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The Acute Effect of Traumatic Brain Injury (TBI) on Motor Power Output

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The Acute Effect of Traumatic Brain Injury (TBI) on Motor Power Output

A THESIS

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Abstract

The Acute Effect of Traumatic Brain Injury (TBI) on Motor Power Output

There has be an increased interest in the chronic effects of traumatic brain injury (TBI) in contact sports from both research and nonscientific fields alike. The chronic effects can only be minimized by understanding the physiological processes and acute effects associated with a TBI that ultimately give rise to chronic effects. Sports-related TBIs are assessed with computerized neuropsychological test batteries and self-reported symptoms. A quantitative measure of neural activity acutely post-TBI may offer information to diminish premature return-to-play, and subsequent neural injuries. Thirteen collegiate football players initially participated in baseline testing where motor power output was measured with electromyography (EMG) contemporaneous with peak torque output, total work, and average power output via the HUMAC Norm isokinetic dynamometer (IKD). One participant successfully completed the study after sustaining a concussion during spring football. He returned for identical testing procedures at 24 hours and 7 days post-TBI. The subject had a significant decrease across all measurements at 24 hours post-TBI. Neural activity and power outputs had improved at 7 days post-TBI, but had not returned to baseline. This case study highlights the appearance of electrophysiological dysfunction acutely after TBI, but does not offer information on the impaired mechanism: rate coding, synchronization, or frequency. The acute decrease in power output may be attributed to a decrease in neural signaling following a head injury. Further research should be conducted with more participants to clarify the cause of a significant decline in motor power output immediately after TBI.

CHAPTER ONE: Introduction

Significance

The effect of concussions acquired in contact sports has gained popularity across vast media sources in recent years. What was formerly known as "postconcussion syndrome" is now deemed to be a mild traumatic brain injury (mTBI) (Kain, 2009). Steve Gleason, a former safety for the New Orleans Saints, was diagnosed with Amyotrophic Lateral Sclerosis (ALS) in 2011, at 33 years of age. ALS, more commonly known as Lou Gehrig's disease, is a neurodegenerative disorder that has been correlated with repetitive head trauma present in heavy contact sports. Most recently, the National Football League (NFL) has settled with former players and families who have suffered concussive injuries for $765 million (Costanza et al., 2011).

Repetitive trauma to the head has produced similar characteristics of ALS, and other neurodegenerative disorders, in neuropathological examinations. Hunt and Asplund (2010) state the most common and complex form of TBI is a concussion: "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces," and cause rapid but acute impairment of neurological function (p.6). A concussion allows for an acute interruption of brain function, and is normally caused by an abrupt increase or decrease of acceleration of the cerebellum against the skull (Shaw, 2002). An acute TBI denotes the result of subconcussive and concussive impacts sustained by the head (Hecht, 2002). It is important to note that most TBIs presented in sports are a metabolic injury rather than structural (Lovell, Collins, & Bradley, 2004), and this component is an underlying factor for why computed tomography (CT) and magnetic resonance imaging (MRI) are not useful for diagnostics or assessment of TBI (Hunt & Asplund, 2010).

The United States reports an estimated 1.6 to 3.8 million concussions each year that are induced by sports (Hunt & Asplund, 2010). Boxing, ice hockey, American football, and European football are scrutinized because of their prominence of head injuries from player-to-player contact (Costanza et al., 2011). The study of TBIs is important to gain knowledge of the specific acute and chronic effects. A minor amount of acute effects caused by TBI are known and used as diagnostic tools. Individuals who become concussed usually experience symptoms that are divided into three domains: somatic, cognitive, and sensory (Hunt & Asplund, 2010). Symptoms can include confusion, nausea, headaches, dizziness, irritability, and altered sleep habits (Shaw, 2002).

Purpose

To date there is no knowledge of experimental investigations measuring the integrity and excitability of the corticospinal system *acutely* after a TBI, even more specifically with a single injury episode (Livingston et al., 2010). The purpose of the current study was to determine if TBI sustained in contact sports has an acute effect on motor power output. McCrea et al. (2003) stated that concussions account for 8% of all collegiate football injuries, and Tegner and Lorentzon (1996) reported college football to have a 19% concussion rate per team. This study focused on collegiate football athletes. There is limited data available on motor performance following a concussion, and the establishment of normative values may provide multiple benefits to athletes in all heavy contact sports.

Background

Wall et al., (2006) discussed how TBIs can induce stress and strain to neural and vascular tissues of the brain, and can cause dysfunction that makes the brain vulnerable to subsequent injury. The increased chance of repetitive head traumas after a primary incident usually results

in a second TBI which is commonly referred to as "second impact syndrome." Athletes with repetitive TBI from sports have prematurely ended their career due to signs of permanent brain damage and accompanying cognitive problems. Players may experience issues with concentration, irritability, memory, and personality. Although the acute neuropathological changes that occur due to a TBI infrequently reflect functional damage; chronic symptoms and complications may impair the overall quality of life. Therefore, it is crucial for proper TBI assessment and management to limit the chance of second impact syndrome that commonly results from premature return to play (Hunt & Asplund, 2010).

Chronic effects derived from TBIs initiate with neural dysfunctions that are not symptomatic and only detected through neural examinations, and then progress to retardation of motor functions, cognitive discrepancies, and personality changes (Kain, 2009). The chronic effects of TBIs have also been correlated with the developmental characteristics of chronic traumatic encephalopathy (CTE). This type of neurodegeneration is derived solely from sports and includes TAR DNA-binding protein-43 (TDP-43) positive inclusions and plaque formations and cerebral hemisphere and ventricle atrophy (Hazrati et al., 2013). The ventricular enlargement presented by TBI athletes primarily reveals axonal injury and loss of white matter in the brain (Tremblay et al., 2009). Biomarkers associated with glial proliferation have been found in TBI athletes of amounts that correlate with neurofibrillary tangle (NFT) counts of postmortem Alzheimer's disease brains. Recent research has also shown excess glial biomarkers to be present in deceased NFL athletes that had pathologically verified CTE (Tremblay et al., 2012).

