VESTIBULAR DYSFUNCTION ASSOCIATED WITH CHRONIC TRAUMATIC BRAIN INJURY IN AMATEUR RUGBY PLAYERS

THESIS

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Patricia M. Hill, M.A.

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DEDICATION

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TABLE OF CONTENTS

LIST OF TABLES AND FIGURES

Page

CHAPTER 1

INTRODUCTION

Statement of the Problem

Traumatic brain injuries (TBIs) are the most common cause of death in people under the age of 45 in the Western World.[1] In the United States alone, there are an estimated two million brain injuries each year, of which 350,000 are the result of sports and recreation activities.[1] The type of brain injury that occurs most in athletes is a mild traumatic brain injury (mTBI), commonly referred to as a concussion.[2,3] While most athletes who suffer a concussion will have a complete and rapid resolution of symptoms within days after their injury, it has been proven that concussions may result in permanent damage to neuronal structures in the brain.[4] This permanent cellular damage results in a prolonged or incomplete recovery in a small percentage of athletes who have suffered a concussion.[4]

There is evidence that the cumulative effects of multiple concussions may result in persistent impairments, functional limitations, and even disability long after the initial injury.[4] There is even evidence that participating in sports such as soccer and boxing, which involve repeated subconcussive blows to the head, without ever suffering a

diagnosed concussion, can result in cumulative and long-lasting neurological impairments.[12,42]

The vestibular system is one of the neural systems providing for postural stability and oculomotor control, thereby helping the body maintain its orientation to gravity. Persistent dysfunction of the vestibular system has been linked to an increased risk for other injuries.[5] Despite the fact that impairments in postural stability, including vestibular deficits, have been proven to occur with concussions, the treatment protocol for athletes who have suffered this type of head injury does not commonly address these deficits.[5] The assessment tools outlined in this research may allow better evaluation of the efficacy of vestibular physical therapy, and may help healthcare providers determine when it is truly safe for these athletes to return to play with less risk of further injury. Through these means it may be possible to decrease the severity of deficits incurred by these athletes, and reduce the overall burden placed on the healthcare system.

Purpose

The purposes of this study were 1) to compare the occurrence of vestibular impairments in amateur rugby players to that in a control group of peers, as evidence of Chronic Traumatic Brain Injury in these athletes, 2) to evaluate rugby players for increased vestibular impairment after an acute brain injury, and 3) to assess the efficacy of a customized vestibular therapy protocol on the improvement of vestibular function in athletes after suffering an acute brain injury. The study assessed the vestibular function of the participants on measures of all aspects of the disablement model[94] in athletes with and without a history of brain injury, and after acute brain injury. Measures of impairment included the Romberg and tandem Romberg sway analyses, the Modified

Combined Test of Sensory Interaction in Balance (MCTSIB), and the Dynamic Visual Acuity Test (DVA). Measures of functional limitation included the Dynamic Gait Index (DGI), and the Patient-Specific Function Scale (PSFS). The Dizziness Handicap Inventory (DHI) was used as a measure of disability.

Rationale

Both within the world of athletics and in the general population, there is a great deal of interest in the study of traumatic brain injury (TBI). The principal causes of TBI in the Unites States are vehicular accidents, falls, acts of violence, and sports injuries.[7] The highest incidence of TBI occurs in people between the ages of 15 and 24, with males being twice as likely to suffer a TBI than females.[7] In children and young adults, TBI is the leading cause of long-term disability and death.[7] Each year, there are approximately 300,000 people admitted to the hospital for mild or moderate brain injuries.[7] Although brain injuries related to sports and recreation account for only three percent of these hospital admissions, it is estimated that approximately ninety percent of the athletic-related brain injuries are mild and may go unreported.[7] These head injuries can be divided into two categories; open head injuries (those resulting in an opening of the cranium), and closed head injuries (those involving injury to the cranium and its internal structures without an opening). Concussions are the most common type of head injury seen in athletics, and are usually the result of a closed head injury.[2,3]

In a 1997 report, the Centers for Disease Control and Prevention[8] stated that concussions related to athletic activity had reached epidemic proportions in the United States.[8] Throughout the first half of this century, the mild brain injury resulting from a concussion, was commonly believed to have no long-term sequelae.[4] Over the past

several decades, mild brain injuries have received a much deserved increase in attention and scrutiny. With the growing interest in both competitive and recreational sports, it is vital that the scientific and medical communities come to a consensus on the best way to identify, diagnose, and treat the mild brain injury resulting from concussion.[3,4]

Significance

While there is an extensive body of research exploring the effects of concussions on athletes,[1-3,9-18] controversy still exists regarding the assessment and treatment of these athletes.[5,19-36] There has been little consensus as to how to properly diagnose and categorize these injuries as mild, moderate, or severe concussions. There is also a great deal of controversy as to when it is safe for these athletes to return to play. One aim of this study was to provide a valid and easily replicated battery of tests to be used by healthcare and athletic professionals to evaluate the impairments, functional limitations, and disability of post-concussion athletes. Tests currently conducted in most medical settings are often unable to identify any lingering vestibular impairments that may exist in an athlete who has suffered a concussion. With proper recognition and subsequent treatment of the vestibular impairments occurring after a concussion, it may be possible to return athletes to competition with less chance of future injury.

Most concussion research conducted to this point has focused primarily on participants in American football.[22,23] There is a small body of research exploring head injuries occurring in other sports such as soccer, ice hockey, and gymnastics.[9] This study is one of the few to examine the effects of head injury in the sport of rugby. Rugby is a full-contact sport that is the second most-played sport in the world, and one that is rapidly growing in popularity in the United States. In the United States, rugby is played at the

recreational or amateur level, and there is frequently a lack of sufficient, expedient medical care available to its participants. Because of the lack of convenient medical treatment, many rugby players who suffer head injuries return to play without appropriate assessment or treatment. A second aim of this study was to provide invaluable information as to the effects of concussion on rugby players, and to provide the governing body of the sport with evidence on which to base their regulations regarding the treatment of players with concussions.

CHAPTER II

LITERATURE REVIEW

The Study of Concussions in Athletes

There have always been risks associated with participation in physical activities. With the rise in popularity in the United States of such sports as football, hockey, rugby, soccer, etc., interest in the study of athletic injuries has skyrocketed. These sports are no longer played only on the professional level, but are also enjoyed at the recreational level by both men and women, from childhood through adulthood. All sports, including rugby, are being encouraged in children at earlier and earlier ages. It is imperative that the potential long-lasting effects of injury at an early age be determined to insure the age-appropriateness of various sports.

In the United States, the sport most often associated with brain injury is American football.[3] This association has meant that the sport of football has also been the most studied with regard to brain injuries.[3] As early as 1904, President Theodore Roosevelt expressed his concern over the number of killed or paralyzed participants in American football.[3] President Roosevelt's threat to ban football, lead to the establishment of the National Collegiate Athletic Association (NCAA). The NCAA was charged with providing a set of rules for safe competition.[3] Beginning in 1931, the

American Football Coaches Association began recording the number of football related fatalities through annual surveys.[9] In 1965, this survey information was transferred to the University of North Carolina at Chapel Hill. Using this survey information, the NCAA began keeping records of catastrophic football injuries through the National Center for Catastrophic Sports Injury Research (NCCSIR).[34,9,23]

From the data collected by the NCCSIR, Torg et al in 1990,[37] calculated the per year incidence of intracranial hemorrhage, intracranial deaths, cervical spine fractures, dislocations and subluxations, and permanent quadriplegia for high school and college football players.[37] While such sports as gymnastics and ice hockey have higher incidence rates (percentage of players injured) of severe head or spine injury than football, the large number of players participating in football in the United States results in football being associated with the largest total number of catastrophic brain and spine injuries.[4,23] An average of 1.8 million people play football each year in the United States, in high school, college, professional, or recreational programs.[23] During the years of the survey, between 1945 and 2000, there was only one year in which a fatal brain injury did not occur in the United States, as a result of a football-related brain injury.[22] A total of 714 athletes died during this time period; 492 (69%) of these deaths were attributed to head and brain injuries, 75% occurred in high school players, 15.5% in recreational players, 6.9% in college players, and only 2.6% in professional players.[22,23] The risk of serious injury increases with level of play due to the increased speed and energy of collisions at higher levels, however the number of participants also decreases at these higher levels, making the rate of incidence higher in the younger populations.[9,22] Non-fatal, mild brain injuries are much more common than fatal injuries, and it was estimated that 5.6% of all high

school football players will suffer a concussion in any one season, and that up to 20% will suffer at least one concussion during their careers.[9,25] One of the most shocking statistics reported by both Gerberich et al,[25] and Guskiewicz et al, [2] was that of those high school players suffering a concussion, 30% to 69% returned to play in the same game.

In 1964, the incidence of football-related fatalities peaked at 30, with 3.52 deaths per 100,000 participants.[23] It was through the work of Schneider in the 1960's, that preventative measures such as protective equipment, were identified and put into practice. Schneider's work eventually lead to the evolution of helmet design, reducing the number of fatalities to 2.0 per 100,000 participants by 1970, and to between 0.6 and 1.10 per 100,000 participants, in the years that followed.[22,23] The incidence of catastrophic injury remained high during the 1970's due to an increase in the number of cervical spine injuries related to a dangerous tackling technique known as "spearing".[3] Rule changes made by the NCAA in 1976 prohibited initial contact with the head or face, and as expected, reduced the number of cervical spine injuries.[3,22,23]

Despite a decrease in the number of fatalities associated with sports-related brain injury, the prevalence of mild brain injury resulting from concussions is still of serious concern. The growing number of recreational participants in sports such as football, hockey, rugby, soccer, and many others, makes it imperative that all members of the health professions be aware of the signs and symptoms, associated risks, and rehabilitation options for athletes suffering from mild brain injury as a result of a concussion.

The 1997 report filed by the CDC[8] provided information regarding the prevalence of head injury in the United States related to participation in a variety of activities. There

are between 300,000[8] and 350,000[1] sports-related head injuries in the United States every year. The types of brain injuries that occur related to sports include concussions, second-impact syndrome, post-concussion syndrome, and intracranial hemorrhage, of which concussion is the most common.[8] While the prevalence of concussion in American football has been discussed, it is important to recognize the rate of occurrence of mild brain injury in other athletic populations.

Ice hockey players are also very susceptible to mild brain injuries resulting from concussions.[9] There are two main mechanisms of injury which can result in concussion for a hockey player: 1) collision with another player, and 2) collision with the boards. Helmet use was mandated in Canada in the 1970's, which dramatically reduced the number of serious head injuries. Recently, the incidence of concussion in Canadian intercollegiate ice hockey players was reported to average three to four concussions per team during the season.[38]

Between the years 1983 and 2000, cheerleading accounted for half of the catastrophic athletic injuries to women, and 70% of the catastrophic injuries to high school-aged female athletes.[22] During this time period, cheerleading-related head injuries resulted in one fatality, one permanent disability, and 11 temporary disabilities.[22] The rate of serious injuries in these athletes was 1.03 per 100,000 participants, almost twice that of American football players, and much higher than other women's sports such as track and field, softball, swimming, or basketball.[4,22]

Soccer is widely considered the most popular sport in the world. The Federation Internationale de Football Association (FIFA) estimated in 2001, that one in twenty-five people worldwide participate in soccer,[39] with over sixteen million people in the United

States playing soccer at some point in their lifetime.[40] Head and facial injuries account for 4.9% to 22% of all soccer injuries, 20% of which are concussions.[41] The rate of concussion in soccer was found to be equal to that in American football.[42] Over a ten year period, Barnes,[43] found that the odds of sustaining a concussion were 50% for male soccer players and 22% for female soccer players. The majority of these concussions resulted from contact with another player.[43]

Other sports such as bicycling, baseball, softball, wrestling, skiing, gymnastics, and equestrian sports report rates of head injury as high as 22%.[9] Rugby is a sport that has not been given adequate attention is the United States related to the prevalence of serious injury. However, the sport has been rapidly growing in popularity in the United States since the 1960's, and there are currently 50,000 people making up seven competitive territories across the country.[44] Several of the characteristics of the sport of rugby make it imperative to have data regarding the incidence and outcome of brain injury: 1) there is an increasing number of men, women, and adolescents now playing the game, 2) rugby is a contact sport involving frequent collisions between players and collisions between players and the ground, and 3) rugby is played largely at the recreational level where medical attention may not be readily available or actively sought.

There are currently no certification requirements for someone to be able to coach a rugby team at any level of competition in the United States. There are three levels of certification available through weekend courses for those coaches who are willing and able to participate. These courses provide coaches with minimal information regarding the treatment of injured players. In the Level I certification program coaches are

provided with an emergency action plan in the event of a player being injured while on the field. The emergency action play involves not moving the player, protecting them, and calling for appropriately trained medical assistance when needed. To determine when an athlete is ready to return to play after a serious injury, coaches are instructed to defer to a physician. Serious injuries are said to include unconsciousness, concussion, surgery, and any injury requiring the player to miss more than seven consecutive days of practice. When a player has suffered a concussion, coaches are provided with guidelines for return to play. These guidelines, however, are based simply on the determined severity of the concussion, the patient's report of symptoms, and the number of prior concussions sustained by the player; they do not take into account any objective measures of postural stability or neuropsychological function.[45]

Despite the prevalence of this type of brain injury in athletes in the United States, there is still a lack of scientific research regarding the assessment and treatment of the impairments resulting from concussions. There are currently several theories as to the mechanism by which the brain is actually injured during a concussion and without this knowledge it becomes difficult to establish tests to accurately capture the deficits seen in these patients. It is important that a comprehensive battery of tests be established to insure appropriate treatment of this population, addressing all of their deficits, and allowing them to return to their activity without any increased risk of further injury.

The Pathophysiology of Concussions

Concussions are encompassed in the broader category of closed head injuries. In athletes, these injuries are divided into two categories: severe head injury and mild head injury.[3] Severe head injury describes an injury that may result in skull fracture,

intracranial hemorrhage, diffuse axonal injury, post-traumatic hydrocephalus, or epilepsy. The concussions more commonly seen during athletics fall into the category of mild head trauma, also known as mild traumatic brain injury (mTBI), or cerebral concussion.[3] The defining characteristics of a mTBI are 1) a period of unconsciousness or diminished consciousness lasting no longer than 20 minutes, 2) a Glasgow Coma Scale (GCS) score greater than 12, and 3) neuroimaging that is negative for intracranial hemorrhage at the time of examination by the physician. Post-traumatic amnesia (PTA) may be present, but can last no longer than 24 hours.[18,24]

Initially, it was thought that a concussion produced only a temporary disturbance of brain function due to neuronal, chemical, or neuroelectrical changes, without any lasting changes in the gross structure or function of the brain.[4] Early studies showed that the number of symptoms resulting from concussions was small and typically resolved between five and ten days post-injury.[20,46,47]

The injury occurring with mTBI depends on the type and amount of force that is imparted to the cranium.[3] Most closed head injuries are the result of a combination of two types of forces: acceleration-deceleration and rotational. Acceleration-deceleration or linear forces are generated when the head is traveling at some velocity and comes into sudden contact with a relatively fixed object. In athletic sports, such as rugby, this fixed object is typically another player or the ground. Activities such as blocking and tackling often result in the generation of these types of linear forces. Injury to the brain typically results from the rapid deceleration of brain tissue, as the frontal or temporal lobes impact the bony projections of the anterior or middle cranial fossae.[3] These injuries tend to

result in what is known as a focal lesion, localized to the anatomical area of impact, with deficits being noted in relation to the function of that area of the brain.

Rotational or angular forces are generated when a force applied to the cranium results in rotational movement of the brain within the cranium. The fixation of the brain at the foramen magnum causes a relative tethering of the brainstem as it passes through the tentorial hiatus.[3] The application of these rotational forces to the brain can result in a variety of injuries ranging from concussion to diffuse axonal injury (DAI).[3] This type of injury can also occur from activities such as tackling, however the best example of this mechanism of injury is the "hook" punch in boxing which delivers a rotational force to the cranium via contact with the mandible.[3]

In 2003, Okonkwo[1] described three types of injury to brain tissue that may be caused by a closed head injury. The first is a focal injury, similar to that described above, which is typically caused by a mechanical insult to the skull leading to the formation of an intracranial hematoma. The hematoma leads to localized cell necrosis of the brain parenchyma, with observed deficits relating to the function of the anatomical structures in that area.[1] While rarely occurring in athletes, these types of isolated focal neural deficits may be seen in athletes suffering from concussions, making it important to be able to recognize all of the signs and symptoms that may indicate a cerebral concussion.

The second type of injury is diffuse axonal injury, which can result from either acceleration/deceleration forces, or rotational forces, and does not require a mechanical insult to the cranium. Diffuse axonal injury is associated with coma, severe disability, and in the most extreme cases, death.[48] Diffuse axonal injury may present as a progressive process during the hours following an acute injury with patients

demonstrating a wide variety of neurological signs and symptoms. It was formerly believed that the severed appearance of certain axons following a traumatic brain injury was the result of the shear forces produced by the initial impact. Recent research suggests that DAI is not directly attributable to the initial impact, but that the initial injury results in the activation of a chain of secondary injury mechanisms which culminates in the formation of a disconnected swollen axonal stub.[1]

Okonkwo also describes a third category of tissue injury resulting as a secondary response to a mechanical insult to the brain.[1] Closed head injuries are thought to elevate levels of excitatory amino acids which then elevate the influx of sodium and calcium ions into neuronal cells.[49,50] This influx of intraneuronal calcium results in a cascade of secondary injury mechanisms that can eventually lead to organelle failure, neuronal somatic cytoskeletal damage, and neuronal death.[49,50]

This excitotoxic damage has been implicated in the cognitive declines and increased risk associated with repetitive traumatic brain injury, known as second-impact syndrome.[4,51,52] Second-impact syndrome occurs when a second brain injury is sustained prior to a complete resolution of the symptoms from the primary head injury.[4] In the days immediately following a concussion, living brain cells may be extraordinarily vulnerable to changes in cerebral blood flow, intracranial pressure, and repeated concussion.[4] The occurrence of a second-impact syndrome is relatively uncommon, and its existence has been questioned.[2,20,21] Despite the controversy existing around second-impact syndrome, the extreme, rapid, and often fatal neurological decline which defines this syndrome has been observed in some patients following repeated head trauma, and is therefore a cause of concern and illustrates the need for further research.[3,4,53]

Regardless of the existence of a true second-impact syndrome, research has conclusively found that once an athlete suffers a concussion, they are four to six times more likely to suffer a second concussion.[25,54] In 2002, Collins et al[55] found that after a third concussion, athletes presented with more severe on-the-field concussion markers such as loss of consciousness, the presence of amnesia, and the number of post-concussion symptoms.[55]

As of today, there are no neuroanotomical or physiologic measurements used to determine the precise degree of injury or the severity of metabolic dysfunction associated with a concussion. It is, therefore, currently difficult to determine when this dysfunction has completely subsided and when it is safe for these athletes to return to play without an increased risk of a repeated concussion and potential for further injury.[4]

While the majority (eighty percent or more) of athletes recover from concussions within five to ten days, the existence of a post-concussion syndrome has been postulated within the past several years. Post-concussion syndrome (PCS) is thought to have both organic and psychological factors.[56] In 2003, Cantu[4] published a checklist of post-concussion signs and symptoms which included drowsiness, memory problems, nausea, poor balance and coordination, irritability, anxiety, depression, blurred vision, and fatigue (Appendix A).[4,56] While the research in this area is largely inconclusive, there is evidence that the level of psychological distress is strongly related to the persistence of PCS symptoms.[57] This evidence indicates that a psychological component may be helpful in the rehabilitation of patients with persistent PCS symptoms.

In addition to the cumulative effects of concussion seen in second-impact syndrome, there is also evidence that the cumulative effects of even subconcussive head

injuries can result in long-term neurological impairments. This phenomenon, known as chronic traumatic brain injury or CTBI, has been primarily documented in boxers, however, a 1998 study by Matser et al,[42] found neuropsychological impairments and neurological abnormalities associated with CTBI in both active and retired professional soccer players. The rationale behind Matser's study was that soccer players frequently suffer concussions during practices and games, and are also susceptible to frequent minor subconcussive head injuries through the action of heading the ball.[42]

In 1999, Matser et al,[12] reported the results of a nine month study of amateur soccer players from three different teams in the Netherlands, comparing them to a group of control athletes including distance runners and swimmers. The study examined the performance of the athletes on 16 neuropsychological tests and found that the amateur soccer players exhibited impaired performance on tests of planning and memory. Among the soccer players tested, only twenty-seven percent had ever incurred a concussion, and twenty-three percent had suffered between two and five concussions. Matser et al[12] concluded the number of concussions recorded in amateur soccer players was inversely related to their performance on some neuropsychological test. They also concluded that participation in a sport such as soccer even without incurring a concussion was related to impaired performance on memory and planning tests, when compared to athletes such as runners and swimmers.[12] If activities such as heading the ball in soccer can result in neurological impairment, it is possible that participation in sports such as rugby, which involve frequent collisions between players, may also result in some degree of neurological dysfunction.

The Classification of Concussions

The definition and grading of concussions has been a source of controversy among medical professionals and healthcare providers for many years. The word concussion comes from the Latin "concussus", meaning "to shake violently".[4] The term "concussion" was initially defined by the Congress of Neurologic Surgeons in 1964 as "any head injury leading to mental status alterations, with or without a loss of consciousness".[19] The hallmark symptoms of concussion are confusion and amnesia. However, patients may exhibit a wide range of cognitive, emotional, sensory, and motor disturbances.[3]

The symptoms that may appear in the early phase after a concussion (minutes to hours) include headache, dizziness, vertigo, a lack of awareness of surroundings, nausea, and vomiting.[19] Later symptoms (appearing in days to weeks) may include persistent low-grade headache, lightheadedness, poor attention and concentration, memory dysfunction, irritability and low frustration tolerance, intolerance of bright lights or difficulty focusing vision, intolerance of loud noises, tinnitus, anxiety, depressed mood, or sleep disturbances.[19]

There are a multitude of differed schemes used to classify the severity of concussions. A system proposed by Cantu in 1986 was adopted by the American College of Sports Medicine, and is widely accepted by physicians.[3] The Cantu grading system designates players displaying only transient amnesia (lasting less than thirty minutes) as mildly concussed, those players displaying longer periods of amnesia (up to twenty-four hours) as moderately concussed, and those players with any loss of consciousness as severely concussed.[4]

In 1991, the NCAA adopted a more conservative grading scheme proposed by the Colorado Medical Society in response to several deaths related to head-injury in high school football players. The Colorado grading scheme places any player suffering amnesia in the moderately concussed category.[58]

In 1997, the American Academy of Neurology presented another classification scheme in an attempt to standardize concussion assessment.[3] This classification scheme differentiates between mild and moderate concussions based on the duration of mental confusion rather than the presence of amnesia, with a loss of consciousness still rendering a severe classification.[3]

The lack of consensus among both scientists and medical professionals demonstrates the lack of evidence-based recommendations available to practitioners when assessing and treating patients suffering from concussions. In the absence of evidence-based guidelines, clinicians are left to manage their patients through clinical judgment and common sense.[21]

The Assessment of Concussion in Athletes

There are three health outcomes that need to be addressed after an athlete has suffered a concussion. First, the medical status of the injured player must be assessed to rule out the presence of any neurosurgical emergencies such as an epidural, subdural, or intracerebral hemorrhage.[19] Second, potential catastrophic outcomes related to the swelling of the brain must be prevented. Finally, the cumulative effects of repeated concussions should be avoided.[19]

In 1997, McCrory outlined a protocol for these three phases of acute evaluation. The immediate management of the injury should employ the basic principles of first aid

following the order of Danger, Response, Airway, Breathing, and Circulation, or DR ABC.[21] Once the athlete is determined to be medically stable, the physician or trainer on the field should perform a thorough cervical spine examination. If there is no injury to the cervical spine, the patient may be safely moved to the sideline for a more thorough medical evaluation.

During the next phase of treatment, referred to by McCrory as "Early Management", the athlete is questioned for the presence of any of the common concussion symptoms including headache, dizziness, blurred vision, and nausea.[21] The priorities in the early management phase of treatment are the establishment of an accurate diagnosis, and the exclusion of a catastrophic intracranial injury.[21] A standard means of establishing the presence or absence of a concussion has been the questioning of the injured player regarding their orientation to name, date, and the location of the event. This type of questioning has been shown to be unreliable following a concussion, due to the fact that this aspect of memory remains fairly intact with this type of brain injury.[59] Questions requiring the recall and application of recently acquired information are more reliable in the diagnosis of a concussion. The Standardized Assessment of Concussion (SAC) developed by McCrea, et al in 1997,[28] includes six different categories: 1) orientation, 2) immediate memory, 3) concentration, 4) delayed recall, 5) a neurological screening, and 6) exertional maneuvers (Appendix B).[28] The SAC is now a widely used instrument for the acute evaluation of concussed athletes. During early management the concussed athlete may also be referred for diagnostic tests including a computerized tomography scan, a magnetic resonance image scan, and/or electronystagmography. The

presence of any of the following signs and symptoms would indicate the need for immediate referral to an emergency treatment facility of a neurosurgical center.[21]

Table 1: Indications for urgent referral[21]

Fractured skull
Penetrating skull trauma
Deterioration in conscious state following injury
Focal neurological signs
Confusion or impairment of consciousness >30 minutes
Loss of consciousness >5 minutes
Persistent vomiting
Increasing headache post-injury
Any convulsive movements
More than one episode of concussive injury in a single match
Any assessment difficulty (e.g. an intoxicated patient)
Head injuries in children
High-risk condition (e.g. hemophilia, anticoagulant use)
Inadequate post-injury supervision
Injury that results from a high-risk mechanism (e.g. high-velocity impact, missile injury)

McCrory refers to the "late management phase" as the stage in which the previously injured athlete is seeking advice regarding return to play.[21] The priorities in this stage are to assess the athlete's recovery and to apply appropriate return-to-sport guidelines.[12] Unfortunately, there is a wide range of ideas as to when it is appropriate for players to return to play after a concussion. While there is a lack of consensus on the period of rest time required before concussed athletes should return to play, there is agreement on the fact that players should be completely symptom-free before returning to their sport.[4,21] This again establishes the need for comprehensive and highly sensitive objective tests, capable of detecting minor impairments in these individuals.

Without objective tests and measures, athletic trainers and clinicians are often forced to rely solely on a subjective report of the athlete's symptoms to determine when it is safe for them to return to play.[29,30] Most athletes are clearly eager to return to competition and may, therefore, underreport their symptoms, leading to a false conclusion that the concussion has resolved. The signs and symptoms of a concussion are often quick to resolve and may go undetected by a clinician, again leading to a false determination that it is safe for the athlete to return to play.[29,20] In addition to deficits in cognition such as decreases in attention span, memory, and concentration which are often noted after a concussion, studies have also shown a diminished ability to maintain equilibrium.[20,24,29,30]

The most recent position statement of the National Athletic Trainer's Association (NATA) recommends the inclusion of a postural assessment in the post-injury evaluation of a concussed athlete. Rather than using a grading scale to determine when it is safe for a concussed athlete to return to play, the NATA suggests focusing on the athlete's symptoms, neuropsychological function, and postural stability.[31]

Clinical tests, such as the Romberg sway analysis, have traditionally been used to assess disequilibrium in patients with balance disorders.[29,30,60,61] An alternative objective measure of postural stability was proposed by Guskiewicz et al in 1997,[29] for the assessment of mild traumatic brain injury in athletes. A sensory organization test (SOT) was performed using NeuroCom's Equitest and Smart Balance Master to evaluate balance in athletes after they had suffered a concussion.[29] The SOT is a form of Computerized Dynamic Posturography (CDP) which utilizes six conditions to challenge the visual, somatosensory, and vestibular systems during static stance.

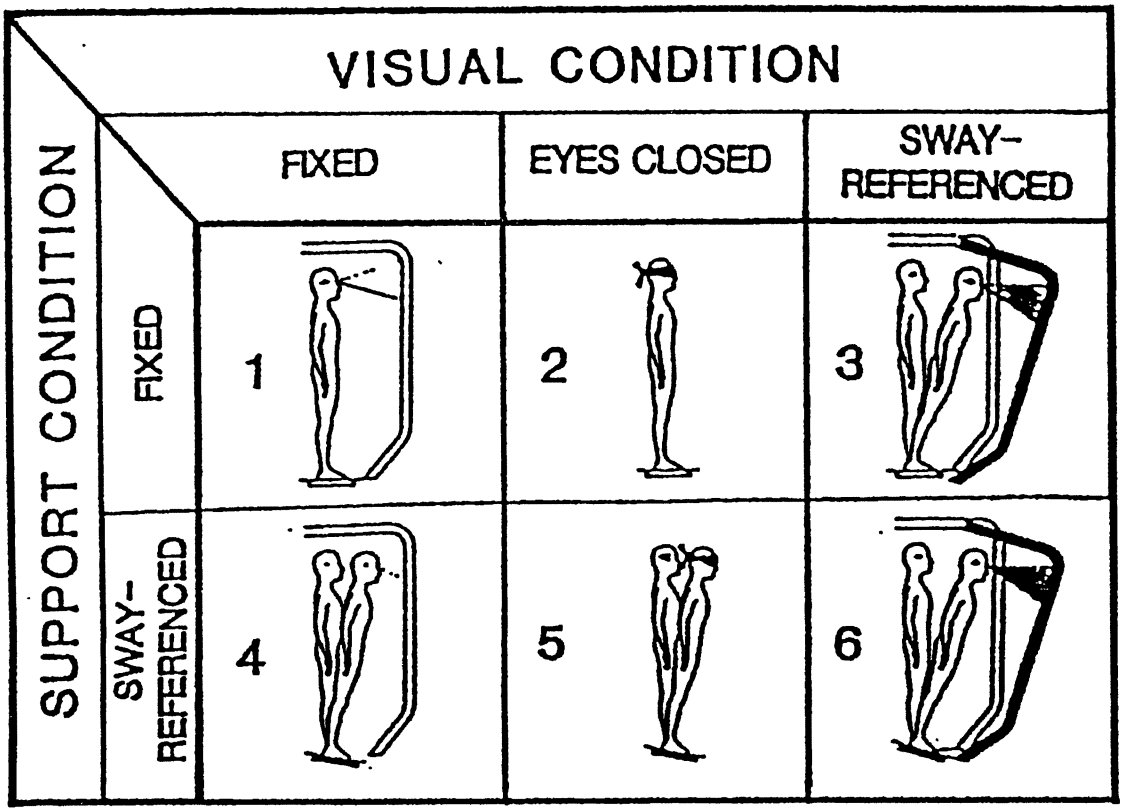


Figure 1: The six testing conditions used during the sensory organization test (SOT).[29]

The first three conditions are all performed on a stable surface, allowing the somatosensory system to provide correct information to the central processing center about the orientation of the body. The first condition allows all of the subject's senses to provide correct input to their central processing centers. The second condition removes vision, forcing the subject to rely on their somatosensory and vestibular systems to remain balanced. The third condition is sway-referenced, meaning the vestibular and somatosensory systems are providing correct information; however the visual environment surrounding the subject moves proportionally to the natural sway of the subject, providing conflicting visual information that the patient is not moving. The last three conditions are performed on an unstable surface, which moves proportionally to the sway of the subject, thus reducing somatosensory input to the central processing center.

The fourth condition allows the visual and vestibular systems to provide correct information to the central processing center. The fifth condition removes vision, forcing the patient to rely solely on their vestibular system to provide correct information. The sixth and final condition is again sway-referenced, with the central processing center of the subject receiving incorrect information from the visual surround that they are not moving, and correct information from their vestibular system that they are moving, again creating sensory conflict.

In the Guskiewicz, et al study,[29] athletes who had suffered a concussion were found to have decreased stability until approximately three days post-injury. Recovery of prior function was not fully achieved until ten days post-injury. The instability noted in these injured athletes was indicative of a dysfunction in the vestibular system. Despite the fact that vestibular deficits have been recorded in athletes who have suffered a concussion, these deficits are still not being addressed in their treatment.

While CDP requires the use of computerized force plates, a simpler, more cost-effective method of assessing balance in athletes was proposed by researchers at the University of North Carolina (Chapel Hill, NC).[30] The Balance Error Scoring System (BESS) is a clinical balance test that can be performed on the sideline, at the time of injury, requiring only a 10cm thick piece of medium-density foam and a stop watch. Scores on the BESS have been found to correlate significantly with force plate sway measures, in both healthy subjects and concussed athletes.[37, 62] While the BESS was able to measure disequilibrium in concussed athletes one day post-injury, it is possible that clinical balance tests, including the BESS and the Romberg sway analysis, may not be

sensitive enough to pick up on any persistent disequilibrium or vestibular dysfunction that might be present in a highly functioning population of athletes.

In addition to being at increased risk for a second concussion during this time of persisting clinical signs, a lack of adequate and correct vestibular input to the central nervous system has been implicated as a cause of various orthopedic injuries.[5] It is known that sports and other activities requiring a great deal of neuromuscular control depend on sensory input from the visual, vestibular, and somatosensory (proprioception) systems.[5] If a persistent dysfunction in the vestibular system is not addressed following a concussion, the athlete may return to play too soon, and be at increased risk for further injury. For this reason, it is important for clinicians to be able to assess when the vestibular system has fully recovered after a concussion. It may become a viable option to have baseline measures of an athlete's vestibular function recorded prior to their participation in competition. If that athlete was then to suffer a concussion, the medical professionals and therapists working with that athlete could be certain as to when their patient had returned to their prior level of function.

The Treatment of Concussion in Athletes

After a concussed athlete has been medically stabilized and their head injury has been assessed and diagnosed, there is little that is done to aid recovery. While there are many rehabilitation programs for persons with TBI, there is a lack of evidence regarding their effectiveness.[7] There is also a deficiency of rehabilitation programs that specifically target the athletic population. The majority of treatments studied in the past have focused on the neuropsychological impairments present in patients with more severe brain injuries. The cognitive impairments that may be present following a concussion include

memory impairment, intellectual compromise, or disturbances of executive functions.[63] A second group of symptoms that may present following a brain injury includes headache, dizziness, poor concentration, irritability, anxiety, depression, blurred vision, and fatigue.[63] These symptoms fall under the previously discussed category of post-concussion syndrome (PCS). Most rehabilitation for persons with PCS focuses on the treatment of the cognitive, behavioral, and psychological difficulties associated with their injury.[62] While sometimes indicated, these treatments are not aimed at the treatment of competitive athletes in preparation for their return to competition. Among other special considerations, athletes will generally have different expectations of outcome from their treatment than the average patient.[6] These expectations may include a higher final level of function, conditioning considerations, patient motivations, and a faster return to expected function.[6]

In 2003, Beazell et al[6] published extensive guidelines for the evaluation and rehabilitation of athletes with head and neck injuries. The evaluation of the post-concussion athlete included a postural assessment, range of motion measurements, assessment of the integrity of the ligaments and vascularity of the cervical spine, and an extensive neurological exam.[6] The treatment prescribed following this examination involved management of acute symptoms, strengthening of the postural and cervical muscles, mobility exercises for the upper thoracic spine, and sport-specific training prior to the athlete's return to sport.[6] Once again, the possibility of vestibular deficits was not considered during either the examination or the treatment of the athlete following a concussion.

The Physiology of Balance

To maintain postural control and balance both in static positions, and while performing dynamic activities, the body relies on input from three sensory systems: visual, somatosensory (proprioception), and vestibular. People rely most heavily on input from the visual system to maintain balance and postural stability at all times. The visual system provides the body with a reference position for vertical, orientation to the horizon, and information regarding our motion with respect to other objects in the environment.

The somatosensory system provides information to the central nervous system about the relationship of different body segments to each other, and about the body's position relative to its supporting surface, such as the velocity and direction of postural sway. Second to the visual system, the somatosensory system is the predominant source of sensory information during static balance.

The vestibular system, is the predominant source of sensory input during dynamic balance. The vestibular system provides us with information regarding the body's orientation in space, acceleration and tilt of the head, and provides input to reflexes controlling eye-head coordination as well as balance strategies.[64]

The Anatomy and Physiology of the Vestibular System

The vestibular system consists of two main components, peripheral sensory organs, and a central processing center. The peripheral sensory organs are located in the inner ear and are made up of membranous and bony labyrinths, and contain motion sensing hair cells (Figure 2).[64]

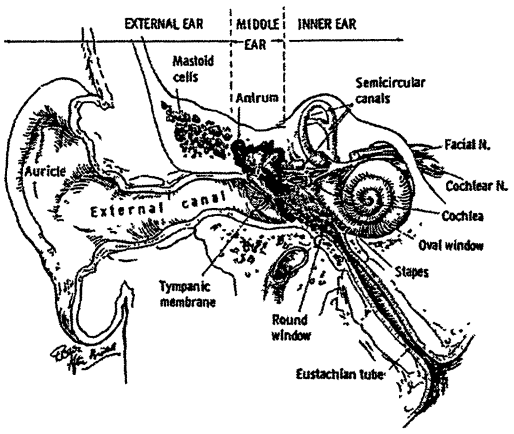


Figure 2: Anatomy of the peripheral vestibular system.[64]

The bony labyrinth consists of the semicircular canals, the cochlea, and a central chamber called the vestibule (Figure 3).[64] Perilymphatic fluid (similar in composition to cerebrospinal fluid, CSF) fills the bony labyrinth and communicates freely with the CSF of the subarachnoid space via the cochlear aqueduct.

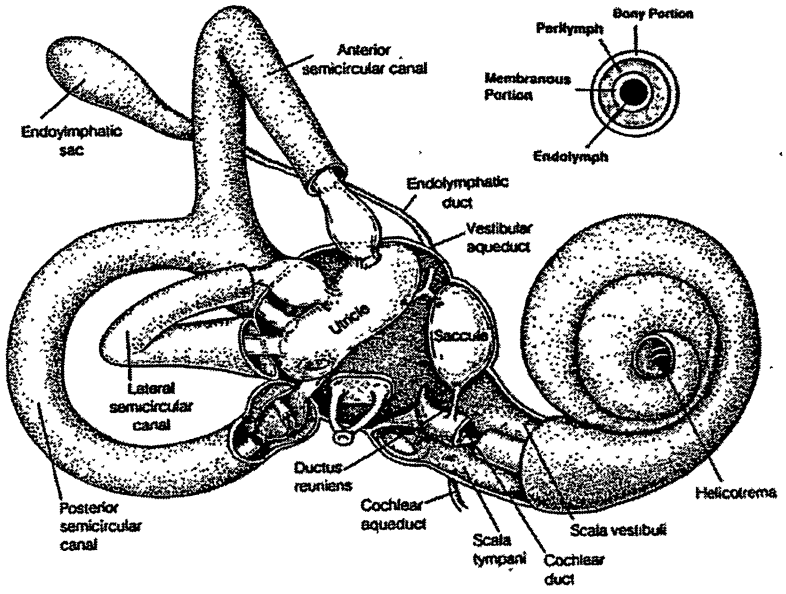


Figure 3: The membranous and bony labyrinths of the vestibular system.[64]

The five sensory organs of the vestibular system are located in the membranous labyrinth which is suspended in the bony labyrinth by fluid and connective tissue. The

five sensory organs are the membranous portions of the three semicircular canals and the two otolith organs, the utricle, and the saccule. The membranous labyrinth is filled with endolymphatic fluid which is similar in its electrolyte composition to intracellular fluid.[64] The specialized hair cells of the vestibular system are located in the two otolith organs, and in the ampullae, which are enlarged regions at the end of each semicircular canal. In the ampullae, the hair cells rest on a supportive network of blood vessels, nerve fibers, and connective tissue called the crista ampullaris. Specialized hair cells, or maculae, are also located on the medial wall of the saccule, and the floor of the utricle. Each of the hair cells is innervated by an afferent neuron, and when the head is moved, the hair cells bend with the movement of the endolymphatic fluid, causing either an increase or decrease in the firing rate of the vestibular nerve. The otolithic membranes contain calcium carbonate crystals called otoconia, which shift with head movement, again causing the afferent neurons of the maculae to fire, enabling the vestibular system to detect motion, as well as the direction of the pull of gravity.[64]

The semicircular canals of the vestibular system provide information as to the velocity and direction of head movements. There are three semicircular canals located in the inner ear, positioned at ninety degree angles from each other (Figure 4).

