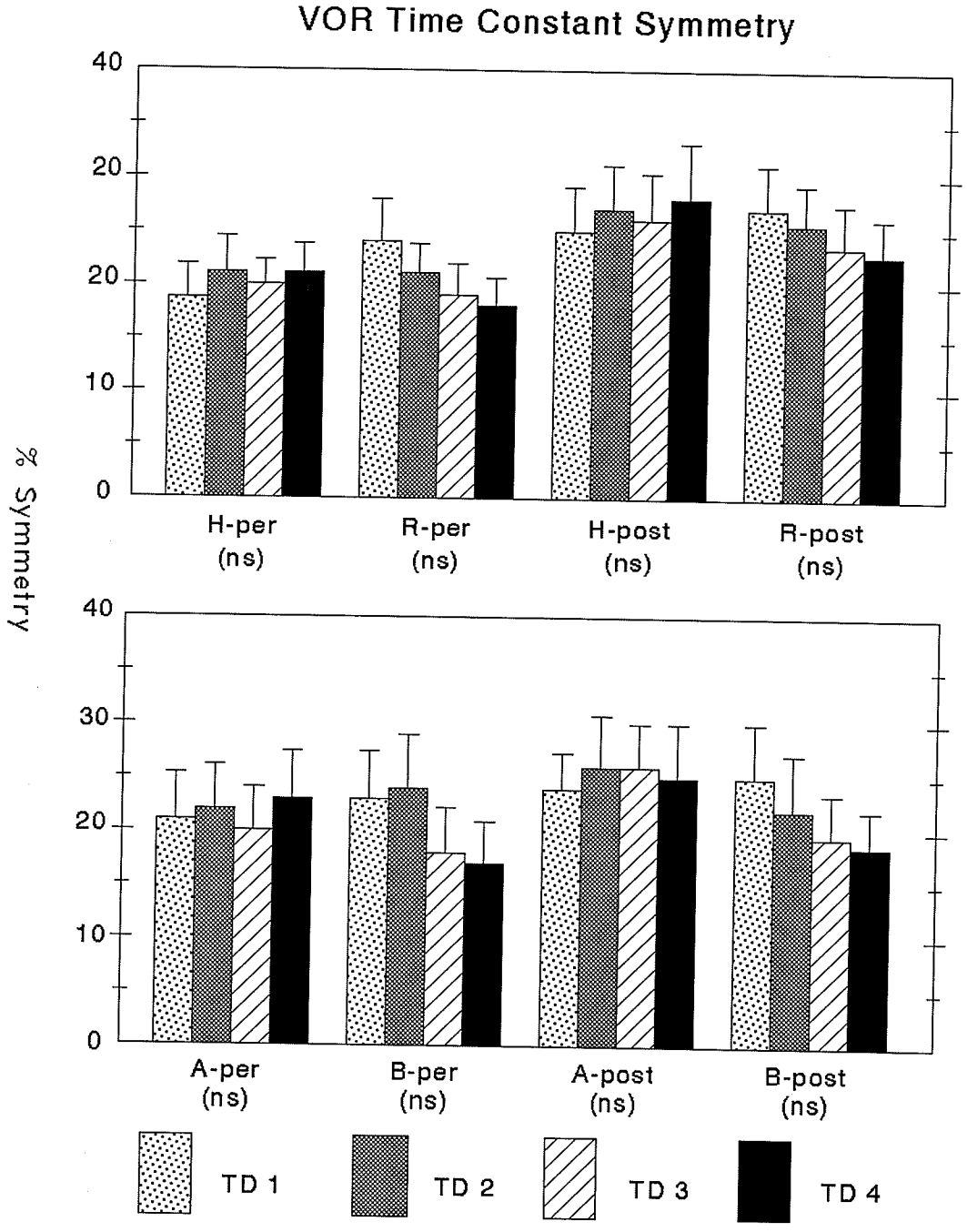

Figure 11 B. **Group means and SEM of VOR time constant symmetry scores to 60°/sec. chair rotation over four test periods. For each group - Home group (H), Reh group (R), A group (A) and B group (B), and for per-rotatory (per) and postrotatory (post) responses - results of statistical analysis (*p* values) are presented, below, in parentheses (ns = no significance).**

Although there was no significant difference observed for any of the groups there was a trend toward a decrease in postrotatory VOR time constant symmetry scores in the Reh group and B group which was not noted for the Home group or A group.

5.4.4. RESULTS AT 120°/SEC. CHAIR ROTATION

For the chair rotation test at 120°/sec., four subjects in the Reh group and two subjects in the Home group (who were in the A-B grouping) became extremely nauseated and asked to stop the test. Since both CW and CC chair rotations at 120°/sec. could not be completed on TD 1, no further attempt was made to test these subjects at 120°/sec. on subsequent test days. Thus, in addition to exclusion of the two subjects diagnosed with BPPV and the subject with no VOR response, this reduced the sample size to seven in the Reh and Home groups, and four in the A and B groups. Statistical analysis of the data to 120°/sec. rotation revealed no significant change in the VOR gain symmetry scores of VOR time constant symmetry scores over the four test periods in either the Reh group or Home group. However, the mean VOR gain symmetry scores and mean VOR time constant symmetry of the Reh group did decrease over the four test periods as observed for the 60°/sec. chair rotations. The mean per-rotatory VOR symmetry scores for the Reh group decreased from the initial value of 15% on TD 1 to 8.5% on TD 4. The mean per-rotatory VOR time constant symmetry scores for the Reh group decreased from the initial value of 21% on TD 1 to 16% on TD 4. The mean VOR gain symmetry scores and mean VOR time constant symmetry of the Home group did not decrease over the four test periods. Given the small sample size in the A and B groups, no statistical

analysis was performed.

5.4.5. OKN GAIN

Group means of OKN gain symmetry scores and SEM at 60°/sec. chair rotations, for the four test days are presented in Figure 12.

There were no significant changes in OKN gain symmetry scores in any group over the four test periods (TD 1 - TD 4). This is not an unexpected finding as the subjects original OKN scores were close to symmetry.