Prior studies conducted on TBI have enabled researchers to understand how the injury is acquired biomechanically. Post, Hoshizaki, and Gilchrist (2011) found TBI reconstruction to be limited to measuring the peak linear and angular acceleration loading curves. Currently, it is

unclear how loading curves contribute to neural tissue damage. However, Guskiewicz et al. (2007) observed no rapport among the rotational and linear magnitudes of impact and symptomatology or neuropsychological function, but reported an average magnitude for TBI impacts to register at 102.8 g. The average speed reported to deliver a concussion is 9.3 ± 1.9 m/s, while an impact speed of $\geqslant 7$ m/s administers 90 g and has the ability to deliver a concussion (Guskiewicz et al., 2007).

In vivo animal studies, as well as *in vitro* human studies have shown repetitive TBI to be more detrimental than a single occurrence (Weber, 2007). Concussions are no longer deemed a "reversible injury." Hazrati et al. (2013) has hypothesized for subconcussive and concussive impacts to be cumulative. Iverson, Brooks, Lovell, and Collins (2006) found athletes with prior TBIs to be at a greater risk for future TBIs, and present enhanced on-field characteristics with more profound changes in memory and performance with slowed recovery times. Although most programs require a baseline test battery, further research may allow for normative quantative values to be established, and help athletic trainers and medical professionals assess each TBI on an individual basis. Improved concussion management can prevent further injury to the athlete, and the possible chronic effects that may diminish quality of life.

A large amount of research examines the changes in athletes' performance in efforts to establish baseline data and normative values to compare injured athletes' performance. ImPACT 2.0 is a neuropsychological test battery. The computerized assessment measures different aspects of cognitive function: memory, reaction time, processing speed and attention (Iverson et al., 2006). The test is administered to athletes prior to the start of the season, and again to athletes who sustain a TBI. A "pre-determined" recovery period for a sports-related TBI has been established based on this traditional assessment approach (Schatz, Pardini, Lovell,

Collins, & Podell, 2006). Self-reported symptoms and neurospsychological testing have been accepted as the standard for sports-related TBI injuries; although there is no definitive assessment method currently available, (Livingston et al., 2010).

Although technology exists to potentially measure neurological factors, they are not feasible to conduct acute assessment for return to play purposes. Functional MRI (fMRI) has been the focus of previous research studies to determine its use in evaluating head trauma. fMRI has shown abnormal activation patterns that are closely related to symptomology in individuals months after the sustained injury (Henry, Tremblay, Boulanger, Ellember, & Lassonde 2010). Magnetic resonance spectroscopy (MRS) has shown to distinguish neural damage that has not been visible with MRI via chemical changes in the brain (Henry et al., 2010). MRS has allowed researchers to show that there appear to be neurometabolic abnormalities present at 30 days post-TBI for first time concussed individuals and longer than 30 days for individuals with more than one concussion (Henry et al., 2010). Transcranial magnetic stimulation (TMS) has shown differences in latency and amplitude of event-related potentials (ERP), and altered intracortical inhibition in athletes who sustained a TBI (Henry et al., 2010). Motor evoked potentials (MEPs), have the ability to provide a direct physiological assessment of the structure of the motor cortex pathways, and have shown acute electrophysiological abnormalities post-TBI in college athletes (Livingston et al., 2010). Livingston et al. (2010) also showed that athletes with more than two concussions showed dysfunction of the motor system with examination of the cortical silent period duration (a measurement of corticospinal inhibitory mechanisms). Studies have shown athletes with TBI to have decreased eye and arm motor control, coordination and postural control, or balance (Heitger et al., 2007; Schneiders, Sullivan, Gray, Hannond-Tooke, & McCrory, 2010). Henry et al. (2010) shared that although there are numerous associated

symptoms that accompany a TBI, there has yet to be a consistent pattern of injury that can be used to detect injury through neuroimaging.

Hypothesis

The current study hypothesized that post-injury assessment would show an acute decrease in motor power output of concussed athletes. The null hypothesis was there would be no acute decrease in motor power output of concussed athletes.

Operational Definitions

Operational definitions are further defined below:

- motor power output describes the electrical signals the brain sends via motor neurons to the muscles in order to produce movement or work measured as power output;
- power output is the measure of force or work produced by muscles;
- total work for this study can be defined as the total amount of force produced in ft-lbs throughout a range of motion;
- traumatic brain injury (TBI)is a metabolic event that occurs due to an abrupt acceleration or deceleration of the head, and can result in an impairment of neurocognitive functions; used interchangeably with mild TBI (mTBI), concussion, and head trauma;
- extensors are skeletal muscles that increase joint angles;
- flexors are skeletal muscles that decrease joint angles;
- loading curve represents the duration, or time, which a specific force is applied;
- Von Mises stress is assessed to determine if designs (protective equipment) can withstand a given load or stress that is applied.

- Root Mean Square calculates the "amplitude" of raw electrical data.

Assumptions

Assumptions for the study included:

- that each player gave their maximum effort throughout the range of motion of the HUMAC Norm;
- the participant was assumed to be correctly diagnosed with a concussion prior to post-TBI testing;
- the EMG data was assumed to not be disturbed by the computers in the lab, or the HUMAC Norm.

Delimitations

The University of Central Oklahoma's (UCO) football players were the participants of a spring-season long study. The delimitations for the study included:

- subjects were required to be listed on the 2013 UCO Football roster as of September 1, 2013;
- subjects were all male;
- only participants that completed baseline testing and sustained a TBI within the spring football season would be used for data analysis.

Limitations

The study had several limitations:

- UCO athletes may not have experienced any head trauma over the stated season;
- data could only be compared if athlete subjects experienced head trauma;
- study sample size was small;

- subjects served as their own control group,
- the study was conducted over a short period of time;
- researcher could not control for internal metabolic components.

Variations in subjects' medical history may have affected the outcome of the study and could not be controlled for due to the nature of the study. Previous research has suggested that genetic traits can affect the way an individual is affected by TBI (Costanza et al., 2011). Diet and exercise have shown to also have an effect on TBI via the amount of brain-derived neurotropic factor (BDNF) produced. BDNF is a growth factor with neuroprotective properties, and an individual with increased levels of BDNF will be affected differently by TBI than an individual with lower levels (Griesbach, 2011). Genetics and growth factors were not considered for this study.