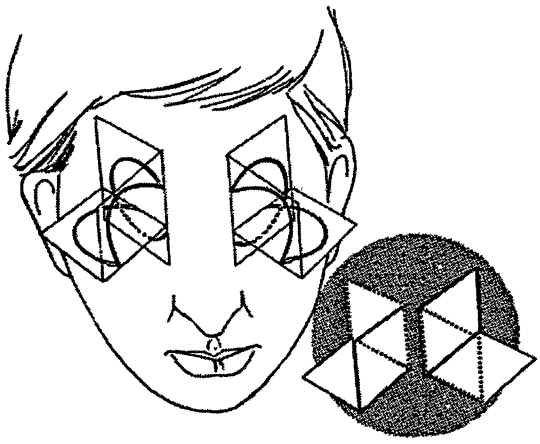


Figure 4: Spatial orientation of the semicircular canals.[64]

The layout of the canals allows the detection of linear motion in the horizontal and vertical planes, as well as angular motion. The anterior and posterior canals are positioned vertically and detect rotational movements in the sagittal and frontal planes. The horizontal canals detect motion in the horizontal plane. The orientation of the canals also matches the orientation of the extraocular muscles, allowing simple reflexive connections between the sensory neurons of the canals and the motor output neurons to the ocular muscles (Figure 4).[64]

The otoliths respond to linear acceleration of the head as well as static tilt of the head with respect to gravity. Acceleration of the head causes the otoconia in the otolithic membranes to shift, producing shear forces which are transformed from mechanical impulses to neural impulses via the maculae. In the upright position, the saccule is vertical and senses acceleration along the occipitocaudal and anterior-posterior axes. When upright, the utricle is positioned horizontally, and senses acceleration along the horizontal and anterior-posterior axes. Tilt of the head with respect to gravity is also registered in the utricle and the saccule. As the head tilts, either laterally, forwards, or

backwards, shear forces in the utricle increase, causing excitation of the vestibular nerve, while shear forces decrease in the saccule.[64]

The afferent neurons of the peripheral vestibular sensory organs provide input to a component of the vestiublocochlear nerve, the vestibular nerve. The vestibular nerve transmits afferent signals through the internal auditory canal, along with the cochlear nerve, the facial nerve, and the labyrinthine artery.[64] Signals from the vestibular nerve then enter the brainstem at the pontomedullary junction, and communicate with the vestibular nuclear complex (VNC) and the cerebellum. The VNC is located primarily in the pons and consists of four major and seven minor nuclei (Figure 5).[64]

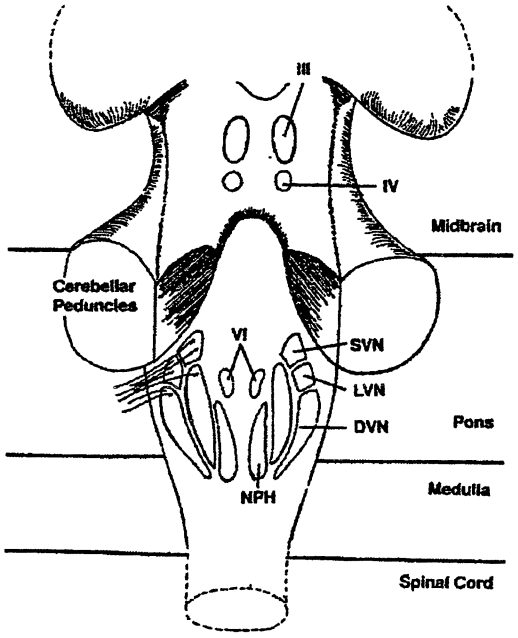


Figure 5: The vestibular nuclear complex with the cerebellum removed.[64]

These nuclei process afferent information from the peripheral sensory organs of the vestibular system, and organize reflexive motor output via the vestibulo-ocular reflex (VOR), and the vestibulo-spinal reflexes (VSR). The VOR generates eye movements of equal magnitude and in the opposite direction of head movements, allowing vision to

remain focused while the head is in motion. For example, if the head moves thirty degrees to the right, the VOR will signal the extraocular muscles to compensate by moving the eyes thirty degrees to the left, thereby allowing the person's vision to remain focused in the center of the visual field. The VOR is triggered during activities generating head movements at a frequency of two Hertz of greater. The activity of walking generates a frequency of head movements of approximately three Hertz, which is one of the reasons that the vestibular system is the primary source of sensory input during dynamic activities.[64] The VSR generates compensatory body movements, which maintain head and postural stability while the body is in motion. The VSR can work through multiple movement strategies to recover balance after a perturbation and to prevent falls.[64]

There is a large degree of communication between the vestibular nuclear complex and the cerebellum, with information traveling in both directions. The cerebellum functions as an inhibitor, to refine the motor output that is triggered by the vestibular reflexes, much in the way that the motor cortex functions to modify spinal reflexes via the corticospinal tract. The cerebellum is also thought to play a large role in the acquisition of adaptive changes in VOR, and can play a role in the recovery of vestibular function after damage has occurred to a peripheral structure.[64]

In addition to its role in the vestibular system, the cerebellum along with the basal ganglia and dorsal (posterior) columns, works with the motor cortex to produce coordinated movement responses.[66] The cerebellum is thought to function as an error-correcting system, by which commands generated by the motor cortex are compared to the actual movement output of the target muscles. The cerebellum receives continual

feedback from peripheral sensory neurons regarding posture and balance, position, rate, and intensity of movement, and is able to modify or correct ongoing movements through the activation or inhibition of specific muscle groups.[66] In addition to this feedback mechanism of movement control, the cerebellum participates in a feedforward control loop, in which communication occurs between the cerebellum, motor cortex, and basal ganglia, prior to movement to prepare the involved sensorimotor systems, and to allow for anticipatory adjustments to postural activity.[66] These mechanisms are vital for the maintenance of postural stability and balance during the challenging dynamic activities seen in athletics.

The information provided by the vestibular system is used in conjunction with input from the visual and somatosensory systems, to maintain balance in both static and dynamic activities. The vestibular system serves four important roles that contribute to postural stability, and to the maintenance of equilibrium and body alignment on unstable surfaces; 1) sensing and perceiving self-motion, 2) orienting to the vertical, 3) controlling the center of mass, and 4) stabilizing the head.[64]

Pathology and Dysfunction of the Vestibular System

Vestibular dysfunction is commonly divided into two categories: distortion and deficiency. A deficiency implies a reduction or absence of input from the peripheral sensory organs. The chief complaints of a person with a deficiency of the vestibular system would commonly be unsteadiness and instability. A distortion means that the system is getting sufficient input from the sensory organs, but the signal is somehow distorted or is not being interpreted correctly by the central processing centers. Distortion

will result in inappropriate motor responses to a given stimulus, presenting as vertigo or ataxia.[64]

Vestibular dysfunction can result from damage occurring at any point along the vestibular pathway, from the peripheral sensory organs, to the central processing centers in the brainstem and cerebellum. Unilateral lesions of the peripheral system result in an imbalance of the normally symmetric inputs from the left and right labyrinths. The affected side will exhibit a decreased neural firing rate, generating the illusion of head movement when there is none. This input to the vestibular system causes the vestibular reflexes (VOR and VSR) to fire, resulting in inappropriate movements, vertigo, nystagmus, and postural instability.[64]

In patients who have suffered mild traumatic brain injury, inner ear concussion is the most common vestibular sequelae. Symptoms of this injury can include high frequency sensorineural hearing loss, benign paroxysmal positional nystagmus and vertigo (BPPN/V), postural dyscontrol, and gait ataxia.[67]

Sensorineural hearing loss can be caused by mechanical injury to the cochlea through a temporal bone fracture, barotraumas, or noise, and by damage to the vestibulocochlear nerve, which carries information pertaining to both the vestibular and auditory systems.

Nystagmus is defined as involuntary rhythmic oscillations of the eyes. There is generally a fast phase in one direction, followed by a slow phase in the opposite direction. Nystagmus is named according to the fast phase of the movement, so that if the eyes move rapidly to the right and then at a relatively slower speed back to the left, the nystagmus would be referred to as right-beating. Physiological nystagmus can be

produced be vestibular or visual stimulation, or upon extreme lateral gaze (end-point nystagmus).[64] In patients with a vestibular disorder, these involuntary oscillations of the eyes, may appear with or without external stimulation, and would be referred to as a pathological nystagmus.[64] Lesions in both the peripheral and central vestibular systems, as well as lesions of the central nervous system, can cause pathological nystagmus. If there is a problem in the peripheral sensory organs of the vestibular system, placing the head in certain positions can invoke the nystagmus, which is then referred to as benign paroxysmal positional nystagmus (BPPN).

Vertigo is described as an illusion of rotational movement specific to vestibular dysfunction, causing the patient to feel as if the room is spinning, and is a result of pathological nystagmus. Benign paroxysmal positional vertigo (BPPV) can result from a traumatic insult to the brain if the acceleration of the head is such that the otoconia dislodge from the otolithic membrane. If the otoconia remain free-floating in the semicircular canals, the BPPV is referred to as canalithiasis, with transient symptoms of vertigo brought on by changes in position such as rolling over in bed, lying down, or looking up or down. If the otoconia become adhered to the cupula, a gelatinous membrane in the posterior canal, the ampulla becomes sensitive to gravity, resulting in BPPV known as cupulolithiasis. Cupulolithiasis will manifest as persistent vertigo, that is brought on by changes in position similar to canalithiasis.[64]

Table 2: The frequency of complaints in patients with BPPV.[64]

Poor Balance	57%
Sense of rotation (vertigo)	53%
Trouble Walking	48%
Lightheaded	42%
Nausea	35%
Queasy	29%

Spinning inside head	29%
Sense of tilt	24%
Sweating	22%
Sense of floating	22%
Blurred vision	15%
Jumping vision	13%

Postural dyscontrol and ataxia are the result of inappropriate motor outputs from the vestibular system, and can be caused by damage to the central vestibular processors in either the vestibular nuclear complex, or the cerebellum.[64]

Many of the signs and symptoms that are indicative of potential problems in the vestibular system, can also indicate a lesion in the central nervous system. It is therefore, important to rule out any central nervous system involvement. The primary method of testing the central nervous system is through an oculomotor screen. The patient can be observed for the presence of spontaneous nystagmus, a deficiency in smooth visual pursuits, a deficiency in saccadic eye movements, and an inability to converge and diverge their vision on an object.

Spontaneous nystagmus is tested by observing a patient in normal room lighting, and may be noted in the acute stage of patients with unilateral peripheral vestibular dysfunction. If the symptoms persist past the first several days post-injury, it may indicate a lesion of the central nervous system.

Smooth pursuits involves the ability of the patient to track an object smoothly with their eyes. The object is typically moved in an H-like pattern through the entire visual field, while the patient's head remains stationary. An inability of the eyes to follow the object could indicate a peripheral dysfunction of cranial nerves III,IV, and VI, which are responsible for the oculomotor muscles controlling the eye movements. The

presence of saccadic (jumping) eye movements, direction changing, or gaze-evoked nystagmus could be indicative of a lesion of the central nervous system. Gaze-evoked nystagmus occurs when the eyes have moved thirty degrees eccentrically, while end-point nystagmus can be found in healthy individuals at the extreme end range of their motion and should not be a cause for concern.[64]

Saccadic eye movements are tested by having the patient alternately focus on either two horizontal or two vertical targets, which are placed approximately fifteen to twenty centimeters apart, within their visual field. If the patient is unable to focus on the targets without multiple corrective movements, there may be a lesion in the central nervous system.

An inability to converge gaze on an approaching target can indicate either a lesion in the peripheral oculomotor nerves, or in the cerebellum, which is responsible for coordinated movements.

Rehabilitation of the Vestibular System

Rehabilitation of the vestibular system was pioneered by Cawthorne[68] and Cooksey[69] in the 1940's, for the treatment of patients with unilateral vestibular deficits and post-concussive disorders.[68,69] Early vestibular rehabilitation focused on adaptation via the substitution of other sensory and motor strategies for the malfunctioning vestibular system, and habituation of the vestibular system to lessen the symptoms of vertigo.[70,71]

Vestibular adaptation refers to long-term changes in the vestibular system's response to sensory input.[64] Adaptation is an important part of normal development and maturation, and also comes into play during the body's response to disease and injury. It

is thought that the vestibular system is malleable, and can be modified during the early stages of recovery after an acute unilateral vestibular loss.[72-74]

Vestibular habituation is based on the neurological theory that repeated exposure to a noxious stimulus will eventually result in a reduction of the pathological response to that stimulus. Habituation exercises involve repeatedly placing the patient in positions that invoke their symptoms of vertigo to allow the nervous system to become accustomed to the noxious stimulus. In 1980, Brandt and Daroff reported success in relieving positional dizziness in patients through repeated general habituation exercises.[75] Norre[76] followed this work in 1987, and reported even greater success with a customized program of habituation exercises which targeted the particular positions and movements which invoked dizziness in their patients.[76]

The recovery of a stable gaze after either a unilateral or bilateral vestibular loss is thought to include the recovery of the VOR itself, as well as changes in the intensity and direction of saccadic eye movements (target to target), central preprogramming, visual tracking mechanisms (smooth pursuits), and limiting activities that involve movements of the head.[79-80] All of these mechanisms of recovery can be stimulated and enhanced through vestibular rehabilitation exercises and patient education.

Neither the eye-head movement exercises developed by Cawthorne[68] and Cooksey,[69] nor the habituation exercises developed more recently are specifically designed to address the balance problems that are noted in many vestibular patients. The recovery of postural stability depends upon the re-establishment of the VOR and VSR, as well as compensatory activation of the visual and somatosensory systems. Because postural stability depends on the function of all of these systems, it becomes difficult to

isolate the function of the VSR.[71] Each sensory system appears to have an optimal movement frequency range throughout which it serves as the primary mechanism for maintaining balance. The vestibular system has been found to function optimally at both high and low frequencies, however it is the sole stabilizing mechanism at high movement frequencies.[81-83] To date, research has supported these two mechanisms as the primary mechanisms for the recovery of postural stability: 1) improving vestibular responses, and 2) increasing reliance on visual and somatosensory cues.[71,77,80,84-86]

Adaptation was shown to be context specific with regard to frequency, meaning that the greatest improvements in function were seen at the training frequency.[87] For this reason, it has been suggested that vestibular adaptation exercises should be performed across a wide range of functional frequencies.[89] As previously stated, a normal gait pattern involves head movements with a frequency of approximately three Hertz, and the VOR is triggered by head movements of two Hertz of greater. Exercises designed to retrain the VOR and VSR must employ head movements at a minimum frequency of two Hertz, or must be performed during gait to insure the activation of the vestibular reflexes.[71]

Initial exercise programs consisted of head and eye movements performed in sitting and standing, and during various dynamic balance activities.[71] In the 1980's, vestibular therapy was advanced to include activities involving gaze stability during head movements (VOR training), and vestibulo-spinal reactions (VSR). Current vestibular rehabilitation protocols employ a variety of static and dynamic exercises to enhance adaptation within the vestibular system, and to teach alternative strategies for balance, using sensory input other than that obtained via the vestibular system.[70]

Studies on the efficacy of vestibular rehabilitation have shown that most patients treated with vestibular exercise protocols experienced at least mild improvements of their symptoms, with a few patients displaying a dramatic improvement or complete resolution of their symptoms.[70,88-90] In 1998, Cowand et al[70] administered the Dizziness Handicap Inventory (DHI) to 37 patients before and after vestibular physical therapy. There was a significant improvement in test scores after therapy in 78% of the subjects tested. The habituation exercises performed in the Cowand et al study, resulted in a significant improvement in the physical and functional activity subscores of the DHI, but did not significantly influence the emotional subscore.[70] Cowand et al[70] also found that patients with peripheral lesions demonstrated greater improvements than those patients who had displayed central vestibular dysfunction. This last finding supported the earlier work of Telian et al,[88] which stated that patients with bilateral peripheral vestibular dysfunction showed a minimal response to vestibular physical therapy.[88]

There are several factors that may affect the final outcome of a patient receiving vestibular physical therapy.[71] Studies have shown the following factors to be indicative of a more complete recovery in patients with vestibular dysfunction: 1) a stable, unilateral deficit, 2) symptoms provoked by movement, 3) less severe initial disability, or 4) a more recent time of onset.[71] Recovery may be delayed if the person has movement restrictions in the cervical spine, or if their visual input is diminished. Age-related changes in the vestibular, visual, and somatosensory systems can also affect the ability of the vestibular system to recover from injury.

A 1990 study by Shepard et al[91] found that patients with vestibular dysfunction related to head injury had a poorer prognosis than patients with a unilateral peripheral

vestibular dysfunction.[91] This study proposed that the decreased efficacy of treatment in patients with a head injury could have been related to the presence of injury to both the peripheral and central vestibular apparati.[91] Vestibular deficits may persist longer in head-injured patients due to the injury of central structures that would normaly assist with the processes of compensation and adaptation.

A better response to vestibular physical therapy was noted in patients who were given a customized, monitored therapy program,[73,92,93] versus patients who were given adapatation exercises and a home exercise program. A complete remission of symptoms was found in 85% of patients who received a customized exercise program, with exercises based on specific deficits that were noted during their testing. Only 65% of the patients performing a generic vestibular exercise program on their own, at home, showed a complete remission of their symptoms.[73,92,93]

A Disablement Model for the Treatment of Concussions

The disablement model was first proposed by sociologist Saad Nagi in 1965,[94] and has been adopted by the American Physical Therapy Association as the preferred approach to the evaluation and treatment of patients. Nagi's disablement model sought to outline the major pathways that lead from a disease or active pathology to the functional consequences which ultimately caused disability in the affected person. The purpose of the disablement model was two-fold; 1) it could be used to predict the effects of various disease processes and/or injury on physical function and individual roles in daily life, and 2) it could be used to identify personal and environmental factors that could either positively or negatively influence the patient's recovery.[95] The four components of the Nagi model were: 1) Active pathology, which involved the interruption of normal cellular

processes by a trauma, metabolic imbalance, disease process, etc…; 2) Impairment, which referred to a loss of abnormality at the tissue, organ, or body system level as a result of the preceding pathology; 3) Functional limitation, which represented decreased performance of the person as a whole, resulting from the previously noted impairments; and 4) Disability, defined as a restriction or inability to perform socially defined roles and tasks as a result of the preceding functional limitations.[94]

A 2000 study, performed by Gill-Body et al,[96] examined the relationships described in the Nagi disablement model, related to impairments, functional limitations, and disability in people with vestibular dysfunction. Impairments studied by Gill-Body et al[94] included complaints of nausea, oscillopsia (the illusion of objects moving in the environment), dizziness, vertigo, unsteadiness during standing and walking, and motion intolerance.[97] The functional limitations noted in the vestibular patients included difficulty with lower-extremity dressing, walking, driving, and other tasks involving head movements.[98] The disability noted in these patients involved an inability to work, and a reduced participation in social and leisure activities.[99] The tests commonly used clinically to measure balance impairments and functional limitations were able to explain between 12% and 78% of the variance in scores on the Dizziness Handicap Inventory (DHI), a commonly used measure of disability in patients with balance disorders.[96]

In a study examining the effects of a vestibular rehabilitation protocol on measures of postural stability and self-reported handicap, Murray et al,[100] found significant improvements in average stability scores, and in average scores on the DHI after a four-week period of therapy.[100]

The link between balance impairments and overall disability lends support to the argument that the balance dysfunction in these patients must be addressed to decrease the disability associated with vestibular dysfunction, and return people to their previous roles in society. The findings that vestibular rehabilitation can restore normalcy at the levels of impairment, functional limitation, and disability, speak to the efficacy of this type of physical therapy. The previously stated research supports the application of the disablement model to the treatment of vestibular dysfunction in athletes.

CHAPTER III

RESEARCH QUESTION

1) Will athletes participating in the sport of rugby show any signs of Chronic Traumatic Brain Injury (CTBI), as evidenced by poor performance on vestibular testing when compared to a group of control subjects?
2) Will rugby players who have suffered acute head injuries show improved vestibular function in response to a customized vestibular therapy protocol?

CHAPTER IV

HYPOTHESES

1) The rugby players tested in this study, despite the presence or absence of a history of head injury, will show signs of CTBI, as evidenced by poor performance on vestibular testing when compared to a group of control subjects.
2) The number and severity of head injuries previously sustained by the participating rugby players will be inversely proportional to their performance on the vestibular tests.
3) Rugby players who have suffered acute head injuries will show diminished vestibular function immediately after their injury, and will display improved vestibular function after receiving customized vestibular physical therapy.

CHAPTER V

METHODS

Subjects

The participants for this study were obtained via samples of convenience through the volunteer participation of both male and female members of local area rugby clubs, and members of the physical therapy program at Texas State University-San Marcos. Volunteer athletes were accepted for the study if they were between the ages of 18 and 45 and were actively playing rugby. Volunteer athletes were excluded from the study if they were unable or unwilling to come to the Texas State University-San Marcos Physical Therapy Department for assessment and treatment purposes, or if they had been diagnosed with a concussion, vestibular disorder, or traumatic injury to the ankle or knee within three months prior to the study. Twenty-eight rugby players participated in the study, fifteen of these participants had no history of head injury, and thirteen had sustained head injury or injuries prior to the study. In the group with a history of head injury, four were females and nine were males. In the group with no history of head injury, six were females and nine were males.

Volunteers for the control group were accepted if they were between the ages of 18 and 45, and were excluded if they had ever played rugby, had ever suffered a head injury, or if they had suffered a traumatic injury to the ankle or knee within three months prior to the study. Seventeen volunteers from the physical therapy department at Texas State University-San Marcos were accepted for participation in the study, fourteen were females and three were males (Table 3).

There were four participants who sustained a head injury prior to undergoing their initial assessment at the physical therapy clinic at Texas State University-San Marcos. Because there was no baseline data on these participants which could be compared to the control group, their information was not included in the above mentioned participant groups, and the results of their testing was included in this study in the form of case study presentations.

Experimental Design

The study followed a pre-test, post-test, quasi-experimental design, with a control group. All members of the control group underwent testing at the physical therapy clinic at Texas State University-San Marcos during February, 2004. The participating athletes underwent testing at the physical therapy clinic at Texas State University-San Marcos during March and April of 2004. The athletes were then monitored until October, 2004 for the occurrence of any head injuries. If a participating rugby player suffered a head injury, he/she was asked to return to the physical therapy clinic at Texas State University-San Marcos for evaluation.

Table 3: Demographics of subject groups: Group 1 = control group of peers, Group 2 = Rugby players without a history of brain injury, Group 3 = Rugby players with a history of brain injury.

Group	Females	Males	Avg. Age	Yrs. Playing Rugby	Avg. # Brain Injuries
1 (control)	14	3	26	0	0
2	6	9	29	1.9	0
3	4	10	30.5	4.0	1.86

Instrumentation

All of the objective measures used during this research have been validated for use in the screening and assessment of balance disorders and vestibular dysfunction. All of the tests are simple clinical tests that require little training to administer or to score. The data obtained on the control group of healthy participants was used to obtain a measure of intra-rater, test-retest reliability on all of the tests that were administered. In the control group, the only measure that displayed variability was the Modified Combined Test of Sensory Interaction in Balance (MCTSIB). An intra-class correlation was performed on the results of three consecutive tests of the MCTSIB on three subjects, and the intra-rater reliability was determined to be R=0.911 (95% CI=0.396-0.998) when measuring postural sway velocity in degrees per second, and R=0.958 (95% CI=0.648-0.999) when measuring the percentage explored of the limits of stability (LOS).

The measures of impairment that were administered during this study included the Romberg and Tandem Romberg Sway Analyses, the clinical test of Dynamic Visual Acuity (DVA), and the MCTSIB. The measures of functional limitation that were assessed included the Dynamic Gait Index (DGI) (Appendix C), and the Patient-

Specific Function Scale (PSFS) (Appendix D). The Dizziness Handicap Inventory (DHI) (Appendix E) was administered as a measure of disability.

The Romberg sway analysis is an easily performed, qualitative test of balance. The Romberg sign was initially described by Moritz Heinrich Romberg in 1846, in his published clinical observations of patients with neurological disorders.[60] A positive Romberg sign involved an increase in postural sway when a patient was asked to stand with their eyes closed, and was initially attributed to a dorsal column lesion, tabes dorsalis.[61] The first application of Romberg's test for the assessment of vestibular disease was by Barany in 1910.[101] A simple Romberg's test was found to be insensitive in the detection of minor chronic vestibular lesions, and a sharpened or tandem Romberg was proposed by Fregly in 1974.[102] Fregly noted that patients with either bilateral or unilateral vestibular deficits were unable to sustain an upright position with their feet in tandem and their eyes closed for 30 seconds. Both the Romberg and Tandem Romberg are still commonly used as a clinical tool for the screening of balance disorders related to vestibular dysfunction. There is still a great deal of variability in how the Romberg is administered and scored, the methods used during this research are described in Test Administration.

The clinical test of DVA is a simple procedure that can be used to assess vestibular function, specifically the function of the VOR.[64] The DVA measures visual acuity during horizontal head movements; its sensitivity for determining vestibular deficits is approximately 85%, and its specificity is 55%.[103] While the computerized test of DVA has been found to have higher sensitivity and specificity (97% and 94%, respectively for those under 65 years of age), the clinical test is easy

to administer and is considered sufficiently reliable for use in guiding the treatment of patients with vestibular disorders.[103] DVA was tested by the principal investigator with the use of a Snellen ten foot eye chart (Appendix F), the methods used during this research are described in Test Administration.

The MCTSIB is similar to the SOT discussed previously, and quantitatively tests the function of each of the sensory systems, visual, somatosensory, and vestibular, that contribute to balance during quiet stance. The Combined Test for Sensory Interaction in Balance (CTSIB) was developed by Shumway-Cook and Horak, in 1986,[102] and includes six conditions, similar to those used in the SOT. The modified CTSIB (MCTSIB) eliminates the two conditions which produce visual conflict with a sway-referenced visual surround, as these conditions were found to produce redundant results.[105] In addition to the high intra-rater reliability reported earlier, the CTSIB has a reported test-retest reliability of 0.99, in young healthy adults.[105] The CTSIB has been reported to be 90% sensitive and 95% specific in the assessment of adults with vestibular disorders.[106] The methods used to perform the MCTSIB during this research are described in Test Administration.

The Dynamic Gait Index (DGI) was originally developed to assess dynamic postural stability in older adults who were at risk for falling.[107] The test consists of eight tasks such as walking at different speeds, walking with head movements and walking over and around obstacles. The test is scored on a four-level ordinal scale, with a zero implying an inability to perform the task and a four implying an unflawed performance of the task. The DGI has a maximum possible score of 24, and a score of 19 or less has been linked to an increase in fall risk in older adults.[107] The DGI has

been shown to be valid in the discrimination of patients with vestibular disorders who report falls and those that do not.[108] The reliability of the DGI on a population of people with vestibular disorders, was tested by Wrisley et al in 2003,[109] and the test re-test reliability of the DGI was found to be 0.68, considerably lower than that found earlier by Shumway-Cook et al (0.98).[107] Despite the lower reliability of the DGI in populations with vestibular disorders, this test is a simple, inexpensive, and valid way to screen patients for vestibular dysfunction. The precise methods of performing the DGI are discussed in Test Administration.

The PSFS was developed by Stratford et al in 1995, as a means of measuring a patient's function and related disability.[110] The PSFS is designed as an outcome measure, to be used in conjunction with condition-specific measures.[111] The test-retest reliability coefficient of the PSFS is R=0.84 (95% CI= 0.78-0.88).[111] The validity of the PSFS as determined by its sensitivity to change is R=0.77 (95% CI=0.61-0.89).[111] The PSFS was suggested to be a practical and efficient tool for the documentation of changes over time that are often missed with generic health status measures.[111] The method of administering the PSFS in this study will be discussed in Test Administration.

The DHI was developed as a disability measure by Jacobson and Newman in 1990,[112] to assess a patient's perception of the impact of imbalance and dizziness on various components of their life. The DHI is composed of 25 questions, divided into three categories examining the self-reported impact of dizziness and imbalance on emotional, functional, and physical aspects of the patient's life. Internal consistency reliability for the DHI was determined to be 0.89, and the test-retest reliability was

calculated to be R=0.97.[113] The DHI was found to significantly correlate with computerized posturography tests,[113] validating its use on patients with balance disorders of a vestibular nature.

During the initial screen the participant's blood pressure was measured using a standard aneroid sphygmomanometer and stethoscope. The participant's height and weight was obtained through self-report. The visual/oculomotor screen was conducted by the principal investigator using a ball point pen as a visual target.

Test Administration

Approval for the use of human subjects in this study was obtained from the Institutional Review Board at Texas State University-San Marcos, in San Marcos, Texas. The location of testing was in the physical therapy clinic at Texas State University-San Marcos, in San Marcos, Texas. Upon their first visit to the lab the participants were asked to sign an informed consent form (Appendix G); a copy of this signed form was then given to the participant. An identification number was assigned to each participant to be used on all data collection sheets, in order to insure anonymity throughout the course of the study.

The volunteers for the control group were obtained through a verbal announcement to the physical therapy department at Texas State University-San Marcos. To obtain participants for the athlete groups, a message was sent via email to all of the collegiate and club rugby teams in the Austin, San Marcos, and San Antonio areas, which explained the study and included a copy of the informed consent form (Appendices G and H). The principal investigator then visited each of

the local teams to explain the study in greater detail, and to schedule volunteer participants for their initial assessment.

All participants received an initial assessment at Texas State University-San Marcos to determined baseline values of balance and vestibular function, and to rule out any exclusionary criteria. The initial assessment consisted of the following: 1) completion of a personal information and medical history intake form (Appendix I), 2) blood pressure measurement, 3) visual/oculomotor screen, 4) Romberg sway analysis, 5) Tandem Romberg sway analysis, 6) DGI, 7) DVA, and 8) MTCSIB.

A blood pressure recording was taken prior to any testing, to provide a baseline value for the participant. This information was to be used in the event that the participant suffered a head injury. A significant drop in blood pressure, post-concussion, could be indicative of an intracranial hemorrhage and would indicate the need for referral of that patient for emergency medical treatment. The visual/oculomotor screen consisted of testing of the peripheral visual field, smooth pursuits, saccadic eye movements, convergence/divergence, and observing for the presence or absence of spontaneous nystagmus. The visual/oculomotor screen was performed to rule out the presence of any deficits that may have indicated the presence of a central nervous system lesion, and a need for referral to a medical professional for assessment.

As discussed previously, there is a large degree of variability in the method of performing a Romberg sway analysis. For the purposes of this research, during the Romberg and Tandem Romberg sway analyses, the participants were asked to stand, unsupported, with their feet together (Romberg) or in tandem (Tandem Romberg),

their arms crossed over their chest, and their eyes closed. For the Tandem Romberg, the participants were allowed to stand with either their right foot or the left foot in front, according to their preference. The test was quantitatively scored on the percentage out of 30 seconds that the participant was able to maintain their balance, without taking a compensatory step in any direction. The participant was allowed two attempts at both the Romberg and tandem Romberg, and the best of the two scores was recorded on the data sheet for each test (Appendix J). Both tests were administered by the principal investigator, with the use of a stopwatch.

For the administration of the DGI, the principal investigator measured a twenty-foot path on the floor, marked by tape at either end. The test was then administered according to the instructions provided. A standard shoe box was used as the obstacle for the participant to step over, two standard six-inch soccer cones were used for the participants to step around, and a set of five six-inch steps in the hallway of the first floor of the Texas State Health Science Center were used for the stair climbing activity. The participants were closely supervised by the principal investigator during all activities to minimize any potential safety risks. The total score out of a possible 24 points was recorded on each participant's data sheet.

Prior to assessing DVA, the participant's static visual acuity (SVA) was evaluated by having him/her sit in a chair placed ten feet from a Snellen eye chart positioned at eye level on the wall, and recording the lowest line that could be read. The participant was then instructed on the procedure of the DVA; they were asked to close their eyes and relax their neck to allow the principal investigator to tilt his/her head slight forward, aligning the horizontal semicircular canals with the horizontal plane.

The principal investigator then gently oscillated the participant's head horizontally, within an arc of 30 degrees, at a rate of two Hertz, for a duration of five seconds. While continuing to oscillate the participant's head, the principal investigator then asked the participant to open his/her eyes and read the lowest line possible on the Snellen eye chart. Performance on the test was then scored as the difference between the number of the line read statically and the line read dynamically. If a line was read with more than two letter errors, the participant was scored at the line above that which was read. A difference of greater than two lines was considered failure of the test. The use of prescription eye glasses or contacts was noted, and the participant was asked to wear their corrective eye-wear during the test.

The MCTSIB was conducted by the principal investigator using the Smart Balance Master which is a computerized force plate developed by NeuroCom, Inc. Information regarding the participant's age and height was entered into the computer for purposes of comparing the participants' scores to age-related norms. The participants were then asked to stand on the force plate and their feet were aligned according to the procedure outlined by NeuroCom. The participants completed three ten second trials in each of four conditions; 1) standing on a firm surface with eyes open, 2) standing on a firm surface with eyes closed, 3) standing on a foam surface with eyes open, and 4) standing on a foam surface with eyes closed. The participants were also assessed in two conditions with the addition of a head-shaking component, one on a firm surface and one on foam, both with the eyes closed. The participants were asked to shake their head side to side within a 30 degree arc, similar to that used during the test of DVA. A demonstration of the head-shake was provided, and a

metronome set at one Hertz was used to help the participants maintain their rate of oscillation. While these final two conditions have not been validated, the purpose of the head-shake was to provide the central processing center with incorrect vestibular information that the subject was actually moving, while in fact they were standing still, thus creating sensory conflict. The DVA and the MCTSIB produce dizziness in some people, so these tests were performed last to eliminate the influence of any symptom provocation on the rest of the tests results.

The values that were analyzed from the results of the MCTSIB were the sway velocities (in degrees per second) of the first four conditions, the percentage of the limits of stability (LOS) which were explored during the first four conditions, and the composite sway velocity (in degrees per second) of the first four conditions. Appendix K displays a sample of the results obtained from the MCTSIB.

The results of these tests provided the baseline values for each of the participating athletes should they suffer a head injury and require further assessment, and were compared to the values obtained for the control group. Before completion of the initial screen, the participants were provided with a handout entitled "How Do I Know If I Have A Concussion" (Appendix L) to familiarize them with the symptoms of a concussion. The participating athletes were then followed throughout the rugby season from February, 2004, until October, 2004, and were asked to contact the principal investigator via telephone or email, if they suffered any type of rugby-related head injury.

Once a participant suffered a head injury, they were asked to return to the physical therapy clinic at Texas State University-San Marcos within 48 hours of their

injury, to assess the severity of the injury. The post-injury assessment included the following: 1) subjective questioning regarding the mechanism of injury and any existing symptoms (Appendix M), 2) blood pressure measurement, 3) visual/oculomotor screen, 4) neurological screen (Appendix M), 5) cranio-vertebral screen (Appendix M), 6) Romberg sway analysis, 7) Tandem Romberg sway analysis, 8) DGI, 9) DVA, 10) MCTSIB, 11) PSFS, and 12) DHI.

The post-concussion evaluation included the addition of the PSFS and the DHI. The instructions for both of these tests were provided at the conclusion of their objective testing, and the participants were asked to complete the forms and return them to the principal investigator at that time.

For the PSFS the participants were asked to identify three activities of daily living (ADLs), and three rugby-related activities, with which they were having difficulty as a result of their concussion. The participants were then asked to rate their ability to perform the tasks on a scale of zero to ten, with zero being unable to perform the task at all, and ten being able to perform the task as they had prior to their injury. The participant's scores for each set of three activities (out of 30 possible points) was then recorded on the data sheet. The participants were then asked to complete the DHI according to the directions provided. For every "Yes" response provided, the participant was given a score of four points. For every "Sometimes" response, they were given a score of two points, and for every "No" response, they were given a score of zero points. The total points for each subscale, as well as the overall total out of 100 points, was then calculated and recorded on the data sheet.

If any of the above listed tests showed positive results relative to the baseline measurements taken at the initial assessment, the participant was referred to a physician and physical therapist for a medical diagnosis. The evaluation performed by the physician and physical therapist included the following tests: 1) Audiogram, 2) Electronystagmography (ENG), 3) VORTEQ assessment of vestibular reflexes and oculomotor function, and 4) Equitest; computerized dynamic posturography.

The audiological assessment was performed to identify the presence of any sensorineural hearing loss. Hearing loss and dizziness can often accompany each other as symptoms of various vestibular disorders including Meniere's disease, labyrinthine fistula, ototoxicity, and acoustic neuroma.[115, 116] It is thus necessary to rule out the presence of any of these disorders, in order to assess the vestibular dysfunction that is truly the result of the concussion for which the participant is being evaluated.

Both the ENG and VORTEQ assessments were performed using software (version 8.0) designed by Micromedical Technologies, Inc. Chatham, Illinois. Electronystagmography (ENG) is a method of quantitatively measuring the pathologic nsytagmus that may be present with dysfunction of the vestibular system,[117, 118] and provides a permanent record of eye movements which can be compare to normative data from a population. A spontaneous or positional nystagmus of greater than four degrees per second is commonly considered a sign of vestibular impairment.[118, 119] The VORTEQ, standing for VOR Technology, is a test of the vestibulo-ocular reflex during active head rotations both in the horizontal and vertical directions. The participant was seated with a target placed at eye level on the

wall in front of them, as specified by the directions accompanying the VORTEQ (Appendix N). Electrodes were attached to five locations on the participant's face to measure the activity of the oculomotor muscles. The participants were asked to shake their heads from side to side with increasing frequency, while maintaining the target in their visual field. The participants completed three trials of 30 seconds in both the horizontal and vertical planes, and were then scored on the coordination of their eye and head movements.

The first score reported by the VORTEQ is gain, the ability of the eyes to move in the opposite direction of the head movement but with the same magnitude as the head movement, thus allowing the eyes to stay focused on the target in front of them. For every degree the head moves to one direction, the eyes must move an equal number of degrees in the opposite direction. If this is performed perfectly, the VORTEQ would measure a gain of 1.00. If a gain of less than 1.00 were reported, it would indicate that the eyes were not moving as much as the head, and if a gain of more than 1.00 were recorded it would indicate that the eyes were moving more than the head. Scores falling outside the normal range for gain would indicate a deficit in some aspect of the VOR.

The second score reported by the VORTEQ is asymmetry, which measures the coordination between the left and right eyes during the head movement. The values for asymmetry are reported as percentages of weakness, so that a value of 2% for the left eye would indicate that the left eye is functioning at 98% of the strength of the right eye.

The third and final value reported by the VORTEQ is phase, which is a measure of the delay in the time component of the VOR. The phase refers to the amount of time required for the received stimulus (vestibular simulation through rotation of the head) to result in a physiological output (compensatory movement of the eyes). The VORTEQ reports these values as degrees of either lag or lead, meaning that the eyes are either behind or ahead of the head, at the time when the head and eyes reach their center-most orientation, directly in front of the focal target. Again, a phase value outside of the normal range for either lag or lead, would indicate a dysfunction in some aspect of the VOR.

The Equitest is a computerized version of the SOT, and was designed by NeuroCom, Inc. for use with a Smart Balance Master force plate. The Equitest assesses the function of the patient's visual, somatosensory, and vestibular systems during the six conditions previously described in the SOT.

The tests performed by the physician and physical therapist were more highly diagnostic than those performed in the clinical evaluation of the post-concussion participants. The diagnostic testing was used to confirm any vestibular findings noted during the clinical testing, and to catch any dysfunction that was not identified by the clinical evaluation. The evaluation performed by the physician and physical therapist was also used to determine the sensitivity and specificity of the clinical test battery used in this study in the identification of vestibular deficits in post-concussion athletes.

If it was determined by the physician that the participant had suffered a concussion, the participant was then provided with a customized vestibular physical

therapy exercise program, and he/she was scheduled for follow-up assessments at one week, two weeks, three weeks, and four weeks post-injury. The follow-up reassessments included all of the testing performed during the post-injury assessment. The vestibular physical therapy protocol was customized for each participant based on the specific impairments and functional limitations recorded during their assessment. The customized protocol consisted of five exercises selected from ten commonly used vestibular rehabilitation exercises (Appendix O). These vestibular exercises were performed once each day for the four weeks following the post-injury assessment.

Data Analysis

Statistical analysis was performed on the collected data using the Statistics Package for the Social Sciences (SPSS) (version 10.1 for Windows). The participants were divided into three groups for the purposes of data analysis: 1) rugby players with a history of head injury, 2) rugby players with no history of head injury, and 3) a control group of peers who had never played rugby and had no history of head injury. The performances of the rugby players with and without a history of head injury were analyzed as separate groups in order to evaluate the presence of any vestibular dysfunction that might be attributable to the cumulative effects of subconcussive blows to the head, as described previously in soccer players with CTBI.[12,42]

Significance for all statistical analyses was set at α=0.05. A Pearson correlation coefficient was calculated to determine the nature of the relationship between the performance of the participants on each of the following measures 1) each of the baseline assessment measures, 2) the number of years the participant had played

rugby, 3) the number of head injuries the participant had sustained, and 4) the number of moderate or severe head injuries the participant had sustained, as indicated by a period of lost consciousness.

A two-tailed independent t-test was also performed between the three participant groups, to determine the existence of significant differences between mean scores on each of the four groups of measures listed previously. In addition to comparing the means of the three separate groups, a two-tailed independent t-test was also performed to compare the performance of the entire group of rugby players, regardless of their history of head injury, to the performance of the control group on all of the baseline assessment measures.

CHAPTER VI

RESULTS

The correlational analysis found significant relationships between the number of years the participants had played rugby and the number of head injuries they had sustained (r=0.508, sig.=0.000) (Figure 6), as well as the number of severe head injuries they had sustained as indicated by a period of lost consciousness (r=0.445, sig.=0.002) (Figure 7).

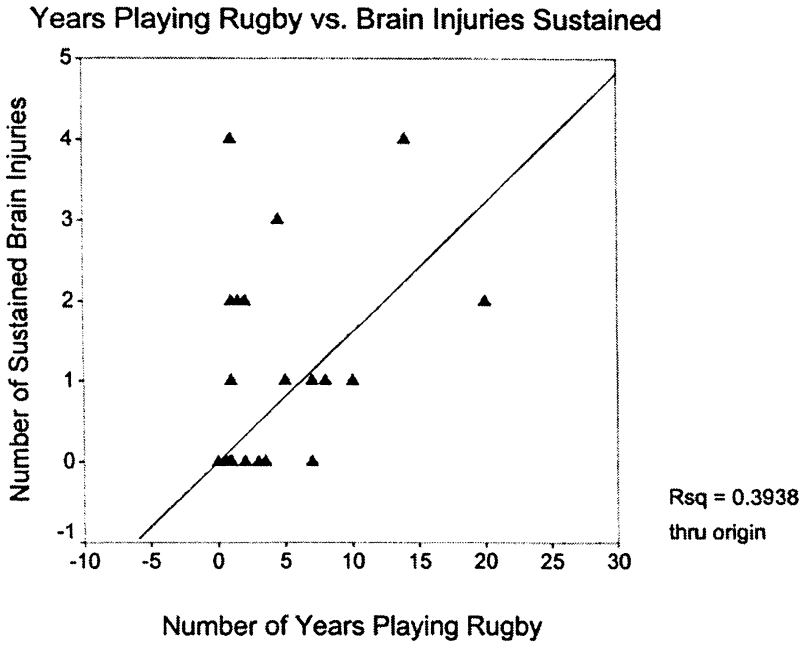


Figure 6: Number of brain injuries sustained relative to the number of years playing rugby.