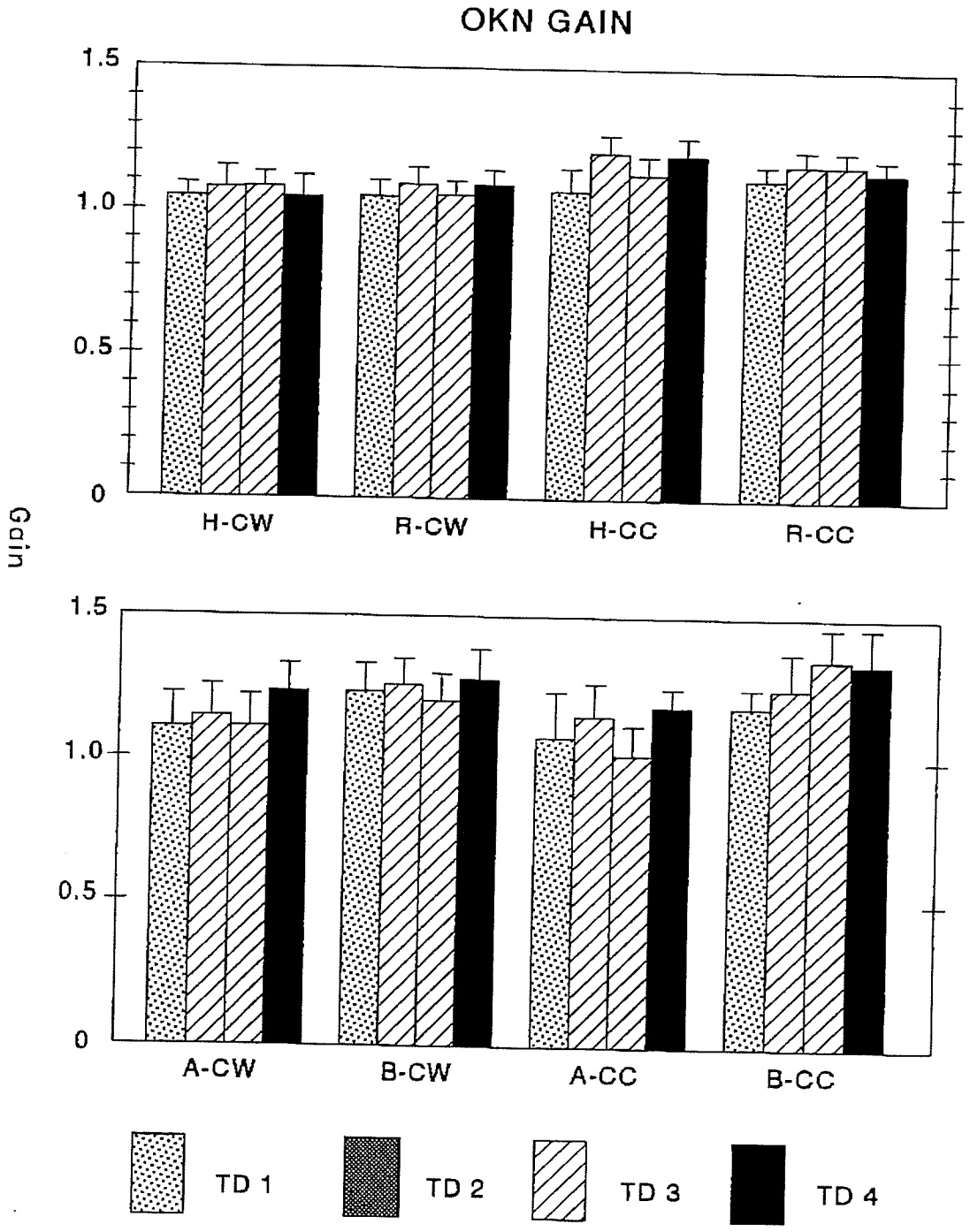

Figure 12. **Group means and SEM of OKN gain symmetry scores to 60°/sec. chair rotation over the four test periods. For each group - Home group (H), Reh group (R), A group (A) and B group (B), for clockwise (CW) and counterclockwise (CC) directions.**

CHAPTER 6-DISCUSSION

6.1. MAIN FINDINGS

The main objective of the present study was to differentiate between two exercise programs in the treatment of patients with chronic peripheral vestibular deficits and to assess whether these exercise programs do enhance adaptive modifications of the VOR gain, VOR time constants and OKN gains, and to assess their effects on postural control under conditions of sensory conflict. The main findings were:

(1) Although the diagnosis differed somewhat between Reh and Home groups the clinical picture as evidenced from: a) duration of symptoms, b) caloric test results, c) outcome measures of balance performance - ES, STS, total A-P COVF displacement, and total shear force, d) VOR gain and time constant symmetry scores, and e) OKN gain symmetry scores, did not. In addition the age, distribution of males and females were similar between the groups.

(2) A significant improvement in all outcomes measures related to balance performance during the sensory organization tests Eqt-3 and Eqt-4 was observed for the Reh and B groups who received the rehabilitation program of exercises . There was virtually no change in these outcome measures for the Home and A groups who received the home program of exercises.

(3) After treatment, balance performance levels of Reh and B group reached normal levels.

(4) There was a significant change in VOR gain symmetry scores over the four test periods in the Reh group and the B group but not the Home group or A

group.

(5) There was no significant change in the VOR time constant symmetry score, although a trend towards reduced VOR time constant symmetry scores was observed over the four test periods for the Reh and B groups, as compared to no change in the Home group and A group.

(6) There was no significant change in OKN gain symmetry scores in any group over the four test periods.

6.1.1. POSTURAL CONTROL/BALANCE PERFORMANCE

Concerning postural control, the body makes use of multiple, partially redundant sources of sensory inputs. This combined with a competent central adaptive capability creates an environment conducive to compensation. Therefore, several adaptive mechanisms, inherent to the CNS (Precht et al 1981; Precht and Dieringer 1981; Goldberg and Colby 1989; Goldberg et al 1991) could contribute to compensation for the loss or reduction of peripheral vestibular signals. Sensory substitution, or the ability to utilize other sources of sensory inputs to provide information about body orientation relative to the environment has been proposed (Schaefer et al 1979; Schaefer and Meyer 1981; Igarashi 1984; Herdman 1994; Shumway-Cook and Horak 1990; Shumway-Cook and Woolacott 1985). This sensory substitution assists the recovery of postural stability in patients with balance impairment due to vestibular dysfunction.

Regulation of postural equilibrium in standing is dependent on one's ability to maintain control over the position and motion of the total body centre of mass (TCM) relative to the base of support: the feet. No one sense organ is

solely responsible for detecting the position of TCM relative to the base of support, rather this is a function of the organization and integration of multiple sources of sensory inputs. Knowledge of head orientation relative to the gravity vector (otolith end organs) is also not enough for regulating postural stability unless relative trunk and limb segment positions are known. Conversely, knowledge of how body segments are aligned to each other is not sufficient to ensure balance, and external spatial reference is still needed. Visual inputs and/or cutaneous inputs from the feet (support surface inputs) can provide external information on spatial reference for the body orientation and balance. Visual inputs are not present in darkness, and visual information can be misleading as it does not have a fixed frame of reference. For example, optokinetic stimulation can induce sensations of self motion, body sway and loss of balance (Bles et al 1984; Dichgans et al 1973a; Dichgans and Brandt 1978; Dichgans et al 1973b; Lestienne et al 1977). This was very evident in the subjects involved in the rehabilitation program (Reh and B group) who had dramatic postural responses to OKS.

As observed in this study (Figure 7A - 9B), and in other investigations (Black and Nashner 1984; Black et al 1988; Black et al 1983; Goebel and Paige 1989; Voorhees 1989), the ability to maintain balance is significantly affected or lost in patients with peripheral vestibular deficits. This study shows that balance retraining for vestibular patients with long-standing deficits, can aid in resolving sensory conflict conditions.

The sensory conflict situations created by the Equitest amounts to a non-physiological sensory conflict, whereby the subject receives misleading or functionally inappropriate spatial information. In condition Eqt-4, for example, the platform and visual surround are sway stabilized and therefore provide

misleading visual and somatosensory information.

In fact, the patients in this study were able to gain normal levels of balance performance in sensory organization test condition Eqt-3 and Eqt-4. These patients initially had difficulty maintaining balance in standing with eyes closed, standing on a spongy surface, in dark under exposure to OKS, or standing on a balance beam. Their performance under these conflicting conditions was poor. After rehabilitation training, the subjects were able to maintain balance on sponge pads of various thicknesses, and on a narrow beam with eyes closed or when subjected to optokinetic stimulus (OKS).

Concerning the Home group, there was virtually no improvement in performance scores on the Equitest. Their performance levels remained below normative values.

All but three subjects (SS, JW, and AS) in this study had unilateral deficits. It is not known whether the subjects resolved the sensory conflict conditions and destabilizing visual inputs by making use of support surface inputs or whether the remaining, intact vestibular signals also contributed. However, as evident from the results of subject AS who had a total bilateral loss (otoxicity) of peripheral vestibular function, the ability to utilize somatosensory information in the regulation of postural stability under different conditions has limitations. Although subject AS was able to maintain her balance in the Eqt-3 and Eqt-4 test conditions after training, ES during those test conditions was still below the normal range (5th percentile level). Nevertheless, this subject was able to progress to maintaining balance on the sponge pads and narrow beam with eyes closed or when exposed to OKS, for periods up to 30 seconds. Similar residual deficits in balance performance of monkeys (Igarashi 1984; Igarashi et al 1975; Igarashi et al 1981; Igarashi et al 1979; Igarashi et al 1988), and cats

(Marchand and Amblard 1984), with bilateral labyrinthectomies have also been reported. Takimori et al (1985) reported delayed resolution of balance deficits of 3-6 months in his bilateral deficit patients. Further work in human studies are needed to confirm these preliminary findings in patients with total bilateral loss. In any event, the indications that compensation and positive effects of training are limited and delayed in patients with bilateral vestibular loss should be taken into consideration in setting up individual treatment programs. These patients need extensive education and should be made fully aware of the fact that certain conditions (darkness, slippery surfaces etc.) are potentially dangerous. They should be taught preventative measures to protect them against falls and possible injury.