CHAPTER TWO: Literature Review

Head trauma and TBI are thoroughly researched fields; however, there are still many factors not understood. Existing research aims to determine the forces that cause TBI, the physiological processes that occur, the biochemical components, present and non-present symptoms, management practices, and return-to-play criteria. Although different, all areas of concussion research are directed towards understanding post-injury function of the brain, and how to eliminate potential long term effects of TBI. Previous research has validated current concussion assessment methods, threshold ranges, and specified chronic affects that may potentially lead to neurodegenerative diseases. A large number of studies researched for the current study were conducted over the assessment of TBI, the acute effects that follow, and the time of recovery. Mechanical analysis and invasive studies were included to understand the internal functions of TBI. Previous research, over the chronic effects potentially associated with repeated TBI, solidify the importance of the current study. The potential metabolic implications were included for future application.

Biomechanical Measurements

There is minimal data on the biomechanics behind concussions. Guskiewicz et al. (2007) states that through research studies, physicians may be able to better understand and diagnose concussions if they understand the precise movements behind the injury. The purpose of this study was to measure the acceleration of head impacts and their locations in collegiate football players, and to determine if there was a relationship between the two variables and the severity of symptoms present post-injury. The study was designed to focus on three areas affected by concussion: symptomatology, neuropsychology, and postural stability. The study consisted of 88 football players from the University of North Carolina at Chapel Hill, and was conducted

throughout the 2004-2006 Division I seasons. Head Impact Telemetry (HIT) was installed in all subjects' helmets. This instrument allowed for the crowning linear and rotational acceleration to be recorded and analyzed. Any subject who sustained a concussion was tested 24 to 48 hours post injury in symptomatology, postural stability and neuropsychology. The post-concussion data was compared to baseline testing completed prior to the start of the seasons. Players were to report their symptoms with the Graded Symptom Checklist. Postural stability tests were conducted with the Sensory Organization Test (SOT), utilizing a force plate. The Automated Neuropsychological Assessment Metrics (ANAM) battery was used to test subjects' neuropsychological function in seven different areas.

The data was analyzed, and Pearson's correlation coefficients were calculated. There were 13 total concussion sustained in the study, with eleven single concussion subjects, and a standard deviation of ±1.09. The average number of impacts participants were subjected to was 27.7, and the average linear acceleration of hits was 102.8 *g*. The correlations between linear acceleration and the three tested measures showed no significance, and the rotational acceleration showed similar correlation values with no significance. Linear acceleration correlation values were .37 for postural stability, .33 for neuropsychological function, and .01 for self-reported symptoms. Rotational acceleration values were .179 for postural stability, .050 for neuropsychological function, and .198 for self-reported symptoms.

Guskiewicz et al. (2007) found no significant relationship between the magnitude and location of the impact subjects sustained and the symptoms that shortly followed. Although the results of the study showed that head impacts can cause concussion, and the magnitude and location of the hit do not determine the severity of symptoms, larger studies are needed. The study was not able to determine a threshold in regards to linear and rotational acceleration and

concussion occurrence.

Guskiewicz et al.'s study was distinctive from other studies because the HIT system allowed researchers to measure the biomechanics of each hit in a "real time" manner. More studies should be to be conducted to measure the effects of linear and rotational acceleration on concussions.

In a similar study, Deibert and Kryscio (2012) conducted a review with the purpose to measure the number of head impacts athletes sustain, and the magnitude the impacts are applied or received. Previous studies have been conducted to validate mechanical equipment that measures impact velocities and peak linear and angular accelerations. Through the use of Hybrid III crash test dummies it was predicted for NFL players to experience concussion at a linear acceleration of 98 *g*. The HITS technology enabled more sensitive measurements to be taken compared to the the first generation of impact devices. HITS allowed for researchers to turn a football field into an extensive laboratory. A prospective cohort study consisted of 159 Division I contact sport athletes and 45 noncontact sport athletes. The study's design included pre- and post-neuropsychological tests for all 214 athletes. Athletes that played contact sports scored an average of 1.5 standard deviations below their predicted level at post-test, $p = .01$. The authors acknowledge that certain aspects of the study need to accommodate personal history and other limitations, and that the use of neuropsychological tests may not measure change in cognitive function in subjects who do not display signs of concussion. The study concluded that an established number of impacts needed to cause damage cannot be defined at this time.

There are many aspects of head injuries that have yet to be explored through research. The analysis of head trauma is currently limited to computer models and animal based research studies. These types of studies have allowed for research on head injuries to be conducted and

for new protective equipment to be designed. The knowledge of loading curves has improved sports equipment, and allowed improved designs to help protect athletes from head trauma. Post, Hoshizaki, and Gilchrist (2012) conducted a study that was designed around the use of the University College Dublin Brain Trauma Model (UCDBTM). The purpose of this study was to measure what influence the linear and angular acceleration loading curves have on neural tissues. The UCDBTM has been validated through the use of cadavers and neural tissue movement studies. Through formulas, brain tissue and cerebral spinal fluid (CFS) were modeled into a "linearly viscoelastic material." The tissue model was created with more than 10,000 hexahedral elements. Researchers based their loading shape curves on those commonly used in helmet development research studies. The three sample loading curves used in the study were plotted with acceleration over time and the displacement of force. The loading curves consisted of soft tissue and a high absorption of energy from an impact, a loading curve that acted upon a more rigid tissue that would experience an acute captivation of energy, and a final loading curve that represented a plateau effect. Each loading curve model was administered to the UCDBTM through all three axes.

Upon application of the UCDBTM, the results in modeled tissue distortion were measured with Von Mises stress. The stress test results were determined by a direct calculation and not a statistical analysis software program. The results were analyzed and determined to affect peak value, time of peak, and the location of the trauma. The brain stem was most affected when linear acceleration was increased directly in all three axes. This was thought to be due to the different shear, tension, and compression characteristic of the different neural tissue by location. Angular acceleration acted differently in each axis based on what type of tissue it disturbed. Grey matter was affected by angular acceleration in the X and Z axes, and white matter was

affected by angular acceleration in the Y-axis. The white matter of the brain is thought to be more susceptible to shearing accelerations because of its directional structure. The loading curve which contained a late spike in energy absorption produced the most strain and Von Mises stress (ability to withstand a given load) under all conditions. The increased levels of strain and stress were thought to be due to the loading curve having a longer duration to peak. The plateau loading curve produced the least amount of strain and Von Mises stress, and was mainly seen in the brain stem region. The data indicated to researchers that there was a relationship among the shape of the loading curve of forces and the instance of strain and Von Mises stress.