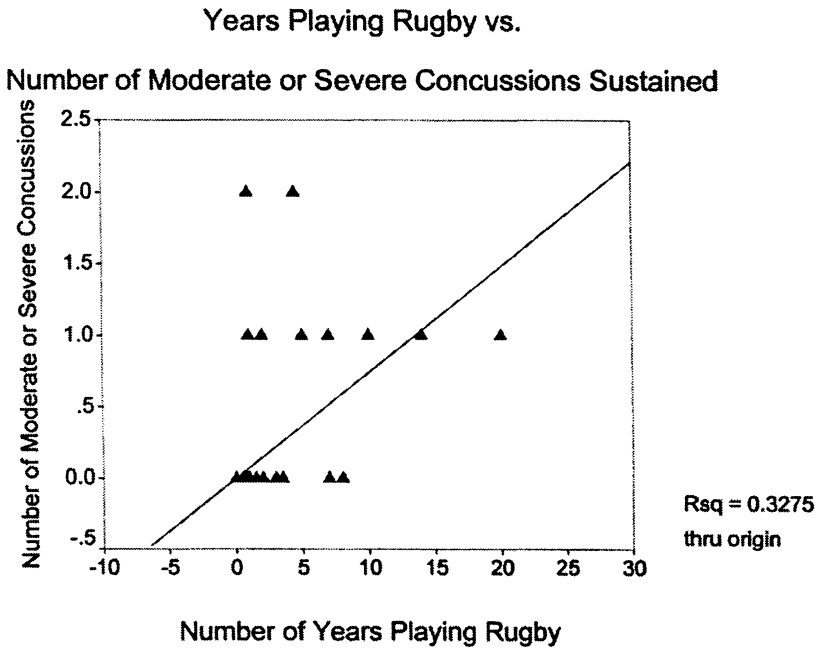


Figure 7: Number of moderate or severe concussions sustained (as defined by a period of lost consciousness) relative to the number of years playing rugby.

The number of head injuries sustained by the participants was found to be inversely related to their ability to perform the Tandem Romberg (r=-0.326, sig.=0.031), indicating that those players who had sustained a greater number of head injuries performed worse on the Tandem Romberg (Figure 8).

The sway velocity recorded during condition six of the MCTSIB (standing on foam surface, eyes closed, with head shake) was found to increase with the number of head injuries sustained by the participants (r=0.383, sig.=0.010) (Figure 9).

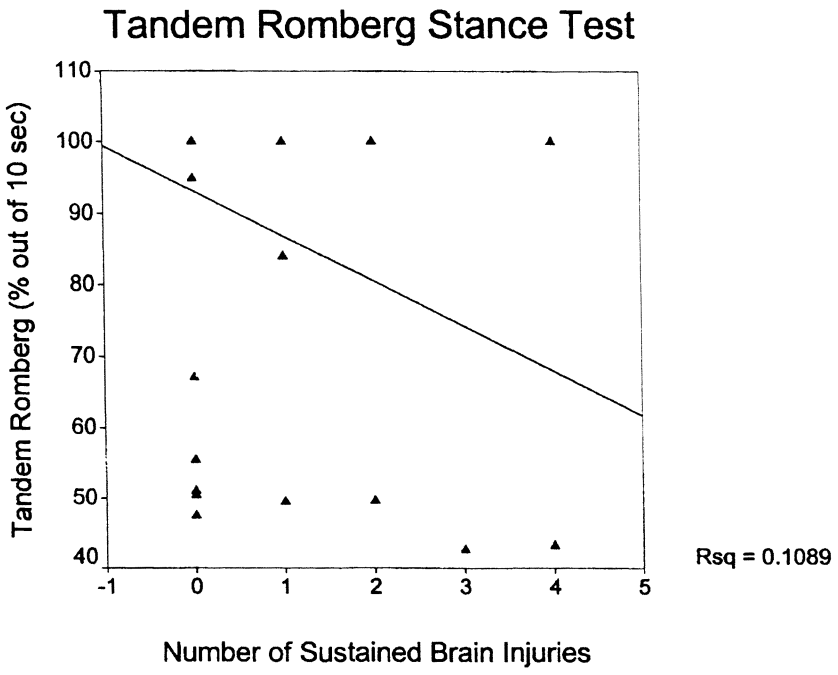


Figure 8: Performance on the Tandem Romberg stance test relative to the number of brain injuries sustained by the participant.

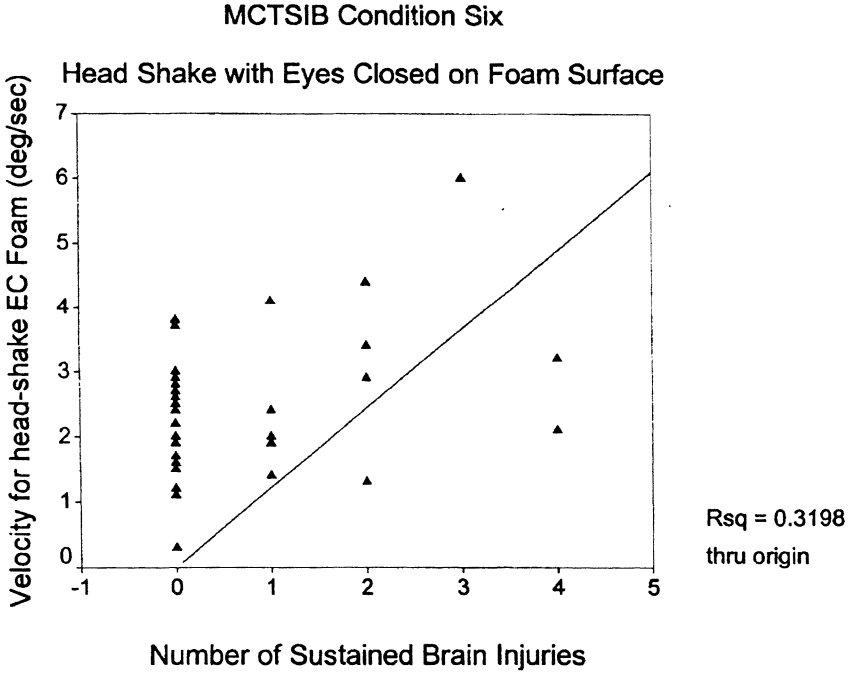


Figure 9: Sway velocity during condition six of the MCTSIB relative to the number of brain injuries sustained.

The sway velocities on both conditions three (standing on foam surface with eyes open) and six, were found to increase with the number of severe head injuries sustained by the participants, as indicated by a period of lost consciousness (r=0.298, sig.=0.050, and r=0.445, sig.=0.002 for conditions three and six, respectively) (Figures 10,11).

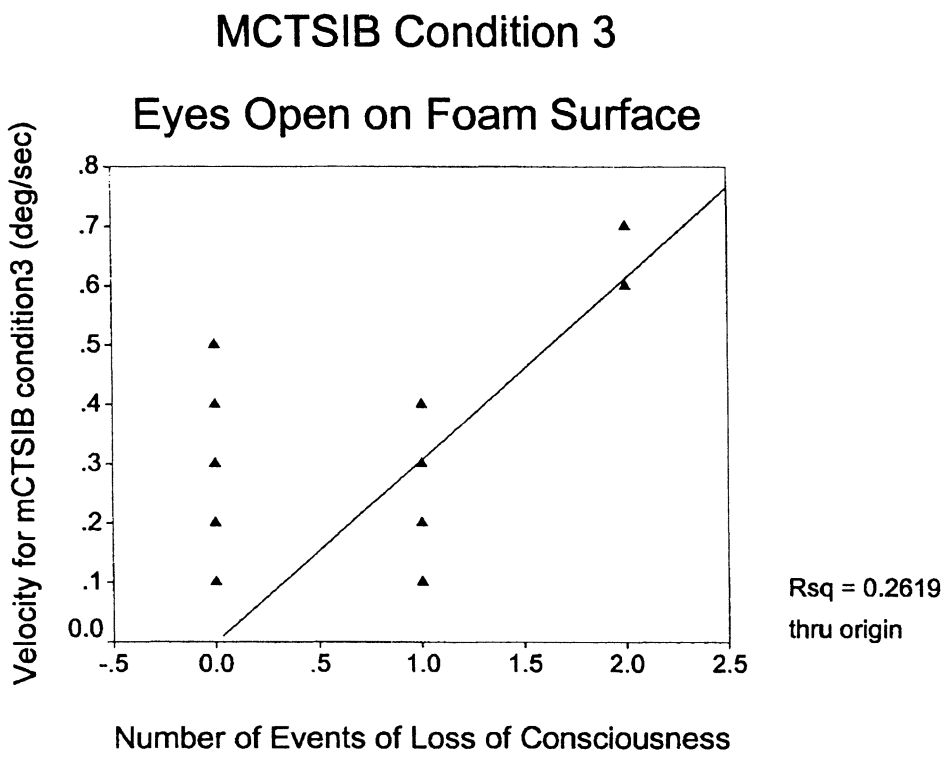


Figure 10: Sway velocity during condition three of the MCTSIB relative to the number of moderate or severe concussions sustained (as defined by a period of lost consciousness).

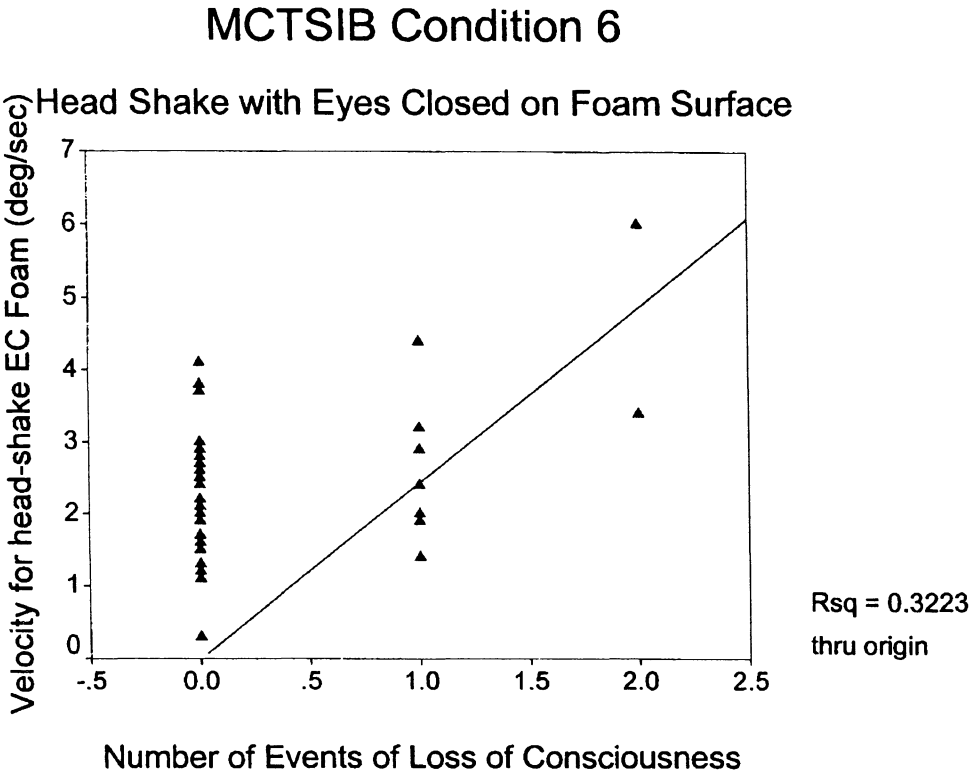


Figure 11: Sway velocity on condition six of the MCTSIB relative to the number of concussions sustained (as defined by a period of lost consciousness).

In comparing the means of the three groups of participants on each of the performance measures, there were significant differences noted on the DVA (α=0.000) (Figure 12), and differences approaching significance on condition six of the MCTSIB (α=0.058) (Figure 13). Both groups of rugby players performed significantly worse on these measures than the control group, and the group of rugby players with a history of head injury performed significantly worse than the group with no history of head injury.

One-way ANOVA

Measure	Sum of Squares	Degrees of Freedom	Mean Square	F	Sig
Romberg	0.000	2	0.000	*	*
Tandem Romberg	1022.071	2	511.035	1.258	0.295
MCTSIB 1 (velocity)	0.133	2	0.066	0.693	0.506
MCTSIB 2 (velocity)	0.026	2	0.013	1.533	0.228
MCTSIB 3 (velocity)	0.012	2	0.006	0.384	0.683
MCTSIB 4 (velocity)	0.052	2	0.026	0.083	0.921
MCTSIB 5 (velocity)	0.152	2	0.076	1.017	0.371
MCTSIB 6 (velocity)	5.617	2	2.809	3.064	**0.058****
Composite velocities 1-4	1.467	2	0733	1.429	0.251
Limits of Stability 1-4	105.073	2	52.537	0.350	0.707
DVA	131.813	2	65.907	44.179	**0.000*****
DGI	0.000	1	0.000	*	*

* Results were identical for all subjects tested.
** Result approaching significant values.
*** Significant result.

Table 4: Results of one-way ANOVA, comparing mean scores of the three independent subject groups on each of the assessment measures.

When the results of the rugby players as a whole (groups 2 and 3 combined) were compared to the results of the control group, the rugby players performed significantly worse on condition 6 of the MCTSIB (sig.=0.039).

Graphic representations of the non-significant correlations and t-test comparisons can be found in Appendix P.

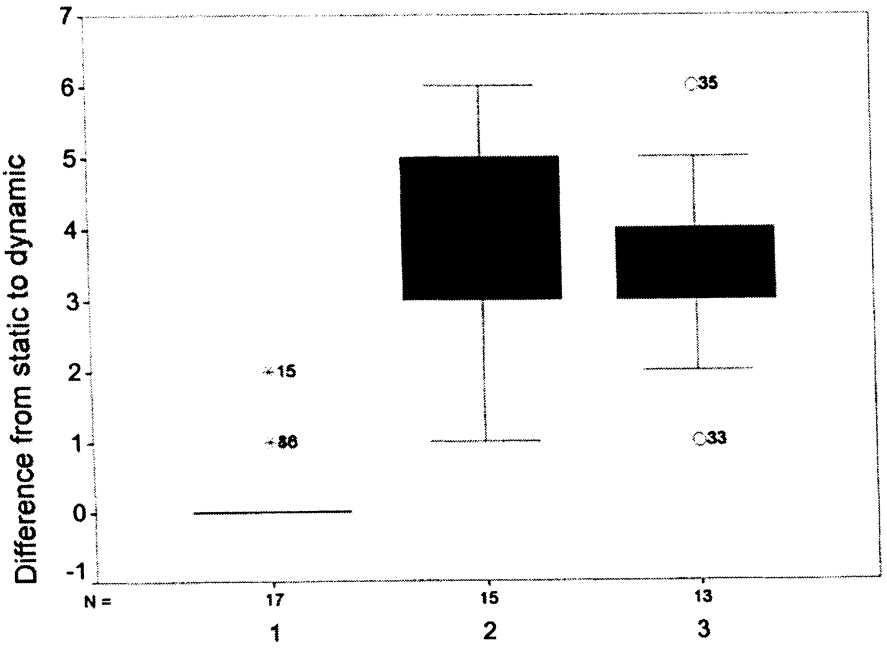


Figure 12: Difference in the number of lines read statically and the number of lines read dynamically during the DVA in each of the three subject groups.

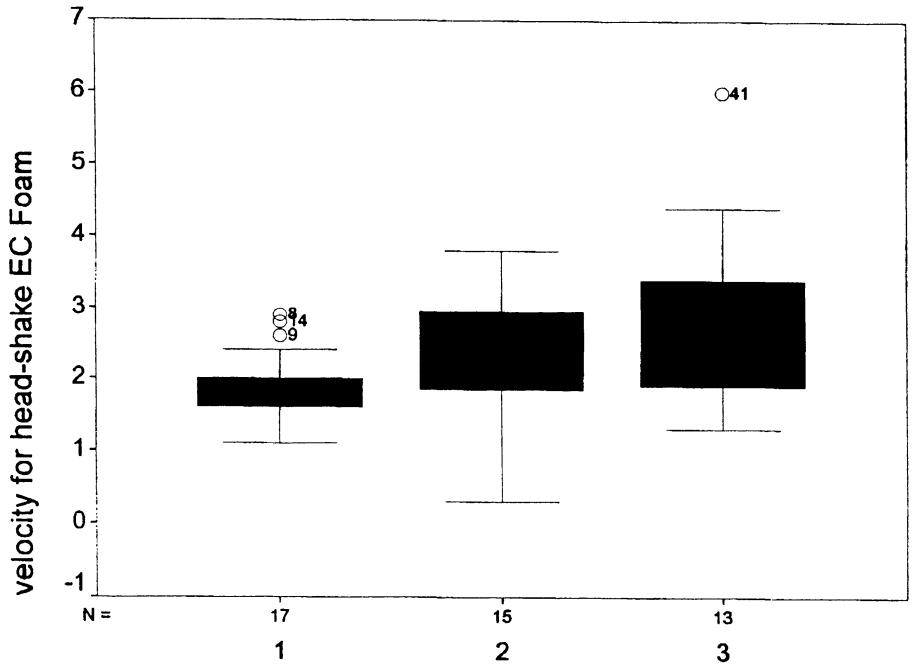


Figure 13: Sway velocities during condition six of the MCTSIB (head shake with eyes closed on the foam surface) for each of the three subject groups.

CHAPTER VII

DISCUSSION

The first hypothesis, that the group of rugby players would show signs of CTBI as evidenced by poorer performance on vestibular tests when compared to a control group of peers, was accepted. The overall results indicate the presence of some long-lasting neurological impairments associated with the cumulative effects of the repeated subconcussive and concussive blows that are commonly suffered by people participating in the sport of rugby. The specific areas of impairment were in the DVA test of the VOR, and the most challenging condition of the posturography assessment. When the group of rugby players as a whole was compared to the control group, the differences were found to be more statistically significant, indicating that a larger sample size might have brought out more significant differences in other measures as well.

The second hypothesis, that the number of MTBIs sustained by the participating rugby players would be inversely proportional to their performance on the vestibular tests, was also accepted. An increased number of MTBIs was related to poorer performance during the tandem Romberg sway analysis, condition 3 of the MCTSIB, and the most challenging condition of the posturography assessment.

These results provide an addition to the Matser et al studies,[12,42] which found persistent neurological impairments in the areas of memory and planning in both professional and amateur soccer players both with and without a history of concussion. It appears that participants in both rugby and soccer are at risk for long-term sequelae associated with Chronic Traumatic Brain Injury.

Not surprisingly, the correlational analysis also found significant relationships between the number of years the participants had played rugby and the number of brain injuries they had sustained. These results could imply that the longer a person plays rugby the more likely they are to suffer some type of traumatic brain injury.

While there is a risk for sustaining long-lasting neurological damage resulting in vestibular impairments, there is also evidence that these impairments may be successfully treated through a customized vestibular therapy protocol. The third and final hypothesis, that the rugby players who suffered acute MTBIs would show decreased vestibular function following their injury, and an improvement in vestibular function following the prescription of a customized vestibular therapy protocol, could not be accepted due to an insufficient number of participants. However, a detailed description of each of the participants who suffered a concussion during the study is included in this study in the form of case studies.

One of these case studies was reassessed throughout several weeks following their initial post-injury assessment and this participant displayed improvements in all vestibular test scores after only a week of performing a home exercise program. Future research should involve a larger sample of post-concussion athletes, randomly divided into a treatment group and a non-treatment control group, to determine if

these improvements in vestibular function were truly the result of treatment or if they were simply the result of recovery time.

The presence of vestibular impairments in rugby players without a history of head injury, may indicate a need for the implementation of vestibular physical therapy exercises into the daily exercise program of these athletes. Future research may investigate the efficacy of such vestibular physical therapy exercises in groups of rugby players and other athletes, and observe the relationship between the vestibular function of these athletes and their risk for other injuries.

During completion of the study there were several limitations that became apparent. The participants were required to be able to travel to San Marcos for their initial evaluation, and for their post-injury follow-up. In addition, the participants who suffered acute MTBIs were required to drive to Austin for the follow-up evaluation with the physician and physical therapist. Because of the need for a physician referral to prescribe a vestibular therapy protocol, the participating subjects were required to either have insurance coverage for the referring physician, or they were required to pay for the physician testing themselves. As many of the participants were college-aged students, the ability to pay for the physician visit was a limiting factor.

The delimitations of the study dealt with the selection of the participant groups. The study was limited to the participation of rugby players, between the ages of 18 and 45, and from the central Texas region. It would be valuable to test participants in other sports, as well as younger and older athletes throughout different regions, for signs of vestibular impairment related to CTBI. This study also focused solely on

vestibular impairments, while there are also many other areas of neurological function that might be affected by CTBI.

Another delimitation of this study was the lack of control for gender, with the participant groups consisting of both males and females, under the assumption that gender would not have an influence on the vestibular function of the participants. The gender breakdown for both the rugby group and the control group was representative of their respective populations. There are more male rugby participants in the central Texas region than females, and the rugby participant group contained more males than females. The population of physical therapists in Texas is predominately female, and the control group of peers, which was drawn from the Texas State University-San Marcos physical therapy department contained more females than males.

Physicians, physical therapists, athletic trainers, neurologists, and neuropsychologists should be collaborating to insure that no element of function is forgotten during the evaluation and treatment of persons suffering from the acute mild traumatic brain injury of a concussion, or from the cumulative effects of repeated subconcussive and concussive blows to the head.

CHAPTER VIII

CASE STUDIES

Case Study 1: 2004AS04

This participant was a twenty-five year-old female who had been playing rugby for a total of four and one-half years, and playing locally for the past six months. In addition to playing rugby, she also lifts weight and either runs or swims three days a week. Her past athletic history included swimming, running track, and playing soccer, basketball, and baseball. At the time of the study she rated herself in excellent condition. Her significant past medical history included surgery on her right foot in 2001 to remove a tumor, a broken right foot in 2002, and arthroscopic surgery on her left knee in 1993. Her history of head injury included three concussions during the rugby seasons of 2002 and 2003. All three of these concussions were diagnosed by a physician, and per patient report, two of them were major and one was minor in severity. The participant met all study criteria in that she had not had any head injuries in the three months prior to the study, and she had not had any injuries to her knees, ankles, or hips in the three months prior to the study.

Upon her initial assessment, in March of 2004, this participant was found to have a normal systolic blood pressure of 108 mmHg, and a diastolic pressure of 64 mmHg. The participant was also clear on all aspects of the visual/oculomotor screen. She

was able to perform the Romberg sway analysis for the full thirty seconds, however she failed the Tandem Romberg with a maximum time of 4.26 seconds. The participant scored a perfect 24 out of 24 points on the DGI, however her DVA was five lines worse than her SVA, indicating a significant VOR deficit. During the first four conditions of the MCTSIB the participant was within normal limits for sway velocities, however her center of gravity was scattered throughout 21% of her limits of stability (Appendix Q). Although there are no normative values for the conditions of the MCTSIB involving a head-shaking component, the participant showed increased sway velocities on both the firm and foam surfaces, and fell during each of the three trials on the foam surface (Appendix Q). This could indicate some dysfunction in the vestibular reflexes which assist with postural stability during dynamic activities.

On June 5th of 2004, this participant suffered a blow to the head during a rugby game. The participant reported being hit by another player's knee while making a tackle. The participant did not report a loss of consciousness at the time of injury, however her symptoms included having spotty vision in both eyes for 10 to 15 minutes after the injury, and feeling foggy-headed for one week post-injury. The participant did not immediately report her injury to the principal investigator, nor did she seek any type of medical treatment. She returned to play rugby the following weekend and suffered a knee injury that ruptured the anterior cruciate ligament of her right knee.

The participant was seen on the 23rd of June for her post-injury evaluation, at the office of the referring physician. At the time of her post-injury evaluation her systolic

blood pressure was 116 mmHg, and her diastolic was 68 mmHg, indicating no sign of intracranial hemorrhage. All screens for cervical spine injury, neurological impairment, and visual/oculomotor impairment were negative. Due to the knee injury that had been sustained the week prior, no standing balance activities were performed including the Romberg and Tandem Romberg, the DGI, and the MCTSIB. The participant showed improved function from her baseline assessment on the DVA, with no differences noted between DVA and SVA, however she did report feelings of dizziness during and after the testing.

Because the participant was unable to undergo any standing balance activities, and because she had reported long-lasting symptoms of foggy-headedness after the injury, she underwent the more highly diagnostic testing provided by the physician. The audiologist was unavailable at the time of her testing, so the participant did not receive a sensorineural hearing assessment, nor did she undergo the Equitest of postural stability due to her recent knee injury. The participant did undergo the ENG assessment of nystagmus and oculomotor function, and the VORTEQ assessment of VOR function (Appendix Q). The participant showed no signs of spontaneous or gaze-induced nystagmus in either the horizontal or vertical planes, and her performance on the random saccades, smooth pursuits, and optokinetic testing were all within normal limits. During the horizontal testing portion of the VORTEQ the participant performed within normal limits for gain, asymmetry, and phase, although she did show some degree of lag and lower gains during higher frequency movements (Appendix Q). As a highly functioning athlete, this participant should display gains closer to 1.00 than the average person. During the vertical testing portion of the

VORTEQ the participant was unable to complete the testing due to complaints of headache caused by the rapid vertical movement. It is not uncommon for patients with vestibular impairments to have increased symptoms as a result of VOR testing, and the inability of this participant to complete the vertical component of this testing may have indicated some VOR dysfunction.

The PSFS and the DHI were not used on this participant because she was unable to define any functional activities of daily living or any rugby-related activities that she had been unable to perform as a result of her head injury.

This participant was scheduled to return for a repeat of the ENG and VORTEQ diagnostic testing one week from the evaluation. At that time, the vertical component of the VORTEQ was to be retested and an appropriate vestibular physical therapy protocol was to be designed for her. However, the knee injury that was sustained following her head injury, took precedence for the participant and she was immediately scheduled for reconstructive knee surgery and was thus unable to continue with any aspect of this study.

Case Study 2: 2004LS39

This participant was a twenty-seven year-old male who had been playing rugby with a local area team for the past seven years. The participant reported some history of head injuries, however, none of them had been previously diagnosed by a physician as concussions. He was unable to participate in the initial assessment prior to suffering a head injury on the 18th of May, 2004, however he contacted the principal investigator immediately after his injury and was scheduled for a post-injury

evaluation. The participant was seen for his post-injury evaluation on the 19th of May, 2004, in the office of the referring physician.

The participant reported that immediately after his injury he suffered from blurry vision lasting for several hours, and that from the time of injury to the time of evaluation he had suffered a persistent headache and had been unable to sleep. At the time of his post-injury evaluation, the participant had a systolic blood pressure of 128 mmHg and a diastolic pressure of 86 mmHg. While there was no baseline measurement with which to compare these values, they fell within a normal range and did not show the dramatically low blood pressure indicative of an intracranial hemorrhage. The screens for cervical spine injury, neurological impairment, and visual/oculomotor impairment were all negative. The participant was able to complete both the Romberg and Tandem Romberg stance tests for the maximum amount of time, however he did struggle to maintain his balance during both conditions. The participant completed the DGI with no difficulties and also passed the DVA test, with a one-line difference between his DVA and SVA.

Because the post-injury evaluation was performed in the physician's office, the additional diagnostic testing was performed on this participant (Appendix R). The participant tested within normal limits for all components of the audiological assessment indicating no sensorineural hearing loss. During the Equitest of postural stability the participant's composite score was within normal limits, however he failed two out of the three trials of condition five. The fifth condition of the Equitest primarily evaluates the function of the vestibular system by removing the participant's visual input (closing their eyes), and reducing their somatosensory input

(allowing the support surface to move with their body sway). During the Equitest, the participant showed appropriate balance strategy responses, however the location of his center of gravity varied along the anterior-posterior plane (Appendix R).

The ENG testing of this participant showed no spontaneous, gaze-induced, or position-induced nystagmus (Appendix R). The oculomotor testing of this participant showed slightly slow velocities in the right eye during the target to target tracking of random saccades, as well as slightly low gains during both smooth pursuits and optokinetic testing (Appendix R). During the horizontal component of the VORTEQ testing the participant showed low gains, especially during lower frequency movements, and showed high phase values during all frequencies (Appendix R). During the vertical component of the VORTEQ testing the participant showed gains that were low but within normal limits, and very low phase values during all frequencies indicating that the eyes were leading the head at each mid-point of the movement. The VORTEQ findings indicated some VOR dysfunction.

The PSFS and the DHI were not used with this participant because he was unable to list any functional activities of daily living, or rugby-related activities that he had been unable to perform since the injury.

Based on his evaluation, the participant received a customized vestibular therapy program to be performed daily at home (Appendix R). The exercises included 1) single-leg stance both with his eyes open and eyes closed, 2) tandem stance both with his eyes open and eyes closed, 3) seated VOR exercises in both the horizontal and vertical planes, 4) dynamic VOR exercises (head shaking while focusing on a stationary target) to be performed while running.

After performing the vestibular physical therapy program for one week at home, the participant returned to the referring physician's office on May 27th, 2004, for reassessment. The results of the reassessment may have been affected by a shoulder injury that the participant received while playing rugby on the 24th of May. The participant was able to complete all required testing, however he did report discomfort while performing the head-shaking actions required during the VORTEQ.

The audiological exam and the ENG were not performed due to the lack of significant findings on the initial post-injury test. The results of the VORTEQ reassessment in the horizontal plane showed an increase in both gain and phase during all tested frequencies, placing the gain within normal limits and the phase above normal limits during some of the tests (Appendix R). The results of the reassessment of the vertical component of the VORTEQ again showed an increase in gain to within normal limits during all frequencies, and a slight increase in phase that was still below normal limits during all tested frequencies (Appendix R).

Upon reassessment of the Equitest, the participant passed all six conditions with no indications of vestibular impairment (Appendix R). The participant also showed improved centralization of his center of gravity within the limits of stability (Appendix R). The participant was instructed to continue with his home exercise program, however he was unable to return for further reassessment.

Case Study 3: 2004CB17

This participant was a twenty-two year-old male who had been playing rugby locally for one year. At the time of his evaluation he was not participating in any other sports, and was not participating in any additional exercise outside rugby

practice twice a week. At the time of his evaluation the participant rated himself as being in good condition. The only significant medical history for this participant was a history of allergies and headaches.

At the time of his initial evaluation this participant had a systolic blood pressure of 130 mmHg and a diastolic of 88 mmHg. The initial screen for visual/oculomotor impairment was negative, and the participant was able to complete both the Romberg and Tandem Romberg stance tests, and the DGI without difficulty. The participant failed the DVA with a six-line difference between his DVA and SVA. During the four conditions of the MCTSIB the participant had sway velocities within normal limits and his center of gravity was within 24% of his limits of stability. During the fifth condition of the MCTSIB which included the head-shaking component the participant was within normal limits for sway velocity, however during the sixth condition he showed increased sway velocities (Appendix S). Once again, there are no normative values for MCTSIB conditions involving a head-shaking component, however increased sway velocities under these conditions might indicate a dysfunction in the vestibular reflexes.

On the 20th of March, 2004, this participant was tackled during a rugby game and hit the back of his head on the ground. There was no period of lost consciousness and the symptoms reported immediately following the injury included drowsiness, feeling in a fog, feeling slowed down, headache, sensitivity to light and noise, and vomiting. The participant reported the injury to the primary investigator the day after it occurred and scheduled an appointment for a post-injury evaluation.

The participant returned to the physical therapy clinic at Texas State University-San Marcos on the 24th of March, for his post-injury evaluation. The screens for cervical spine injury and neurological impairment were negative. The visual/oculomotor screen showed slight dysmetria during saccadic movements. The participant was again able to perform the Romberg and tandem Romberg stance tests, and the DGI with no difficulties. The participant showed a slight improvement in DVA with only a four-line difference between his DVA and SVA. During his reassessment the participant again showed sway velocities within normal limits during the first four conditions of the MCTSIB, and his center of gravity was located within 23% of his limits of stability (Appendix S). During the MCTSIB conditions with the head-shaking component, the participant was again within normal limits during condition five, but showed increased sway velocities during the sixth condition (Appendix S). The participant scored 24 out of 30 points on the PSFS for activities of daily living, and 30 out of 30 possible points on the rugby-related PSFS. On the DHI, the participant had a composite score of 10 out of 100 possible points, with two points in each of the functional and emotional categories, and six points in the psychological section of the survey.

The participant was unable to schedule time for diagnostic evaluation by the physician and physical therapist, and also lacked insurance to pay for the visit, so the principal investigator was unable to provide the participant with a vestibular physical therapy treatment protocol. The participant was asked to return to the physical therapy clinic at Texas State University-San Marcos for a follow-up assessment in one week.

The participant returned for his follow-up assessment on the 29th of March, 2004. At the time of the follow-up the only symptoms reported by the participant were headache, and sensitivity to light. All screens for cervical spine injury, neurological impairment, and visual/oculomotor impairment were negative. The participant was able to complete the Romberg and Tandem Romberg, and the DGI with no difficulties. The participant again failed the DVA, with a five-line difference between his DVA and SVA. There were no significant changes in sway velocities or location of the center of gravity on any of the MCTSIB conditions, with the sixth condition still showing a marked increase in sway velocity (Appendix S). At the time of the follow-up, both the PSFS and the DHI showed perfect scores with no deficits. The participant's performance during the follow-up assessment was not significantly different from that of the initial assessment, so no further follow-up visits were scheduled.

Case Study 4: 2004DW07

This participant was a twenty year-old male who had played rugby locally for two years. The participant stated that his athletic history included sports such as soccer, baseball, and tennis. At the time of his evaluation the participant stated that he was in excellent physical health, and that he exercised four days per week; swimming in addition to rugby practice. The participant had a significant medical history with several fractures and head injuries, and a recent history of dizziness, blackouts, and headaches. The participant had suffered two previous head injuries, the first of which occurred in September of 2003, and resulted in a loss of consciousness for ten seconds, per participant report. The second occurred in March of 2004, and resulted

in no loss of consciousness but a loss of memory for approximately fifteen minutes. The participant did not seek medical evaluation or treatment for either of his previous head injuries.

The participant was unable to participate in the pre-injury assessment prior to his second head injury, so no baseline values were obtained for this participant. The participant described the injury as occurring while he was running with the ball and was tackled, causing the back of his head to hit the ground. At the time of his evaluation the participant's systolic blood pressure was 118 mmHg, and his diastolic pressure was 68 mmHg. Without baseline values for comparison, no conclusions could be drawn about his blood pressure, however the values recorded were within a normal range. The visual/oculomotor screen was negative, and the participant was able to complete the Romberg and Tandem Romberg stance tests, and the DGI with no difficulties. The participant failed the DVA, with a four-line difference between his DVA and SVA, indicating a VOR dysfunction. During the MCTSIB the participant failed condition four (eyes closed on foam surface) (Appendix R), with a sway velocity of 2.2 degrees/second, and his center of gravity was scattered throughout 22% of his limits of stability. The participant also showed excessive sway velocities during both head-shaking conditions of the MCTSIB, recording one fall out of the three attempts at condition six (Appendix T).

This participant was a student at the time of his injury and evaluation, and did not have his own health insurance. The physician that was assisting us with our diagnostic testing and evaluation was not covered under the insurance plan of the participant's parents, and the participant was unable to pay for the cost of the

evaluation himself. This participant was therefore, not able to continue with any aspect of this study, however it was recommended by the principal investigator that he seek professional medical attention.

Case Study 5: 2004BN34

This participant was a twenty-seven year-old male who had been playing rugby for a total of seven years and playing locally for the past two years. At the time of his evaluation the participant was not participating in any other sports, but stated that he exercised an average of five days per week, and he rated himself as being in good physical health. The participant's significant medical history included previous head injuries that were undiagnosed by a physician, and arthroscopic surgery on his left knee to repair damaged cartilage in 1995.

This participant was unable to participate in an initial assessment prior to sustaining a head injury during a rugby game on March 28th, 2004. The participant received a blow to the right side of his head above his ear while tackling another player, resulting in a loss of consciousness for approximately thirty seconds. Immediately following his injury, the participant contacted the principal investigator and scheduled a post-injury evaluation. The participant was seen at the physical therapy clinic at Texas State University-San Marcos on the 29th of March, 2004.

At the time of his post-injury evaluation the only symptom reported by the feeling of being "dinged". All screens for cervical spine injury, neurological impairment, and visual/oculomotor impairment were negative. The participant was able to complete both the Romberg and Tandem Romberg stance tests, and the DGI without difficulty. The participant failed the DVA test with a three-line difference between his DVA and SVA.

The sway velocities and limits of stability were within normal ranges for all four conditions of the MCTSIB, including the two additional conditions with the head-shaking component (Appendix U). The PSFS and the DHI were not performed with this participant because he was not having difficulties with any activities of daily living or any rugby-related activities at the time of his post-injury evaluation. Because of the lack of significant findings during the post-injury assessment, no further evaluation or treatment was recommended to this participant.

APPENDICES

Appendix A: Postconcussion Signs and Symptoms Checklist[4]

Bell Rung
Depression
Dinged
Drowsiness
Excess Sleep
Fatigue
Feel "in a fog"
Feel "slowed down"
Headache
Inappropriate emotions or personality change
Irritability
Loss of consciousness
Loss of orientation
Memory problems
Nausea
Nervousness
Numbness/tingling
Poor balance or coordination
Ringing in the ears
Sadness
Seeing stars
Sensitivity to light
Sensitivity to noise
Sleep disturbance
Vacant stare/glassy eyed
Vomiting

Appendix B: Standardized Assessment of Concussion[28]

Table 1 Standardized Assessment of Concussion (SAC)

Orientation: (1 point each)

Month

Date

Day of week

Year

Time (within 1 hr)

Orientation score: 5

Immediate Memory: (1 point for each correct, total over 3 trials)

	Trial 1	Trial 2	Trial 3
Word 1			
Word 2			
Word 3			
Word 4			
Word 5			

Immediate Memory score: 15

Concentration:

Reverse digits. (Go to next string length if correct on first trial. Stop if incorrect on both trials. 1 point each for each string length.)

3-8-2	5-1-8
2-7-9-3	2-1-6-8
5-1-8-6-9	9-4-1-7-5
6-9-7-3-5-1	4-2-8-9-3-7

Months of the year in reverse order. (1 point for entire sequence correct.)

Dec-Nov-Oct-Sep-Aug-Jul

Jun-May-Apr-Mar-Feb-Jan

Concentration score: 5

Delayed Recall: (approximately 5 minutes after Immediat Memory. 1 point each.)

Word 1

Word 2

Word 3

Word 4

Word 5

Delayed Recall score: 5

Summary of total scores:

Orientation	5
Immediate Memory	15
Concentration	5
Delayed Recall	5
Total score	30

The following may be performed between the Immediate Memory and Delayed Recall portions of this assessment when appropriate:

Neurologic Screening:

Recollection of the injury:

Strength:

Sensation:

Coordination:

Exertional Maneuvers:

1 40-yard sprint

5 sit-ups

5 push-ups

5 knee bends

Appendix C: Dynamic Gait Index (DGI)

BOX 15–7 Dynamic Gait Index

1. Gait Level Surface

   Instructions: Walk at your normal speed from here to next mark (20').
   Grading: Mark the lowest category that applies.

   - **Normal:** Walks 20', no assistive devices, good speed, no evidence for imbalance, normal gait pattern.
   - **Mild impairment:** Walks 20', uses assistive devices, slower speed, mild gait deviations.
   - **Moderate impairment:** Walks 20', slow speed, abnormal gait pattern, evidence for imbalance.
   - **Severe impairment:** Cannot walk 20'without assistance, severe gait deviations, or imbalance.

2. Change in gait speed

   Instructions: Begin walking at your normal pace (for 5'), when I tell you "go," walk as fast as you can for 5'. When I tell you "slow," walk as slowly as you can (for 5').
   Grading: Mark the lowest category that applies.

   - **Normal:** Able to smoothly change walking speed without loss of balance or gait deviation. Shows a significant difference in walking speeds between normal, fast, and slow speeds.
   - **Mild impairment:** Is able to change speed but demonstrates mild gait deviations, or no gait deviations but unable to achieve a significant change in velocity, or uses assistive device.
   - **Moderate impairment:** Makes only minor adjustments to walking speed, or accomplishes a change in speed with significant gait deviations, or changes speed but has significant gait deviations, or changes speed but loses balance but is able to recover and continue walking.
   - **Severe impairment:** Cannot change speeds, or loses balance and has to reach for wall or be caught.

3. Gait with horizontal head turns

   Instructions: Begin walking at your normal pace. When I tell you to "look right," keep walking straight, but turn your head to the right. Keep looking to the right until I tell you "look left," then keep walking straight and turn your head to the left. Keep your head to the left until I tell you, "look straight," then keep walking straight but return your head to the center.
   Grading: Mark the lowest category that applies.

   - **Normal:** Performs head turns smoothly with no change in gait.
   - **Mild impairment:** Performs head turns smoothly with slight change in gait velocity (i.e., minor disruption to smooth gait path or uses walking aid).
   - **Moderate impairment:** Performs head turns with moderate change in gait velocity, slows down, staggers but recovers, can continue to walk.
   - **Severe impairment:** Performs task with severe disruptions of gait (i.e., staggers outside 15° path, loses balance, stops, reaches for wall).

4. Gait with vertical head turns

   Instructions: Begin walking at your normal pace. When I tell you to "look up," keep walking straight, but tip your head and look up. Keep looking up until I tell you, "look down." Then keep walking straight and turn your head down. Keep looking down until I tell you, "look straight," then keep walking straight, but return your head to the center.
   Grading: Mark the lowest category that applies.

   - **Normal:** Performs head turns with no change in gait.

- **Mild impairment:** Performs task with slight change in gait velocity (i.e., minor disruption to smooth gait path or uses walking aid).
- **Moderate impairment:** Performs tasks with moderate change in gait velocity, slows down, staggers but recovers, can continue to walk.
- **Severe impairment:** Performs task with severe disruption or gait (i.e., staggers outside 15" path, loses balance, stops reaches for wall).