Subjects were tested using four different sensory tests conditions designed to evaluate balance performance under different sets of sensory conditions described above. The main findings of this study demonstrated a significant improvement in standing balance performance under these conditions, in subjects with chronic peripheral vestibular disorders who received the rehabilitation program, but not the eye-head exercises.

Comparing the results of this study to data from similar programs is not yet possible due to the lack of other published data. The majority of studies have examined the effects of different exercise programs on the alleviation of vertigo. These studies have shown that exercise has a significant effect on reducing the symptom of vertigo in subjects with peripheral vestibular dysfunction. Norré and DeWeerdt (1993, 1994) report a 50% reduction in positive manoeuvres within the first two weeks of training; Hecker, Haug and Herndon (1974) report 84% favourable response to the Cooksey-Cawthorne exercises; Norré et al (1987) report an 85.71% reached scores of zero vertigo

following VHT; Shepard et al (1990) report 87% of the patients experienced at least some reduction in vertigo following treatment.

Only a few human studies have evaluated the effects of rehabilitation on balance performance in subjects with peripheral vestibular dysfunction. Takemori et al (1985) report the use of static posturographic measures for the patients undergoing labyrinthectomy, VIIIth nerve resection, and unilateral chemical ablation with streptomycin sulfate, as well as for those suffering from bilateral peripheral loss of function. They used a generic, noncustomized exercise program for balance retraining. Although, they report a 100% recovery, they only examined static balance performance under relatively simple tasks. They measured movements of centre of gravity while subjects stood quietly with eyes open and closed for 30 sec. This study relied on more dynamic measures of balance as tested by the Equitest conditions. The subjects were tested on the balance performance under conditions of altered sensory input. The altered sensory conditions and dynamic movement of the Equitest add a considerable degree of difficulty to the standing balance performance measures of the subjects in this study as compared to the test conditions reported by Takemori et al (1985).

Shumway-Cook et al (1990) in a case study of one subject, reported effective management of impaired balance with a rehabilitation approach. They reported a return to normal body sway during all six sensory organization tests (Equitest) following six weeks of therapy. This study did not use quantitative measures to compare their rehabilitation approach to other modalities.

A few animal studies in both primates (Igarashi 1974; Igarashi et al 1975; Igarashi et al 1981; Igarashi et al 1979; Igarashi et al 1988) and cats (Marchand and Amblard 1984) also report benefit from exercise therapy for the recovery of

postural and eye movement control following unilateral vestibular lesions. Igarashi et al (1988) reported a significant enhancement of locomotor balance performance was achieved postoperatively in an exercise group of monkeys, as compared to a non-exercise group. They made special note of the type of exercise that facilitate recovery of balance; exercises that were goal oriented and involved agility were superior to continuous constant exercise that were not impulsive or taxing to the animal. This study would appear to confirm Igarashi's findings as the subjects in the Reh (goal oriented) group had a significant improvement in balance performance as compared to the subjects in the Home group.

Lacour et al (1981) showed that the time course of recovery is distinctly delayed for the animals who were restrained postoperatively. They studied the H-reflex, posture and sequences of usual motor activities in neurectomized baboons when the hind limbs of the baboons were restrained post lesion. They suggested that all sensory inputs involved in the perception of space, posture and motion intervene in the recovery process. They reported that the animals needed to be active and that activity has to be started early post lesion to develop compensation. This study would appear to suggest that for humans, activity even in the chronic post lesion state can facilitate recovery of balance.

This study was based on comparing a rehabilitation approach (Reh and B group) to treatment of vestibular dysfunction that was specific to each subject's needs, with a more generic eye-head exercise program (Home and A group). This study supports and extends the findings researchers (Norre 1987; Norre and Beckers 1987 and 1988; Norré et al 1987a and 1987b; Norré and DeWeert 1980a and 1980b; Shepard et al 1990) who used customized exercises based on the Norré positional test battery results to treat patients with peripheral

vestibular disorders.

This study has clearly demonstrated the beneficial effects of this rehabilitation program in improving balance function in subjects with chronic vestibular dysfunction.

6.1.2. VOR

The purpose of the VOR is to maintain visual acuity during head motion, through eye-head coordination. Injury or disorders affecting the semicircular canals will affect the gain and time constant of VOR which in turn, results in loss of eye-head coordination.

Improvements in VOR gain and time constant symmetry or left-right differences in VOR gain and time constant should contribute to recovery of eye-head coordination. Therefore, VOR gain and time constant symmetry scores can be used as outcome measures of compensation or recovery of VOR and consequently eye-head coordination. This is based on theory that compensation can occur due to re-balancing of activity in vestibular nuclei ipsilateral (loss of inputs) and contralateral to lesioned side in the case of unilateral disorders. In fact animal studies which have examined effects of exercise on VOR compensation used VOR and OKN symmetry scores as outcomes measures of compensation (Courjon et al 1977; Dichgans et al 1973a and 1973b; Fetter et al 1988; Fetter and Zee 1988).

This study demonstrates that, in patients with chronic peripheral vestibular deficits, a reduction in left-right differences in VOR gain was induced by the rehabilitation program of exercises where fixation on stationary and moving visual targets was a requirement, but not by an exercise program of eye

and head oscillations where fixation was not a requirement (Home exercise group). This reduction in left-right differences could be due in part to the fact that the rehabilitation program was designed to train the subject to use smooth pursuit and saccades to assist the VOR to maintain focus on objects. In the rehabilitation program the subjects were also trained to suppress the optokinetic stimulus by focusing on a stationary or moving target. The exercises were goal directed in that fixation was an aspect of most of the exercises in the rehabilitation program, including the exercises for balance.

These findings are consistent with the results reported by Mathog and Peppard (1982) on their research with hemilabyrinthomized cats. They showed that daily exercise were effective in facilitating recovery of VOR gain symmetry. Fetter and Zee (1988) in their work on recovery of VOR in monkeys after unilateral labyrinthectomy, suggest that the capability for making corrective adjustments in VOR gain may be lost unless there is active exploration of the environment with its consequent sensory feedback in the critical period immediately following a vestibular lesion. Fetter et al (1988) report that the geniculostriate pathways play an important role in providing the necessary error signals to stimulate adaptive change in the gain of VOR. They state that visual experience is critical for compensation of abnormally reduced VOR gain following unilateral labyrinthectomy in monkeys. Maioli and Precht (1985) observed that, in hemilabyrinthectomized cats, forced oscillations in front of a pattern wall (OKS) had little effect on the lesion induced VOR gain asymmetry. Taken together, these findings and other research in the study of visually mediated adaptive gain control mechanism support the view that exercise or activities that produce error signals (varying degree of retinal image slip) that can be used to indicate the performance of the oculomotor system during head

movements are required to facilitate recalibration of left-right differences in VOR gain (Monnier et al 1970; Paige 1983; Robinson 1976 and 1981). Regarding recovery of oculomotor function following an insult to the peripheral vestibular system, besides the process of sensory substitution, visual information (Precht et al 1981; Precht and Dieringer 1981), cervical proprioception (Kasai and Zee 1978; Robinson 1976 and 1981) and other somatosensory inputs (Bles et al 1984), it is also possible that a recalibration of left-right difference in VOR gain could contribute to recovery of gaze stabilization. Further investigations will be required to determine the impact of VOR gain recalibration on gaze stabilization.