The linear acceleration was much higher than that of the rotational acceleration in the study (Post, Hoshizaki, & Gilchrist, 2012); therefore, it cannot be concluded that strain and Von Mises stress are caused by rotational acceleration. The researchers were not able to take human variation into account for this study. Researchers claimed that imminent research studies should focus on the limitations of studies and try to gain a more profound knowledge of exactly how loading curves cause deformations in the brain and TBI. Finite element models allow for science to gain a sense of how changes in the brain occur upon impact, but they will never fully be able to represent the human brain.

Physiological Processes

Weber (2007) based a study on the conclusions from previous studies of animal models that simulate TBI in humans. Previous studies have predicted that athletes involved in contact sports would experience repetitive TBI, while other studies have found cellular markers associated with Alzheimer's Disease (AD) to be more dense in subjects when TBI is repeated. These ideas may predict an association between repetitive head trauma and neurodegeneration. Weber used a model that simulates a stretched-induced mechanical injury on neurons and glia

cells that were grown in a culture. The model is used to represent a type of damage displayed by TBI. Weber (2007) observed cumulative damage when cultured neuronal cells received repetitive stretch injuries every hour and every 24 hours. Cultured cells released an injury biomarker when stretch injury was received twice, but was not released upon initial injury. The study was simulated to show the mechanisms of cellular dysfunction in the brain post-concussion and repetitive TBI.

Effects of a TBI

Casson, Pellman, and Viano (2009) reviewed the research studies funded by the National Football League (NFL) and NFL Charities. The article reviewed the recent studies conducted within the NFL in efforts to help neurologists, in the clinical setting, better understand the mTBI they are faced with weeks and months post-injury. The review consisted of laboratory and on-field conditions. Researchers discussed studies conducted over biomechanics, epidemiology, repetitive concussion injuries, players not allowed to return-to-play within seven days, return-to-play on the day of injury, neuropsychological tests, concussion management guidelines, and chronic traumatic encephalopathy. The review represented a collection of studies that may serve as a reference for neurologists.

Gavett, Stern, and McKee (2011) conducted a study with a purpose to show the association among contact sports and chronic traumatic encephalopathy (CTE; a form of neurodegeneration). CTE is thought to be caused by repetitive head injury, and symptoms are habitually displayed post-career. CTE was first studied among boxers in 1973, with the same behaviors reported in athletes of high-contact sports: American football, soccer, and hockey. *In vivo* methods included 12 brains of the 321 professional American football players who died between February 2008 and June 2010. Brains were exposed to neuropathological analysis at Boston University Center

for the Study of Traumatic Encephalopathy (BU CSTE), and all 12 brains possessed CTE. The study considered the 309 unexamined brains not to contain signs of CTE, and concluded a relative risk of 3.7% of CTE for a professional American football player. *In vitro* methods consisted of magnetic resonance images to detect changes in white matter integrity of the brain, and magnetic resonance spectroscopy to observe changes in molecular abnormalities within the brain. The mean age of athletes who suffer from CTE was 42.8 years, with a standard deviation (SD) of 12.7. The average number of years CTE requires to show visible symptoms was 8 years after the individual's retirement (SD=10.7), with a mean duration of 17.5 years (SD=12.1). CTE is similar in nature to other neurodegenerative diseases, but has specific characteristics that allow for its diagnosis. The direct relationship between repetitive head injuries and CTE is not understood in its entirety, and examination of the brain is the only tool available for diagnostics. Further research will allow for biomarkers to be identified, diagnosis of CTE, and therapies to be developed to retard or reverse the damage of CTE. The limitations of the study included the small sample size of brains examined at BU CSTE, and the ability to only test brain tissue in an *in vivo* manner. Although the tested sample size was small, the study was able to portray the duration of sport activity and the onset of CTE.

Second Impact Syndrome

A study sponsored by the National Collegiate Athletic Association (NCAA), conducted by McCrea et al. (2003), discussed the lack of data to support proper return-to-play assessment. The purpose of the study was to examine and understand the effects and recovery of concussion and symptoms in athletes. The study included 1,631 football athletes from Division I, II, and III colleges across the United States, and was conducted over a three year period. There were 56 individuals who served as matched controls to the injured athletes for year one. Limited

resources did not allow for a control group through the second and final year of the study. There were 94 athletes who sustained a concussion within the duration of the study, and 79 of the concussed athletes finished the study. These individuals and matched control subjects were tested directly after injury with the Graded Symptom Checklist (GSC), Standardized Assessment of Concussion (SAC), and the Balance Error Scoring System (BESS), and then followed with assessments at three hours, 1, 2, 3, 5, 7, 9, and 90 days post-concussion. The subjects were assessed with a neuropsychological test battery on post-injury test days 2, 7, and 90. Multivariate regression models and recovery curves were plotted in graphs with SPSS software. Only 86% of the data was completed due to loss of experimental subjects, but baseline scores in data were determined not to differentiate between players allowing data to be considered randomized. The analysis of data showed a significant difference between concussed athletes and aged matched controls, $p < .05$. Concussed athletes showed to have a mean GSC score 20.93 points higher than non-concussed controls, a mean SAC score 2.94 points lower than controls, and a mean BESS score 5.81 points above controls, $p < .05$. Athletes required different recovery times for the different functions assessed post-concussion, but no significant changes were present in concussed athletes at the time of assessment on day 90 except for one measure. Concussed subjects had lower performance on verbal fluency at day 90 post-concussion when compared to control subjects. This study highlighted the variability of athletes' recovery from a concussion. It revealed that the normal guideline of a seven day recovery rate may not be acceptable in treatment of concussions in all athletes. Each TBI should be assessed on an individual basis and an athlete should not be allowed to return-to-play after seven days if any symptoms are present. The study was limited to collegiate football players and should be applied to more diverse sport population to ensure data are externally valid.