5. Gait and pivot turn

Instructions: Begin walking at your normal pace. When I tell you, "turn and stop," turn as quickly as you can to face the opposite direction and stop.
Grading: Mark the lowest category that applies.

- **Normal:** Pivot turns safely within 3 seconds and stops quickly with no loss of balance.
- **Mild impairment:** Pivot turns safely in >3 seconds and stops with no loss of balance.
- **Moderate impairment:** Turns slowly, requires verbal cuing, requires several small steps to catch balance following turn and stop.
- **Severe impairment:** Cannot turn safely, requires assistance to turn and stop.

6. Step over obstacle

Instructions: Begin walking at your normal speed. When you come to the shoe box, step over it, not around it, and keep walking.
Grading: Mark the lowest category that applies.

- **Normal:** Is able to step over box without changing gait speed; no evidence for imbalance.
- **Mild impairment:** Is able to step over box, but must slow down and adjust steps to clear box safely.
- **Moderate impairment:** Is able to step over box but must stop, then step over. May require verbal cuing.
- **Severe impairment:** Cannot perform without assistance.

7. Step around obstacles

Instructions: Begin walking at your normal speed. When you come to the first cone (about 6' away), walk around the right side of it. When you come to the second cone (6' past first cone), walk around it to the left.
Grading: Mark the lowest category that applies.

- **Normal:** Is able to walk around cones safely without changing gait speed; no evidence of imbalance.
- **Mild impairment:** Is able to step around both cones, but must slow down and adjust steps to clear cones.
- **Moderate impairment:** Is able to clear cones but must significantly slow speed to acomplish task, or requires verbal cuing.
- **Severe impairment:** Unable to clear cones, walks into one or both cones, or requires physical assistance.

8. Steps

Instructions: Walk up these stairs as you would at home (i.e., using the rail if necessary). At the top, turn around and walk down.
Grading: Mark the lowest category that applies.

- **Normal:** Alternating feet, no rail.
- **Mild impairment:** Alternating feet, must use rail.
- **Moderate impairment:** Two feet to stair, must use rail.
- **Severe impairment:** Cannot do safely.

Appendix D: Patient-Specific Function Scale (PSFS)

Patient Specific Functional Scale

ID: _____ Date of Initial Eval: ___/___/_____

Date of Post-Injury Eval: ___/___/_____

Initial Post-injury Assessment:

I am going to ask you to identify up to three activities that are important to your everyday activities that you are unable to do, or are having difficulty with as a result of your head injury?
(e.g. getting dressed, walking, driving, working, exercising, etc...)

I am also going to ask you to identify up to three activities that are important to your ability to play rugby that you are unable to do, or are having difficulty with as a result of your head injury?
(e.g. running, cutting, passing, tackling, rucking, mauling, kicking, etc...)

On the first table below, please use the Patient-Specific Activity Score to rate your current difficulty with the everyday activities that you stated above.

Patient-Specific Activity Scoring (point to one number):

0 (unable to perform) 1 2 3 4 5 6 7 8 9 10 (able to perform at same level as before injury)

Activity	Post-Injury	Follow-up 1	Follow-up 2	Follow-up 3	Follow-up 4
1					
2					
3					

On the second table below, please use the Patient-Specific Activity Score to rate your current difficulty with the rugby related activities that you stated above.

Activity	Post-Injury	Follow-up 1	Follow-up 2	Follow-up 3	Follow-up 4
1					
2					
3					

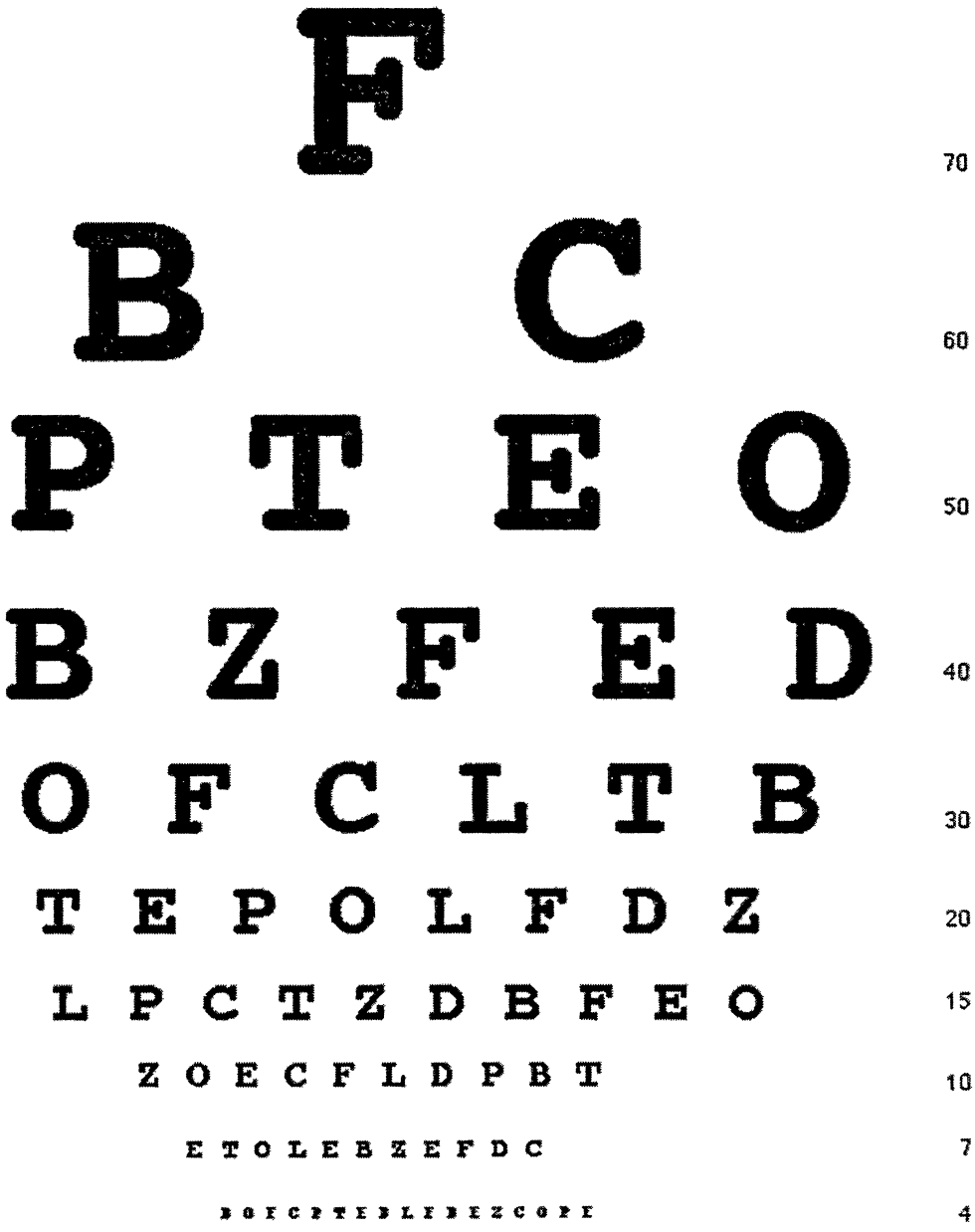
Appendix E: Dizziness Handicap Inventory (DHI)

Dizziness Handicap Inventory

Instruction: The purpose of this scale is to identify difficulties that you may be experiencing because of your dizziness or unsteadiness. Please answer "yes", "no", or "sometimes" to each question. *Answer each question as it pertains to your dizziness or unsteadiness problem only.*

ITEM	RESPONSE
P1. Does looking up increase your problem?	
E2. Because of your problem, do you feel frustrate?	
F3. Because of your problem, do you restrict your travel for business or recreation?	
P4. Does walking down the aisle of a supermarket increase your problem?	
F5. Because of your problem, do you have difficulty getting into or out of bed?	
F6. Does your problem significantly restrict your participation in social activities such as going to dinner, the movies, dancing, parties, etc...?	
F7. Because of your problem, do you have difficulty reading?	
P8. Does performing more ambitious activities like sports, dancing, household chores such as sweeping or doing dishes increase your problem?	
E9. Because of your problem, are you afraid to leave your home without having someone accompany you?	
E10. Because of your problem, have you been embarrassed in front of others?	
P11. Do quick movements of your head increase your problem?	
F12. Because of your problem, is it difficult for you to do strenuous housework or yardwork?	
P13. Does turning over in bed increase your problem?	
F14. Because of your problem, is it difficult for you to do strenuous housework or yardwork?	
E15. Because of your problem, are you afraid people may think you are intoxicated?	
F16. Because of your problem, is it difficult for you to go for a walk by yourself?	
P17. Does walking down a sidewalk increase your problem?	
E18. Because of your problem, is it difficult for you to concentrate?	
F19. Because of your problem, is it difficult for you to walk around your house in the dark?	
E20. Because of your problem, are you afraid to stay at home alone?	
E21. Because of your problem, do you feel handicapped?	
E22. Has your problem placed stress on your relationships with members of your family or friends?	
E23. Because of your problem, are you depressed?	
F24. Does your problem interfere with your job or household responsibilities?	
P25. Does bending over increase your problem?	

Appendix F: Snellen 10-foot Eye Chart



Appendix G: Consent Form

Consent Form

The Effects of Vestibular Therapy on the Post-Concussion Athlete

You are being invited to participate in a study on the effects of vestibular therapy on the post-concussion athlete. My name is Patricia Hill, and I am a graduate student in the Physical Therapy Department of Texas State University, at San Marcos, under the supervision of Diana Hunter, PhD. I am also a rugby player for the Austin Women's Rugby Team. We are interested in how vestibular therapy may help athletes who have suffered concussions return to their sport faster, healthier, more confident, and with less chance of future injury.

The vestibular system is one of the ways we maintain our balance. It helps let us know when we are upright and in line with gravity, and when our bodies are accelerating. When a person suffers a traumatic injury to the head and/or neck, the vestibular system may be damaged causing problems such as dizziness, nausea, double-vision, etc... Vestibular therapy involves retraining this system through repetitive movements of the head and eyes while sitting still and while performing various activities.

If you decide to participate in this study you will be joining several other rugby players from various local area teams. You understand that participation in this study will involve one initial visit to the Physical Therapy Clinic at Texas State University in San Marcos for a screening; followed by several weekly visits to the same clinic should you suffer a head injury during the spring or summer rugby seasons, from February, 2004 to August, 2004. The initial screening will last approximately 1 hour and will include a test of your vision, several balance tests, and a test of your vestibular system, which involves a computerized measurement of your balance in various situations. If you should suffer a head injury during the coming spring or summer rugby seasons, the follow-up visits would consist of an initial visit within 48 hours of your injury to determine the severity of the injury, followed by an appointment (at your earliest convenience) with a physician of your choice for confirmation of the diagnosis of the head injury. We will then see you for weekly visits at the clinic in San Marcos for repeated testing of your balance and your vestibular system, until it is determined that you have recovered to your previous testing levels. During the weeks after your injury you will be asked to perform specific exercises to improve the function of your vestibular system. These exercises will be done for 20 minutes, twice each day.

None of your person information, including your name, will be stored with any of the collected data. Any personal information will remain locked in the office of Dr. Diana Hunter. An identification number will be assigned each of the participants in this study. Any information that is obtained in connection with this study will be kept confidential and will be disclosed only with your permission. Some of the data collected during your participation may be used in the future for further research or educational purposes, in which case you would be referred to only by your identification number, and your identity would not be disclosed.

Potential risks: There is minimal risk involved in the activities described. During the initial assessment you will be asked to perform activities involving standing on a balance platform, performing a visual screen, jogging a short distance, and standing with your feet together and your eyes closed. As with any physical activity there is minimal risk of

falling. A gait belt will be used to minimize the risk of falling. If you suffer a head or neck injury during the season, and chose to continue with our study, the balance and vestibular testing may increase some of your symptoms such as dizziness, nausea, and double-vision, and there will be an increased risk of falling during the testing activities. Experienced assessors will administer the test in a controlled environment in order to minimize any risk to you. There is a chance that you may disagree with the recommendations that we are making to you regarding your ability to return to play at which point you may chose to terminate your participation in the experiment. If at any point during any of the testing or treatments you wish to terminate your participation in the experiment, you may do so. There is also a risk of losing confidentiality of your information. Coding all data with letters and numerals and keeping all participants' names on a separate sheet available only to the investigators directly involved with this study will minimize this risk. All computer and paper records are archived indefinitely, but they are stored with no individual identifying information.

Benefits: The initial assessment of your vestibular system can tell us if you fall within typical ranges of performance. This may alert you to a deficit caused by a previous head or neck injury. If you suffer a head or neck injury during the coming season, our post-injury assessment can tell you the severity of your injury, which can then be confirmed by a physician of your choice. Following the diagnosis of a concussion, you will receive several physical therapy sessions at no cost to you. These therapy sessions may help you return to play faster, with more confidence, and with less chance of future injury. Any and all data recorded during your participation in this study may be made available to you at your request.

You are making a decision whether or not to participate in our study. Your signature indicates that you have read the information provided and have decided to enroll in our study. Should you choose to discontinue participation in this study, you may withdraw at any time after signing this form.

If you have any questions during or after your participation in this study you may contact me (Patricia Hill) at 512-565-0743, ph1038@txstate.edu, or my supervisor, Dr. Diana Hunter at 512-245-3517, dh11@txstate.edu.

You will be provided with a copy of this form for your records.

Name of Participant	Phone Number	Email

Signature of Participant	Date

Signature of Investigator	Date

Appendix H: Informational Email

Research Opportunity

Hello Fellow Rugby Players and Coaches:

My name is Patricia Hill and I am a graduate student in the Physical Therapy Department at Texas State University, in San Marcos. I am also a member of the Austin Valkyries Women's Rugby Team, and a coach of the Texas State Women's Rugby team. I am currently conducting research in partial fulfillment of a Master of Science degree, to examine the effects of a specific physical therapy protocol on athletes after they have suffered a concussion. I am looking for volunteers to participate in this study. I require as many participants as possible, and I am looking within the Austin, San Marcos, and San Antonio areas.

The study will involve a minimum of 1 visit to the Physical Therapy clinic at Texas State University, for a series of tests lasting approximately 1 hour. I will then be following each of the participants via phone and email conversations to monitor any head injuries that occur during rugby games and/or practices. If you suffer a head injury of any kind, I would ask that you continue your participation in this study, by returning to the Physical Therapy clinic in San Marcos as soon as possible after your injury, and once a week for 4 weeks thereafter.

The only thing that will preclude your participation in this study is a significant injury to a foot, ankle, knee or hip within the past 3 months (ie. you had to miss more than 4 weeks of play).

If you have been playing rugby for any length of time, I'm sure you have had some kind of experience with head injuries, either personally, or through a teammate. The diagnosis and treatment of the mild head injury that occurs with a concussion is an area of research that is still not well understood. The research we wish to perform could be a vital tool in establishing objective measurements that can determine when it is safe for an athlete to return to competition after a head injury, as well as treatment protocols that can help the athlete return to play faster and with less chance of future injury. We ask for your participation in this important area of study.

I am attaching a copy of the consent form to provide you with more information about what will be required of you, if you should choose to participate. I would appreciate it if you would forward this information on to the rest of your teams. I would like for a representative from each team to reply to me and let me know what your practice schedule will be over the next two weeks, so that we can schedule a time (approximately 15 minutes) during which I can visit with your team. At that time we will discuss any questions they might have regarding the study, and we will set up times for the volunteers to come to the physical therapy clinic at Texas State for their first visit.

Thank you,
Patty

Appendix I: Personal Intake Information and Medical History

Personal Information Intake Form

(to remain confidential, accessible only to the primary investigator and supervising professor)

Name: _____ ID: _____

Date of Initial Screen: ___/___/_____ Time: ___:_____ AM

___:_____ PM

Date of Birth: ___/___/_____ Current Age: _____

Male Female

Preferred mode of contact: Phone: _____

Email: _____

Best time to contact: ___:_____ AM

___:_____ PM

Emergency contact information:

Name: _____

Phone number: _____

Relation: _____

Occupation: _____

Marital Status: single married divorced widowed

Do you have any children? _____ How many? _____

If so, what are their ages? _____

Sport-Related History

ID:_____ Date of Initial Eval: ___/___/_____

Current Rugby Team: _____

What division is this team? (collegiate, club, etc...) _____

Years w/ team: _____

Years playing rugby: _____

Position/s played: _____

Most common position/s: _____

Do you currently participate in any other sports or activities? What?

_____

_____

Throughout your past, in what other sports or activities have you participated? _____

Medical History

ID:_____ Date of Initial Eval:___/___/_____

General Health Status:

1. Please rate you health: Excellent Good Fair Poor
2. Have you had any major life changes during the past year? (e.g. new baby, job change, death of a family member, etc...) Yes No

Social/Health Habits:

1. Smoking: Currently Yes # pack per day: _____
   No
   Past Yes Year you quit: _____
   No
2. Alcohol:
   How many days per week do you drink beer, wine, or other alcoholic beverages, on average? _____

   If one beer, one glass of wine, or one cocktail equals one drink, how many drinks do you have on an average day? _____

3. Exercise: Do you exercise beyond normal daily activities and chores?
   Yes Describe the exercise _____
   How many days per week do you do exercise? _____
   For how many minutes, on an average day? _____
   No

Family History (Indicate whether mother, father, brother/sister, aunt/uncle, or grandmother/grandfather, and age of onset if known)

Heart Disease: _____
Hypertension: _____
Stroke: _____
Diabetes: _____
Cancer: _____
Psychological Problems: _____
Arthritis: _____
Osteoporosis: _____
Other: _____

Contact Information: Patricia M. Hill 512-565-0743, PH1038@txstate.edu
Dr. Diana Hunter 512-245-3517, DH11@txstate.edu

Medical/Surgical History

Please check if you have EVER had:

___ Arthritis
___ Broken bones/fractures
___ Osteoporosis
___ Blood Disorders
___ Circulation/vascular problems
___ Heart Problems
___ High Blood Pressure
___ Lung Problems
___ Stroke
___ Diabetes/high blood sugar
___ Low blood sugar/hypoglycemia
___ Head injury
___ Skin Diseases
___ Other; describe _____

___ Multiple Sclerosis
___ Muscular Dystrophy
___ Parkinson Disease
___ Seizures/epilepsy
___ Allergies
___ Develop./growth problems
___ Thyroid problems
___ Cancer
___ Infectious Disease (TB, Hep)
___ Kidney Problems
___ Repeated Infections
___ Ulcers/stomach problems
___ Depression

Within the past year have you had any of the following symptoms?

___ Chest Pain
___ Heart Palpitations
___ Cough
___ Hoarseness
___ Shortness of breath
___ Dizziness or blackouts
___ Coordination problems
___ Weakness in arms/legs
___ Loss of balance
___ Difficulty walking
___ Joint pain/swelling
___ Pain at night

___ Difficulty sleeping
___ Loss of appetite
___ Nausea/vomiting
___ Difficult swallowing
___ Bowel problems
___ Weight loss/gain
___ Urinary problems
___ Fever/chills/sweats
___ Headaches
___ Hearing problems
___ Vision problems
___ Other; describe _____

Have you ever had surgery? Yes No

Describe:	Date:
_____	___/___/_____
_____	___/___/_____
_____	___/___/_____
_____	___/___/_____
_____	___/___/_____

Medications

1. Are you currently taking any prescription medications? Yes No
   If yes, please list: _____
2. Do you take any of the following non-prescription medications?
   - ___ Advil/Aleve
   - ___ Antacids
   - ___ Ibuprofen/Naproxen
   - ___ Antihistamines
   - ___ Aspirin
   - ___ Decongestants
   - ___ Herbal supplements
   - ___ Tylenol
   - ___ Other: _____
     _____

Study-Relevant Criteria

Have you ever had a concussion/s that was diagnosed by a physician?

How did it/they happen? _____
_____

When did it/they happen? _____
_____

What did you do to get better? _____
_____

Have you had any head injuries in the past 3 months (even if you didn't see a doctor for it)?

Yes No ; Describe _____
_____

In the past 3 months have you had any injuries to your knees, ankle, or hips?

Yes No ; Describe _____
_____

Are you currently playing rugby? Yes No

If no, why not? _____
_____

Would you be willing to return to the physical therapy clinic at Texas State University in San Marcos, if you were to suffer a head injury during the next 6 months? Yes No

Appendix J: Data Sheet for Initial Screen

Data Sheet: Initial Screen

ID: _____ **Date of Initial Eval:** ___/___/_____
Height _____ **Weight** _____ **BP** ____/____

Visual/Oculomotor Screen: Peripheral Visual Field R_____ L_____
Smooth Pursuits Horiz + - _____ Saccades Horiz + -_____
Vert + - _____ Vert + -_____
Diag + - _____ Diag + -_____
Convergence/Divergence _____
Spontaneous Nystagmus (sitting) _____
Romberg Sway Analysis: Time _____ sec %30sec _____
Tandem Romberg: Time _____sec %10sec _____
Dynamic Gait Index:

Gait Level Surface	____(0-3)
Change in gait speed	____(0-3)
Gait w/ horizontal head turns	____(0-3)
Gait w/ vertical head turns	____(0-3)
Gait and pivot turn	____(0-3)
Step over obstacle	____(0-3)
Step around obstacles	____(0-3)
Stairs	____(0-3)
Total	____/24

Dynamic visual acuity: Do they require glasses or contacts? Yes No
If yes, are they using them during this test? Yes No
Line read static: _____
Line read dynamic: _____
Modified CTSIB:

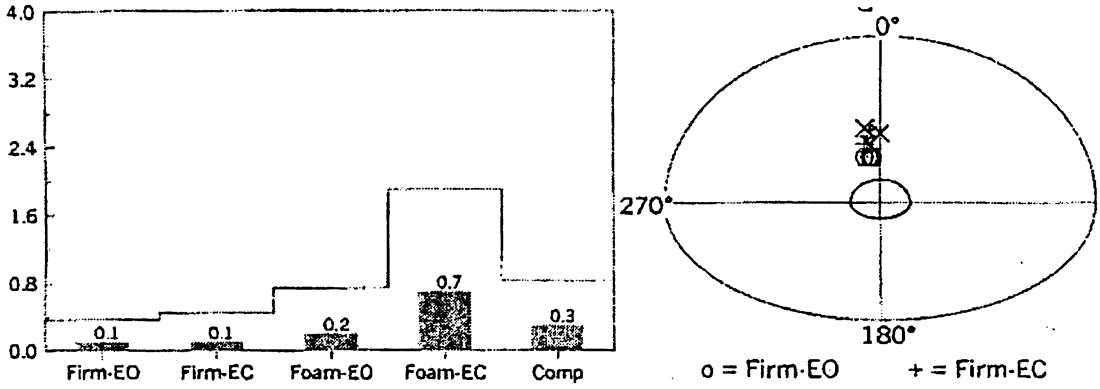
Condition	%LOS	COG sway vel	WNML
1. EO/Firm			
2. EC/Firm			
3. EO/Foam			
4. EC/Foam			
5. HS/EC/Firm			X
6. HS/EC/Foam			X

Composite Sway Velocity = _____

Signature of Primary Investigator: _____

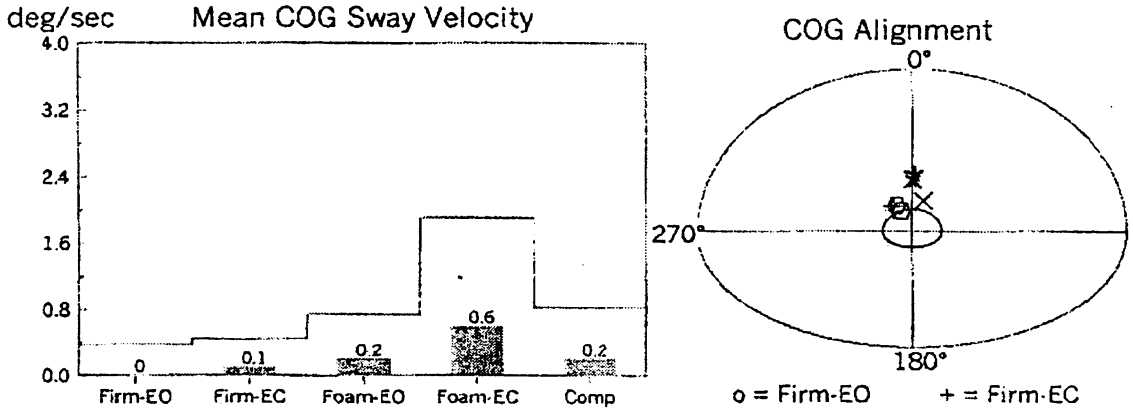
Signature of Supervising Therapist: _____

Appendix K: Sample Data Obtained During a MCTSIB



Data Range Note: NeuroCom Data Range: 20--39

Post Test Comments:



Data Range Note: NeuroCom Data Range: 20--39

Post Test Comments:

Appendix L: "How Do I Know If I Have A Concussion" Take Home Sheet

How do I know if I have a concussion?

Concussions fall under the broader category of closed-head injuries. Within the category of closed-head injuries there are both mild and severe degrees of brain injury. Concussions are categorized as mild traumatic brain injuries or MTBIs.

The most commonly recognized indications that a person has suffered a concussion are:

1) Periods of disorientation or confusion following a blow to the head
2) Periods of amnesia or short-term memory loss following a blow to the head
3) A loss of consciousness immediately following a blow to the head

While the presence of these signs might indicate a moderate or severe concussion, there are many other signs and symptoms that might be present following a mild or moderate concussion that did not result in a loss of consciousness or amnesia.

The following is a list of other symptoms that might indicate that a person has suffered a concussion following a trauma to the head; these symptoms may appear immediately following the injury or within hours to days afterwards.

Feeling of having bell rung
Depression
Feeling of being dinged
Drowsiness
Excess sleep
Fatigue
Feel "in a fog"
Feel "slowed down"
Headache
Inappropriate emotions or personality change
Irritability
Loss of consciousness
Loss of orientation
Vomiting
Vacant stare/glassy eyed
Sleep disturbance
Sensitivity to noise
Sensitivity to light
Seeing stars
Sadness
Ringing in the ears
Poor balance and coordination
Numbness/tingling
Nervousness
Nausea
Memory problems

If you experience a blow to the head during any game or practice situation, and you are experiencing any of the above listed symptoms, please call us to schedule an evaluation. It is important that you be evaluated by either our staff or a physician following an injury to the head, regardless of how minor you think the injury.

The results of any and all testing that is performed following an injury will remain confidential, to be disclosed only at your request.

Appendix M: Data Sheet for Post-Injury Follow-Up

Data Sheet: Post-MTBI

ID: _____ Date of Initial Eval: ___/___/_____
Date of Post-Injury Eval: ___/___/_____

Date of Injury: ___/___/_____
How did the injury occur: _____
Have you sought medical care for this injury: Yes No
If yes, did a physician provide you with a diagnosis? Yes No
If yes, what was the diagnosis? _____
What was the date of your doctor's visit? ___/___/_____
What was the name of the physician? _____
Are you taking any medications for this injury: Yes No
If yes, what are you taking? _____
Since your injury have you had any of the following: (please indicate if you had the symptom initially or if you continue to have it)

Initially	Currently	
___	___	Bell rung
___	___	Depression
___	___	Dinged
___	___	Drowsiness
___	___	Excess sleep
___	___	Fatigue
___	___	Feel "in a fog"
___	___	Feel "slowed down"
___	___	Headache
___	___	Inappropriate emotions or personality change
___	___	Irritability
___	___	Loss of consciousness
___	___	Loss of orientation
___	___	Memory problems
___	___	Nausea
___	___	Nervousness
___	___	Numbness/tingling
___	___	Poor balance and coordination
___	___	Ringing in the ears
___	___	Sadness
___	___	Seeing stars
___	___	Sensitivity to light
___	___	Sensitivity to noise
___	___	Sleep disturbance
___	___	Vacant stare/glassy eyed
___	___	Vomiting

BP _____/_____

Neuro Screen: **Reflexes** Brachio _____ Patellar _____
Biceps _____ Achilles _____
Triceps_____ Hoffman's _____
Myotomes Intact Yes No
If no, nerve root(s) _____
Dermatomes Intact Yes No
If no, nerve root(s)

Cranio-vertebral Screen: Alar Ligament Intact Yes No
Transverse Ligament Intact Yes No
Vertebral Artery Intact Yes No

Romberg Sway Analysis: Time _____ sec %30sec _____
Tandem Romberg: Time _____ sec %10sec _____
Dynamic Gait Idex:

Gait Level Surface	_____(0-3)
Change in gait speed	_____(0-3)
Gait w/ horizontal head turns	_____(0-3)
Gait w/ vertical head turns	_____(0-3)
Gait and pivot turn	_____(0-3)
Step over obstacle	_____(0-3)
Step around obstacles	_____(0-3)
Stairs	_____(0-3)
Total	**_____/24**

Dynamic visual acuity: Do they require glasses or contacts? Yes No
If yes, are they using them during this test? Yes No
Line read static: _____
Line read dynamic: _____

Visual/Oculomotor Screen: Peripheral Visual Field R_____ L_____
Smooth Pursuits Horiz + - _____ Saccades Horiz + -_____
Vert + - _____ Vert + -_____
Diag + - _____ Diag + -_____
Convergence/Divergence _____
Spontaneous Nystagmus (sitting) _____

Modified CTSIB:

Condition	LOS	COG sway vel	WNML
1. EO/Firm			
2. EC/Firm			
3. EO/Foam			
4. EC/Foam			
5. HS/EC/Firm			X
6. HS/EC/Foam			X

Composite Sway Velocity = _____

PSFS:

IADL score: _____/30

Rugby-related score: _____/30

DHI: Composite: _____/100

Functional: _____/36

Psych: _____/28

Emotional: _____/36

Date of next scheduled visit: ___/___/_____ Time: ___:_____ AM/PM

Date of visit with MD: ___/___/_____ Time: ___:_____ AM/PM

Signature of Primary Investigator: _____

Signature of Supervising Therapist: _____

Data Sheet: Follow-up

ID: _____ Date of Initial Eval: ___/___/_____

Date of Post-Injury Eval: ___/___/_____

Date of Follow-up Eval: ___/___/_____

Date of Injury: ___/___/_____

Are you taking any medications for this injury: Yes No

If yes, what are you taking? _____

Are you still having any of the following:

___ Bell rung
___ Depression
___ Dinged
___ Drowsiness
___ Excess sleep
___ Fatigue
___ Feel "in a fog"
___ Feel "slowed down"
___ Headache
___ Inappropriate emotions or personality change
___ Irritability
___ Loss of consciousness
___ Loss of orientation
___ Memory problems
___ Nausea
___ Nervousness
___ Numbness/tingling
___ Poor balance and coordination
___ Ringing in the ears
___ Sadness
___ Seeing stars
___ Sensitivity to light
___ Sensitivity to noise
___ Sleep disturbance
___ Vacant stare/glassy eyed
___ Vomiting

Have you been able to complete your vestibular rehab program? Yes No

If no, why not? _____

If yes, how often? _____ For how long? _____

Have you been keeping up with the log of your rehabilitation? Yes No

If no, why not? _____

BP _____/_____

Neuro Screen: **Reflexes** Brachio _____ Patellar _____
Biceps _____ Achilles _____
Triceps_____ Hoffman's _____

Myotomes Intact Yes No
If no, nerve root(s) _____

Dermatomes Intact Yes No
If no, nerve root(s)

Cranio-vertebral Screen: Alar Ligament Intact Yes No
Transverse Ligament Intact Yes No
Vertebral Artery Intact Yes No

Romberg Sway Analysis: Time _____ sec %30sec _____
Tandem Romberg: Time _____ sec %10sec _____

Dynamic Gait Idex:

Gait Level Surface	____(0-3)
Change in gait speed	____(0-3)
Gait w/ horizontal head turns	____(0-3)
Gait w/ vertical head turns	____(0-3)
Gait and pivot turn	____(0-3)
Step over obstacle	____(0-3)
Step around obstacles	____(0-3)
Stairs	____(0-3)
Total	____/24

Visual/Oculomotor Screen: Peripheral Visual Field R_____ L_____
Smooth Pursuits Horiz + - _____ Saccades Horiz + -_____
Vert + - _____ Vert + -_____
Diag + - _____ Diag + -_____
Convergence/Divergence _____
Spontaneous Nystagmus (sitting) _____

Dynamic visual acuity: Do they require glasses or contacts? Yes No
If yes, are they using them during this test? Yes No
Line read static: _____
Line read dynamic: _____

Modified CTSIB:

Condition	LOS	COG sway vel	WNML
1. EO/Firm			
2. EC/Firm			
3. EO/Foam			
4. EC/Foam			
5. HS/EC/Firm			X
6. HS/EC/Foam			X

Composite Sway Velocity = _____

PSFS:

IADL score: _____/30

Rugby-related score: _____/30

DHI: Composite: _____/100

Functional: _____/36

Psych: _____/28

Emotional: _____/36

Have you returned to playing rugby? Yes No

If no, why not? _____

If yes, are you having any difficulties? _____

_____

Date of next scheduled visit: ___/___/_____ Time: ___:____ AM/PM

Date of next visit with MD: ___/___/_____ Time: ___:____ AM/PM

Signature of Primary Investigator: _____

Signature of Supervising Therapist: _____

Appendix N: Patient Preparation for the VORTEQ

Taken from Micromedical Technologies Version 8.0 Software Quick Start Guide[112]

Patient Preparation:

- Electrodes: horizontal and vertical recording possible (5 leads), wires over ears to reduce lead movement during testing
- Headband: Click tight. Check sensor orientation.
- Video: goggles snug on face
- Distance: 39 inches from light bar. Measure it!
- Light Bar: calibrate EOG before each VORTEQ test

Testing:

- Instruct patient to stare at the light bar stimulus during head movement.
- Have patient shake head no (horizontal test) and yes (vertical tests)
- Head movement: about +/- 10 degrees (+/- 5 degrees at higher frequencies).
    - Note: excessive head movement reduces maximum frequency patient can produce.
- Frequencies performed do not have to match beeper frequencies for analysis, but if they do not perform higher frequencies you will have fewer data points on the summary.
- Perform and analyze three tests horizontal and three tests vertical. After analyzing a test you can press F9 to view test results with normative lines (from test schedule) to determine if results are within normal or abnormal thresholds.
- The 1 Hz test is the easies to perform and most patients should have a normal gain at 1 Hz. Higher frequencies are usually the ones that are the most sensitive to pathology.
- Repeat test if patient did not understand or made a mistake (moved eyes off target, moved head during calibration, did not perform required frequencies).
- If the patient can not hear the beeper, tap on their shoulder to pace them.
- If the first test did not go well for what ever reason, repeat it. Remind them to look at the light bar during the test and try to keep that light steady in their vision.

Results:

- Expect false positive rate to be lower than false negative rate. In other words, CNS compensation may mask a unilateral lesion or a patient may not desire to shake their head at a rate that produces symptoms (and abnormal Vorteq findings).
- Vorteq gain is the most sensitive parameter of the three for identifying vestibular dysfunction. If the patient shakes their head with vigor and can not maintain the target on the fovea then Vorteq gain will be reduced. If gain is normal at 1 Hz but decreases below normative thresholds at the higher frequencies this is also a common but abnormal peripheral vestibular finding. Abnormally low gain at all frequencies is indicative of a bilateral vestibular loss. High gain may be an EOG

calibration artifact. The test should be repeated with a new EOG calibration. Consistently high gain suggests overcompensation by the CNS.

- Vorteq phase is a measure of the timing relationship between head and eye movement. If the head moves equally and opposite the eyes, then phase will be zero. Phase is a measure of central vestibular velocity storage. Normal gain but abnormal phase may thus be of central origin.
- Gain asymmetry is a measure of the difference in gain with head moving right versus moving left. If the patient produced adequate head velocity during the test and the symmetry is abnormal this would correspond to a significant unilateral lesion. However, a unilateral lesion may be masked if the patient does not shake their head with vigor.

Interpretation:

- Always combine Vorteq results with the patient's history, a clinical examination, and other test results before making a diagnosis.