6.1.3. VOR TIME CONSTANT

In response to step changes in head velocity the semicircular canals respond by producing an exponentially decaying change in neural firing in the vestibular afferent fibres which outlasts the period of acceleration. The time constant of the exponential is approximately seven seconds in total. That is, the firing rate decays to 32% of the maximal initial amount in seven seconds. This is due to the mechanical nature of the semicircular canals, in particular to the dynamics of cupula displacement during acceleration, and is referred to as the peripheral time constant. A central mechanism, referred to as velocity storage, prolongs the peripheral time constant with a central time constant of approximately twenty seconds so that the nystagmus produced by the acceleration has this time course (Kandel et al 1991).

In addition to inputs from semicircular canals, the velocity storage mechanism receives information about head velocity derived from several kinds of motion sensors, including somatosensory and otolithic information (Hain

1986).

The findings in this study did not demonstrate a significant effect of exercise on VOR time constant symmetry scores. However, a trend towards reduced VOR time constant asymmetry was observed over the four test periods for the Reh and B groups, as compared to no change in the Home or A group. These findings are similar to those found in investigations on monkeys by Fetter and Zee (1988a and 1988b) and Paige (1983) and in human subjects by Allum (1988), Fetter and Dichgans (1991) , and Jenkins (1985) with unilateral peripheral vestibular lesions. It should be noted that these studies examined recovery of VOR time constant over time with no intervention. They found a general lack of recovery of VOR time constant or a persistent phase shift to low frequency sinusoidal rotations. Researchers have suggested that this indicates a disabling of the velocity storage mechanism (Fetter and Dichgans 1991; Jenkins 1985; Raphan et al 1977). This central mechanism, presumably consisting of positive feedback loops within the vestibular nuclei (Buettner et al 1978; Honrubia et al 1984) would account for the prolonged VOR time constant above the values predicted from cupulo-endolymph mechanisms. Due to it's neural complexity, the velocity storage mechanism may be more unyielding to adaptive modifications following the loss of vestibular inputs as compared to that of the VOR gain. It is also possible that the subjects in this present study, due to the chronic nature of their vestibular disorders, have reached the maximum possible level of their recovery.

Fetter and Zee (1988) have proposed other factors that may occur to inhibit recovery of the VOR time constant. They propose that the presence of spontaneous nystagmus could be interpreted by the CNS as if a persistent rotational stimulus was occurring, and this may lead to habituation of the VOR

time constant.

6.1.4. OKN

The optokinetic system uses visual information to compliment the vestibulo- ocular reflex. The optokinetic system compensates for the defects in the vestibular system by using the visual motion of head movement to drive the eyes. During a prolonged unidirectional head movement, the VOR will adapt and can no longer compensate for these head movements. The optokinetic reflex (OKN) then kicks into play. This reflex utilizes a continuous retinal signal rather than a phasic signal from the labyrinth to sense head movement. The eye automatically starts to track a target as the head moves in order to keep the image stationary on the retina. Optokinetic nystagmus is induced by having a subject observe a rotating striped drum as in the methods used in this study. The eyes move slowly in the direction of the drum movement (slow phase) and then rapidly in the opposite direction (fast phase) (Kandel et al 1991).

There is very little information in the literature on human or animal studies on the effects of peripheral vestibular dysfunction on the optokinetic reflex system.

Keller and Precht (1979) studied OKN in cats and discussed the difficulty of measuring the OK system which operates as a closed-loop feedback system in contrast to the open-loop VOR . They state that the OK velocity tracking of the eyes builds up slowly with an approximate exponential time course. Similar behaviour was also reported in the cat Evinger and Fuchs (1978), in monkeys Cohen et al (1977), and in the rabbit Colliwyjn (1969). Keller and Precht (1979) noted that the direct OK pathways, which are probably shared with the VOR

pathways from the central vestibular neurons on to the eye velocity output, are not modified during VOR recalibration. They felt their OK results provide additional evidence that major changes must occur at the level of central vestibular neurons to support the hypothesis that the OK and VOR pathways peripheral to the vestibular nucleus are shared. They felt their results support the concept of a parallel modifying projection to the central vestibular neuron rather than a modification of these neurons themselves, because the same vestibular neurons receive both the head velocity signal and the OK signal. Kellar and Precht (1979) felt that since a subset of these neurons show a modification for head velocity signals, they would be expected to show the same change for the OK inputs. Their study did not support this theory as they did not observe a change in the gain of their OK response results.

Precht et al (1981) in their study on hemilabyrinthectomized cats report that the slow phase velocity of the OKN was strongly impaired when directed away from the lesion and that this deficit compensated with time after lesion. They felt that central vestibular neurons on the lesioned side would normally participate in generating optokinetically driven opposite eye movements by an increase in firing of type I neurons. They felt it is understandable that a strong reduction in resting rate of the neurons (on the lesioned side) in the acute state also leads to a smaller sensitivity of the vestibular neuron to optokinetic stimuli and therefore, to a poorer OKN. They concluded that all the animals in their study showed a recovery of the VOR when measured in the light as apparently, optokinetic mechanisms were able to substitute for the vestibular defects still persisting although the optokinetic system itself was also strongly impaired in the acute stage post lesion.

The results of this study showed no significant changes in OKN gain

symmetry scores in any group over the four test periods. Basically the OKN values for the subjects in this study were normal to begin with, as can be seen by the values of OKN for each subject listed in Table 2. Therefore, the results are reflective of the type of patients in this study.

CHAPTER 7- CONCLUSION

As stated previously peripheral vestibular impairment can cause a number of disturbing signs and symptoms; disequilibrium, blurred vision, disorientation, vertigo and nausea. These sensory disturbances and motor deficiencies in turn cause dysfunctions in many activities of every day existence in both activities of daily living and in social interactions. There has been little help available from traditional medical treatments for these patients. Medication and surgical procedures have had limited success for this population. Research on humans has developed exercise programs that address the aspects of vertigo and dizziness for vestibular disorders. Animal research has examined motor performance post unilateral and bilateral induced lesions and the effect of exercise on that motor performance. Very little work has been done to develop and evaluate rehabilitation programs to address the issues of impaired balance in humans.

The results of this study demonstrated a significant positive effect of the rehabilitation exercise on postural stability under dynamic conditions and on left-right differences in VOR gain. Subjects who initially were unable to maintain their balance in conditions of sensory conflict, after twelve weeks of retraining were able to walk on a narrow beam with their eyes closed or when subjected to optokinetic stimulation. They scored well within the fifth percentile on their balance performance tests. Whereas the Home exercise group did not improve their postural stability or their left-right differences in VOR gain. There could be a number of reasons for the different results: 1) subjects in the Home program may not have exercised as regularly as the Reh group; 2) subjects in the Home program may not have progressed their exercises at the same rate as the Reh

group; 3) subjects in the Home program may have discontinued exercises when they experienced dizziness and nausea whereas, the Therapist encouraged the Reh group to continue exercising; and 4) the subjects in the Home group did not have access to the more difficult balance exercises and the optokinetic stimulus due to safety factors.

It was observed that the patients reported a decrease in their symptoms of dizziness when they were able to focus on the moving laser targets while moving their head in different directions. These goal directed eye-head coordination exercises, once mastered by the patient, appeared to be the key to decreasing their dizziness. It was also noted that the patients all complained of an increase in symptoms of dizziness when they were progressed to activities with optokinetic stimulations. These symptoms resolved very quickly in all cases with repeated exposure to the stimulus.

It was observed that the patients made steady progress in their standing balance when they started the sessions with biofeedback which was designed to assist them in developing an awarness of their CFP. Once the patients were able to control their sway with eyes closed using the audio portion of the feedback apparatus, we noted that they progressed quite quickly through the various balance tasks. It was again noted that these patients regressed for a short period of time when exposed to balance activities with optokinetic stimulation.

The one patient in the study with bilateral involvement did not respond as well as the patients with unilateral involvement. She did make considerable progress in her balance reactions but did not reach the fifth percentile level. It was necessary to provide this patient with instructions on how to make her home setting safe. She was advised to use night lights and to be careful of throw rugs

on polished floor surfaces. She was advised to use a walking aid when walking outside during the winter and to dry off her boots to prevent slipping.