McClincy, Lovell, Pardini, Collins, and Spore (2006) stated that issues associated with return-to-play guidelines are frequently discussed and researched because of the immense effects second impact syndrome can have on an athlete. The purpose of the study was to examine concussion recovery time in diverse athletes at the high school and collegiate level for a duration of 30 months. Researchers gathered baseline scores from affiliated schools for 104 athletes who sustained a concussion. Baseline scores were obtained in offseason with the ImPACT battery. The concussed athletes were 87.5% male and had a mean age of 16.11 years (SD ± 2.22). Subjects completed the ImPACT battery three times after concussion. The normal test times for subjects post-concussion were two days, 1 week, and 2 weeks. Data were analyzed with a MANOVA, and a Bonferroni post hoc test was conducted to determine significance between baseline and post-injury test scores. There was a significant difference of the symptom total score at baseline and post-tests, $F(3, 309) = 72.03$, $p < .01$. There was significance in the pairwise comparisons among baseline data and tests scores recorded on the second day post-concussion, and also one week post-concussion, $p < .00$. Significance was not found among baseline test scores and those achieved at the third week post-concussion. The ImPACT battery reports post-concussion effects present 7 to 14 days after the injury occurs. Athletes at the collegiate level showed less recovery time than high school athletes. The authors concluded that this study correlates with other research, and return-to-play decisions should be made on a case-by-case basis.

A review conducted by Lovell, Collins, and Bradley (2004) discussed new developments in return-to-play management criteria after sports-related concussion. Rodent models show metabolic dysfunction caused by TBI, but cannot be studied in human subjects. Animal models have shown metabolic cascades to be detrimental to neural function, and have a potential

duration of two weeks.

The on-field assessment of concussions can be complicated, and no athlete should be allowed to return-to-play if diagnosed with concussion symptoms. Amnesia, of any degree, has recently shown to serve as a post-injury symptom indicator. There are four more current guidelines for an athlete's return-to-play: Cantu, Colorado guidelines, American Orthopedic Society for Sports Medicine (AOSSM) guidelines, and the American Academy of Neurology (AAN). These types of neuropsychological tests are a large aspect of the assessment of concussions. Tests can be compared with an athlete's own baseline data to determine changed nervous activity. In addition to all return-to-play assessment methods, concussions should be managed on an individual basis. Authors concluded that age, history of concussion, and grade of concussion should all be included in return-to-play assessment. There are many types of concussion evaluations, but none measure motor power output.

TBI Assessment

Hunt and Asplund (2010) discussed current techniques in concussion assessment and management. Concussion rates in sports have increased to an estimated 1.6 to 3.8 million in the United States every year. The combination of investigation and concussion management is crucial to reduce the occurrence of injury, and to ensure vital recovery time. The current and most common form of concussion assessment is a multidimensional method. This method includes neuropsychological and stability, as well as reported symptoms. A physical evaluation is commonly conducted immediately post-injury on the sidelines, and includes history, nervous, motor, and sensory assessment. The costs of imaging techniques are expensive, and not feasible unless used in research. Another common assessment relies on an athlete's self-reported symptoms. There are three main scales used in sport concussion management: the Graded

Symptom Checklist (GSC), Post Concussion Symptom Scale (PCSS), and the Head Injury Scale. The two most common postural stability tests are the Balance Error Scoring System (BESS) and the Neurocom Sensory Organization Test. These two assessments are recommended to be used in conjunction with one another to gain the most accurate post-concussion results. There are imaging techniques that can be used in certain concussion cases, but these tools of assessment are rarely used because concussions are usually considered a functional disorder and not structural.

Educational interventions have shown to be most effective in regards to treatment options for concussions. There are a limited amount of pharmaceuticals that may help in certain cases of concussions, but are rarely used and should only be done so under the supervision of a medical professional. Hunt and Asplund's (2010) article discussed the most practiced methods of return-to-play after a concussion. Return-to-play should be addressed with a multifaceted approach, and should include comparison to baseline measurements. Researchers concluded with recommendations for the rehabilitation process. All parties involved with the concussed athlete should be made aware of the potential occurrences that follow post-injury.

A study supported by the Centers for Disease Control and Prevention examined the post-concussion recovery rate of different motor functions. Parker, Osternig, van Donkelaar, and Chou (2007) studied the effect concussion had on motor performance because normal concussion assessment tests do no measure motor performance and there is little published data on the topic. The purpose of the study was to determine the relationship between cognitive function and dynamic motor function in concussed subjects. The study consisted of 29 NCAA male and female concussed athletes, and 29 matched controls. Concussed subjects were tested within 48 hours of TBI in gait stability and neuropsychological function. Participants were also tested on

days 5, 14, and 28 post-injury. Gait stability was analyzed with three-way ANOVAs, and two-way ANOVAs were conducted to measure neuropsychological function. Pearson's correlation coefficient was calculated to measure the relationship between the two measured variables. There was a low to moderate, significant relationship between the sway velocity and reaction time, r = .32, p = .02, and also for the reaction time and dual task sway, r = .40, p = .00, but there was not a significant difference among other tested components of the gait stability and neuropsychological tests. There was an independent relationship between test variables and recovery time shown in recovery curves. Concussion history was not measured in the study, and could act as a limitation. Results of the study showed minimal relationship among gait stability and neuropsychological tests. The authors concluded that to adequately measure the effects of concussion a multifactorial approach should be applied in the assessment.

In relation to other studies, Hinton-Bayre and Geffen (2002) examined the severity of TBI and neuropsychological test scores. Robert Cantu guidelines, the Colorado guidelines, and the American Academy of Neurology (AAN) all recommend prolonged return-to-play in relation to the severity of concussion grade. The study consisted of 175 rugby players and 29 concussions. The study was conducted over three seasons, and had a final number of 21 concussed athletes. A control group was established, but details were not explained in the publication. Athletes completed the grading systems for AAN, Cantu, and the Colorado Medical Society. No athletes sustained a grade 3 concussion, and the measurements of the study resulted in no relationship between the severity of concussion and the level of cognitive impairment. The statistical test utilized in the publication was the calculation of a standardized Z score. Authors concluded that return-to-play assessment should follow guidelines that require additional recovery time with increased grade of concussion.