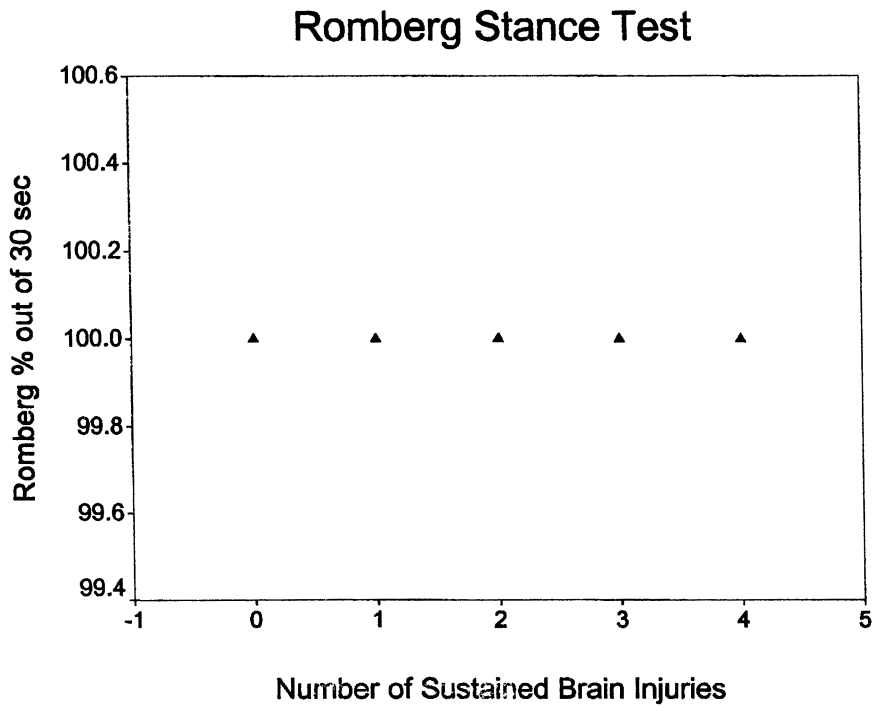
Appendix O: Vestibular Rehabilitation Exercises

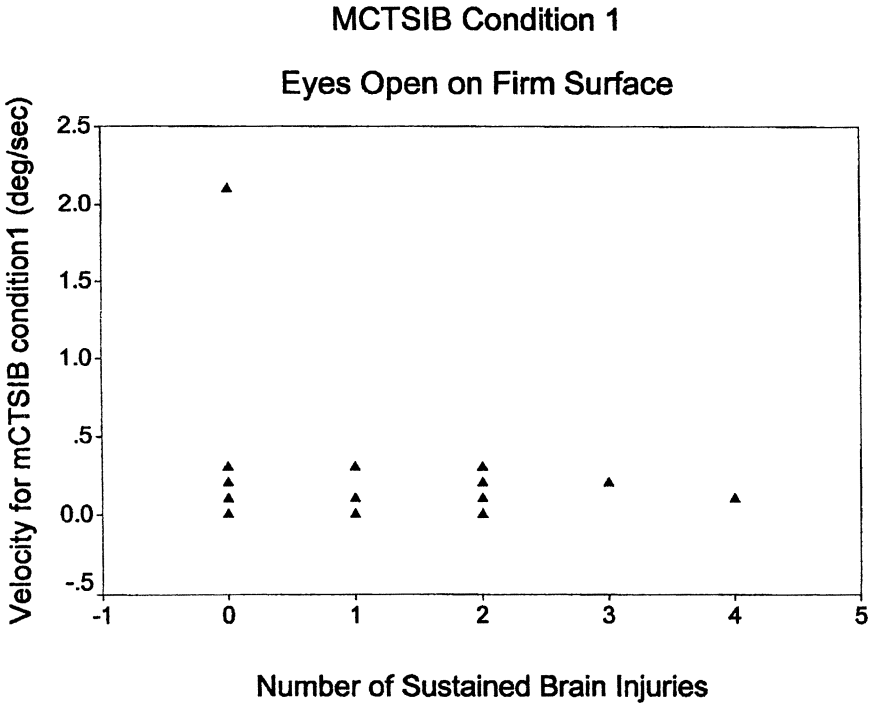
Customized Treatment Plan: Will consist of five of the following exercises

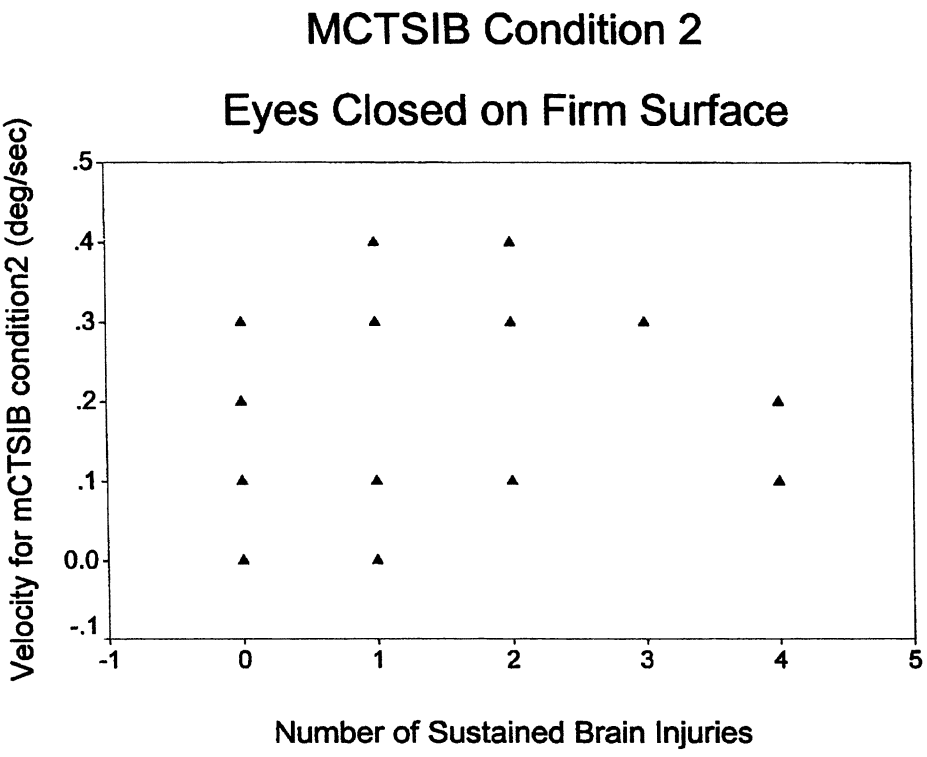
1) Horizontal head movements
2) Saccades
3) Smooth pursuits
4) Visual tracking with head movements to targets
5) Focusing while turning head
6) Ankle sways (eyes closed)
7) Ball circles
8) Single leg stance (SLS) on firm/foam; eyes open/eyes closed
9) Tandem stance; eyes open/eyes closed
10) Gait with head movements (horiz., vert., diagonal)

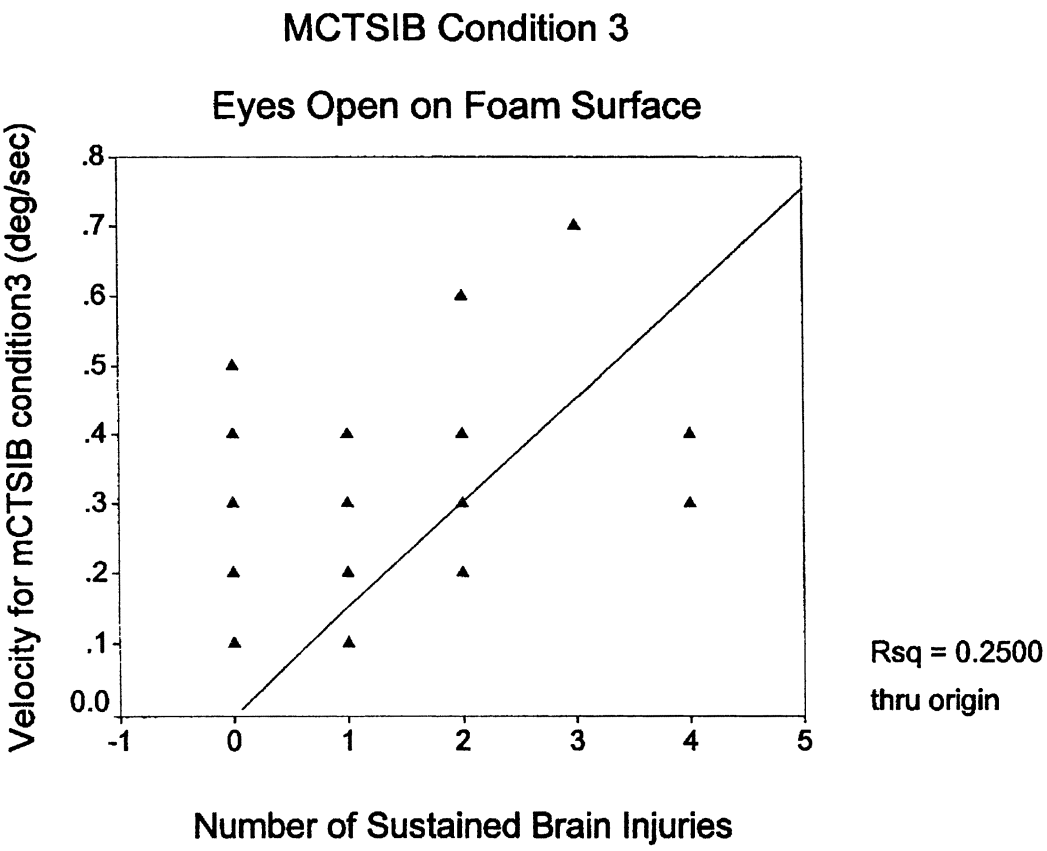
Appendix P: Graphic data of non-significant results not included in text

Correlations Between the ***Number of Brain Injuries Sustained*** and Assessment Results



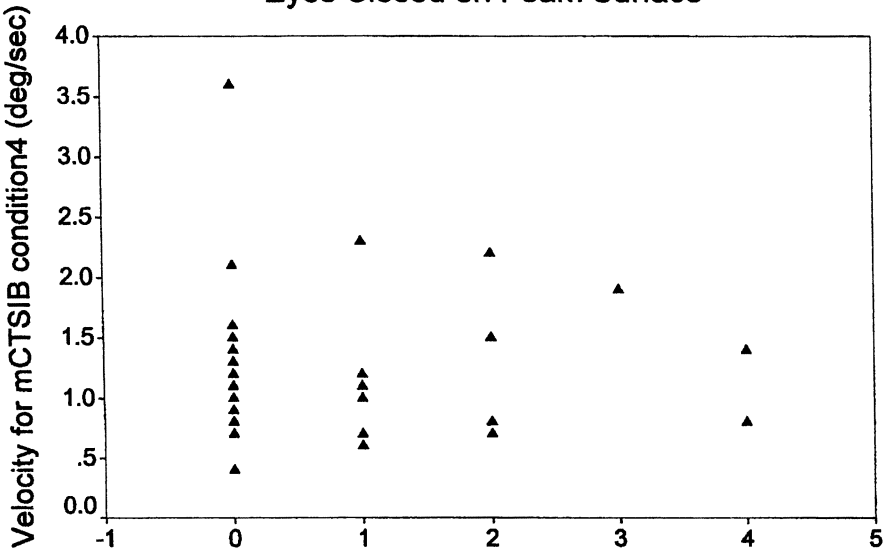



MCTSIB Condition 2
Eyes Closed on Firm Surface
Velocity for mCTSIB condition2 (deg/sec)
.5
.4
.3
.2
.1
0.0
-.1
-1
0
1
2
3
4
5
Number of Sustained Brain Injuries


MCTSIB Condition 3
Eyes Open on Foam Surface
Velocity for mCTSIB condition3 (deg/sec)
.8
.7
.6
.5
.4
.3
.2
.1
0.0
-1
0
1
2
3
4
5
Number of Sustained Brain Injuries
Rsq = 0.2500
thru origin

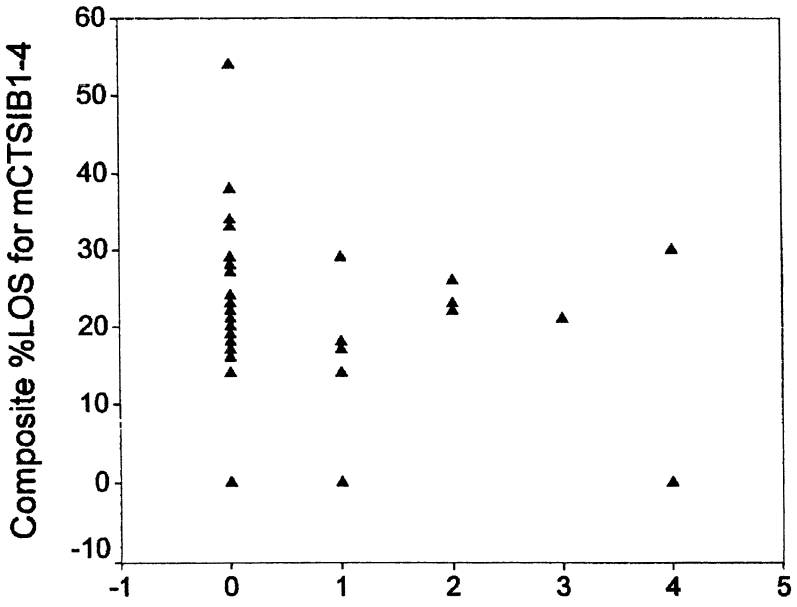
MCTSIB Condition 4

Eyes Closed on Foam Surface



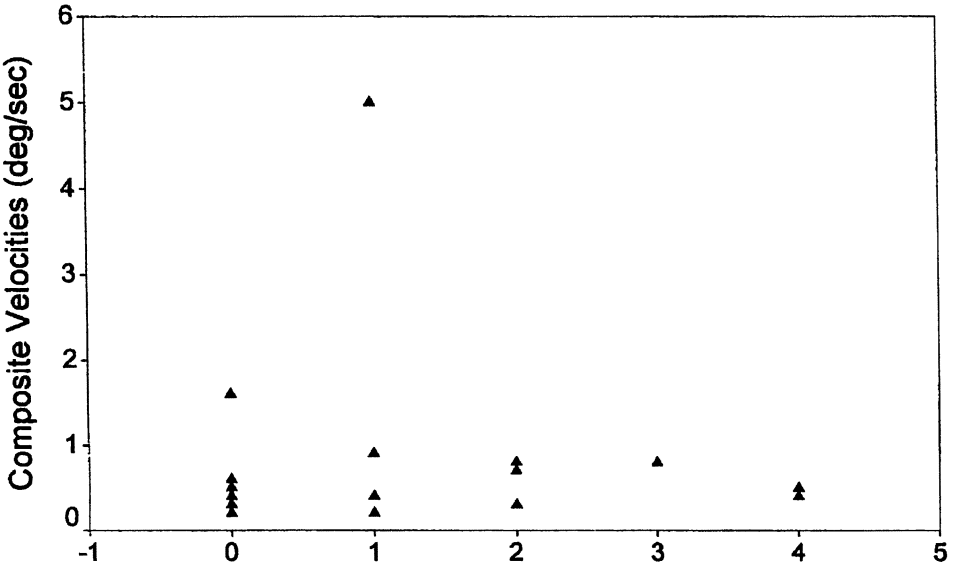
MCTSIB Limits of Stability

Conditions 1 through 4



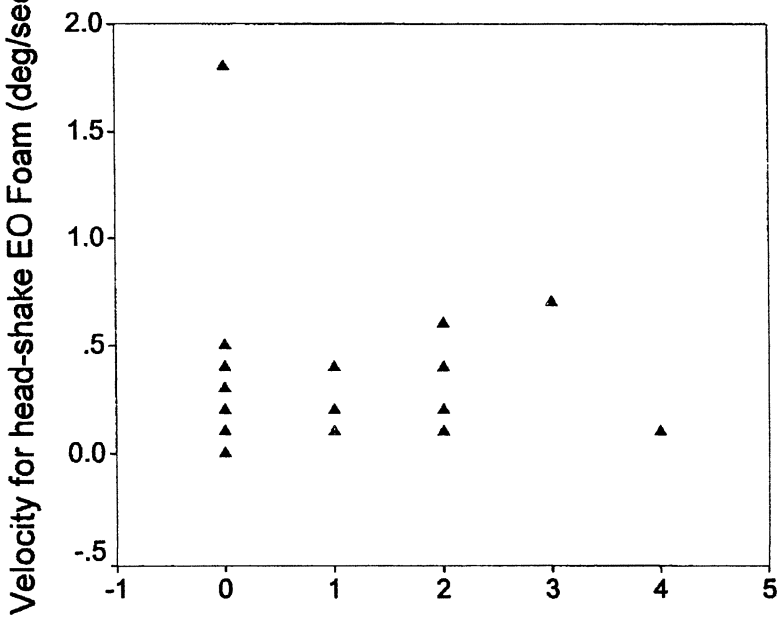
MCTSIB Composite Velocities

Conditions 1 through 4

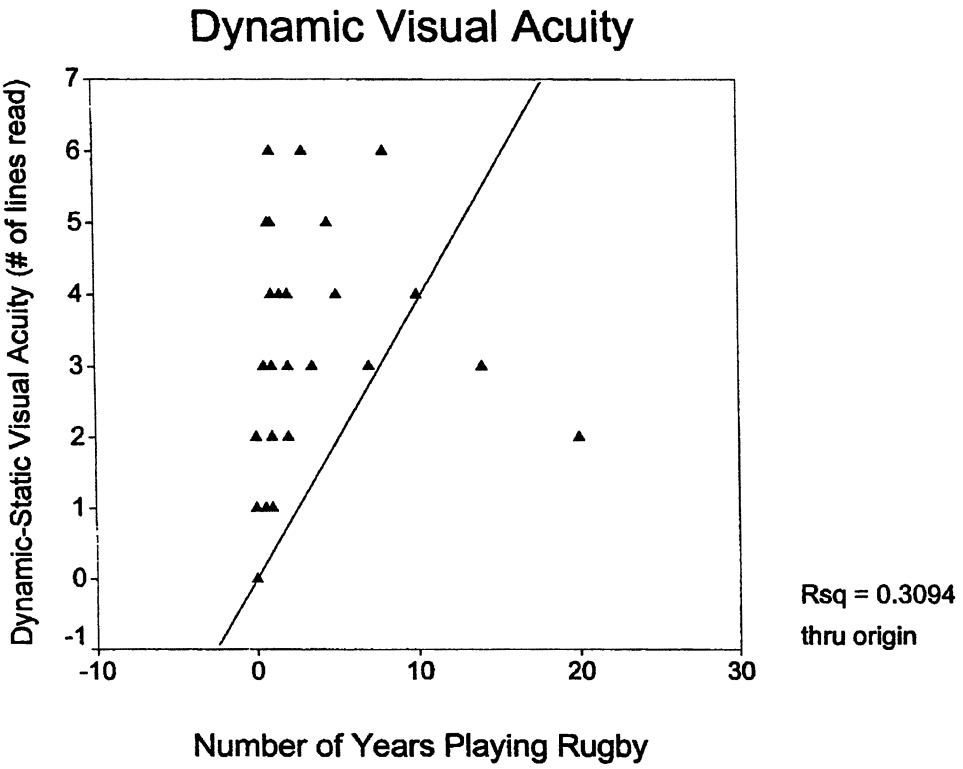


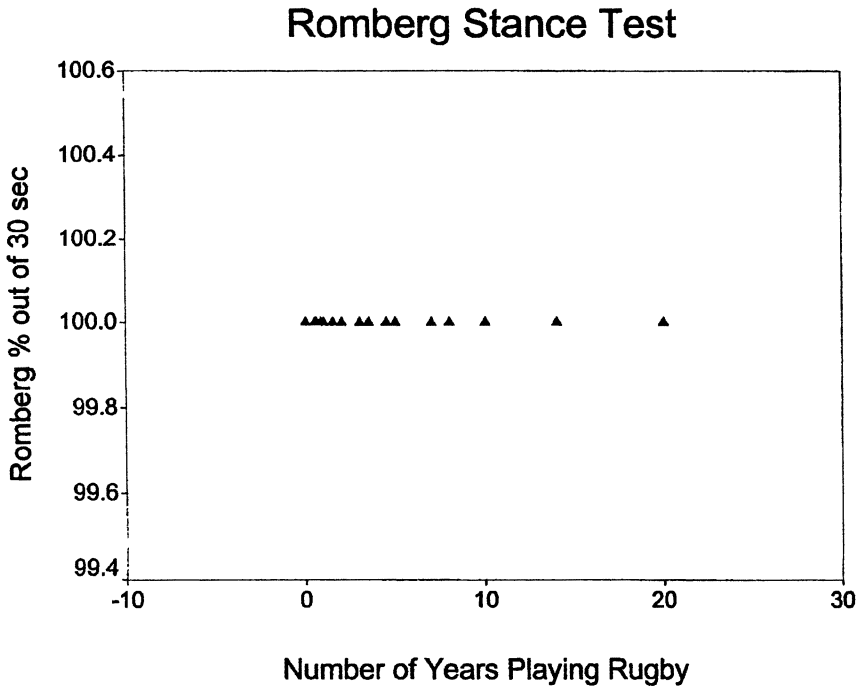
MCTSIB Condition Five

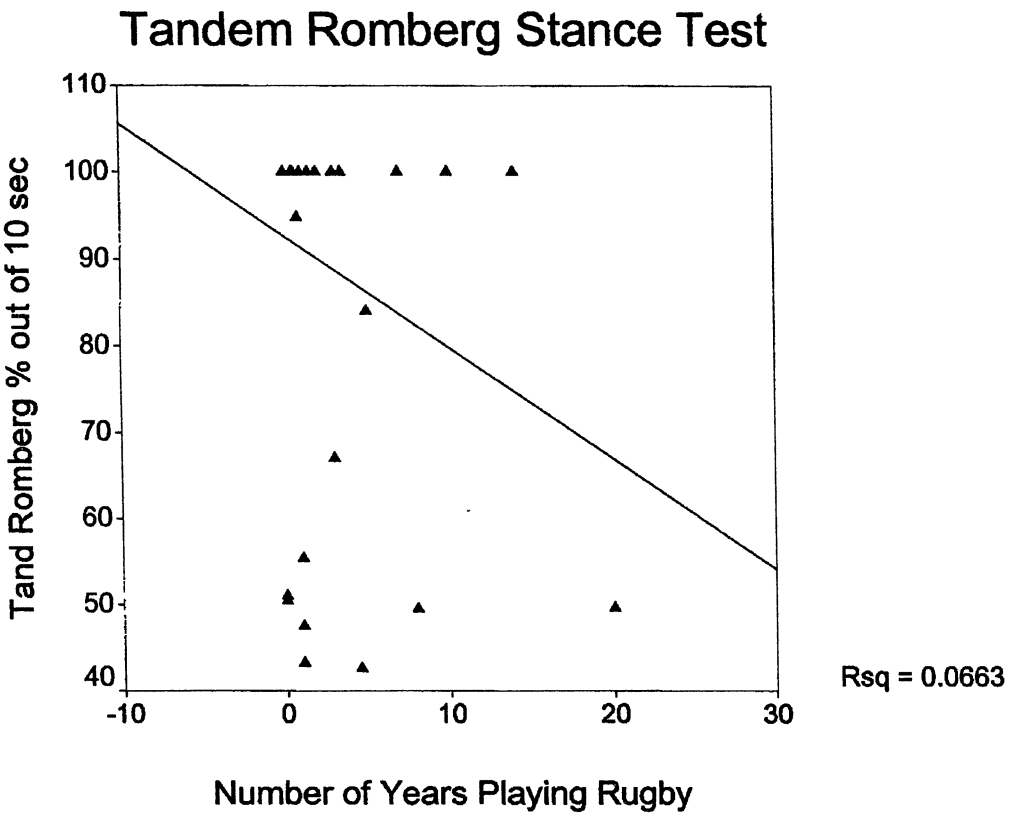
Head Shake with Eyes Open on Foam Surface

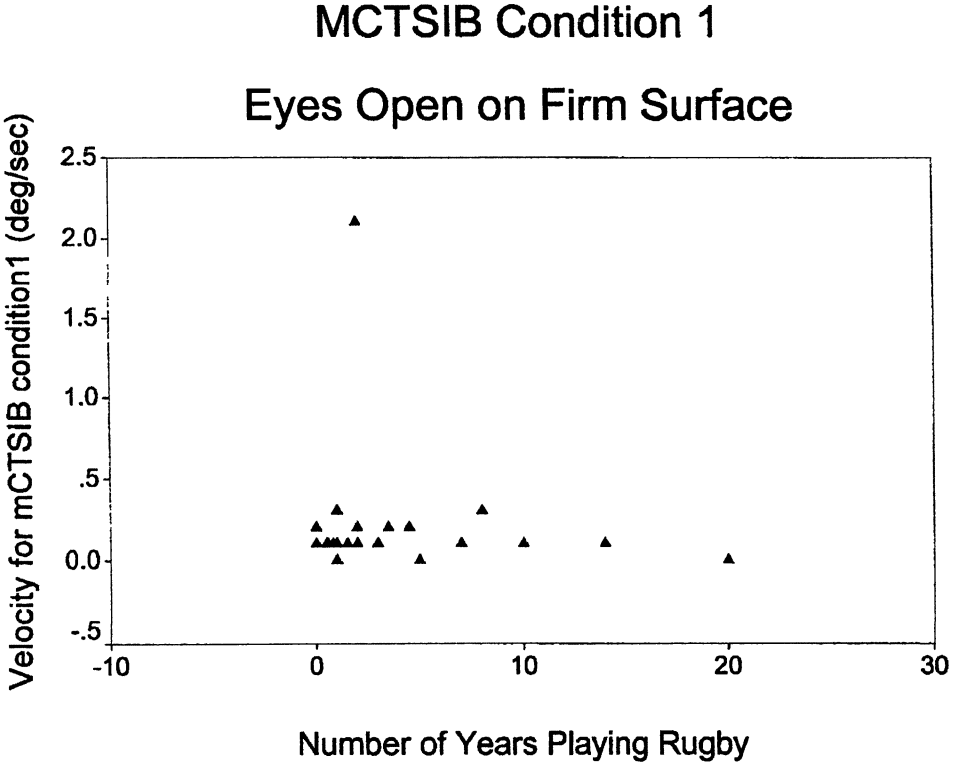


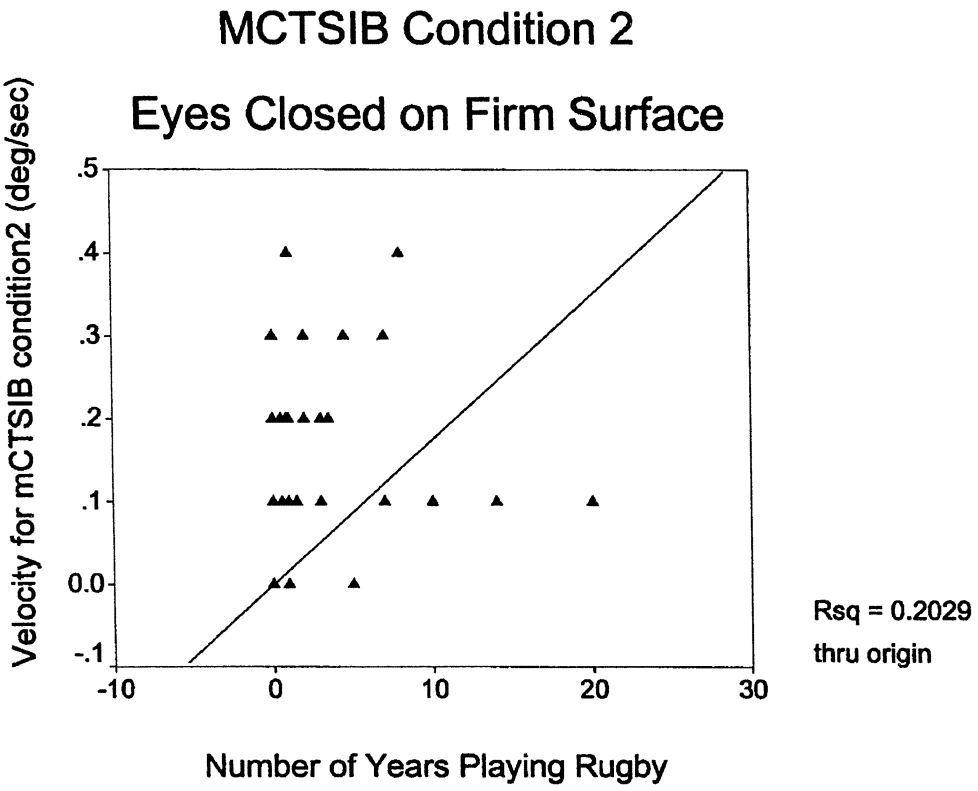
Correlations Between the ***Number of Years Playing Rugby*** and the Assessment Results

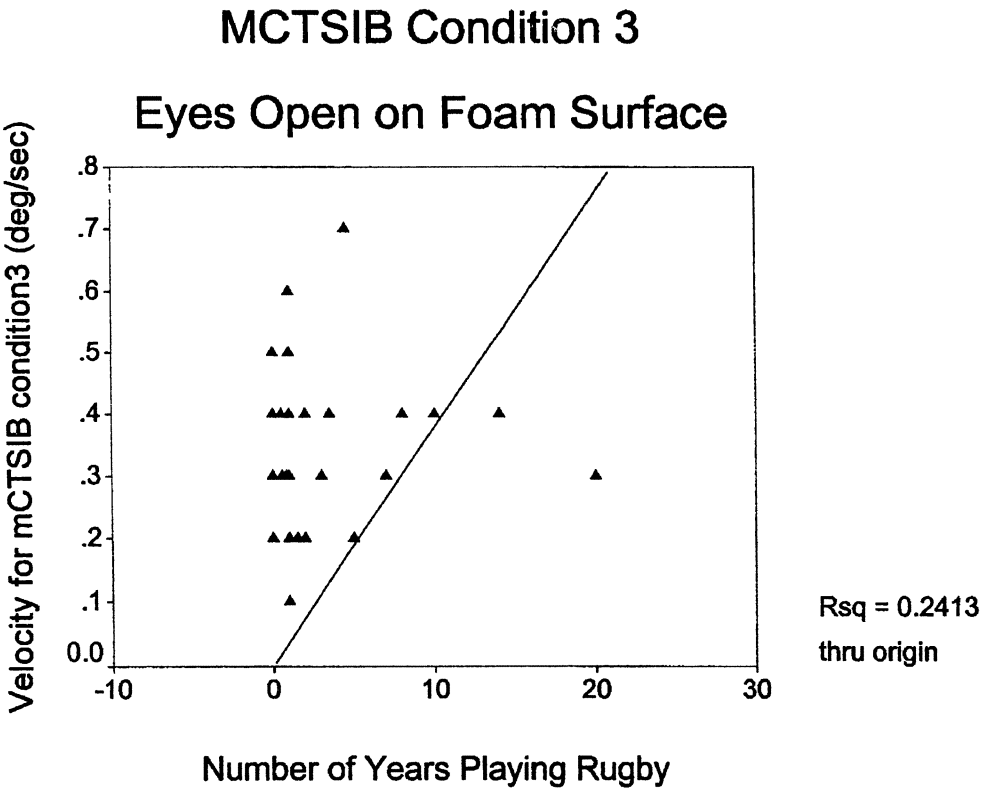


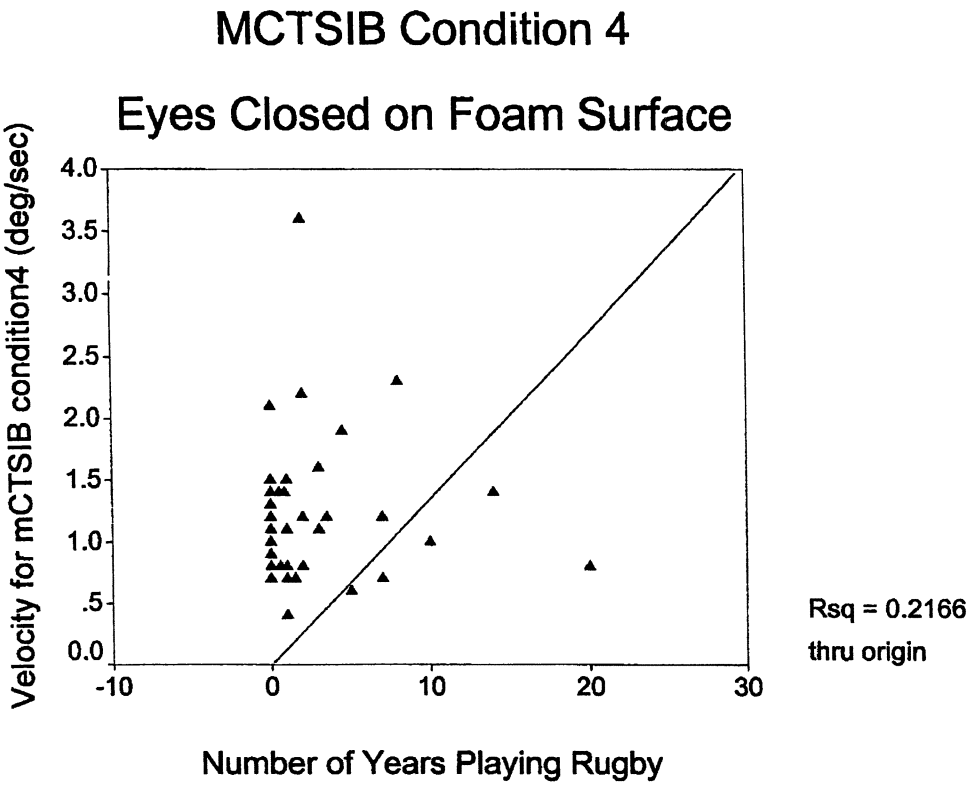


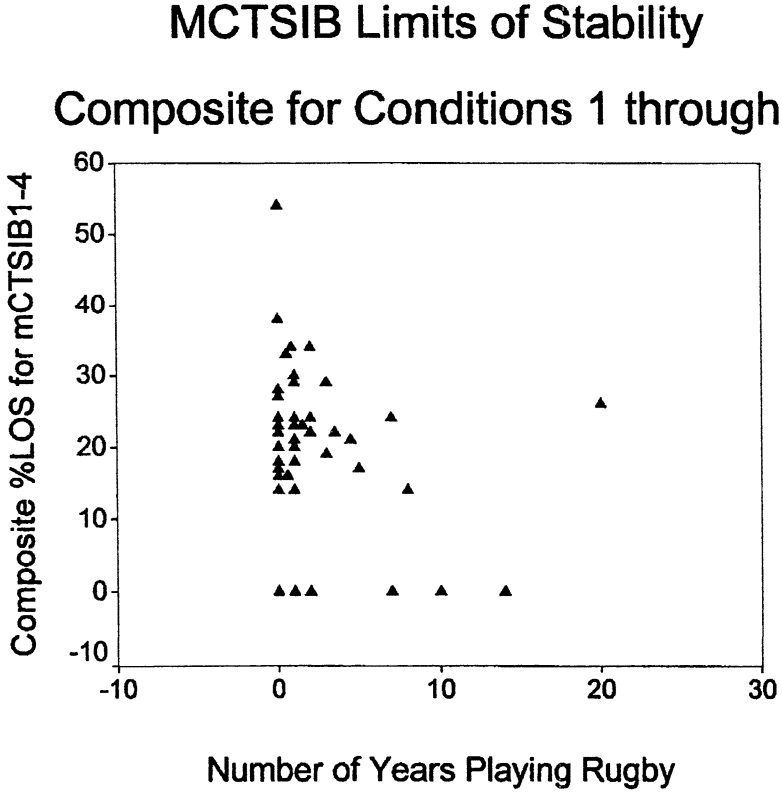

Tandem Romberg Stance Test
Tand Romberg % out of 10 sec
110
100
90
80
70
60
50
40
-10
0
10
20
30
Rsq = 0.0663
Number of Years Playing Rugby


MCTSIB Condition 1
Eyes Open on Firm Surface
Velocity for mCTSIB condition1 (deg/sec)
2.5
2.0
1.5
1.0
.5
0.0
-.5
-10
0
10
20
30
Number of Years Playing Rugby


MCTSIB Condition 2
Eyes Closed on Firm Surface
Velocity for mCTSIB condition2 (deg/sec)
.5
.4
.3
.2
.1
0.0
-.1
-10
0
10
20
30
Number of Years Playing Rugby
Rsq = 0.2029
thru origin

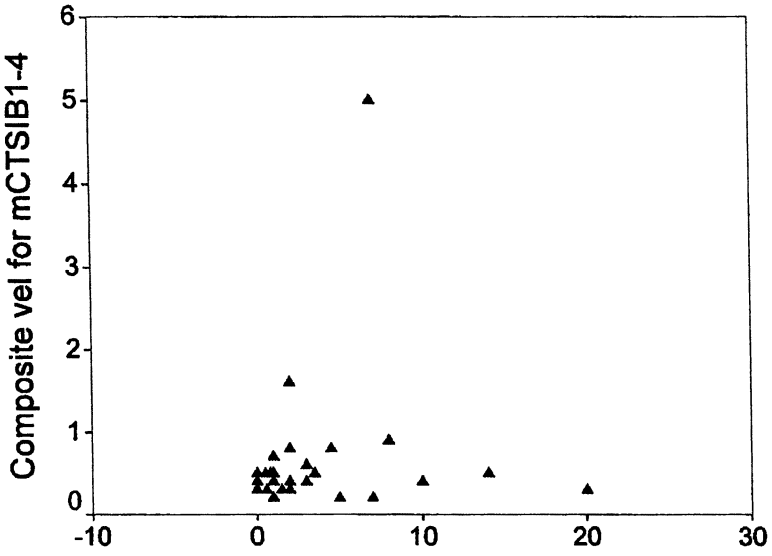

MCTSIB Condition 3
Eyes Open on Foam Surface
Velocity for mCTSIB condition3 (deg/sec)
.8
.7
.6
.5
.4
.3
.2
.1
0.0
-10
0
10
20
30
Number of Years Playing Rugby
Rsq = 0.2413
thru origin


MCTSIB Condition 4
Eyes Closed on Foam Surface
Velocity for mCTSIB condition4 (deg/sec)
4.0
3.5
3.0
2.5
2.0
1.5
1.0
.5
0.0
-10
0
10
20
30
Rsq = 0.2166
thru origin
Number of Years Playing Rugby


MCTSIB Limits of Stability
Composite for Conditions 1 through 4
Composite %LOS for mCTSIB1-4
60
50
40
30
20
10
0
-10
-10
0
10
20
30
Number of Years Playing Rugby

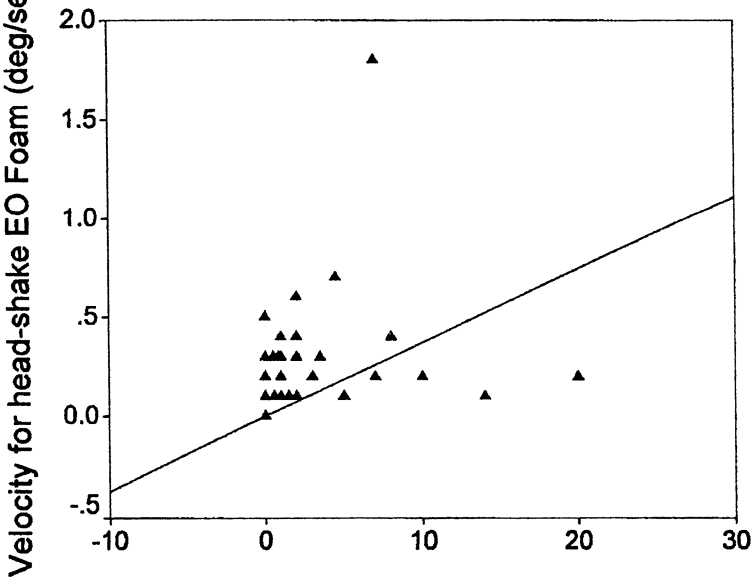
MCTSIB Composite Velocities

Conditions 1 through 4



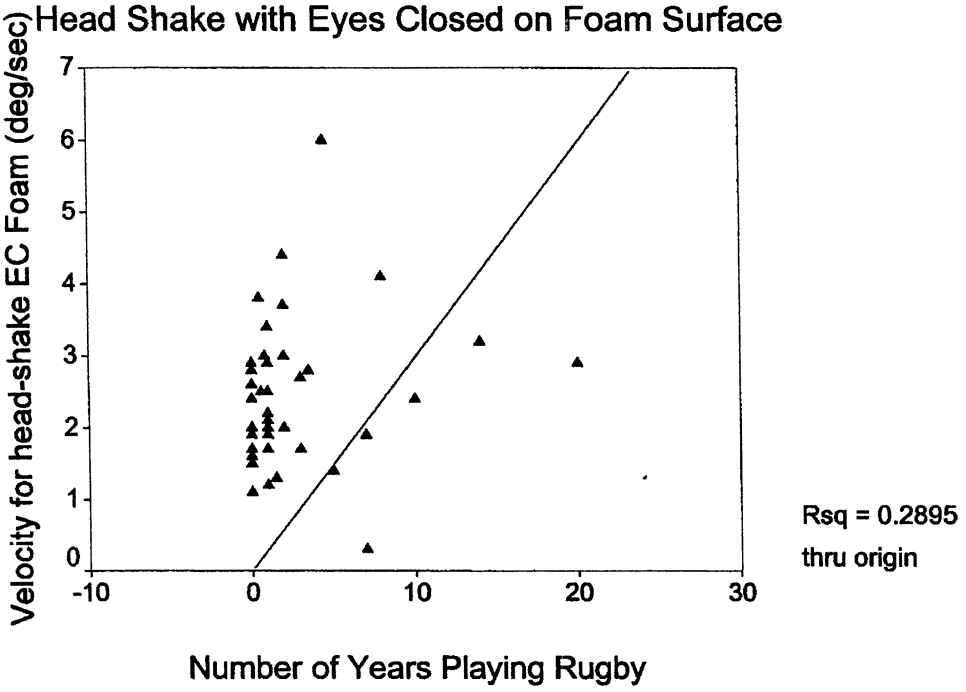
MCTSIB Condition 5

Head Shake with Eyes Open on Foam Surface

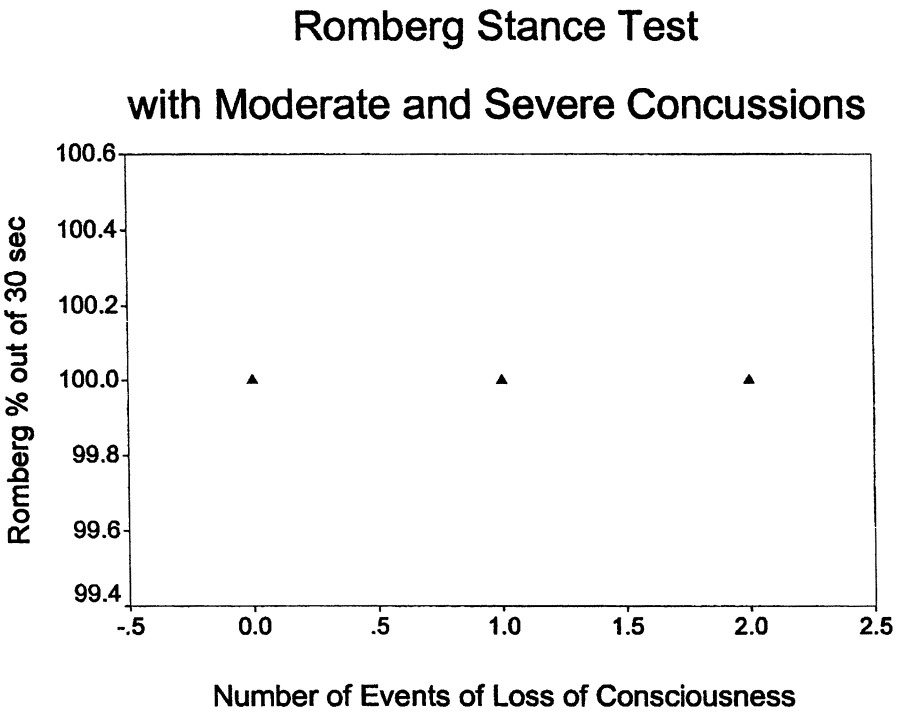


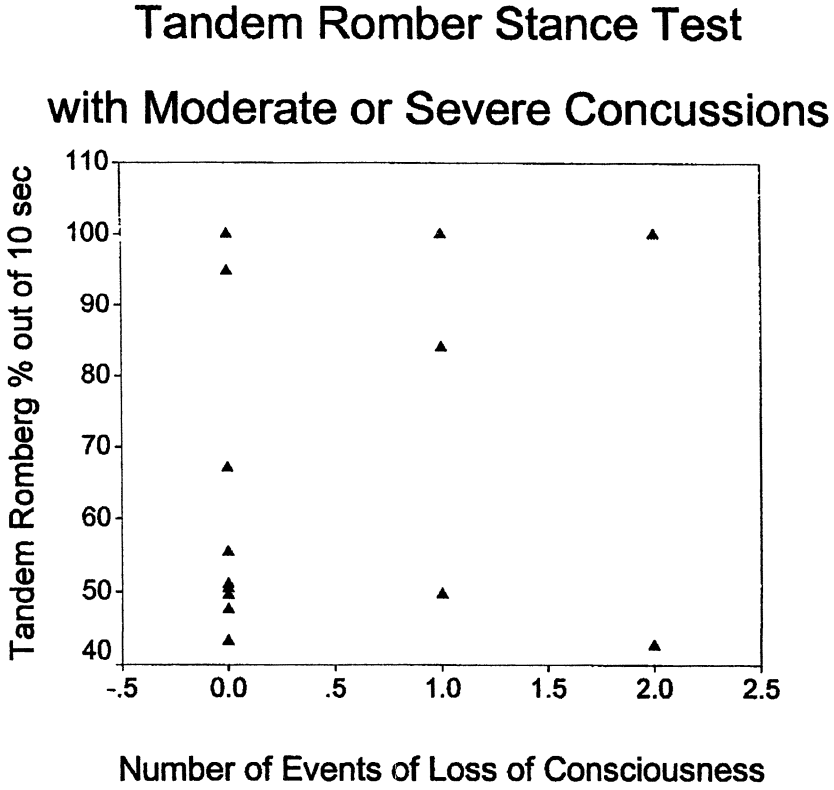
Rsq = 0.2092
thru origin

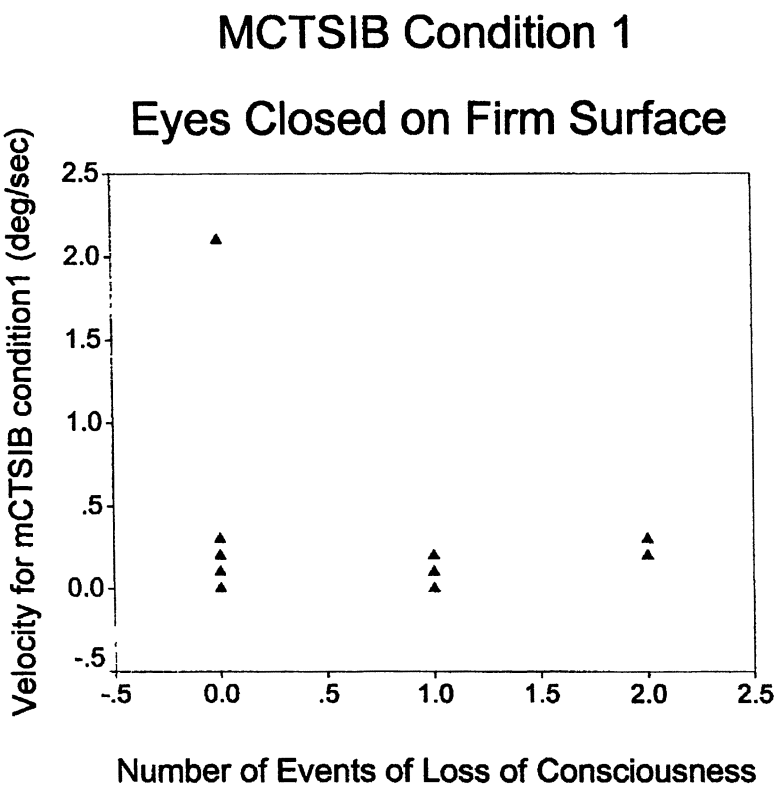
MCTSIB Condition 6

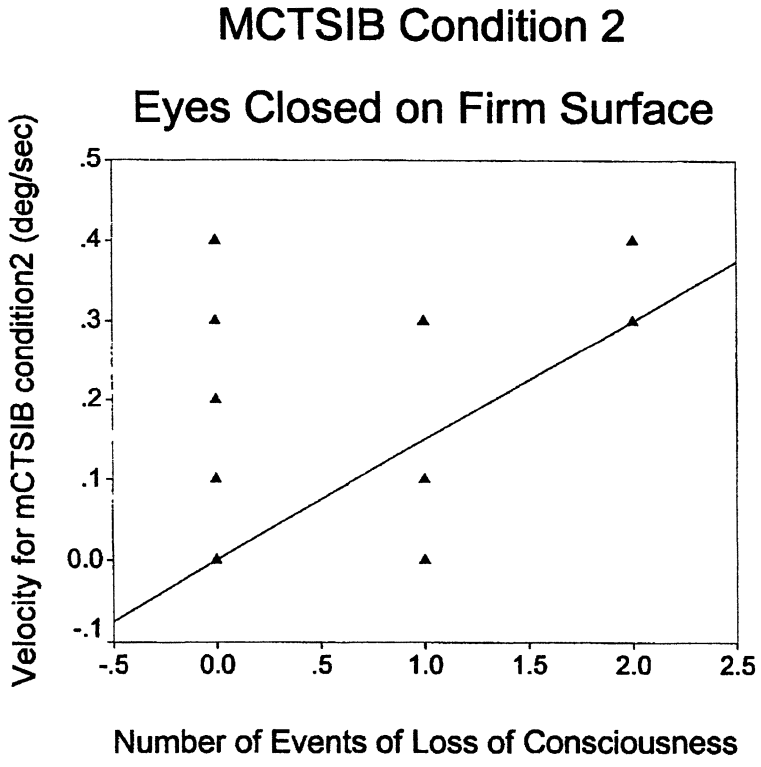


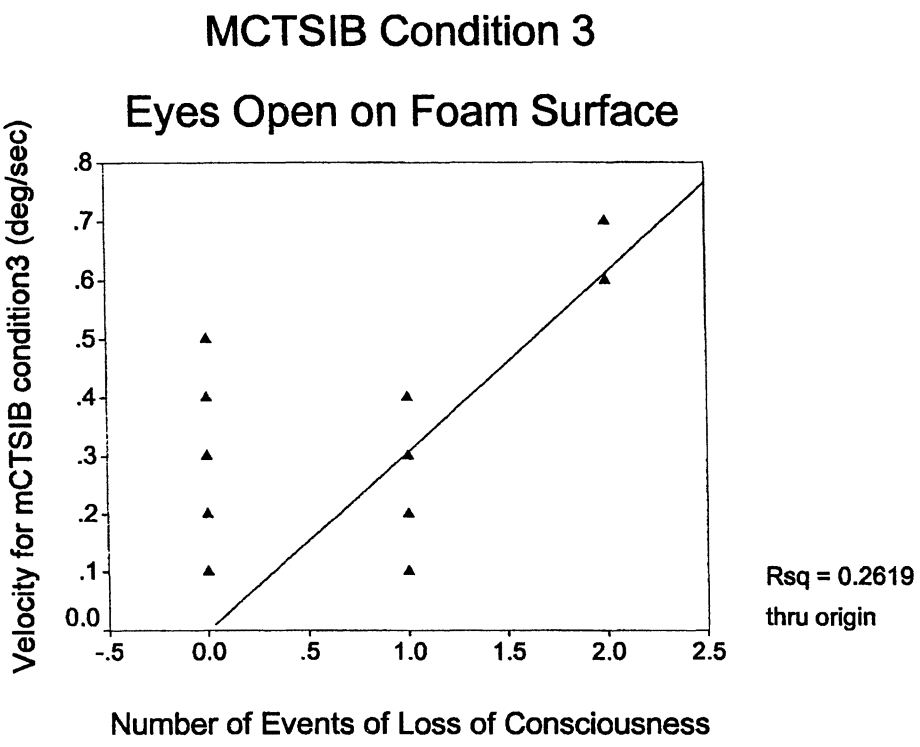
Correlations Between the ***Number of Moderate or Severe Concussions*** and the Assessment Results