There was no significant effect on the VOR time constant symmetry score although there was a trend towards symmetry in the Reh and B group but not in the Home group. These findings are consistent with other research of the VOR time constant in both animal and human studies.

The study showed no significant findings in the OKN gain symmetry scores for any of the exercise groups. These results could be reflective of the chronicity of the disorders tested.

The major benefit to the patient population studied was the positive outcomes of the patients following the rehabilitation program. Although this program is relatively more expensive to provide in the hospital setting a treatment effect was demonstrated with this program whereas, there was no treatment effect observed in the Home exercise group. Some of the exercises in the rehabilitation program would be difficult to dublicate in a home setting due to safety issues. It was important to have the therapist close to the patient when practicing balance activities on the balance beam and when progressing to optokineticstimulation training. The other benifit for the patient treated in the rehabilitation group was the encouragement and knowledge of the Physical Therapist who progressed the patient according to their level of tolerance. A follow-up study could look at how to decrease the cost to the health care system by identifying whether the exercises used in this rehabilitation program could be safely adapted to a home program.

The costs of setting up this rehabilitation program in a treatment facility is relatively inexpensive as the equipment used in the treatment is readily available in most clinic settings. Space requirements for this rehabilitation

program was also quite economical.

Thus, even in patients with chronic vestibular dysfunction, compensation for the loss or disruption of peripheral vestibular inputs can be effectively induced by exercises. The exercises need to provide sensory feedback appropriate for behavioral changes involving sensory substitution, or sensory motor reorganization in the case of postural control, or recalibration of VOR gain.

Further work needs to done to look at subjects with bilateral lesions and central lesions to examine whether this rehabilitation exercise program could enhance improvement in postural control for this population. Work also needs to be done to examine whether this rehabilitation program has an affect on subject's perceptual manifestations and to identify whether improvements in balance performance levels relate to improved function.

CHAPTER 8 - REFERENCES

1. Allum J.H.J., Keshner E., Honeggar F., Pfaltz C.R. Organization of leg-trunk-head equilibrium movements in normals and patients with vestibular deficits. Prog. in Brain Res. 76: 277-290, 1988.

2. Allum J.H.J., Yamane M., Pfaltz C.R. Long-term modifications of vertical and horizontal vestibulo-ocular reflex dynamics in man. I. After acute unilateral peripheral vestibular paralysis. Acta Otolaryngol.; 105: 328-337, 1988.

3. Amblard B., Cremieux J., Marchand A.R., Carblanc A. Lateral orientation and stabilisation of human stance: static versus dynamic visual cues. Exp. Brain Res. 61: 21-37, 1985.

4. Baloh R.W. Dizziness, hearing loss, and tinnitus: The essentials of neurology. Philadelphia; F.A. Davis Co., 1984.

5. Baloh, R.W., Honrubia V. Clinical neurophysiology of the vestibular system. Philadelphia: F. A. Davis, 1990.

6. Baloh R.W., Honrubia V., Yee R.D., Langhofer L., Minser K. Recovery from unilateral peripheral vestibular lesion. In: Keller E.L., Zee D.S., (eds.). Adaptive processes in visual and oculomotor systems. Oxford, UK: Pergamon; 349-356, 1986.

7. Berthoz A., Lacour M., Soechting J.F., Vidal P.P. The role of vision in the control of posture during linear motion. In: Granit R., Pompeiano O., (eds). Reflex control of posture and movement. Prog. Brain Res.; 50: 197-209, 1979.

8. Berthoz A., Melville-Jones G. Adaptive mechanisms in gaze control: facts and theories. Amsterdam: Elsevier, 1985.

9. Bles W., De Jong J.M.B.V., De Wit G. Somatosensory compensation for loss of labyrinthine function. Acta Otolaryngol.; 97: 213-221, 1984.

10. Black F.O., Nashner L.M. Vestibulo-spinal control differs in patients with reduced versus distorted vestibular function. Acta. Otolaryngol. (Stockh) 406 (suppl): 110-114, 1984.

11. Black F.O., Shupert C.L., Horak F.B., Nashner L.M. Abnormal postural control associated with peripheral disorders. Progress in Brain Res. 76: 236-275, 1988.

12. Black F.O., Wall G., Nashner L.M. Effects of visual and support surface orientation references upon postural control. In vestibular deficient subjects: Acta Otolaryngol (Stockh) 95: 199-210, 1983.

13. Brodal A. Anatomical observations on vestibular nuclei with special reference to their relations to the spinal cord and cerebellum. Acta Otolaryngol (Stockh). 192: 24, 1964.

14. Buettner U.W., Buettner U., Henn V. Transfer characteristics of neurons in vestibular nuclei of the alert monkey. J. Neurophys.; 41: 1614-1628, 1978.

15. Cawthorne T. The physiological basis for head exercises. J. Chart. Soc. Physiother. 106-107, 1944.

16. Cohen B., Matsuo V., Raphan T. Quantitative analysis of the velocity characteristics of optokinetic nystagmus and optokinetic after-nystagmus. J. Physiol. (Lond); 270: 321-344, 1977.

17. Collewijn H. Optokinetic eye movements in the rabbit: input-output relations. Vision Res.; 9: 117-132, 1969.

18. Collins W.E. Arousal and vestibular habituation. Habituation and vestibular responses with and without visual stimulation. In Kornhuber H.H. (eds). Handbook of Sensory Physiology. Berlin, Springer-Verlag, 1974.

19. Cooksey F.S. Rehabilitation in vestibular injuries. Proc. R. Soc. Med. 39: 273-275, 1946.

20. Courjon J.H., Jeannerod M., Ossuzio I., et al. The role of vision in compensation of vestibulo-ocular reflex after hemilabyrinthectomy in the cat. Exp. Brain Res. 28: 235-248, 1977.

21. Craik R.L., Cozzens B.A., Freedman W. The role of sensory conflict on stair descent performance in humans. Exp. Brain Res. 45: 399-409, 1982.

22. Currie J.N., Matsuo V. The use of clonazepam in the treatment of nystagmus-induced oscillopsia. Ophthalmology, 93: 924, 1986.

23. Dichgans J., Bizzi E., Morasso P., Talliasco V. Mechanisms underlying recovery of eye-head coordination following bilateral labyrinthectomy in monkeys. Exp. Brain Res. 18: 548-562, 1973a.

24. Dichgans J., Brandt T. Visual-Vestibular interaction: effect of self-motion perception and postural control. In: Held R, Liebowitz H., Teuber H.L., (eds). Handbook of sensory physiology, vol. 8. Heidelberg: Springer, 1978.

25. Dichgans J., Schmidt C.L., Graf W. Visual input improves the speedometer function of the vestibular nuclei in the gold fish. Exp. Brain Res. 18: 319-322, 1973b.

26. Diener H.C., Dichgans J. The role of vestibular, visual and somatosensory information for dynamic postural control in humans. Progress in Brain Res. 76: 253-262, 1988.

27. Diener H.C., Dichgans J., Bacher M. Role of visual and static vestibular influences on dynamic postural control. Human Neurobiology 5: 105-113, 1986.

28. Diener H.C., Dichgans J., Bootz F., Bacher M. Early stabilisation of human posture after a sudden disturbance: influence of rate and amplitude of displacement. Exp. Brain Res. 56: 126-137, 1984a.

29. Diener H.C., Dichgans J., Guschlbauer B., Mau H. The significance of proprioception on postural stabilisation as assessed by ischemia. Brain Res. 76: 253-262, 1988.

30. Diener H.C., Horak F.B., Nashner L.M. Influences of stimulation parameters on human postural responses. J. Neurophysiology. 6, 59: 1888-1905, 1988a.

31. Dietz V., Horstman G.A., Berger W. Interlimb coordination of leg muscle activation during perturbations of stance in humans. J. Neurophysiol. 3, 62: 680-693, 1984.

32. Dix M.R. The physiological basis and practical value of head exercises in the treatment of vertigo. Practitioner 217: 919-924, 1976.