In regards to concussion research, the establishment of baseline data has become a popular field of study. Schneiders et al. (2010) aimed to determine normative values that can determine neurological function after concussion: postural control and motor coordination. The establishment of baseline data for these functions can provide medical and clinical sports professionals with guidelines on how to assess post-concussive cognitive function. The study was comprised of a cross-sectional design with repeated measures. A convenience sample was used of healthy men and women between 15 and 40 years of age. Subjects complete the Tandem Gait test (TG), Finger-to-Nose test (FTN), and a Single-Leg-Stance (SLS) test for time. All tests were completed on a hard and a softer surface, and subjects who failed to complete a test were required to retest. Descriptive characteristics of participants were recorded and analyzed with test scores. The study simply reported mean scores, and did not display results that accounted for personal characteristics of the subjects. The study generated normative data, but should not be used in a clinical environment with TBI patients. The subjects of the study were a convenience sample in efforts to reach the minimum sample size of 120 subjects needed for the study. The data analysis of the repeated measures study was not published, and data was not comparable. The study did not include subjects with a sports-related concussion.

A common method used to assess most sports-related concussions is the ImPACT test battery. Schatz, Pardini, Loveall, Collins, and Podell (2006) conducted a study to examine the sensitivity and specificity of the battery for the assessment of concussion in athletes. The purpose of the study was to determine the efficacy of ImPACT scores and PCSS scores in diagnosis of concussion. The study included 72 high school athletes that suffered from mTBI 72 hours prior to tests. Sixty-six high school athletes, with no history of mTBI, served as the control group for the study. Baseline tests scores were obtained from tests administered in

offseason. An ANOVA was conducted to measure the difference between the groups. The results displayed that concussed athletes were younger than athletes in the control group $F(1, 136) = 5.35$, $p = .02$. A MANOVA was conducted to measure the individual composite scores of the ImPACT battery for the concussed athletes. All composite scores from the ImPACT battery were significantly different from the group of concussed athletes: verbal memory [$F(1, 136) = 16.6$; $p = .00$], visual memory [$F(1, 136) = 34.9$; $p = .00$], reaction time [$F(1, 136) = 43.6$; $p = .00$], processing speed [$F(1, 136) = 61.1$; $p = .00$], and symptom scale scores [$F(1, 136) = 39.6$; $p = .00$]. The authors concluded that the ImPACT test battery serves as an instrument that is sensitive and specific for concussion assessment. The study did not compare concussed athletes' post-test with test scores completed at baseline. The design of the study allowed for concussed athletes to be compared to scores of other athletes with no prior history of concussion. Future studies should compare concussed athletes post-injury ImPACT scores compared to their own baseline test scores.

More invasive forms of research yield results that aid in understanding the chronic effects of head trauma. A fair portion of research is dedicated to the cumulative effects associated with sports-related head traumas. The purpose of a NCAA study conducted by Guskiewicz et al. (2003) was to predict the occurrence of concussion in college football athletes, and the time required to recover. The study was comprised of 4251 player-seasons from 2905 NCAA football athletes. There were a total of 94 concussions included in the assessment. Baseline data was collected at enrollment with the Graded Symptom Checklist (GSC). Upon concussion, the GSC was completed by injured athletes at days 1, 2, 3, 5, and 7 post-concussion. In addition, the athletic trainers were asked to complete a concussion index that would allow for a record of critical information. Data was analyzed with SAS software, and an X^2 test of association was

used to compare GSC scores, $p < .05$. The descriptive analysis showed the rate of occurrence of concussion was 0.81 per 1000 athletes, $p < .05$. Division I athletes experienced a total of 131 concussions. Division II athletes sustained 26 concussions, and Division III accumulated 39 concussions. Athletes who reported a history of three or more concussions was 3.0 times more likely to sustain a concussion, and was associated with a longer recovery time when compared to first time concussion athletes, $p < .05$. The NCAA sponsored study suggests that athletes with a history of prior TBI are at a higher risk to sustain a concussion. Division I athletes sustain more concussions than athletes in other divisions. The study did not discuss potential reasons behind the amount of increased incidents in Division I athletes. However, the difference in mechanical forces of each division could be explored.

Livingston et al. (2010) conducted a preliminary investigation on the abnormalities of motor evoked potential (MEP) after a concussion sustained in a sport. There has been an increase in head injuries sustained from participation in heavy contact sports, and there is an estimated yearly 1.6 to 3.8 million TBI. Concussions are predominantly assessed based on visible symptoms displayed by the athlete. The purpose of this study was to determine if the motor cortex showed acute dysfunction in concussed athletes through MEPs. Authors aimed to look at the abnormalities of concussed athletes' MEPs for a period of 10 days post-concussion through the use of transcranial magnetic stimulation (TMS). MEPs allow for the pathways of the motor cortex to be assessed in a direct manner. The study included 6 female and 12 male Division I athletes from the University of Virginia and Virginia Military Institute. The subjects were divided in two groups; one with three female athletes, and one with six male athletes who sustained a TBI within 24 hours, and a matched control for each concussed subject. A MagStim Novametrix 200 magnetic stimulator was used to conduct the TMS, and three different MEP

thresholds were measured: movement, lower, and upper thresholds. The central motor conduction time (CMCT), or the time elapsed for signals to reach the spinal motor neurons form the motor cortex, was also measured with TMS. The TMS was administered with a coil that was placed at a 45˚ angle to the sagittal plane over the right motor cortex of the brain. The subjects were measured within 24 hours post-injury and again on days three, five, and ten post-injury. Repeated measures ANOVAs were used in SPSS version 17.0 to conduct data analysis. There was a significant difference in the MEP amplitudes between days three and five of post-injury assessment, $F(3, 48) = 3.13$, $p = .04$, and there was a significant difference in MEP latencies between days one and ten, $F(3, 48) = 4.53$, $p = .02$. However, there was no significant difference in CMCT or MEP thresholds, $p > .05$. The study showed a change in MEPs acutely after TBI, and this may represent impairment in the actual neurophysiology and signaling of the brain. Further research is needed using a larger sample size to gain more insight as to how concussion impairs MEP and MEP thresholds.

Other Variables

The purpose of a study conducted by Griesbach (2011) was to determine if exercise after TBI is detrimental to neural plasticity. The University of California at Los Angeles (UCLA) Brian Research Center conducted a study that aimed to measure the amounts of different growth factors present after TBI in mice. The animal model subjects incurred TBI by a fluid-percussion injury (FPI) and via controlled cortical impact (CCI). Subjects that were exposed to exercise prior to FPI or CCI did not have a decrease in the growth factor brain-derived neurotrophic factor (BDNF), and subjects not exposed to exercise prior to TBI did have a sufficient decrease in BDNF. Results showed mice exposed to FPI and acute exercise post TBI showed problems with delayed learning and memory. Mice exposed to FPI with no exercise after injury did not show

same problematic symptoms. The author declared that many factors need to be taken into account when comparing animal and human models, however; this study correlates with published studies with human subjects. The article explained previous research highlighted opportunities for further research in order to identify an exact time frame when exercise should be halted in order to gain maximum health post TBI. In relation to human models, this study may show insight to potential harmful effects of premature return-to-play.