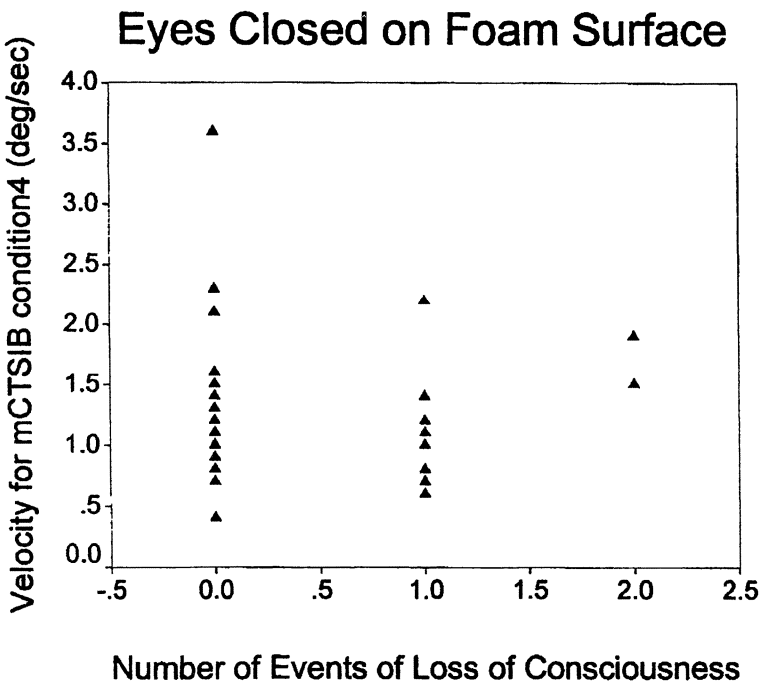




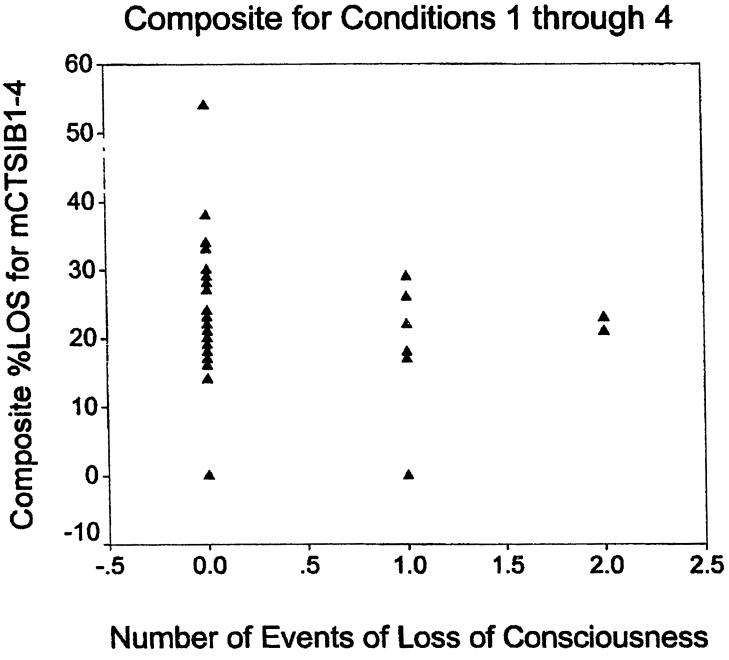

MCTSIB Condition 1
Eyes Closed on Firm Surface
Velocity for mCTSIB condition1 (deg/sec)
2.5
2.0
1.5
1.0
.5
0.0
-.5
-.5
0.0
.5
1.0
1.5
2.0
2.5
Number of Events of Loss of Consciousness


MCTSIB Condition 2
Eyes Closed on Firm Surface
Velocity for mCTSIB condition2 (deg/sec)
.5
.4
.3
.2
.1
0.0
-.1
-.5
0.0
.5
1.0
1.5
2.0
2.5
Number of Events of Loss of Consciousness
Rsq = 0.2483
thru origin

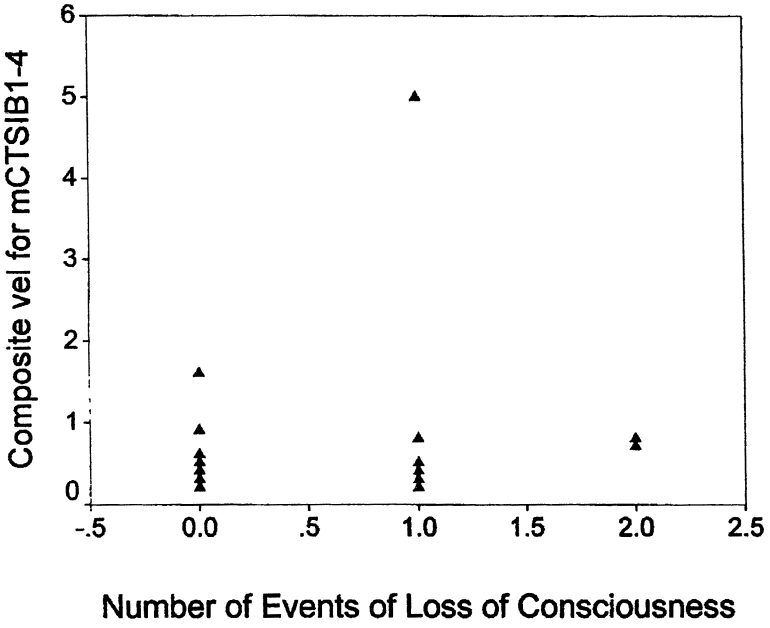

MCTSIB Condition 3
Eyes Open on Foam Surface
Velocity for mCTSIB condition3 (deg/sec)
.8
.7
.6
.5
.4
.3
.2
.1
0.0
-.5
0.0
.5
1.0
1.5
2.0
2.5
Rsq = 0.2619
thru origin
Number of Events of Loss of Consciousness


MCTSIB Condition 4
Eyes Closed on Foam Surface
Velocity for mCTSIB condition4 (deg/sec)
4.0
3.5
3.0
2.5
2.0
1.5
1.0
.5
0.0
-.5
0.0
.5
1.0
1.5
2.0
2.5
Number of Events of Loss of Consciousness

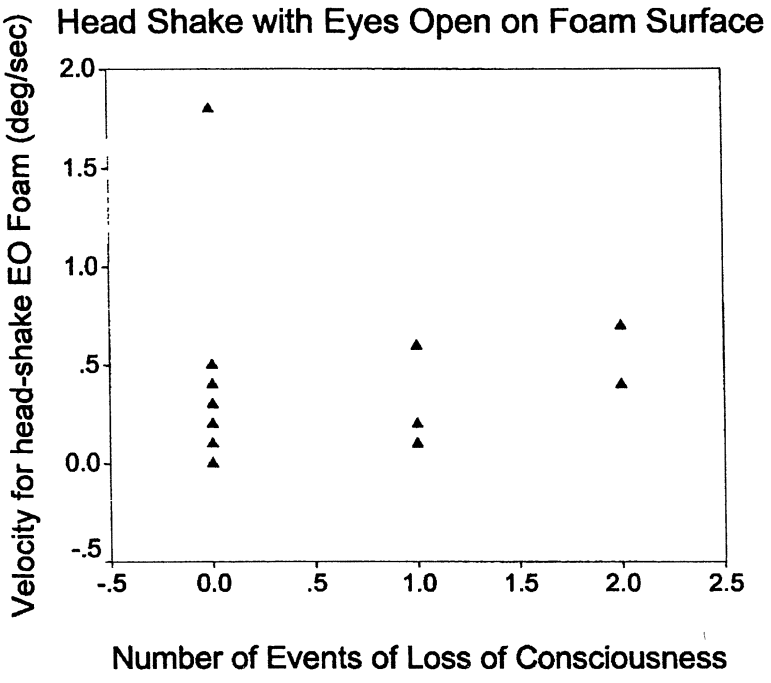
MCTSIB Limits of Stability



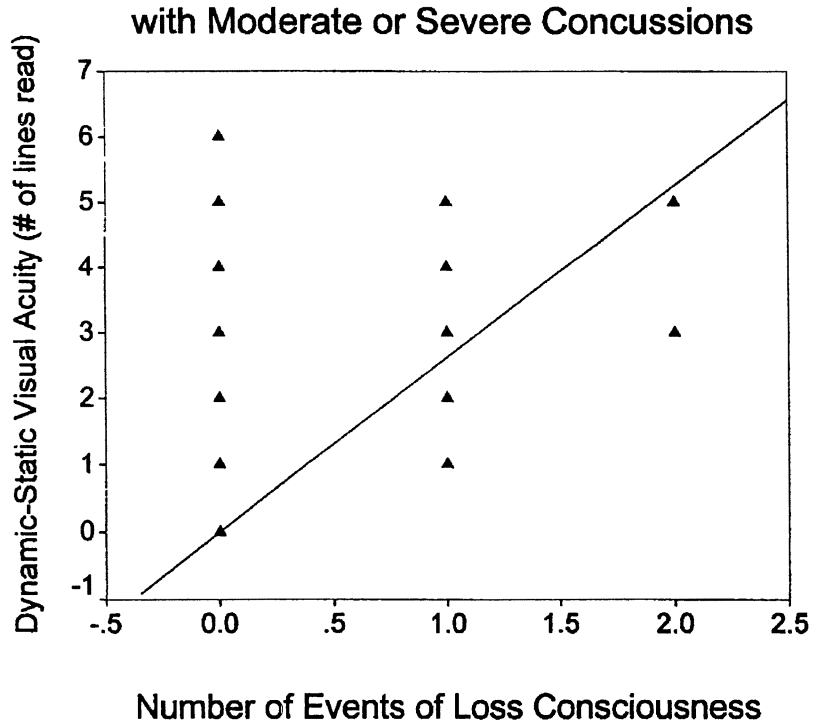
MCTSIB Composite Velocities



MCTSIB Condition 5

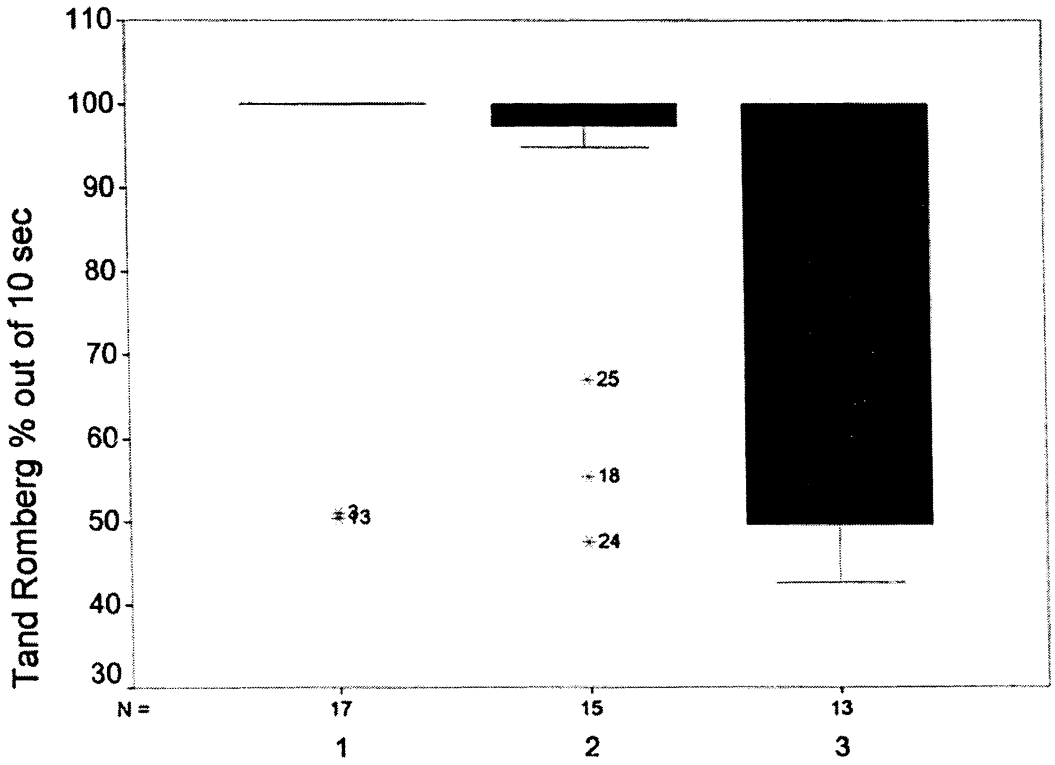


Dynamic Visual Acuity

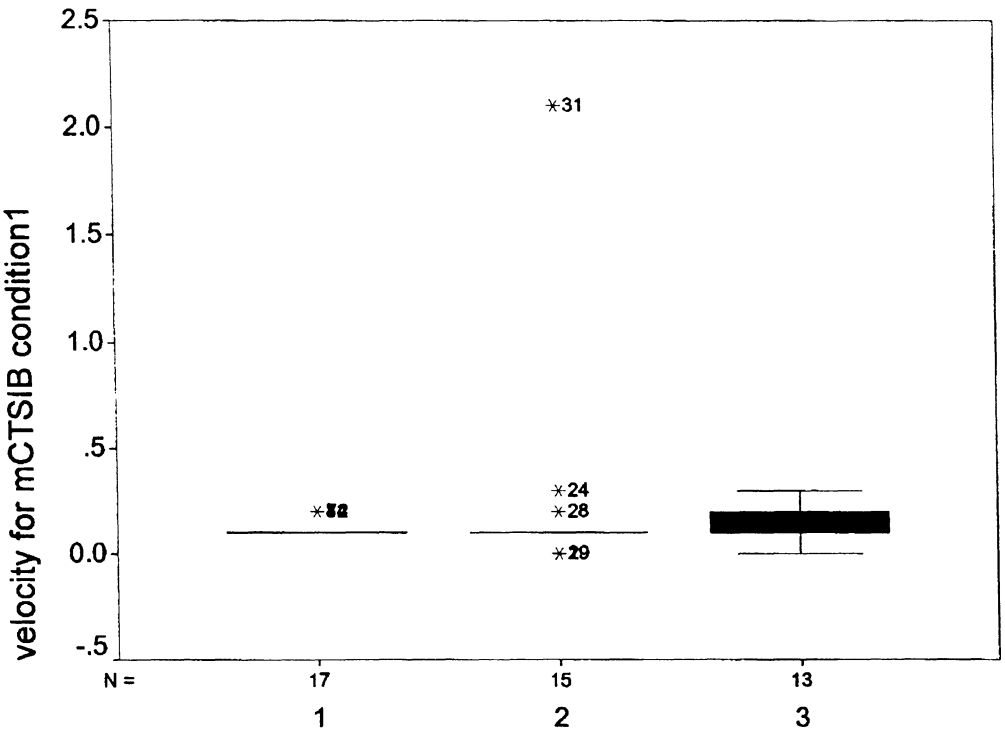


Results of the One-way ANOVA: ***Comparisons Between Each of the Three Subject Groups*** *(Group 1=Control, Group 2=Rugby players with no history of head injury, Group 3=Rugby players with history of head injury)*

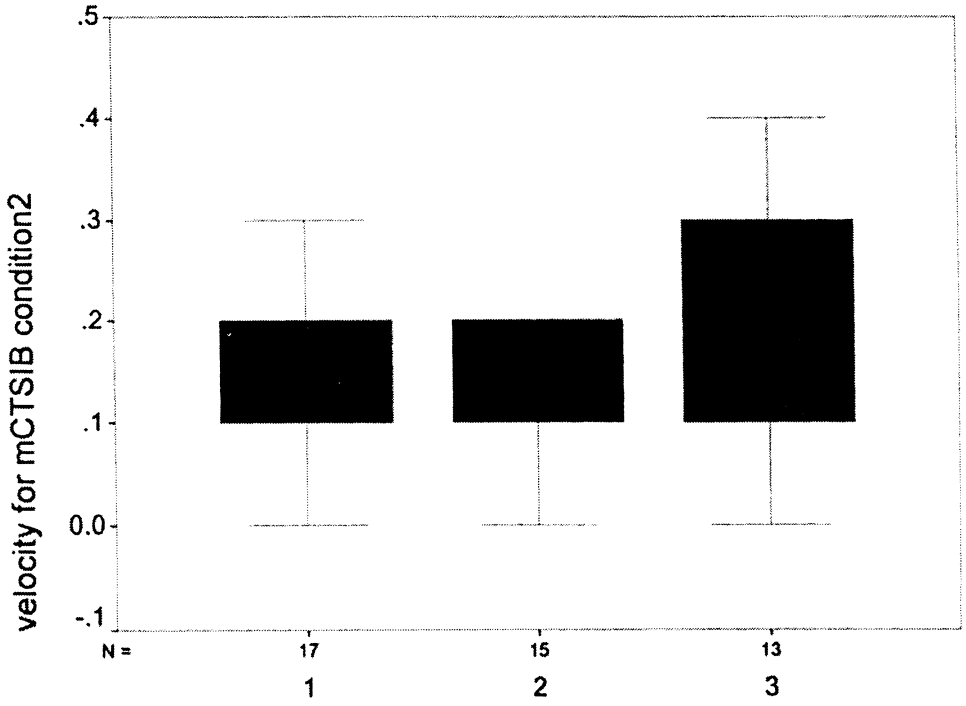
Tandem Romberg



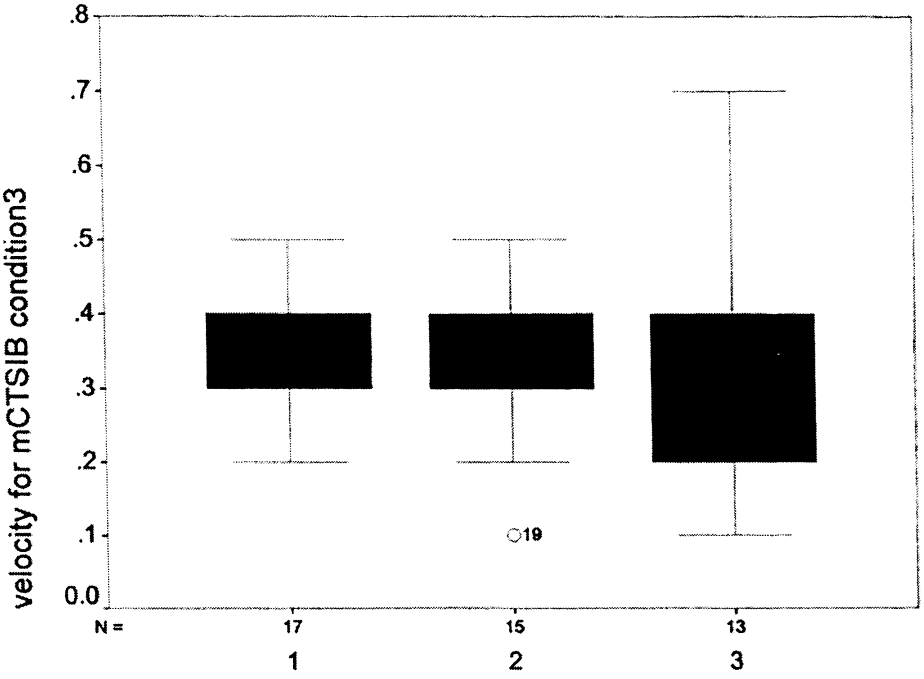
Sway Velocity for Condition 1 of the MCTSIB



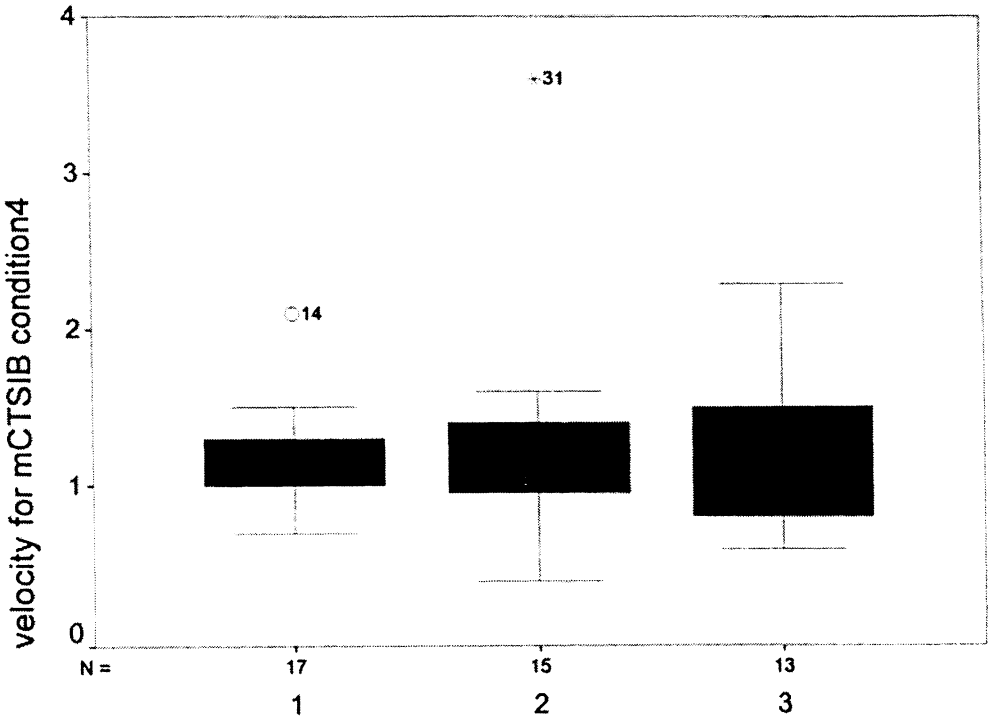
Sway Velocity for Condition 2 of the MCTSIB



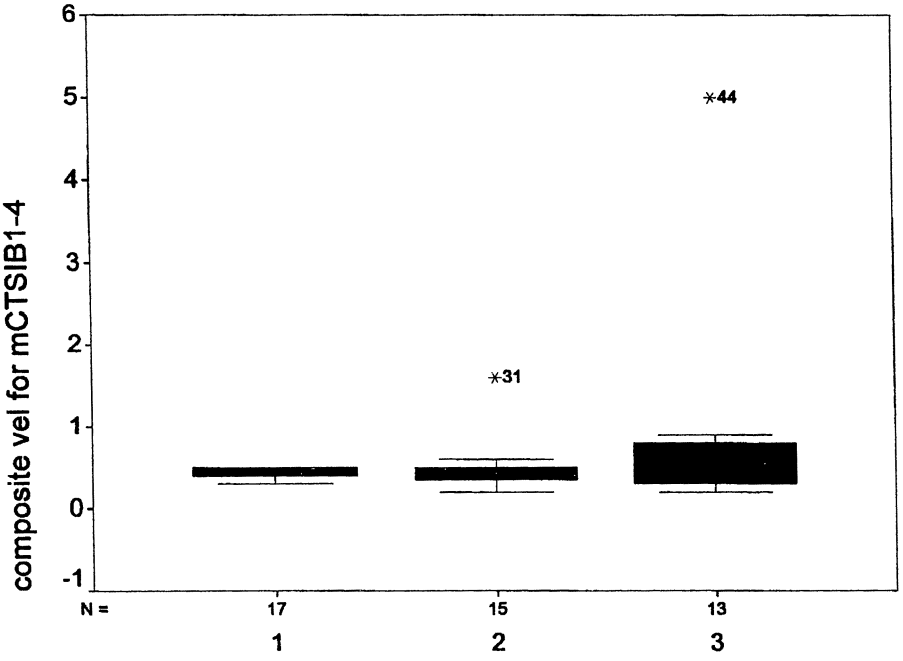
Sway Velocities for Condition 3 of the MCTSIB



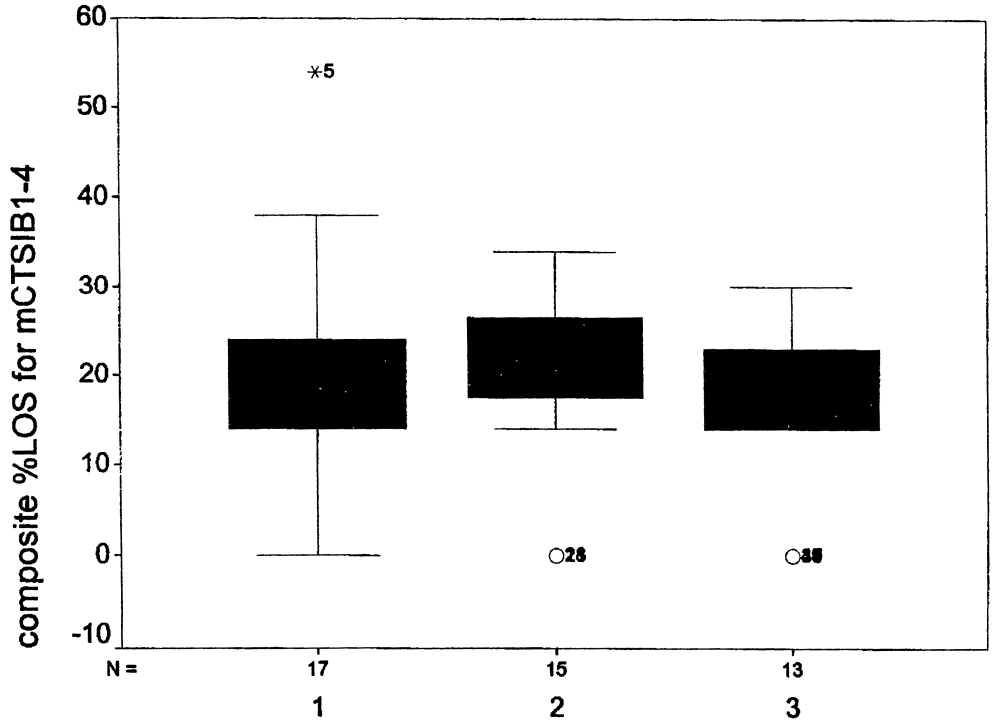
Sway Velocities for Condition 4 of the MCTSIB



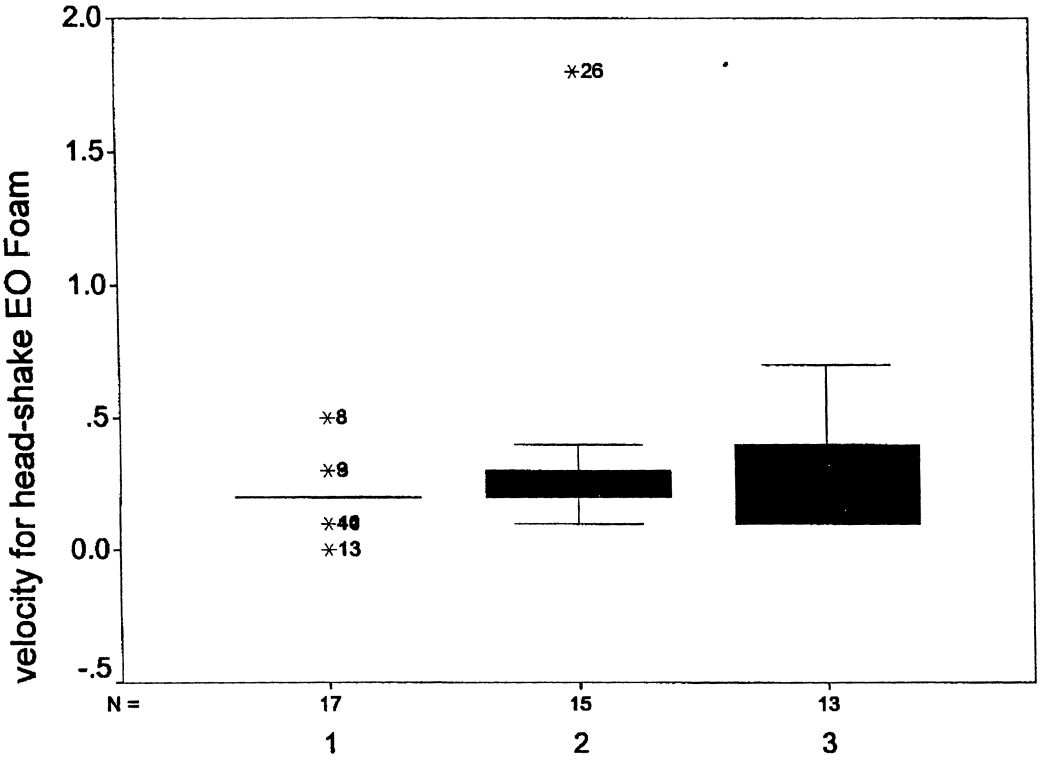
Composite Sway Velocities for MCTSIB Conditions 1-4



Limits of Stability for MCTSIB Conditions 1-4



Sway Velocities for Condition 5 of the MCTSIB



Appendix Q: Data pertaining to case study 1

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

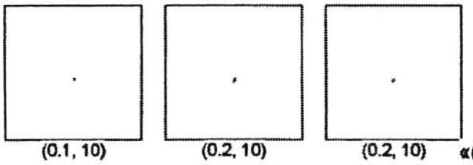
Name:
ID: ATID00252
Date of Birth:
Height: 5'4"

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD252.DRX
Date: 3/15/2004
Time: 19:54:39

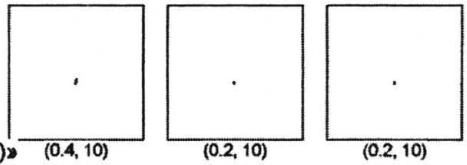
Modified CTSIB

1. Firm--Eyes Open (FIRM-EO)



(0.1, 10) Trial 1 | (0.2, 10) Trial 2 | (0.2, 10) Trial 3

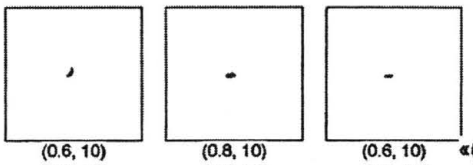
2. Firm--Eyes Closed (FIRM-EC)



«(deg/sec)»

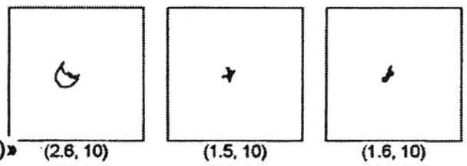
(0.4, 10) Trial 1 | (0.2, 10) Trial 2 | (0.2, 10) Trial 3

3. Foam--Eyes Open (FOAM-EO)



(0.6, 10) Trial 1 | (0.8, 10) Trial 2 | (0.6, 10) Trial 3

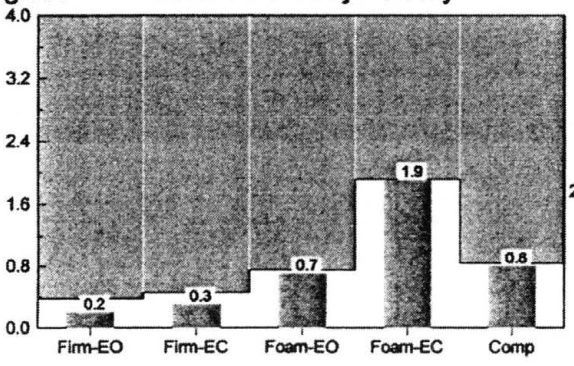
4. Foam--Eyes Closed (FOAM-EC)



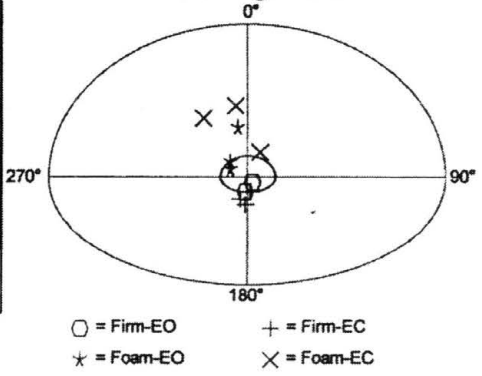
«(deg/sec)»

(2.6, 10) Trial 1 | (1.5, 10) Trial 2 | (1.6, 10) Trial 3

deg/sec — Mean COG Sway Velocity



COG Alignment



COG Alignment:
Scattered, 21%LOS

Data Range Note: User Data Range: 20--39

Post Test Comment:
No Head-shake

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

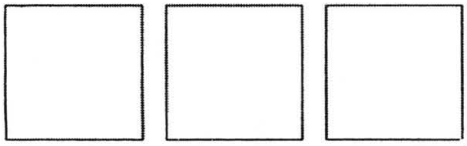
Name:
ID: ATID00252
Date of Birth:
Height: 5'4"

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD252.DRX
Date: 3/15/2004
Time: 20:00:39

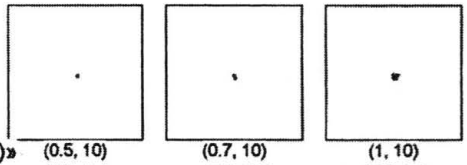
Modified CTSIB

1. Firm--Eyes Open (FIRM-EO)



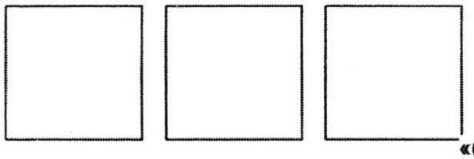
Trial 1 Trial 2 Trial 3

2. Firm--Eyes Closed (FIRM-EC)



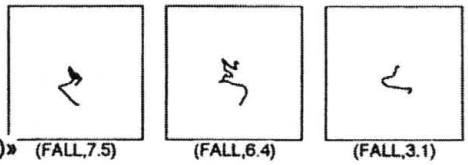
«(deg/sec)» (0.5, 10) (0.7, 10) (1, 10)
Trial 1 Trial 2 Trial 3

3. Foam--Eyes Open (FOAM-EO)



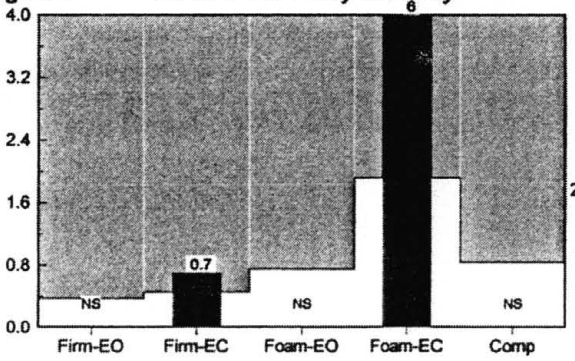
Trial 1 Trial 2 Trial 3

4. Foam--Eyes Closed (FOAM-EC)

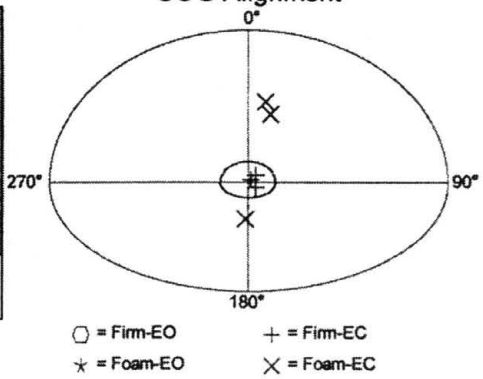


«(deg/sec)» (FALL,7.5) (FALL,6.4) (FALL,3.1)
Trial 1 Trial 2 Trial 3

deg/sec Mean COG Sway Velocity



COG Alignment



COG Alignment:
Scattered, 24%LOS

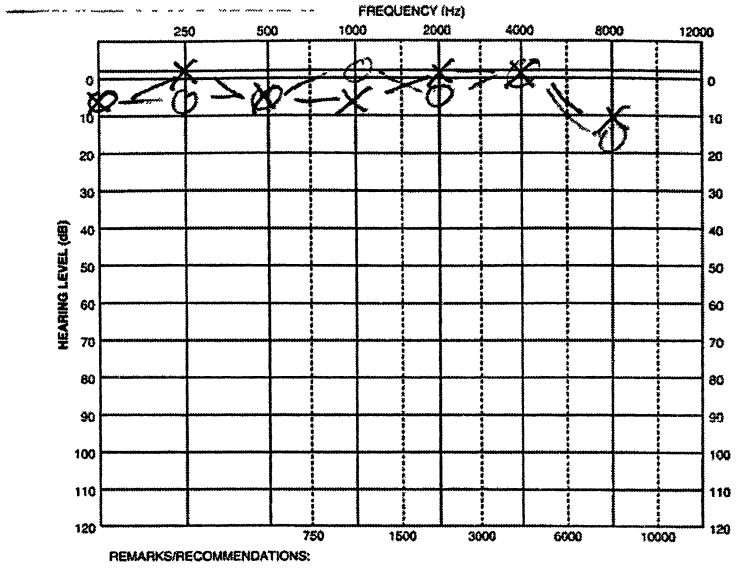
Data Range Note: User Data Range: 20--39

Post Test Comment:
With Head-shake

Appendix R: Data pertaining to case study 2
Initial Post-Injury Assessment

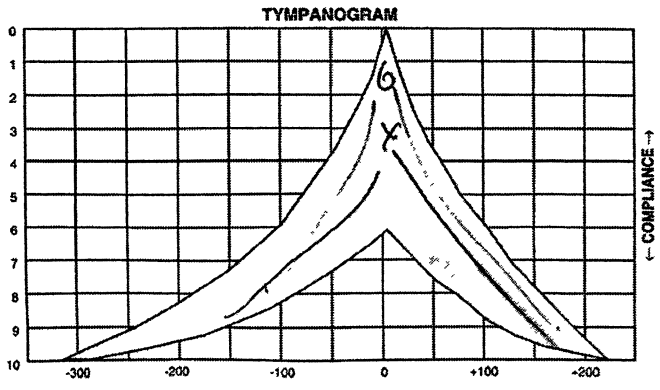
EAR SPECIALISTS OF AUSTIN
12221 N. Mopac Expressway, Building B
Austin, Texas 78758
JAMES V. KEMPER, JR., M.D.