33. Dix M.R. Rehabilitation of vertigo. In Dix MR, Hood JD (eds). Vertigo, New York, John Wiley and Sons, 1984.

34. Dix M.R. The rationale and technique of head exercises in the treatment of vertigo. Acta Oto-Rhino-Laryngologica. Belgica 33, 3: 370-384, 1979.

35. Evinger C., Fuchs A.F. Saccadic, smooth pursuit, and optokinetic eye movements of the trained cat. J. Physiol. (Lond) 285: 209-229, 1978.

36. Fetter M., Dichgans J. Adaptive mechanisms of VOR compensation after unilateral peripheral vestibular lesions in humans. J. of Vestibular Res. 1: 9-22, 1991.

37. Fetter M., Zee D.S., Proctor L.R. Effect of lack of vision and occipital lobectomy upon recovery from unilateral labyrinthectomy in Rhesus Monkey. J. Neurophys. 59: 394-407, 1988.

38. Fetter M., Zee D.S. Recovery from unilateral labyrinthectomy in Rhesus Monkey. J. Neurophys. 2, 59: 370-393, 1988.

39. Flohr H., Bienhold H., Abien W., Macskovics I. Concepts of vestibular compensation. In : Flohr H., Precht W., (eds). Lesion-induced neuronal plasticity in sensorimotor systems. Berlin: Springer-Verlag: 153-172, 1981.

40. Forssberg H., Nashner L.M. Otogenetic development of postural control on man: adaptation to altered support and visual conditions during stance. J. Neurosci. 5, 2: 545-552, 1982.

41. Fox C.R., Cohen H. The visual and vestibular systems. In: Cohen H.(ed). Neurosciences for rehabilitation. Philadelphia: Lippencott: 97-128, 1993.

42. Fuchs A.F. The vestibular system. In: HD Patton, AF Fuchs, P Hille, AM Scher, and R Steiner (eds). Textbook of Physiology, 21st ed. Excitable cells and neurophysiology. Vol. 1. Philadelphia: Saunders: 582-607, 1989.

43. Fuchs A.F, Kaneko C.R.S., Scudders C.A. Brainstem control of saccadic eye movements. Annu. Rev. Neurosci. 8: 307-337, 1985.

44. Goebel J.A., Paige G.D. Dynamic posturography and caloric tests results in patients with and without vertigo. Otolaryngol. Head Neck Surg. 100: 553-558, 1989.

45. Goldberg M.E., Colby C.L. The neurophysiology of spatial vision. In: Boller F, Crafman (eds). Handbook of Neuropsychology, Vol. 2. Amsterdam: Elsevier Science publishers, 301-315, 1989.

46. Goldberg M.E., Eggars H.M., Gouras P. The ocular motor system. In: Kandel E.R., Schwartz J.H., Jessell T.M., (eds). Principles of Neural Science: North Holland, Elsevier, 660-677, 1991.

47. Hain T.C. A model of the nystagmus induced by off-vertical axis of rotation. Biological Cybernetics; 54: 337, 1986.

48. Hecker H.C., Haug C.O., Herndon J.W. Treatment of the vertiginous patient using Cawthorne's vestibular exercises. Laryngoscope; 84: 2065-2072, 1974.

49. Held R., Dichgans J., Bauer J. Characteristics of moving visual scenes influencing spatial orientation. Vision Res. 15: 357-365, 1975.

50. Herdman S.J. Vestibular rehabilitation. F.A. Davis Co., Philadelphia, 1994.

51. Honrubia V., Jenkins H.A., Baloh R.W. Vestibulo-ocular reflexes in labyrinthine lesions: I. Unilateral dysfunction. Am. J. Otolarnygol.; 7: 15-26, 1984.

52. Horak F.B. Clinical measurements of postural control on adults. Physical Therapy, 67, 12: 1881-1885, 1987.

53. Horak F.B., Nashner L.M. Central programming of postural movements: adaptation to altered support surface configurations. J. Neurophysiol. 55: 1369-1381, 1986.

54. Horak F.B., Diener H., Nashner L.M. Postural strategies associated with somatosensory and vestibular loss. Exp. Brain Res. 82: 167, 1991.

55. Horak F.B., Shumway-Cook A., Crowe T.K., Black F.O. Vestibular function and motor proficiency of children with impaired hearing, or with learning disability and motor impairments. Dev. Med. Child Neur.; 30: 64-79, 1988.

56. Horak F.B., Shupert C.L., Mirka A. Components of postural dysfunction on the elderly; a review. Neurobiology and Aging; 10: 727-738, 1989.

57. Horak F.B, Shupert C.L. Role of the vestibular system in postural control. In Herdman S.J. (eds). Vestibular rehabilitation. Philadelphia, F.A. Davis, 22-46, 1994

58. Igarashi M. Vestibular compensation: An overview. Acta Otolaryngol. (Stockh) 406: 78-82, 1984.

59. Igarashi M. Squirrel monkey platform runway test; a preliminary report. Acta Otolaryng.; 77: 284- 288, 1974.

60. Igarashi M., Alford B.R., Kato Y., Levy J.K. Effects of physical exercise upon nystagmus and locomotor dysequilibrium after labyrinthectomy in

experimental primates. Acta Otolaryngol. 79: 214-220, 1975.

61. Igarashi M., Levy J.K., Ouchi T., Reschke M.F. Further study of physical exercise and locomotor balance compensation after unilateral labyrinthectomy in squirrel monkeys. Acta Otolaryngol. 92: 101-110, 1981.

62. Igarashi M., Levy J.K., Takahashi M., Alford B.R., Homick J.L. Effects of exercise upon locomotor balance modification after peripheral vestibular lesion (unilateral utricular neurotomy) in squirrel monkey. Adv. Oto-Rhino-Laryngol. 25: 72-78, 1979.

63. Igarashi M., Ishikawa M., Yamane H. Physical exercise and balance compensation after total ablation of vestibular organ. Prog. Brain Res. 76: 395, 1988.

64. Jenkins H.A. Long-term changes of the vestibulo-ocular reflex in patients following acoustic neuroma surgery. Laryngoscope; 95: 1224-1234, 1985.

65. Kandel E.R., Schwartz J.H., Jessell T.M. Principles of Neural Science. Chapter 33 and 43, North Holland, Elsevier,1991.

66. Kasai T., Zee D.S. Eye-head coordination in labyrinthine-defective human beings. Brain Res.; 144: 123-141, 1978.

67. Keller E.L., Pretch W. Visual-vestibular responses in vestibular nuclear neurons in the intact and cerebellectomized alert cat. Neuroscience; 4: 1599-1613, 1979

68. Keshner E., Allum J.H.U., Pfaktz C.R. Postural coactivation and adaptation in the sway stabilizing responses of normals and patients with bilateral vestibular deficits. Exp. Brain Res. 66: 77-92, 1987.

69. Lacour M., Roll J.P., Appaix M. Modifications and development of spinal reflexes in the alert baboon (papio papio) following a unilateral vestibular neurotomy. Brain Res. 113: 255-269, 1976.

70. Lacour M., Xerri C. Vestibular compensation: New perspectives. In: Flohr H., Precht W., (eds). Lesion induced neuronal plasticity in sensorimotor systems. New York, Springer-Verlag, 1981.

71. Lacour M., Xerri C. Compensation of postural reactions to fall in the vestibular neurectomized monkey. Role of the visual cues. Exp. Brain Res.; 40: 103-110, 1980.

72. Leigh R.J., Sharpe J.A., Ranalli P.J., Thurston S.E., Hamid M.A. Comparison of smooth pursuit and combined eye-head tracking in human subjects with deficient labyrinthine function. Exp. Brain Res.; 66: 458-464, 1987.

73. Leigh R.J., Zee D.S. The neurology of eye movements (eds). F.A. Davis Co., Philadelphia, 1991.

74. Lestienne F., Soechting J.F., Betthoz A. Postural readjustments induced by linear motion of visual scenes. Exp. Brain Res.; 28: 364-384, 1977.