As in previous studies, different elements of subjects' medical and health history can affect the outcome of the experimental measurements. Biochemical components and other internal compounds are hard to manage in a noninvasive study, and could alter results of research. Joseph et al. (2012), examined the effects of docosahexaenoic acid (DHA) and exercise on nervous tissue and sensorimotor learning. DHA is an omega-3 fatty acid that has many important roles in neural function, and in previous research has shown to aid in the repair of neural damage. Exercise has been shown to produce brain-derived neurotrophic factor (BDNF). BDNF serves as a neuroprotective compound which aids in decreased structural damage. The study used animal models that completely severed the spinal columns of mice in the mid-thoracic region. The mice were administered DHA which increased the rate of delivery of BDNF to the spinal cord. Mice were able to plantar flex bottom paws when electrical stimulation was applied below the severed spinal cord. No statistical analyses were discussed, and statistical results were limited to graphs. Authors concluded that a DHA supplemented diet will improve spinal learning, and increase related cellular marker learning as well. The study also found that when BDNF was blocked from the spinal column, mice were not able to improve spinal cord learning. This study shows how diet and exercise may potentially affect the way individuals internally respond to head trauma.

Summary

There are current gaps in concussion-related research. Medical and clinical sports professionals manage concussions with current scientific knowledge, but no specific guidelines or normative data have been established. Concussions are assessed based on visible characteristics and performance of the injured athlete. Resent research has suggested that the acute effects of TBI are more severe and last beyond the visible diagnostic symptoms. A large portion of previous research seems to focus attention on the chronic effects potentially caused by concussions, and repetitive TBI. Efforts in acute phase research and improved diagnostic tools are key to understanding the chronic effects, and ultimately diminishing the chronic life altering effects. To date, there has been no research conducted that has examined the integrity and neural excitability of the corticospinal system upon a TBI. Further research is needed to explore the acute effect on motor power output after a TBI. Flaws in the existing techniques of concussion management, and adequate research will allow for these flaws to diminish and for athletes to return-to-play in a safe manner.

CHAPTER THREE: Methods

Participants

College football athletes at the University of Central Oklahoma (UCO) were recruited. The study, procedures, measurements, and the importance of the study was discussed with the head coach. Athletes were required to be listed on UCO's roster as of September 1, 2013, to participate in the study, ensuring they had passed a physical and obtained medical clearance to play. Athletes with a history of prior concussion were not excluded from the study, and concussion history was documented. A previous study that examined clinical measures of motor performance post-sport induced TBI received 172 volunteer subjects (Schneiders, Sullivan, Gray, Hammond-Tooke, & McCrory, 2010). Based upon the previous study, an estimated sample size of 60-70 participants was calculated with a statistical power of .80, $\alpha = .05$. The estimated sample size was unattainable due to the number of UCO football athletes. However, recruited subjects were limited to UCO football players due to lack of time and sources of subjects, and 82 subjects were recruited via PowerPoint presentation. A total of 55 athletes volunteered for participation in the study, and 13 athletes completed baseline testing. There were two participants who had sustained three prior TBIs, and eight participants who had never sustained a medically diagnosed TBI. Only participants who completed baseline testing and sustained a TBI were used for data analysis. One participant sustained a TBI and completed the study. The TBI participant had sustained two TBIs within the previous nine months.

An application and amendment were submitted to UCO's Institutional Review Board for approval (Appendix A). Athletes who volunteered and were present at baseline testing received an Informed Consent form, and were required to sign and return the form to the principle investigator before participation in the study was allowed (Appendix B). Subjects also reported

their prior concussion and health information prior to EMG measurements (Appendix C).

Instruments

Medical History Survey. Participant health background and previous concussion information was self-reported at the time baseline measurements were obtained. Subjects were asked to complete a descriptive form that included: name, age, number of years active in heavy contact sports, number of medically diagnosed concussions, number of assumed concussions, exercise habits, and dietary supplements (Appendix C). All self-reported data, minus subjects' name, was used upon completion of data collection to explain varied results. A randomized number was assigned to each participant for the protection of their identity and personal information.

Electromyography (EMG). EMG was used to measure motor power output with AD Instruments Lab Chart 7. The device graphically displayed the electrical activity produced by the subjects' skeletal muscles resulting from movement (Appendix D). Previous studies have shown EMG measurements to have reliability through intraclass correlation coefficients in maximal and submaximal contractions over time (Yang & Winter, 1983). The machine produces raw data, recorded in millivolts (mV), which must be converted into the root mean square (RMS) to be able to be measured.

HUMAC Norm Isokinetic Dynamometer. The university also possesses a HUMAC Norm isokinetic dynamometer that ensured the same force was applied by each subject through the range of motion (ROM). The HUMAC Norm measures torque produced by the participant through calculations of applied amount of torque, and the length of the limb used as a moment arm to apply the force. The device provides a print out of torque produced throughout the range of motion for each repetition of movement (Appendix E). The HUMAC Norm was set at

60˚/second for each participant, because subjects apply more torque through a slower range of motion.

Procedures

Upon completion and return of the informed consent and previous medical history survey, each participant voluntarily sat in the HUMAC Norm chair and the principle investigator (PI) secured the safety harnesses. The PI then secured a ground wire around the wrist, heart rate monitor upon the middle finger, and four electrodes on each participant: two upon the vastus lateralis and two upon the vastus medialis. The right leg was tested on all participants, but the self-reported "dominant leg" was noted. Each participant was allowed three practice repetitions to become familiarized with the machine, technique, and movements. After the three practice repetitions, participants then completed two sets of six repetitions with a two minute rest period between sets. Peak torque, total work output, and electrical data from the EMG were recorded and used as baseline measurement information. The baseline data was compiled into one document with Excel, and included the subjects' assigned randomized number. Self-reported information sheets with the subjects' names and assigned number for the study were stored in a separate, locked file.