SEX	AGE	TINNITUS TYPE	DATE
M	21		05. 19. 04
AUDIOMETER (ANSI 1969)	TEST RELIABILITY		REFERRED BY
GSI 61	Good		



SUMMARY OF RESULTS			
TEST	RT	LT	S F.
PTA			
LF PTA			
SRT/SAT	10	15	
SPEECH DISCRIMINATION	90 dB	92 %	96 %
	dB	%	%
	dB	%	%
TYMP			
Speech Stimuli:			

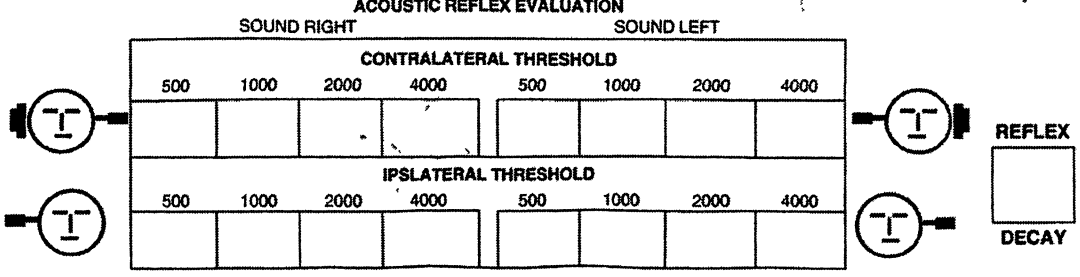
↓ NO RESPONSE
S SOUND FIELD
A S F AIDED
V FOREHEAD
* MASKED



	SYMBOL	
	UNMASKED	MASKED
AIR CONDUCTION RIGHT	O	△
AIR CONDUCTION LEFT	X	□
BONE CONDUCTION RIGHT	<	[
BONE CONDUCTION LEFT	>	]

RIGHT EAR		LEFT EAR	
C_2	1.9 cc	C_2	1.5 cc
C_1	1.0 cc	C_1	1.2 cc

9 10

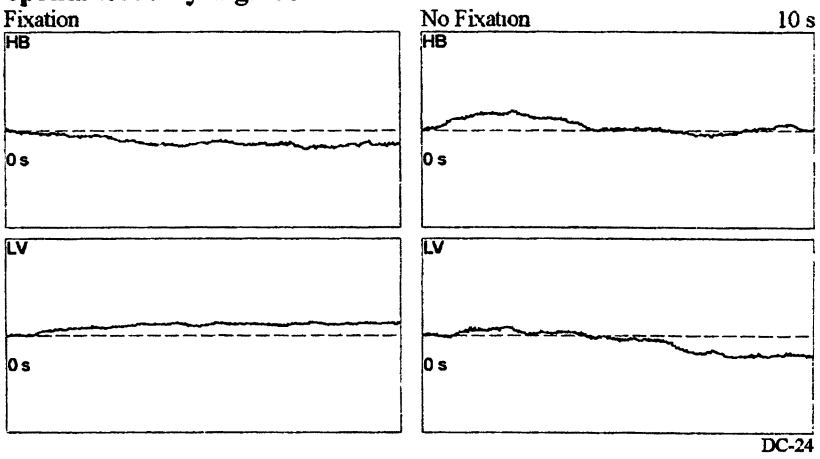


Ear Specialists of Austin
James Kemper, M.D.

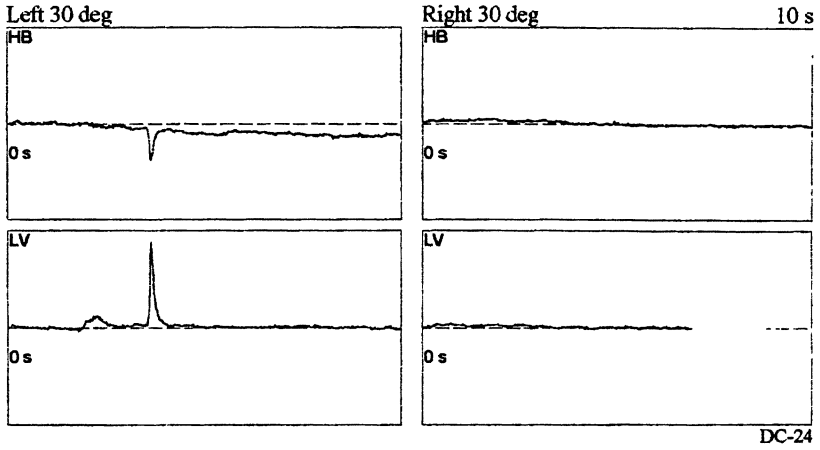
Name:
Age: 27
Visit Date: 5/19/04

10 deg.
1

Spontaneous Nystagmus



Gaze-Horizontal

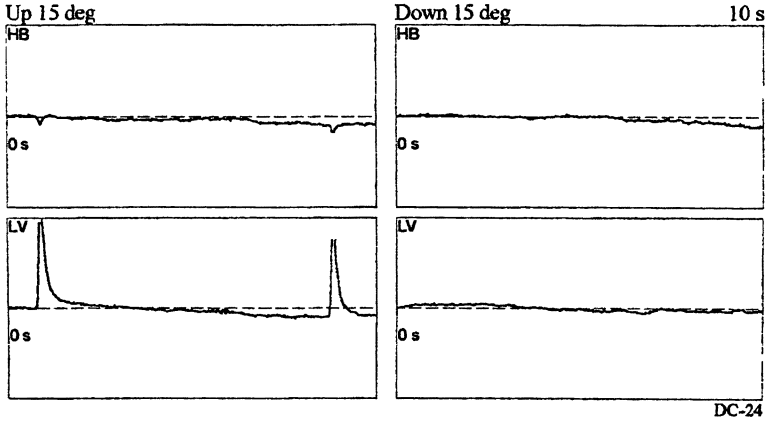


Ear Specialists of Austin
James Kemper, M.D.

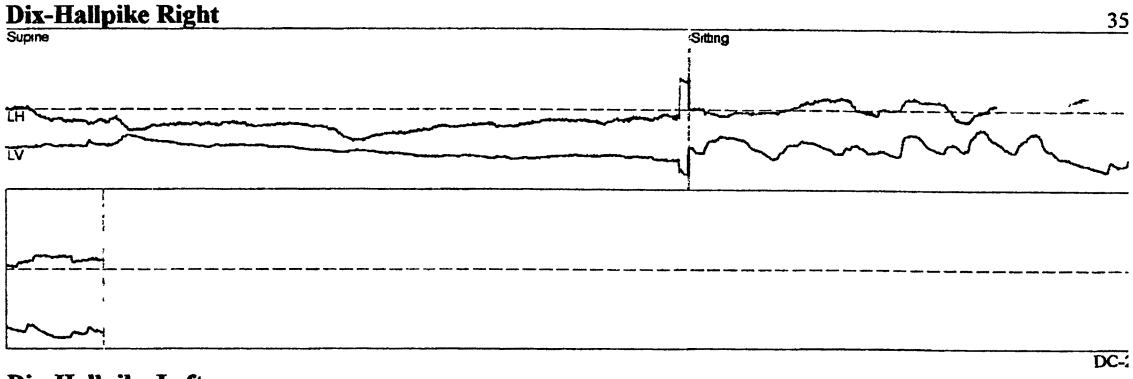
Name
Age: 27
Visit Date: 5/19/04

10 deg.
1 s

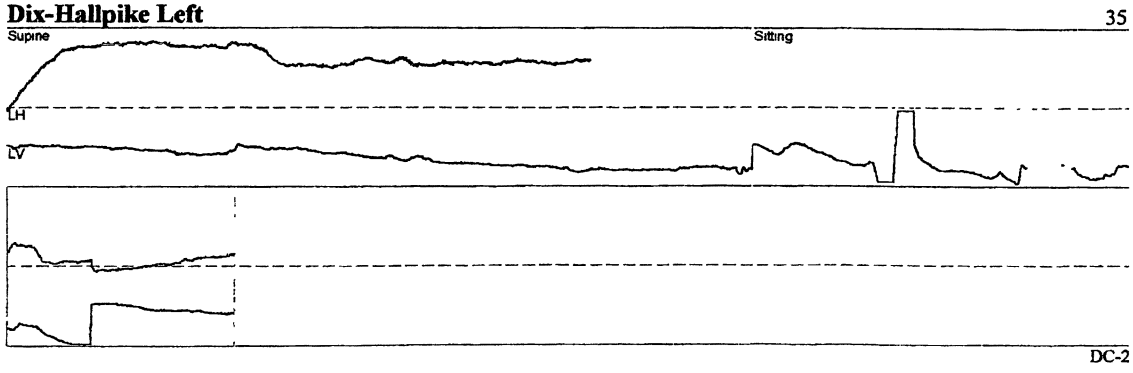
Gaze-Vertical



Dix-Hallpike Right



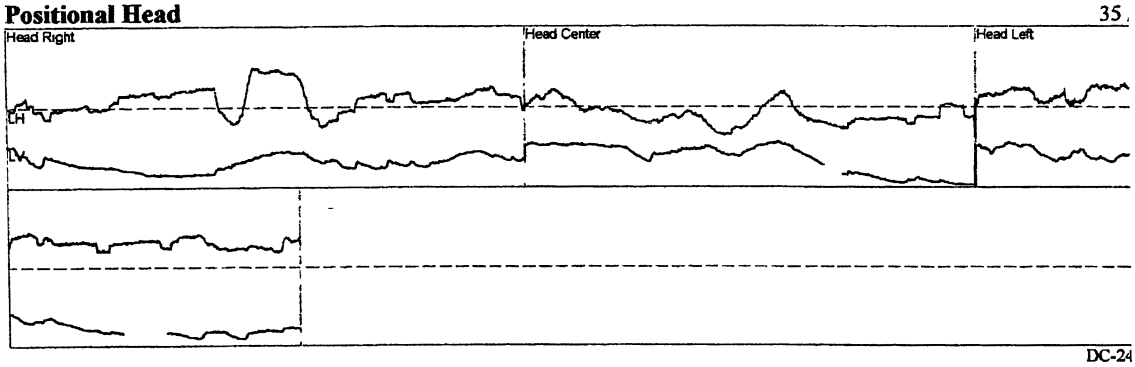
Dix-Hallpike Left



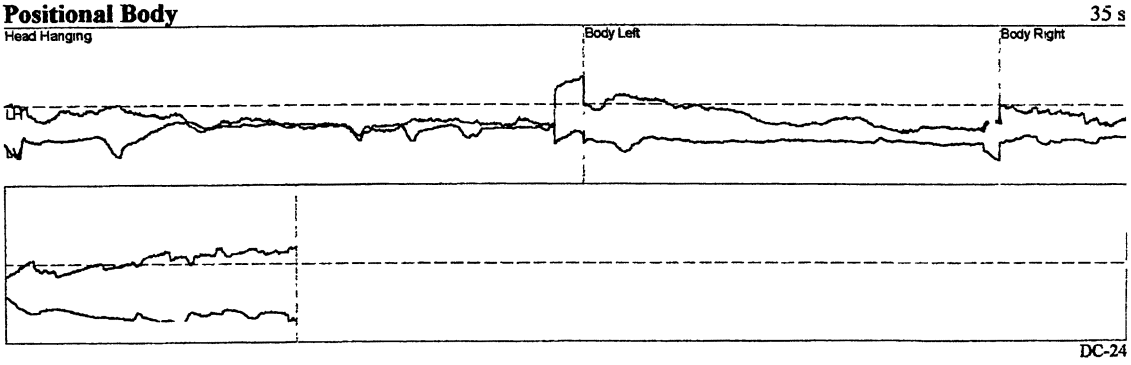
Ear Specialists of Austin
James Kemper, M.D.

Name
Age: 27
Visit Date: 5/19/04

Positional Head 35 s



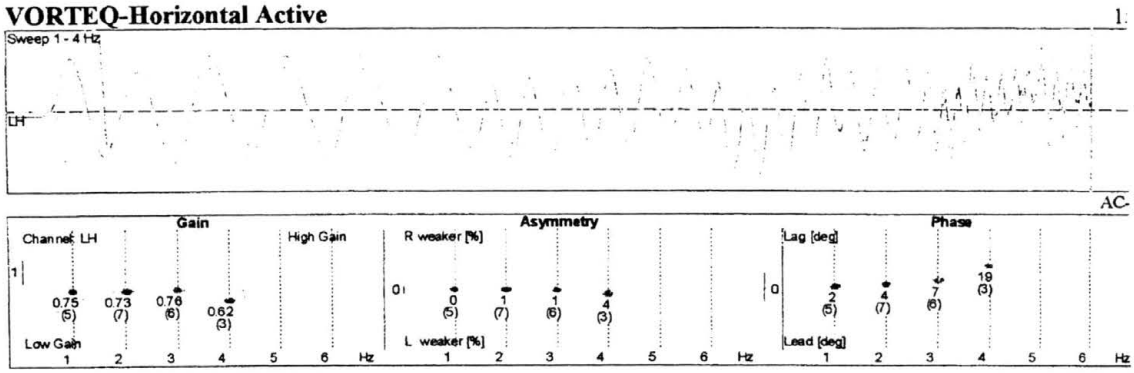
Positional Body 35 s



Ear Specialists of Austin
James Kemper, M.D.

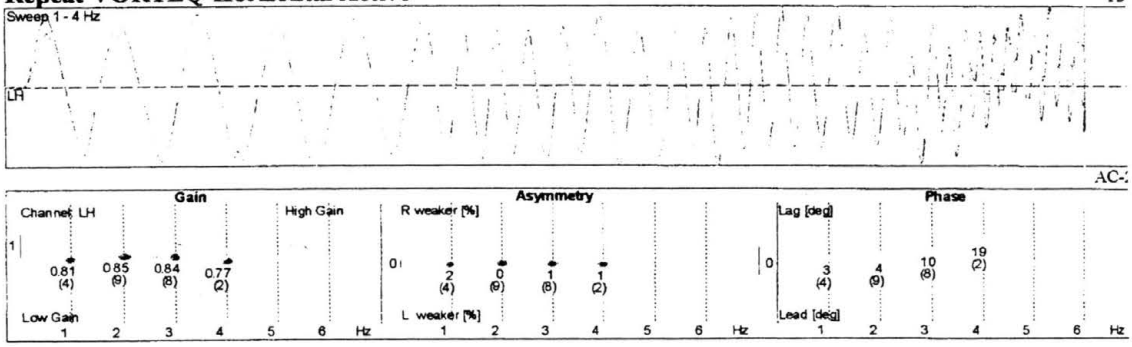
Name:
Age: 27
Visit Date: 5/19/04

VORTEQ-Horizontal Active 1



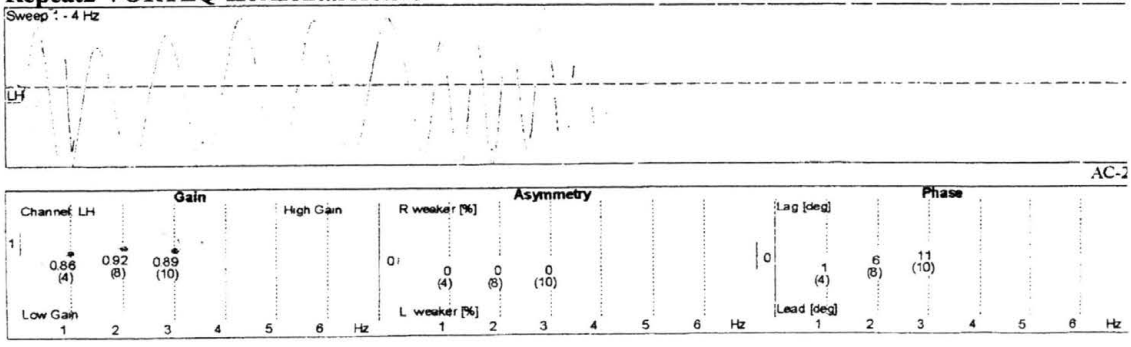
AC-

Repeat VORTEQ-Horizontal Active 15



AC-

Repeat2 VORTEQ-Horizontal Active 15

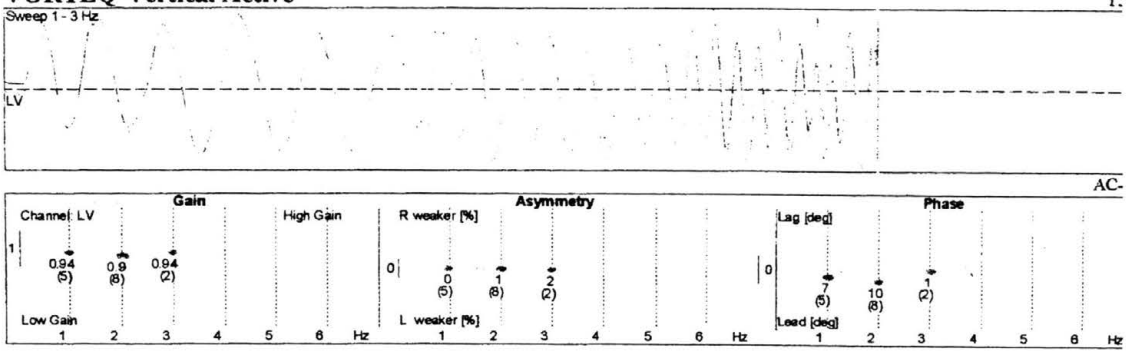


AC-

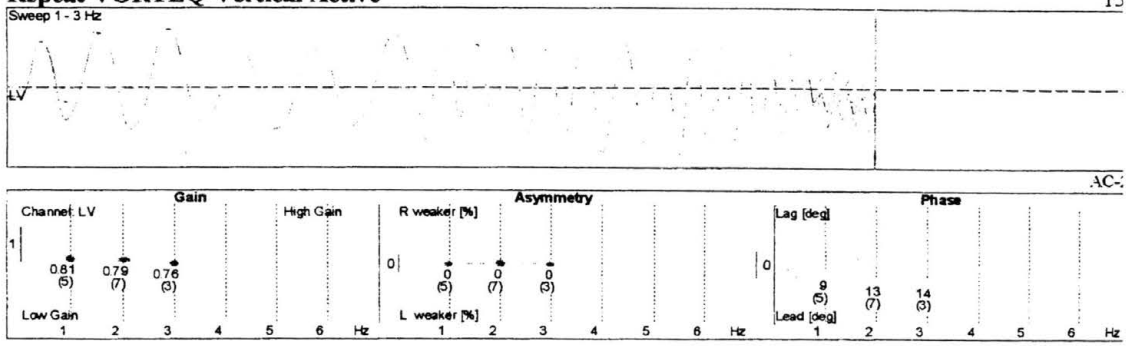
Ear Specialists of Austin
James Kemper, M.D.

Name:
Age: 27
Visit Date: 5/19/04

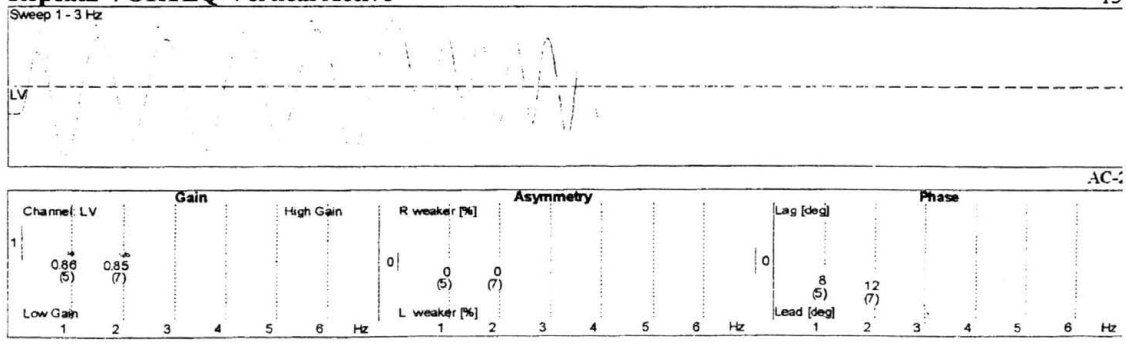
VORTEQ-Vertical Active



Repeat VORTEQ-Vertical Active



Repeat2 VORTEQ-Vertical Active



EQUITEST SUMMARY

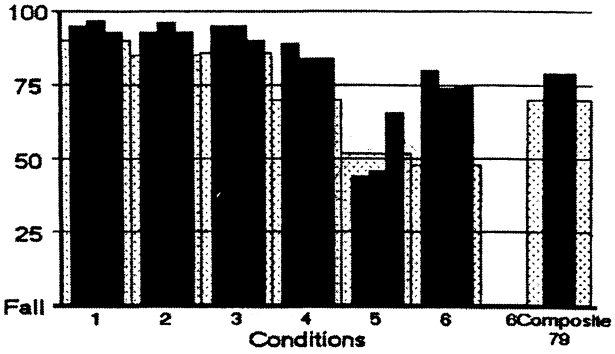
JAMES KEMPER,M.D
EAR SPECIALISTS OF AUSTIN
7000 N. Mopac, Ste. 320
Austin, TX 78731

Patient: I

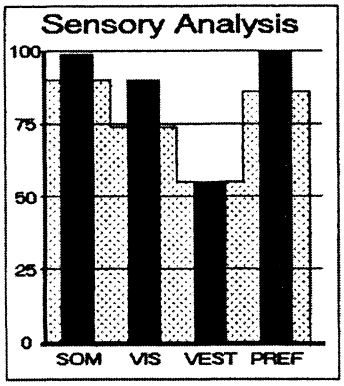
File: 000834A.RAW
Date: May 19 2004
Time: 07:33

Referred By: SELF
Sway-Referenced Gain: 1.00 Operator ID: PH

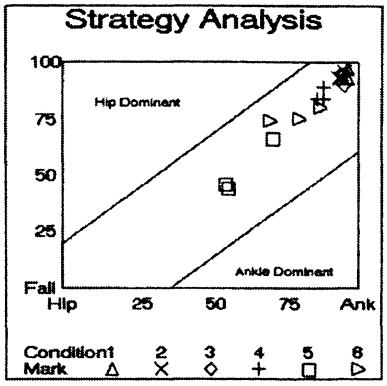
Equilibrium Score



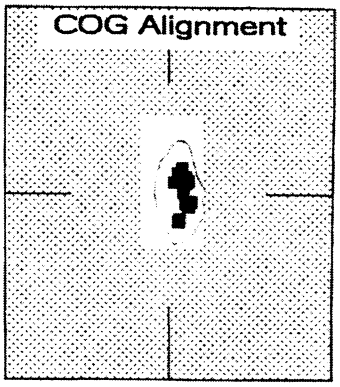
Sensory Analysis



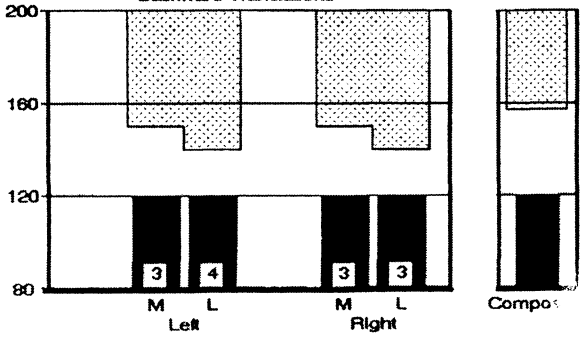
Strategy Analysis



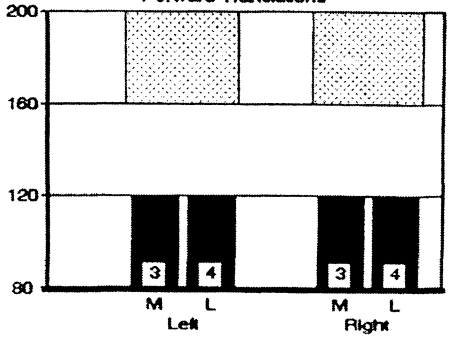
COG Alignment



Latency (msec)
Backward Translations



Latency (msec)
Forward Translations



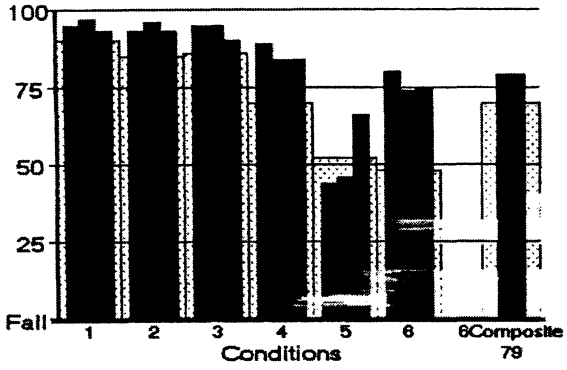
TEST NOTES: NeuroCom Data Range: 20 - 59; Data from EquiTest Version 5.08b

SENSORY ORGANIZATION TEST ANALYSIS

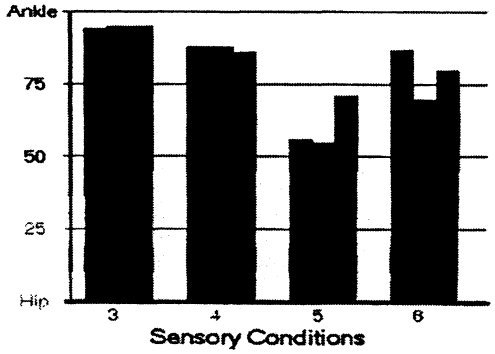
JAMES KEMPER,M.D
EAR SPECIALISTS OF AUSTIN
7000 N. Mopac, Ste. 320
Austin, TX 78731

Referred By: SELF
Sway-Referenced Gain: 1.00
Operator ID: PH
File: 000834A.RAW
Date: May 19 2004
Time: 07:33

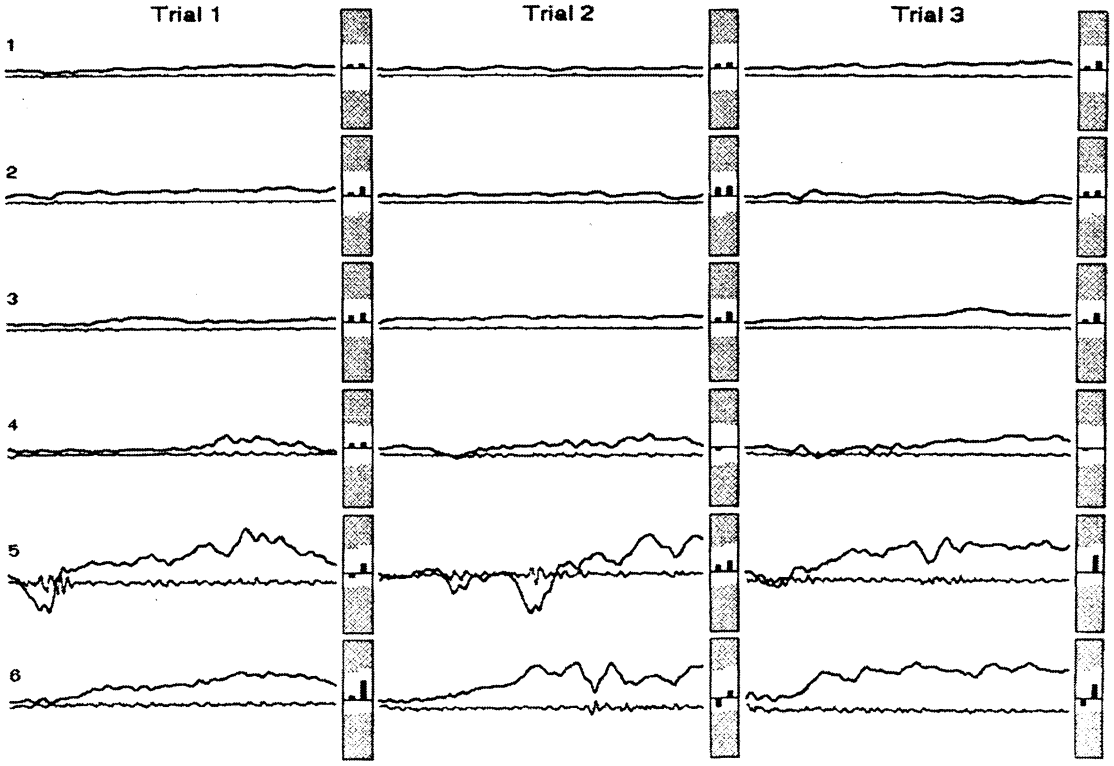
Equilibrium Score



Strategy



Sway, Shear, and Alignment Data



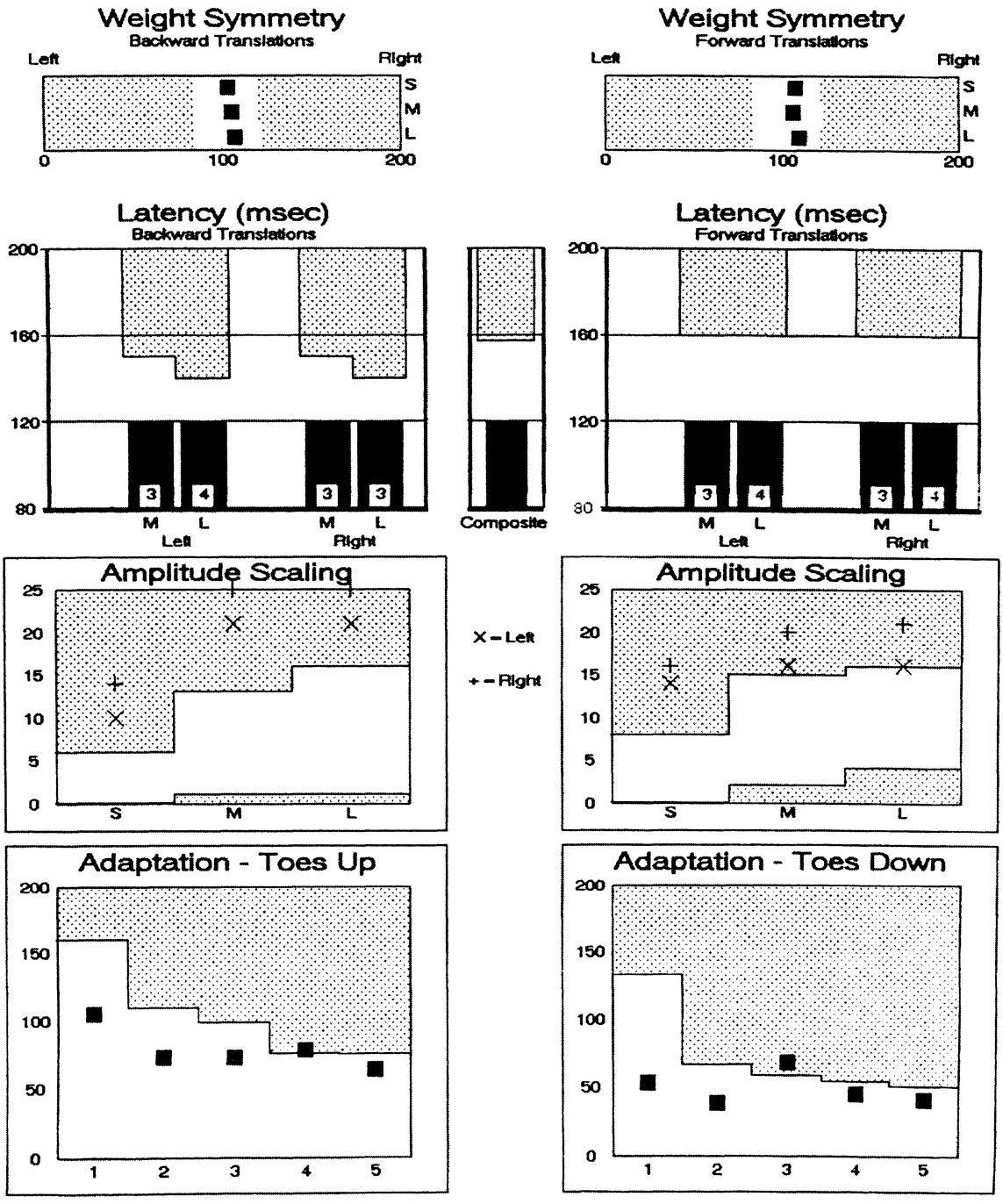
TEST NOTES: NeuroCom Data Range: 20 - 59; Data from EquiTest Version 5.08b

MOTOR CONTROL TEST

JAMES KEMPER,M.D
EAR SPECIALISTS OF AUSTIN
7000 N. Mopac, Ste. 320
Austin, TX 78731

File: 000834A.RAW
Date: May 19 2004
Time: 07:33

Referred By: SELF
Sway-Referenced Gain: 1.00 Operator ID: PH



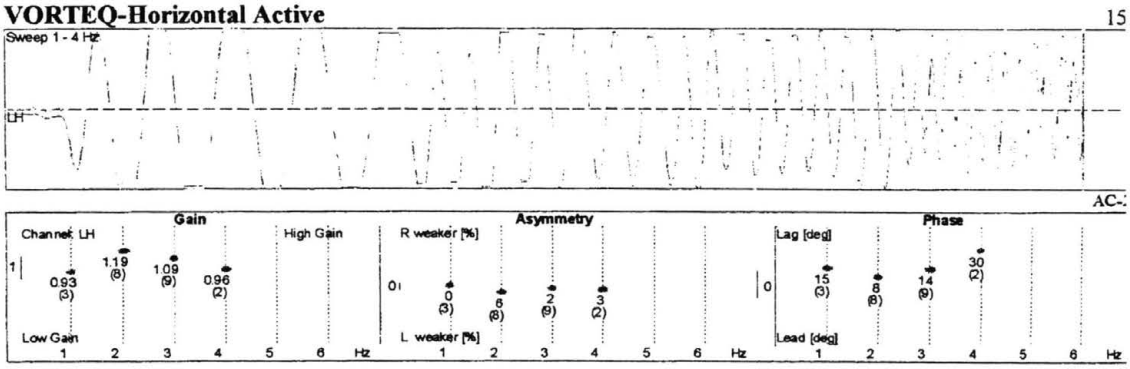
TEST NOTES: NeuroCom Data Range: 20 - 59; Data from EquiTest Version 5.08b

Reassessment After One Week of Customized Vestibular Exercise Program

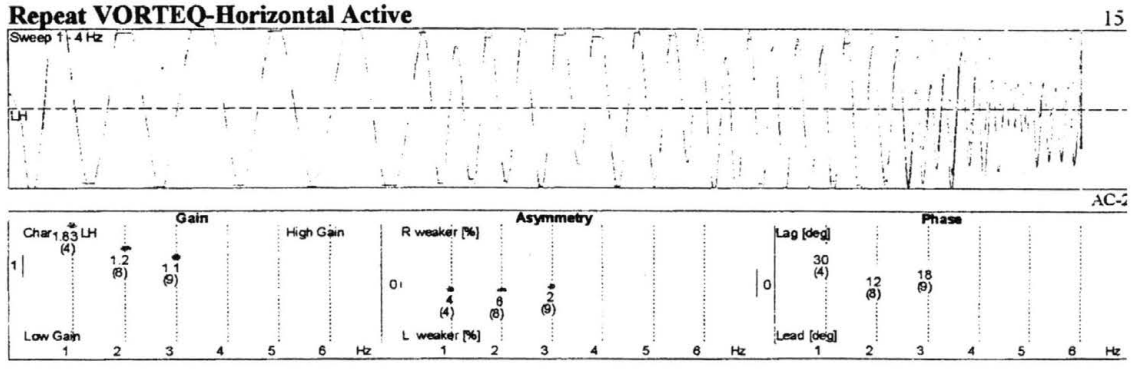
Ear Specialists of Austin
James Kemper, M.D.

Name:
Age: 27
Visit Date: 5/27/04

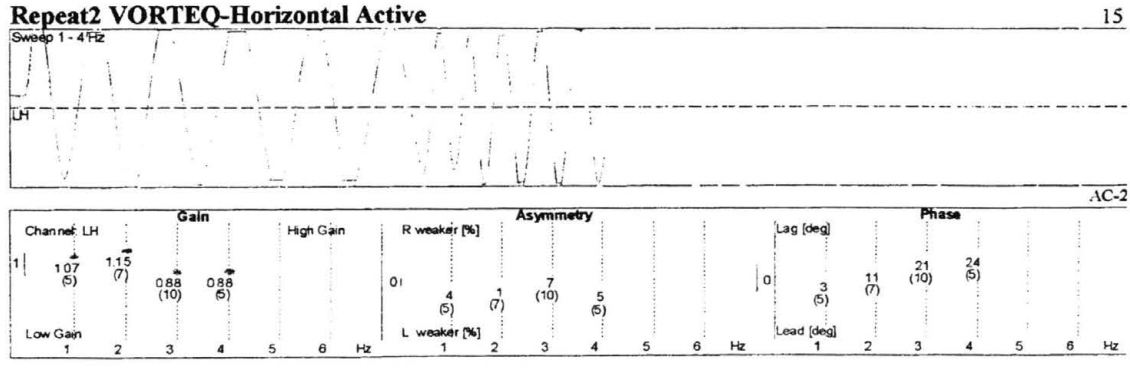
VORTEQ-Horizontal Active



Repeat VORTEQ-Horizontal Active



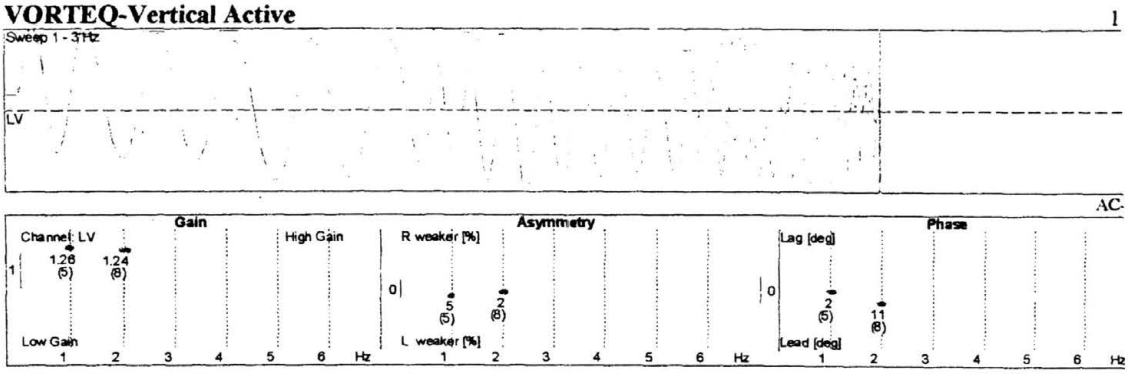
Repeat2 VORTEQ-Horizontal Active



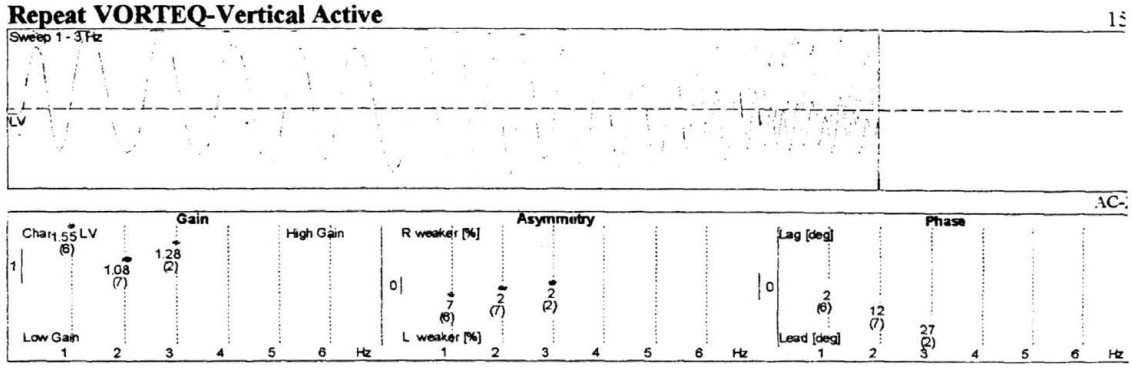
Ear Specialists of Austin
James Kemper, M.D.