75. Maeda M. Mechanisms of vestibular compensation in the unilateral labyrinthectomized cat. Prog. in Brain Res. 76: 385-394, 1988.

76. Maioli C., Precht W. On the role of vestibulo-ocular reflex plasticity in recovery after unilateral peripheral vestibular lesion. Exp. Brain Res. 59: 267-272, 1985.

77. Marchand A.R., Amblard B. Locomotion in adult cats with early vestibular deprivation: Visual cue substitution. Exp. Brain Res. 454: 395, 1984.

78. Mathog R.H., Peppard S.B. Exercise and recovery from vestibular injury. Am. J. Otolaryngol. 3: 397- 407, 1982.

79. Mc Cabe B.F. Labyrinthine exercises on the treatment of disease characterized by vertigo: their physiologic basis and methodology. Laryngoscope; 80: 1429-1433, 1970.

80. Miles F.A., Kawano K. Short-latency ocular following responses of monkey. III. Plasticity. J. Neurophys.; 56: 1381-1396, 1986.

81. Monnier I., Belin I., Polc P. Facilitation, inhibition and habituation of vestibular response. Adv. Otorhinolaryngol. 17: 28, 1970.

82. Nardone A., Giordano A., Corra T., Shieppati M. Responses of leg muscles in human displacement while standing. Brain; 113: 65-85, 1990.

83. Nashner L.M. Adapting reflexes controlling the human posture. Exp. Brain Res.; 26: 59-72, 1976.

84. Nashner L.M. Fixed patterns of rapid postural responses among leg muscles during stance. Exp. Brain Res. 30: 13-24, 1977.

85. Nashner L.M. Strategies for organization of human posture. In: Igarashi M. and Black F.O., (eds). Vestibular and visual control on posture and locomotor equilibrium, Basel, Karger, 1985.

86. Nashner L.M., Berthoz A. Visual contribution to rapid motor responses during postural control. Brain Res. 150: 403-407, 1978.

87. Nashner L.M., Black F.O., Wall G. Adaptation to altered support and visual conditions during stance: patients with vestibular deficits. J. Neurosci. 2, 5: 536-544, 1982.

88. Nashner L.M., Cordo P.J. Relation of automatic postural responses and reaction-time voluntary movements of human leg muscles. Exp. Brain Res. 43: 395-405, 1981.

89. Nashner L.M., Wolfson P. Influence of head position and proprioceptive cues on short latency postural reflexes evoked by galvanic stimulation of the human labyrinth. Brain Res. 67: 255, 1974.

90. Norré M.E. Rationale of rehabilitation treatment for vertigo. Am. J. Otolaryngol; 111: 609, 1987b.

91. Norré M.E., Beckers A. Exercise treatment for Paroxysmal Positional Vertigo: comparison of two types of exercise. Arch. Otorhinolaryngol; 244: 291-294, 1987b.

92. Norré M.E., Beckers A. Vestibular habituation training. Specificity of adequate exercise. Arch. Otolaryngol. Head Neck Surg.; 114: 883-886, 1988.

93. Norré M.E., Forrez G., Beckers A. Posturographic findings in two common peripheral vestibular disorders. J. of Otolaryngol.; 6, 16: 340-344, 1987.

94. Norré M.E., Forrez G., Beckers A. Vestibular compensation evaluated by rotation tests and posturography. Arch. Otolaryngol. Head Neck Surg.; 113: 533-535, 1987.

95. Norré M.E., DeWeerdt W. Treatment of vertigo based on habituation. 1. Physio-Pathological basis. J. of Larngolog. and Otology; 94; 971-977, 1980a.

96. Norré M.E., DeWeerdt W. Treatment of vertigo based on habituation. 2. Technique and results of habituation training. J. of Laryngol. and Otology; 94: 971-977, 1980b.

97. Paige G.D. Vestibular reflexes and its interaction with visual following mechanisms in the squirrel monkey. II. Response characteristics and plasticity following unilateral inactivation of horizontal canal. J. Neurophys.; 1, 49: 152-168, 1983.

98. Pfaltz C.R., Kamath R. Central compensation of vestibular dysfunction: (1) Peripheral lesions. Adv. Otorhinolaryngol.; 30: 335, 1983.

99. Pompeiano O., Brodal A. The origin of vestibulospinal fibers in the cat. An experimental anatomical study, with comments on the descending medical longitudinal fasciculus. Arch. Ital. Biol. ; 95: 166, 1957.

100. Precht W., Maioli C., Dieringer N., Cochran S. Mechanisms of compensation of the vestibulo-ocular reflex after vestibular neurotomy. In: Flohr H., Precht W., (eds). Lesion-induced neuronal plasticity in sensorimotor systems. Berlin: Springer : 221-230, 1981.

101. Pretch W., Dieringer N. Neuronal events paralleling functional recovery (compensation) following peripheral vestibular lesions. In: Berthoz A., Melvill-Jones G., (eds). Adaptive mechanisms in gaze control: facts and theories. Amsterdam: Elsevier: 251-268, 1981.

102. Pukonnen P.T., Courjon J.H., Jeannerod M. Compensation of postural effects of hemilabyrinthectomy in the cat. A sensori-substriatal process. Exp. Brain Res. 28: 249 - 257, 1977.

103. Raphan T., Cohen B., Matsuo V. A velocity-storage mechanism responsible for optokinetic (OKN), optokinetic after-nystagmus (OKAN) and vestibular nystagmus. In: Baker R., Berthoz A., (eds). Control of gaze by brainstem neurons: developments in neurosciences, vol. 1. New York: Elsevier; 37-47, 1977.

104. Robinson D.A. Adaptive gain control of vestibulo-ocular reflex by the cerebellum. J. Neurophys.; 39: 954-969, 1976.

105. Robinson D.A. Control of eye movements. In: Brooks V.B., (eds). Handbook of physiology, Section 1: The Nervous System, Vol. 11. Motor Control, Part 2. Bethesda, Md.: American Physiological Society: 1275- 1320, 1981.

106. Schaefer K.P., Meyers D.L., Wilhelms G. Somatosensory and cerebellar influences on compensation of labyrinthine lesions. In: Granit R., Pompeiano O., (eds). Reflex control of posture and movement. Prog. Brain Res. Vol. 50, Elsevier/ North-Holland Biomed. Press, Amsterdam, New York: 591-598, 1979.

107. Schaefer K.P., Meyer D.L. Aspects of vestibular compensation in guinea pigs. In: Flohr H., Precht W., (eds). Lesion-induced neuronal plasticity in sensorimotor systems. New York: Springer-Verlag: 197-207, 1981.

108. Shepard N.T., Telian S.A., Smith-Wheelock M. Habituation and balance retraining therapy. A retrospective review. Diag. Neurotology; 8: 459-475, 1990.

109. Shumway-Cook A., Horak F.B. Rehabilitation strategies for patients with vestibular deficits. Neurologic Clinics Vol. 8, 2: 441-457, 1990.

110. Shumway-Cook A., Woollacott M.H. The growth of stability: postural control from a developmental perspective. J. Motor Behaviour; 17, 2: 131-147, 1985.

111. Soechting J.F., Berthoz A. Dynamic role of vision in the control of posture in man. Exp. Brain Res.; 36: 551-561, 1979.

112. Talbott R.E. Postural reaction of dogs to sinusoidal motion in the peripheral visual field. Am. J. Physiol.; 239: (Regulatory Integrative Comp. Physiol.8): R71-R79, 1980.

113. Talbott R.E., Brookhart J.M. A predictive model study of the visual contribution to canine postural control. Am. J. Physiol. 239: (regulatory Integrative Comp. Physiol. 8): R80-R92, 1980.

114. Talbott R.E., Humphrey D.R. (eds). Posture and movement. New York: Raven Press, 1979.

115. Takemori S., Ida M., Umezu H. Vestibular training after sudden loss of vestibular function. ORL; 47: 76-83, 1985.

116. Telian S.A., Shepard N.T., Smith-Wheelock M. Habituation therapy for the chronic vestibular dysfunction: preliminary results. Otolaryngol. Head Neck Surg.; 103: 89-95, 1990.

117. Traccis S., et al. Successful treatment of acquired pendular elliptical nystagmus in Multiple Sclerosis with isoniazid and base-out prisms. Neurology; 40: 492, 1990.

118. Wilson V.J., Jones M.J. Mammalian vestibular physiology. Plenum Press, New York, 1979.

119. Woollacott M., Marin O., Nashner L.M. Modifiability of human long latency (90-100ms) muscle responses to postural perturbations by expectancy. Soc. Neurosci. Abstr. 6: 463, 1980.

120. Voorhees R.L. The role of dynamic posturography in neurologic diagnosis. Laryngoscope; 99: 995-1001, 1989.

121. Zee D.S. Perspectives on the pharmacotherapy of vertigo. Arch. Otolaryngol. 111: 609, 1985.

122. Zee D.S. Vestibular adaptation. In Vestibular Rehabilitation. Herdman J.S., (eds). F.A. Davis, Philidelphia; 68-79, 1994.

Appendix 1

INFORMATION AND CONSENT FORM

COMPARISON OF DIFFERENT EXERCISE PROGRAMS IN THE REHABILITATION OF PATIENTS WITH CHRONIC PERIPHERAL VESTIBULAR DYSFUNCTION

INFORMATION

This clinical study is designed to evaluate the effectiveness of rehabilitation treatment for patients with vestibular disease or dysfunction. Your participation may lead to development of a new treatment program for patients with vestibular disorders.

All subjects in this study will undergo some form of rehabilitative treatment. All treatments and training procedures will be conducted under the general supervision of your doctor, Dr. D. J. Ireland. Subjects who agree to participate in this study will be randomly assigned to one of two groups. Group one will be given a series of exercises to perform at home on a regular basis. Group two will be treated at the Physiotherapy Department, Health Sciences Centre, by a licensed Physical Therapist. The treatment period will last between eight (8) and twelve (12) weeks.

You will be asked to undergo a series of clinical tests. You will have already taken some of these tests during the medical examination by Dr. Ireland. From the onset of the study, the clinical tests will be repeated on; week four (4), week eight (8), and week sixteen (16). Follow-up testing will also be performed on the eight and twelfth month. These tests will provide us with information about how effective the various treatment programs are in reducing or alleviating your signs and symptoms. All data collected will be used for analytical purposes and may also serve as documentation in research papers and presentations.

The following is a description of the clinical tests you will be asked to perform:
TEST 1 - You will be asked to sit fully clothed in a chair, and while recording eye position with surface electrodes applied to your temples, you will be asked to 1) shake your head for approximately 20 seconds. 2) View a moving visual scene (alternating white and black vertical strips) that will rotate around you. 3) sit stationary while the chair is rotated for periods of one minute.
TEST 2 - You will be asked to stand on a platform that will move two to four inches forwards and backwards. This will be done with eyes open and closed.

CONSENT

The purpose of the study, the nature of the clinical tests, and my involvement in it have been clearly explained to me.

I hereby consent to act as a subject in this research project.

My participation is voluntary, and I reserve the right to withdrawal from the study whenever I wish and if I do decide to withdrawal my medical care at this hospital will not be affected in any way.

_____ _____

Signature of Participant *Signature of Witness*

_____ _____

Signature of Investigator who explained procedures to participant *DATE*

Appendix 2

ASSESSMENT FORM
VESTIBULAR REHABILITATION STUDY
HEALTH SCIENCES CENTRE

NAME: _____

DATE: _____

SITTING:

ACTIVE MOVEMENTS	WHITE WALL		EYES CLOSED	STRIPED WALL	
	NORMAL	FIXATION		NORMAL	FIXATION
ROTATION (R)					
ROTATION (L)					
NOSE UP					
NOSE DOWN					
EAR (R)					
EAR (L)					

PASSIVE MOVEMENTS	EYES CLOSED
ROTATION (R)	
ROTATION (L)	
NOSE (UP)	
NOSE (DOWN)	
EAR (R)	
EAR (L)	

KEY: ND= NO DIZZINESS
SD= SLIGHT DIZZINESS
MD= MODERATE DIZZINESS
ZD= SEVERE DIZZINESS

COMMENTS:

STANDING BALANCE

I WHITE WALL

HEAD MOVEMENT	AMOUNT OF SWAY	DIZZINESS
HEAD ROTATION (R) NO FIXATION		
HEAD ROTATION (L) NO FIXATION		
NOSE UP NO FIXATION		
NOSE DOWN NO FIXATION		
HEAD ROTATION (R) FIXATION		
HEAD ROTATION (L) FIXATION		
NOSE UP FIXATION		
NOSE DOWN FIXATION		

II STRIPED WALL

HEAD MOVEMENTS	AMOUNT OF SWAY	DIZZINESS
HEAD ROTATION (R) NO FIXATION		
HEAD MOVEMENTS (L) NO FIXATION		
NOSE UP NO FIXATION		
NOSE DOWN NO FIXATION		
HEAD ROTATION (R) FIXATION		
HEAD ROTATION (L) FIXATION		
NOSE UP FIXATION		
NOSE DOWN FIXATION		

III EYES CLOSED

HEAD MOVEMENT	AMOUNT OF SWAY	DIZZINESS
QUIET STANDING		
HEAD ROTATION (R)		
HEAD ROTATION (L)		
NOSE UP		
NOSE DOWN		

IV SPONGE ON WHITE WALL

HEAD MOVEMENTS	AMOUNT OF SWAY	DIZZINESS
EYES OPEN		
HEAD ROTATION (R) NO FIXATION		
HEAD ROTATION (L) NO FIXATION		
NOSE UP NO FIXATION		
NOSE DOWN NO FIXATION		
HEAD ROTATION (R) FIXATION		
HEAD ROTATION (L) FIXATION		
NOSE UP FIXATION		
NOSE DOWN FIXATION		
EYES CLOSED		

V SPONGE STRIPED WALL

HEAD MOVEMENTS	AMOUNT OF SWAY	DIZZINESS
EYES OPEN		
HEAD ROTATION (R) NO FIXATION		
HEAD ROTATION (L) NO FIXATION		
NOSE UP NO FIXATION		
NOSE DOWN NO FIXATION		
HEAD ROTATION (R) FIXATION		
HEAD ROTATION (L) FIXATION		
NOSE UP FIXATION		
NOSE DOWN FIXATION		
EYES CLOSED		

KEY: ND= NO DIZZINESS
SD= SLIGHT DIZZINESS
MD= MODERATE DIZZINESS
ZD= SEVERE DIZZINESS

NS= NO SWAY
SS= SLIGHT SWAY
MS= MODERATE SWAY
ZS= SEVERE SWAY
F= FALL

COMMENTS:

SIGNATURE OF THERAPIST:_____

DATE:_____

Appendix 3

UNIVERSITY OF MANITOBA

FACULTY COMMITTEE ON THE USE OF HUMAN SUBJECTS IN RESEARCH

NAME: Dr. Tony Szturm

OUR REFERENCE: E88:135

DATE: October 4th, 1988

YOUR PROJECT ENTITLED:

Development and evaluation of rehabilitation treatment for patients with vestibular disorders.

HAS BEEN APPROVED BY THE COMMITTEE AT THEIR MEETING OF:

October 3rd, 1988.

COMMITTEE PROVISOS OR LIMITATIONS:

None.

You will be asked at intervals for a status report. Any significant changes of the protocol should be reported to the Chairman for the Committee's consideration, in advance of implementation of such changes.

** This approval is for the ethics of human use only. For the logistics of performing the study, approval should be sought from the relevant institution, if required.

Sincerely yours,

J. P. Maclean, M.D.,
Chairman,
Faculty Committee on the Use of Human
Subjects in Research

JPM/11
TELEPHONE ENQUIRIES:
- Lorraine Lester