Name:
Age:
Visit Date: 5/27/04

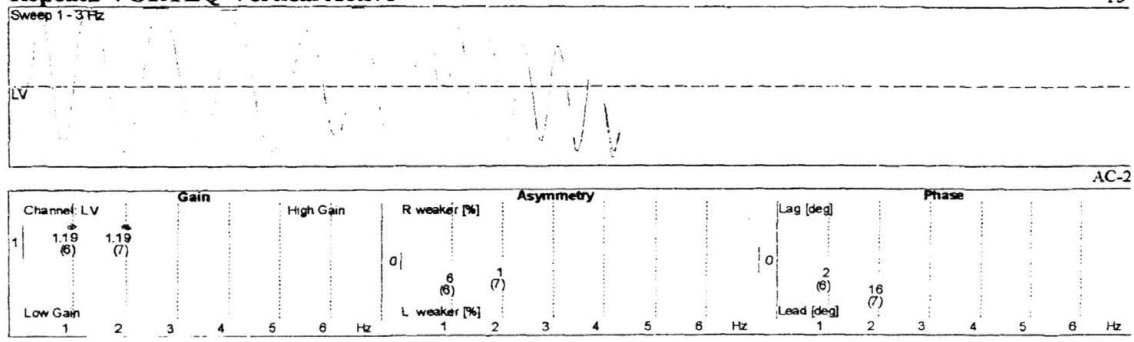
VORTEQ-Vertical Active 1



Repeat VORTEQ-Vertical Active 15



Repeat2 VORTEQ-Vertical Active 15



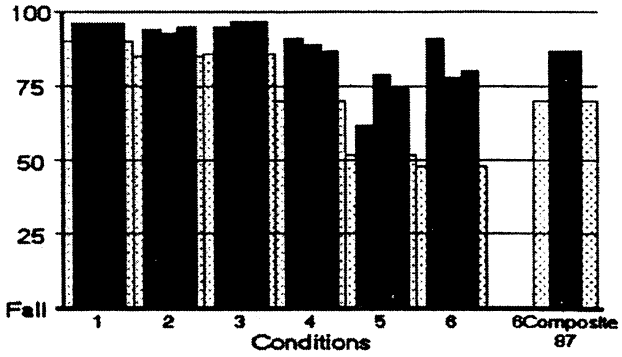
EQUITEST SUMMARY

JAMES KEMPER,M.D
EAR SPECIALISTS OF AUSTIN
7000 N. Mopac, Ste. 320
Austin, TX 78731

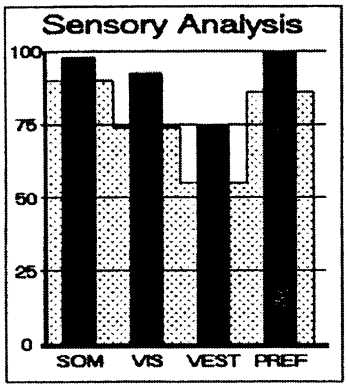
Referred By: KEMPER
Sway-Referenced Gain: 1.00 Operator ID: PH

File: 000838A.RAW
Date: May 27 2004
Time: 12:01

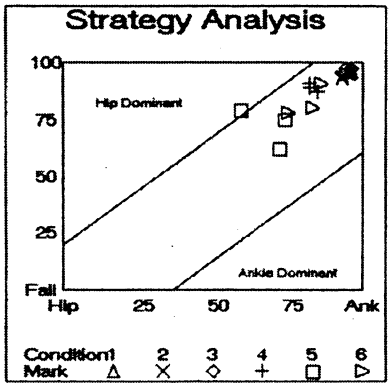
Equilibrium Score



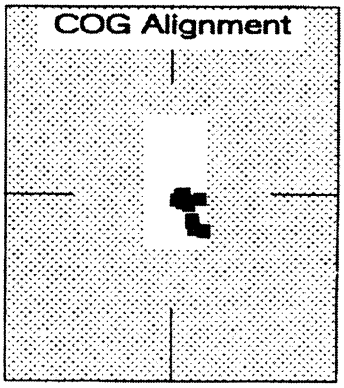
Sensory Analysis



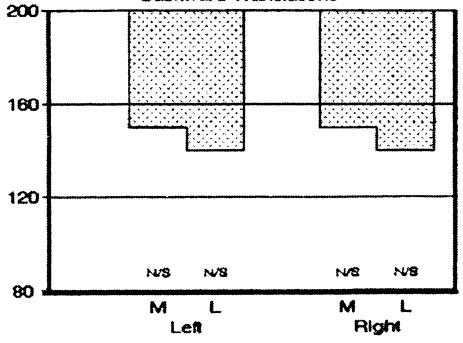
Strategy Analysis

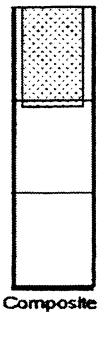


COG Alignment

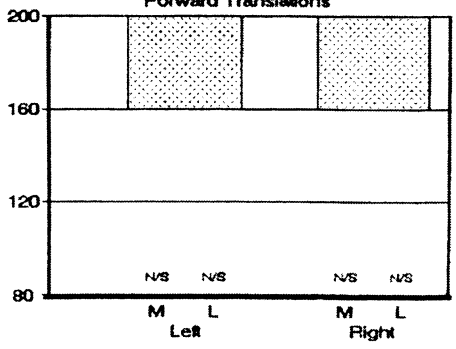


Latency (msec)
Backward Translations





Latency (msec)
Forward Translations



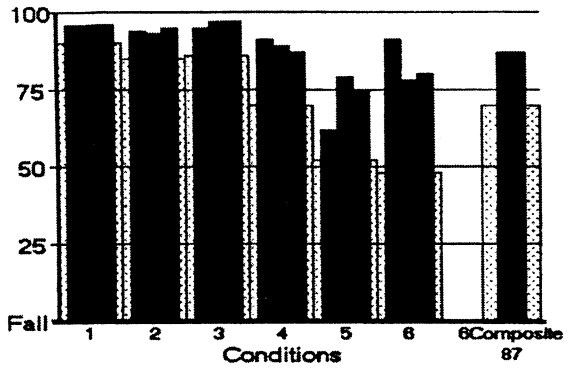
TEST NOTES: NeuroCom Data Range: 20 - 59; Data from EquiTest Version 5.08b

SENSORY ORGANIZATION TEST ANALYSIS

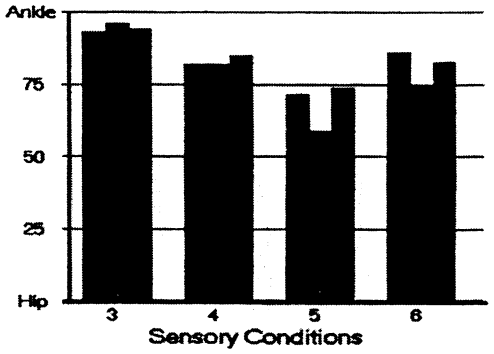
JAMES KEMPER,M.D
EAR SPECIALISTS OF AUSTIN
7000 N. Mopac, Ste. 320
Austin, TX 78731

Referred By: KEMPER
Sway-Referenced Gain: 1.00
Operator ID: PH
File: 000838A.RAW
Date: May 27 2004
Time: 12:01

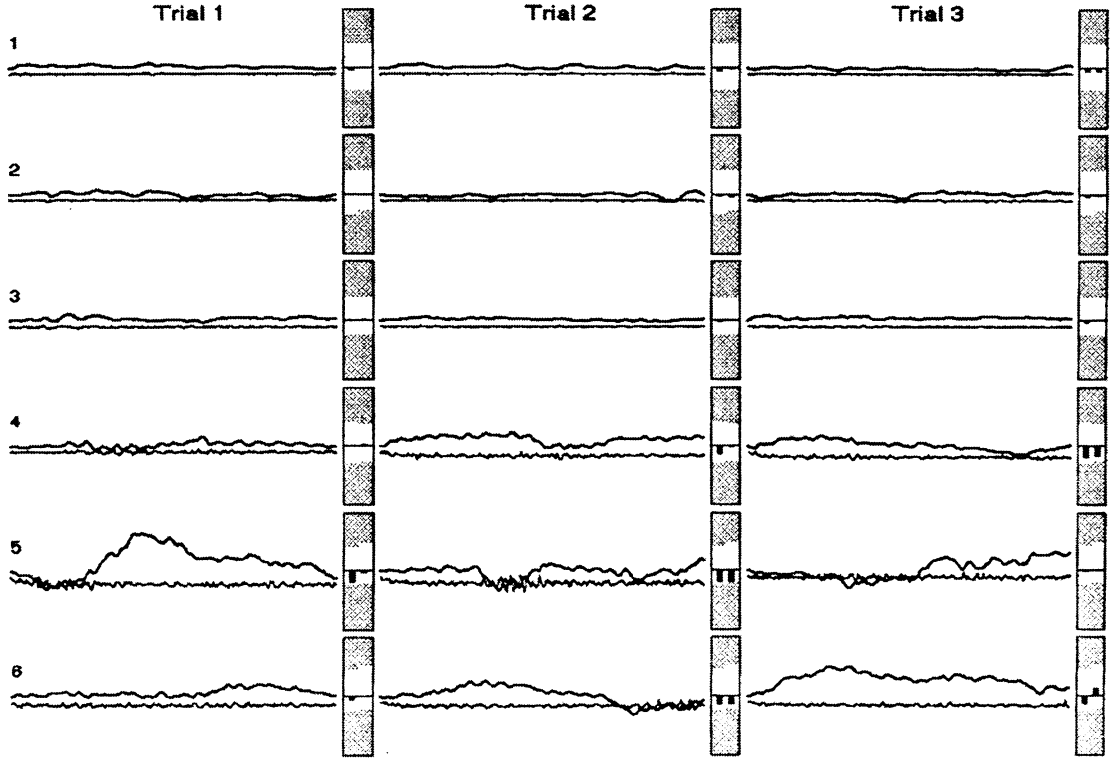
Equilibrium Score



Strategy



Sway, Shear, and Alignment Data



TEST NOTES: NeuroCom Data Range: 20 - 59; Data from EquiTest Version 5.08b

Appendix S: Data pertaining to case study 3
Pre-Injury Assessment

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

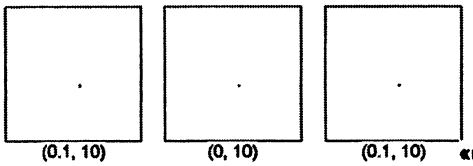
Name:
ID: ATID00260
Date of Birth:
Height: 5'6"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD260.DRX
Date: 3/18/2004
Time: 15:42:14

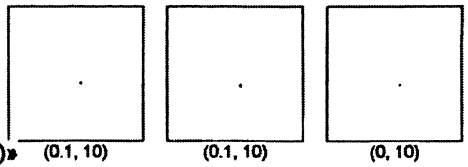
Modified CTSIB

1. Firm--Eyes Open (FIRM-EO)



(0.1, 10) Trial 1 | (0, 10) Trial 2 | (0.1, 10) Trial 3

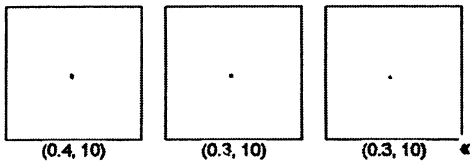
2. Firm--Eyes Closed (FIRM-EC)



«(deg/sec)»

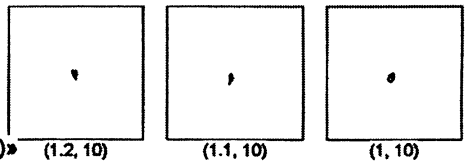
(0.1, 10) Trial 1 | (0.1, 10) Trial 2 | (0, 10) Trial 3

3. Foam--Eyes Open (FOAM-EO)



(0.4, 10) Trial 1 | (0.3, 10) Trial 2 | (0.3, 10) Trial 3

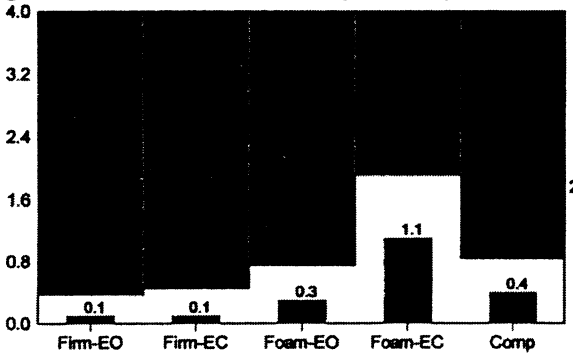
4. Foam--Eyes Closed (FOAM-EC)



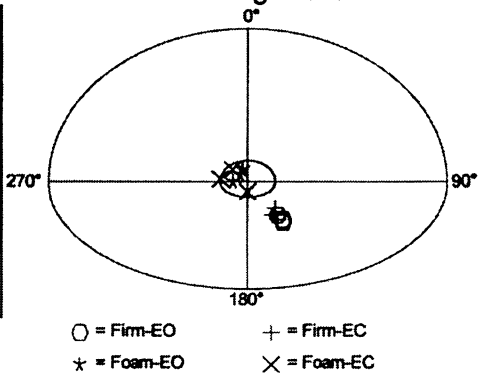
«(deg/sec)»

(1.2, 10) Trial 1 | (1.1, 10) Trial 2 | (1, 10) Trial 3

deg/sec — Mean COG Sway Velocity



COG Alignment



COG Alignment:
Right Back, 24%LOS @153.4 degree

Data Range Note: User Data Range: 20--39

Post Test Comment:
No Head-shake

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

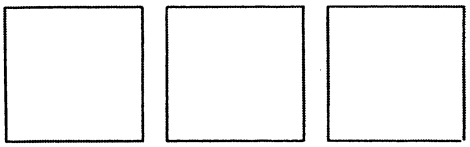
Name:
ID: ATID0026(
Date of Birth:
Height: 5'6"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD260.DRX
Date: 3/18/2004
Time: 15:47:37

Modified CTSIB

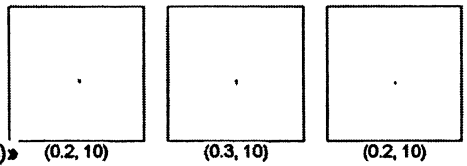
1. Firm--Eyes Open (FIRM-EO)



Trial 1 Trial 2 Trial 3

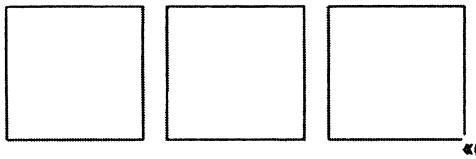
2. Firm--Eyes Closed (FIRM-EC)

«(deg/sec)»



(0.2, 10) (0.3, 10) (0.2, 10)
Trial 1 Trial 2 Trial 3

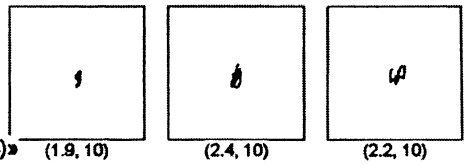
3. Foam--Eyes Open (FOAM-EO)



Trial 1 Trial 2 Trial 3

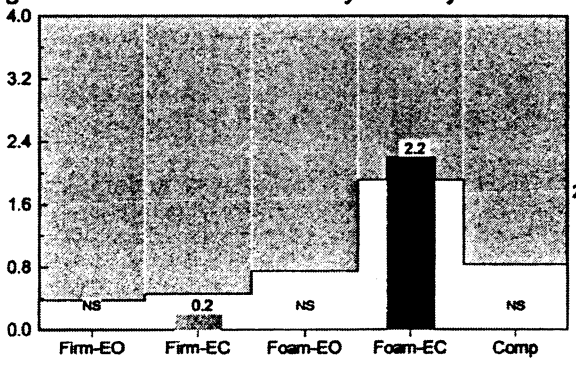
4. Foam--Eyes Closed (FOAM-EC)

«(deg/sec)»

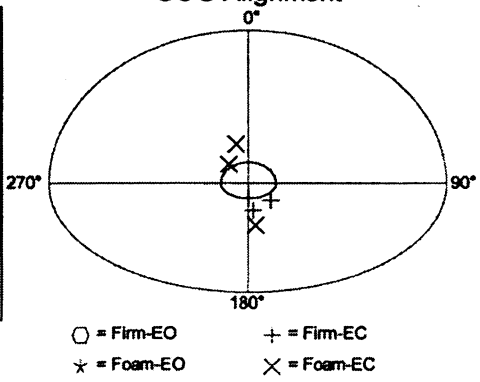


(1.9, 10) (2.4, 10) (2.2, 10)
Trial 1 Trial 2 Trial 3

deg/sec Mean COG Sway Velocity



COG Alignment



COG Alignment:
Scattered, 24%LOS

Data Range Note: User Data Range: 20--39

Post Test Comment:
With head-shake

1st Post-Injury Follow-up Assessment

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

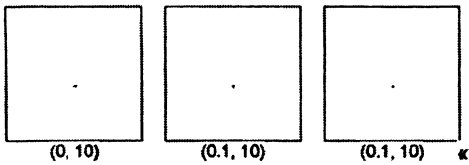
Name:
ID: ATID00260
Date of Birth:
Height: 5'6"

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD260.DRX
Date: 3/24/2004
Time: 16:46:53

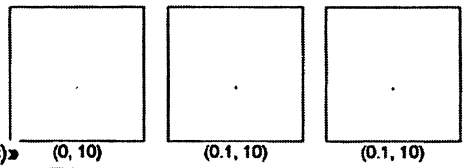
Modified CTSIB

1. Firm--Eyes Open (FIRM-EO)



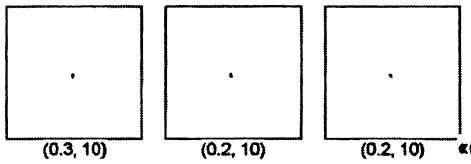
(0, 10) Trial 1 | (0.1, 10) Trial 2 | (0.1, 10) Trial 3

2. Firm--Eyes Closed (FIRM-EC)



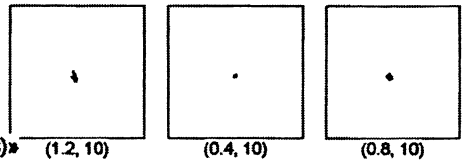
«(deg/sec)» (0, 10) Trial 1 | (0.1, 10) Trial 2 | (0.1, 10) Trial 3

3. Foam--Eyes Open (FOAM-EO)



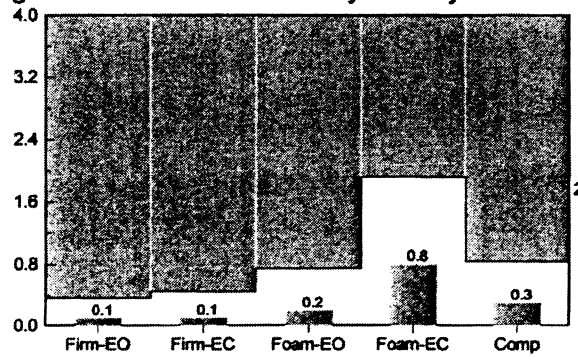
(0.3, 10) Trial 1 | (0.2, 10) Trial 2 | (0.2, 10) Trial 3

4. Foam--Eyes Closed (FOAM-EC)

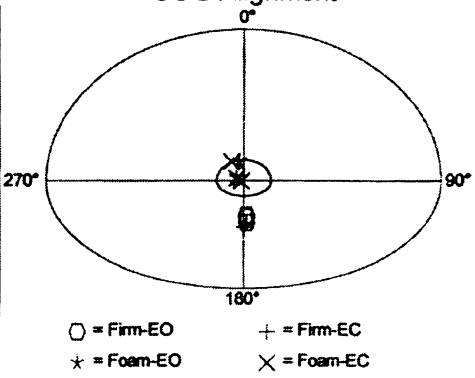


«(deg/sec)» (1.2, 10) Trial 1 | (0.4, 10) Trial 2 | (0.8, 10) Trial 3

deg/sec **Mean COG Sway Velocity**



COG Alignment



COG Alignment:
Back, 23%LOS @187.9 degree

Data Range Note: User Data Range: 20--39

Post Test Comment:
No head-shake

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

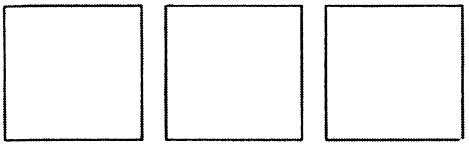
Name:
ID: ATI000200
Date of Birth:
Height: 5'6"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD260.DRX
Date: 3/24/2004
Time: 16:52:37

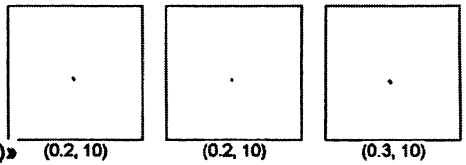
Modified CTSIB

1. Firm--Eyes Open (FIRM-EO)



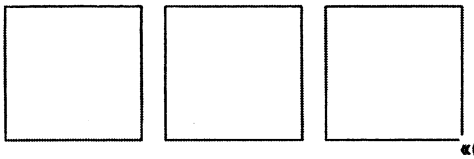
Trial 1 Trial 2 Trial 3

2. Firm--Eyes Closed (FIRM-EC)



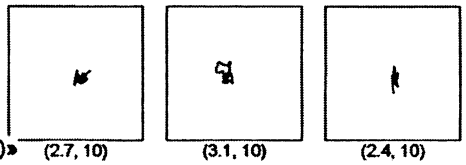
«(deg/sec)» (0.2, 10) (0.2, 10) (0.3, 10)
Trial 1 Trial 2 Trial 3

3. Foam--Eyes Open (FOAM-EO)



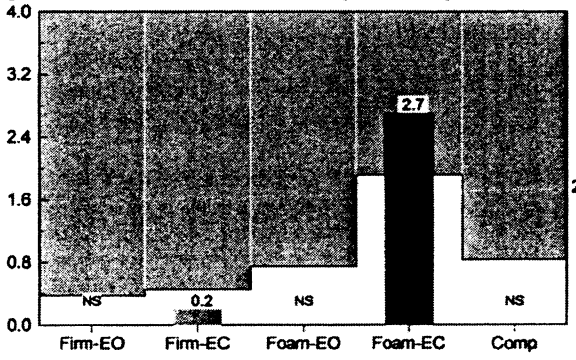
Trial 1 Trial 2 Trial 3

4. Foam--Eyes Closed (FOAM-EC)

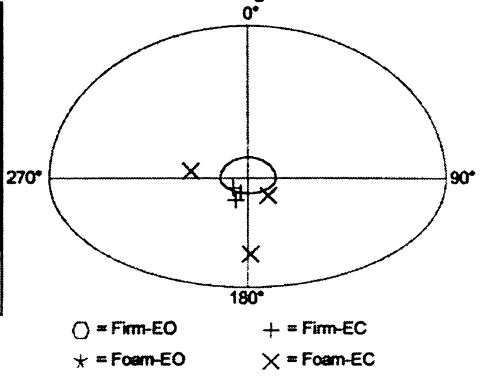


«(deg/sec)» (2.7, 10) (3.1, 10) (2.4, 10)
Trial 1 Trial 2 Trial 3

deg/sec Mean COG Sway Velocity



COG Alignment



COG Alignment:
Left Back, 29%LOS @205 degree

Data Range Note: User Data Range: 20--39

Post Test Comment:
With head-shake

2nd Post-injury Follow-up Assessment

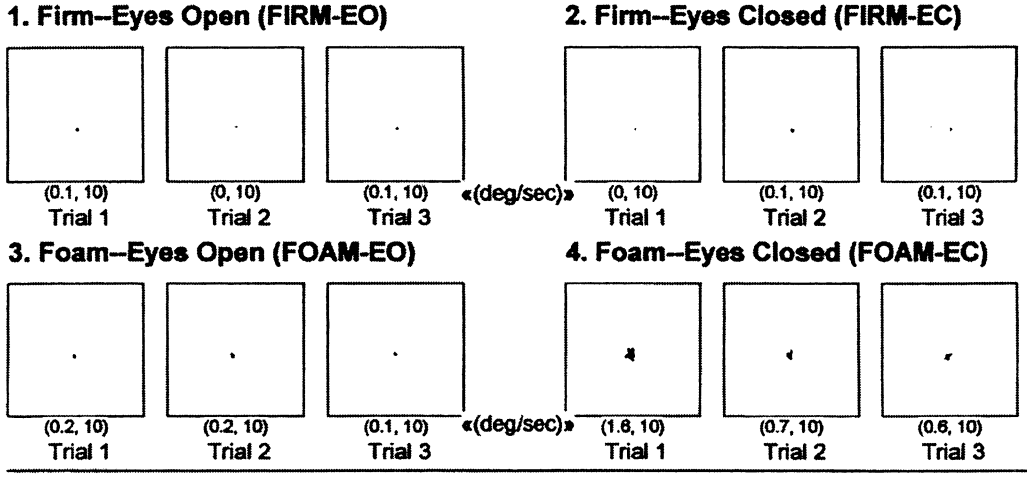
Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

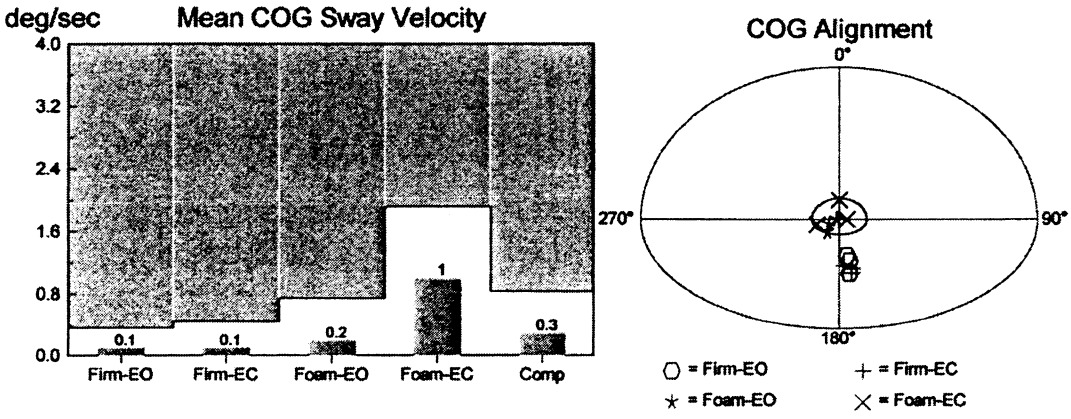
Name:
ID: ATID00260
Date of Birth:
Height: 5'6"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD260.DRX
Date: 3/29/2004
Time: 15:31:30

Modified CTSIB





Data Range Note: User Data Range: 20--39

COG Alignment:
Back, 27%LOS @176.9 degree

Post Test Comment:
No head-shake

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

Name:
ID: ATID00260
Date of Birth:
Height: 5'6"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD260.DRX
Date: 3/29/2004
Time: 15:36:36

Modified CTSIB

1. Firm--Eyes Open (FIRM-EO)

Trial 1 | Trial 2 | Trial 3

2. Firm--Eyes Closed (FIRM-EC)

«(deg/sec)»

Trial 1 (0.1, 10) | Trial 2 (0.3, 10) | Trial 3 (0.1, 10)

3. Foam--Eyes Open (FOAM-EO)

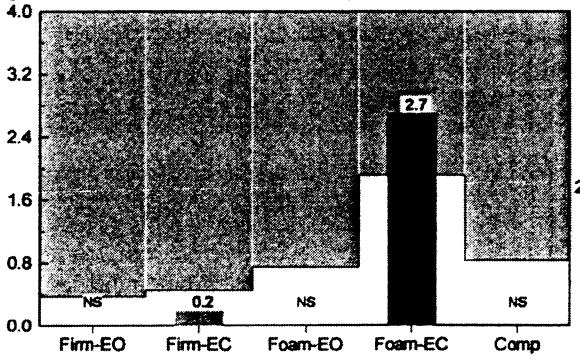
Trial 1 | Trial 2 | Trial 3

4. Foam--Eyes Closed (FOAM-EC)

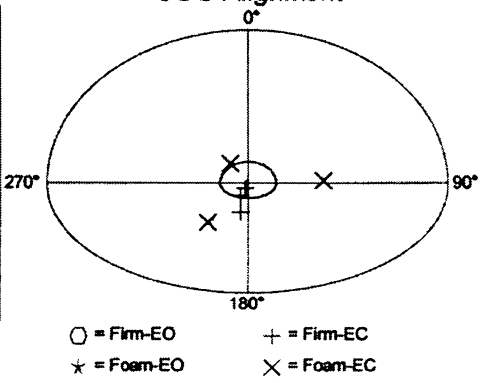
«(deg/sec)»

Trial 1 (2.9, 10) | Trial 2 (2.5, 10) | Trial 3 (2.6, 10)

deg/sec — Mean COG Sway Velocity



COG Alignment



COG Alignment:
Scattered, 24%LOS

Data Range Note: User Data Range: 20--39

Post Test Comment:
With head-shake

Appendix T: Data pertaining to case study 4

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

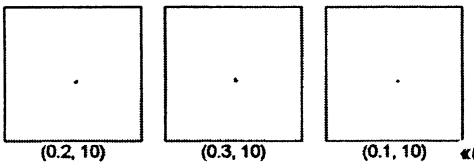
Name:
ID: ATID00255
Date of Birth: 9/27/1983
Height: 6'0"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD255.DRX
Date: 3/16/2004
Time: 17:20:33

Modified CTSIB

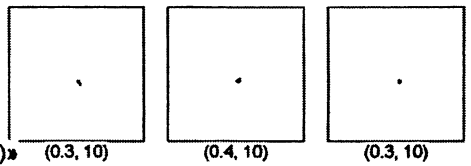
1. Firm--Eyes Open (FIRM-EO)



(0.2, 10) Trial 1 | (0.3, 10) Trial 2 | (0.1, 10) Trial 3

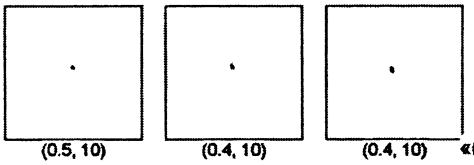
«(deg/sec)»

2. Firm--Eyes Closed (FIRM-EC)



(0.3, 10) Trial 1 | (0.4, 10) Trial 2 | (0.3, 10) Trial 3

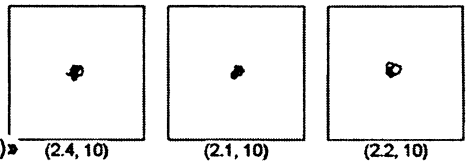
3. Foam--Eyes Open (FOAM-EO)



(0.5, 10) Trial 1 | (0.4, 10) Trial 2 | (0.4, 10) Trial 3

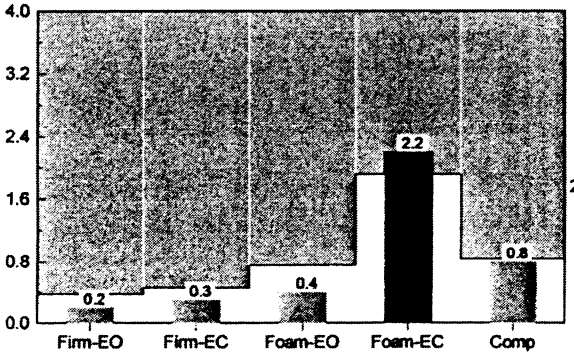
«(deg/sec)»

4. Foam--Eyes Closed (FOAM-EC)

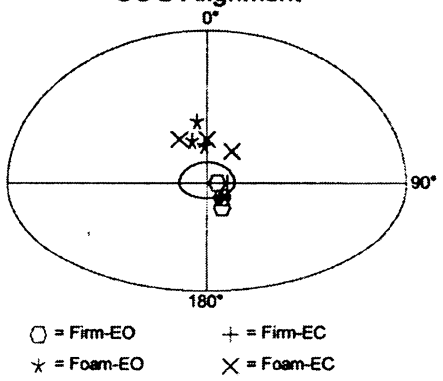


(2.4, 10) Trial 1 | (2.1, 10) Trial 2 | (2.2, 10) Trial 3

deg/sec — Mean COG Sway Velocity



COG Alignment



COG Alignment:
Scattered, 22%LOS

Data Range Note: User Data Range: 20--39

Post Test Comment:
No Head-shake

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

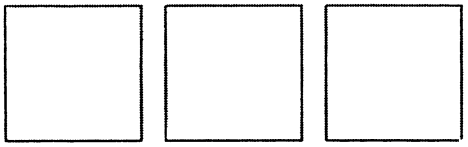
Name: '
ID: ATID00255
Date of Birth:
Height: 6'0"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD255.DRX
Date: 3/16/2004
Time: 17:26:37

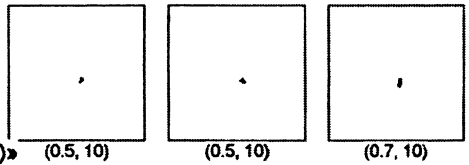
Modified CTSIB

1. Firm--Eyes Open (FIRM-EO)



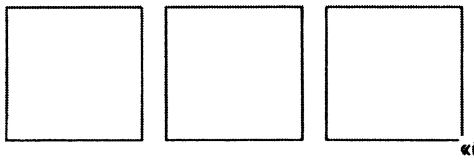
Trial 1 Trial 2 Trial 3

2. Firm--Eyes Closed (FIRM-EC)



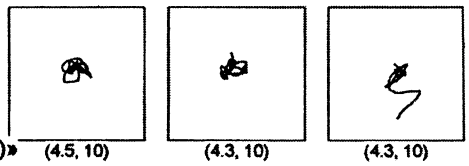
«(deg/sec)» (0.5, 10) (0.5, 10) (0.7, 10)
Trial 1 Trial 2 Trial 3

3. Foam--Eyes Open (FOAM-EO)



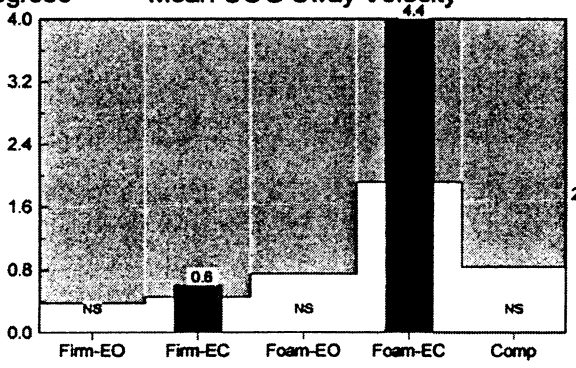
Trial 1 Trial 2 Trial 3

4. Foam--Eyes Closed (FOAM-EC)

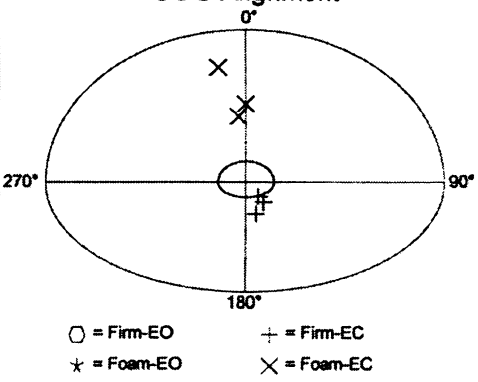


«(deg/sec)» (4.5, 10) (4.3, 10) (4.3, 10)
Trial 1 Trial 2 Trial 3

deg/sec Mean COG Sway Velocity



COG Alignment



COG Alignment:
Scattered, 40%LOS

Data Range Note: User Data Range: 20--39

Post Test Comment:
With Head-shake

Appendix U: Data pertaining to case study 5
Post-Injury Assessment

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

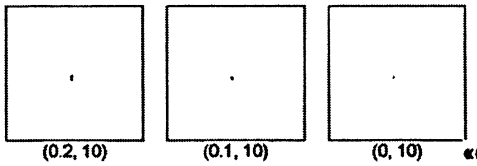
Name:
ID: ATID0026
Date of Birth:
Height: 6'0"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD269.DRX
Date: 3/29/2004
Time: 18:37:19

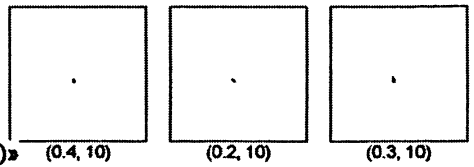
Modified CTSIB

1. Firm--Eyes Open (FIRM-EO)



(0.2, 10) Trial 1 | (0.1, 10) Trial 2 | (0, 10) Trial 3

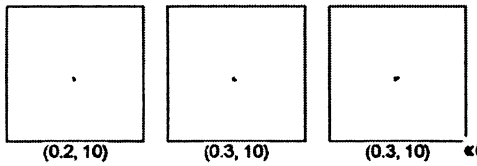
2. Firm--Eyes Closed (FIRM-EC)



(0.4, 10) Trial 1 | (0.2, 10) Trial 2 | (0.3, 10) Trial 3

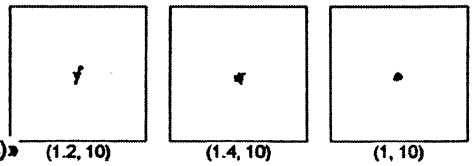
«(deg/sec)»

3. Foam--Eyes Open (FOAM-EO)



(0.2, 10) Trial 1 | (0.3, 10) Trial 2 | (0.3, 10) Trial 3

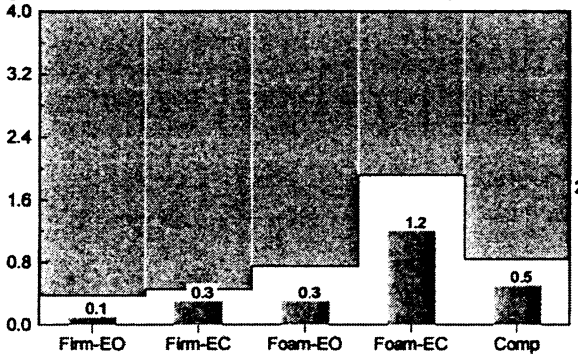
4. Foam--Eyes Closed (FOAM-EC)



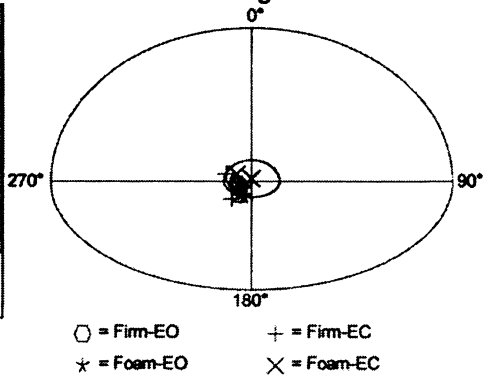
(1.2, 10) Trial 1 | (1.4, 10) Trial 2 | (1, 10) Trial 3

«(deg/sec)»

deg/sec — Mean COG Sway Velocity



COG Alignment



COG Alignment:
Within normal range.

Data Range Note: User Data Range: 20--39

Post Test Comment:
No head-shake

Bridgett Wallace, PT
Balance Therapy of Texas
7000 N. Mopac, Ste. 315*Austin, TX*78731
T: 512-345-4664*F: 512-345-6150

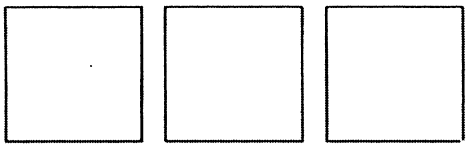
Name:
ID: ATID00269
Date of Birth:
Height: 6'0"
Comments:

Diagnosis: Not Specified
Operator: Hill,Patricia M
Referral Source: Not Specified

File: FD269.DRX
Date: 3/29/2004
Time: 18:46:06

Modified CTSIB

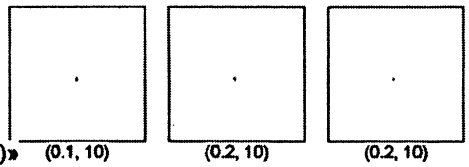
1. Firm--Eyes Open (FIRM-EO)



Trial 1 Trial 2 Trial 3

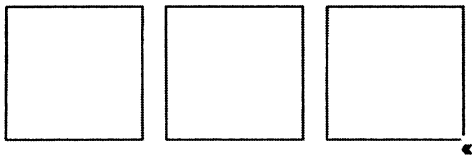
«(deg/sec)»

2. Firm--Eyes Closed (FIRM-EC)



(0.1, 10) (0.2, 10) (0.2, 10)
Trial 1 Trial 2 Trial 3

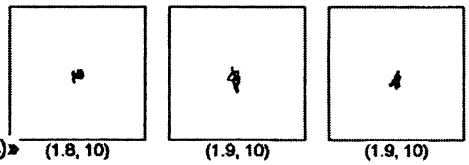
3. Foam--Eyes Open (FOAM-EO)



Trial 1 Trial 2 Trial 3

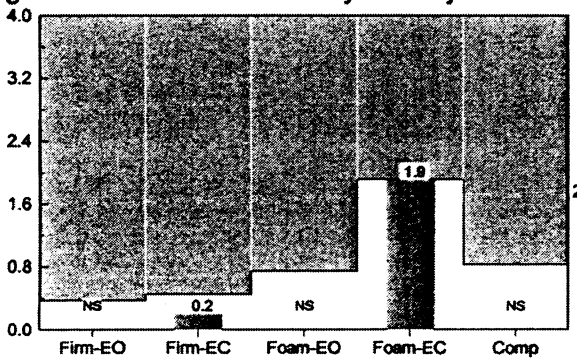
«(deg/sec)»

4. Foam--Eyes Closed (FOAM-EC)

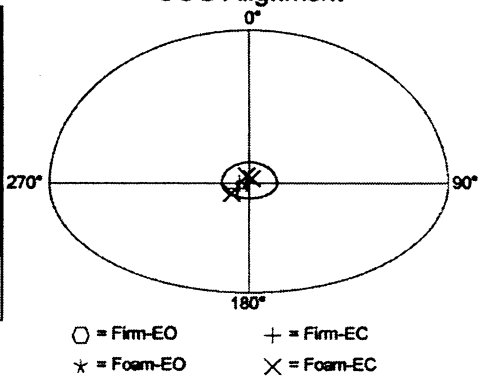


(1.8, 10) (1.9, 10) (1.9, 10)
Trial 1 Trial 2 Trial 3

deg/sec Mean COG Sway Velocity



COG Alignment



COG Alignment:
Within normal range.

Data Range Note: User Data Range: 20--39

Post Test Comment:
With head-shake

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VITA

Patricia Marie Hill was born in McLean, Virginia, on August 24, 1975, the daughter of Martha L. Hill and Jimmie D. Hill. After completing her work at Langley High School in McLean, Virginia, in 1993, she entered the University of Virginia in Charlottesville, Virginia. She received the degree of Bachelor of Arts with a dual major in Biology and Environmental Sciences in May of 1997. In January of 1999, she entered the graduate college at The University of Texas at Austin. She received the degree of Master of Arts in December of 2001, from the Department of Kinesiology, with a focus on Biomechanics. Her master's thesis research at The University of Texas explored the development of standing balance in infants and children. In May of 2003, she began school at Texas State University-San Marcos, and is currently working towards the degree of Master of Science in Physical Therapy.

Permanent Address: 8002 Willet Trail
Austin, Texas 78745

This thesis was typed by Patricia M. Hill.