The participant that sustained a concussion during the 2014 spring season performed the same tests using the HUMAC Norm and EMG post-TBI. The subject served as his own control group, and tests were conducted 24 hours and seven days after the sustained TBI. The results from the EMG were recorded in Excel next to the subject's baseline data. The principle investigator compared baseline measurements with post-injury measurements on TBI subject. The tests were conducted from March 24, 2014 through April 25, 2014.

Statistical Analysis

The raw electrical data recorded with the EMG were calculated into the root mean square (RMS) to allow for motor power output analysis and post-TBI comparison. The HUMAC Norm measured the peak torque and total work produced for both extensors and flexors. The average of the two sets was calculated for motor power output, peak torque, and total work output for baseline, 24 hour post-TBI, and 7 days post-TBI. The percent difference was then calculated to compare motor power output and power output performance post-TBI to assess the null hypothesis that stated a TBI would not have an acute effect on motor power output.

CHAPTER FOUR: Results

The purpose of this study was to determine if TBI produces an acute effect on motor power output. Percent difference was calculated between baseline measures and post-TBI test dates for motor power output, peak torque, and total work. The percent difference within each variable was also premeditated.

Electromyography (EMG)

All recorded data are listed in Table 2 for each test date. Raw data were computed to a usable form and expressed as RMS. RMS was calculated for both channels to determine electrical activity for the vastus lateralis and vastus medialis. Percent difference was calculated to compare differences among test dates, and is presented in Figure 1. With baseline data considered 100%, the participant had vastus lateralis performance of 35.72% and vastus medialis performance of 36.2% at 24-hours after the injury was sustained compared to levels prior to the TBI. Although the athlete's EMG performance increased from 24-hours to 7-days post-TBI, the data showed he had not fully recovered with vastus lateralis performance at 89.30% and vastus medialis performance at 58.38% of baseline.

HUMAC Norm Isokinetic Dynamometer

Data collected with the HUMAC Norm are presented in Table 3 for each test date. The data are individualized for extensor and flexor measures, and also averaged for each measure: peak torque, total work, and average power/repetition. Figure 2 displays the percent difference of peak torque displayed in both extensors and flexors for each test date. Baseline data served as 100%. The subject performed at 58.99% (extensors) and 85.98% (flexors) at 24 hours after he sustained a concussion. He showed similar results with total work performed recorded at 40.43% (extensors) and 63.01% (flexors) at the same test time. Similar to EMG data, the subject

had an increase in performance at the 7-day test: peak torque recorded 85.25% (extensors) and 108.91% (flexors). Although there was a measure that had returned to baseline, the total work performed was still well below baseline at 56.79% (extensors) and 72.33% (flexors) (Figure 3).

CHAPTER FIVE: Discussion

Conclusions

The current study set out to measure the acute effect of TBI on motor power output. Data obtained from EMG showed that there was a decrease in motor power output at seven days post-TBI, which has been shown through previous research to serve as the general timeframe for returning-to-play. The TBI subject was operating at less than 30% of baseline electrical nerve signaling for both vastus measurements when tested 24 hours after the injury. Although the subject had significant improvement, he was still only operating at 58-89% seven days after the sustained TBI. There are different mechanisms that may be responsible for the continued decrease of motor power output. Frequency, rate coding, and synchronization are all responsible for the strength of nerve signal conduction. Data from this study suggests that there may be a retardation or issue with one or multiple nerve signaling components. Additional instrumentation used in the study allowed for more variables to be measured. Total work performed in ft-lbs responded similarly to EMG results; the RMS of electrical data had not returned to baseline at 7 days post-TBI. The decrease in electrical activity (motor power output) may have contributed to the decrease of total work performed (torque). There seemed to be no noticeable pattern in percent improvements of variables.

Limitations

Results from the study are externally valid because of the “real world” design. Internal validity was hindered due to a small sample size, no randomization, and metabolic components that cannot be controlled for.

Future Research

Future studies should be conducted that include more participants to allow for a larger

sample size, and should also include more sports and both genders to ensure testing methods produce similar results. Future research should also investigate to determine if there is a difference among subjects who have previously sustained a TBI and those who have remained concussion free prior to TBI. Similarly, the acute effect a TBI has on motor power output requires further exploration. Although peak torque and total work output measurements were not an original aspect of the study, future research may identify the relationship between peak torque and total work performed from a motor power output perspective and identify the reason for an increase in flexor peak torque while total work output of flexors (and extensors) and motor power output remained below baseline.

References

Casson, I., Pellman, E., & Viano, D. (2009). Concussion in the National Football League: An overview for neurologist. *Physical Medicine and Rehabilitation Clinics of North America, 20*(2009), 195-214. doi: 10.1016/j.pmr.2008.10.006

Costanza, A., Weber, K., Gandy., S., Bouras, C., Hof, P., Giannakopoulos, P., & Canuto, A. (2011). Review: Contact sport-related chronic traumatic encephalopathy in the elderly: Clinical expression and structural substrates. *Neuropathology and Applied Neurobiology* 37, 570-584. doi: 10.1111/j.1365-2990.2011.01186.x

Deibert, E., & Kryscio, R. (2012). How many hits are too many? *Neurology, 78*, 1712-1713. doi: 10.1212/WNL.0b013e31825875f7

Dupuis, F., Johnston, K.., Lavoie, M., Lepore, M., & Lassonde, M. (2000). Concussion in athletes produce brain dysfunction as revealed by event-related potentials. *Clinical Neuroscience and Neuropathology, 11,* 4087-4092.

Gavett, B.F., Stern, R.A., & McKee, A.C. (2011) Chronic traumatic encephalopathy: A potential late effect of sport-related concussive and subconcussive head trauma. *Clinical Sports Medicine* 30(1). doi:10.1016/j.csm.2010.09.007

Griesbach, G. (2011). Exercise after traumatic brain injury: Is it a double-edged sword? *American Academy of Physical Medicine and Rehabilitation* 3, S64-S72. doi: 10.1016/jpmrj.2011.02.008

Guskiewiez, K., McCrea, M., Marshall, S., Cantu, R., Randolph, C., Barr, W., Onate, J., & Kelly, J. (2003). Cumulative effects associated with recurrent concussion in collegiate football players. *Journal of the American Medical Association, 290*, 2549-2555. doi: 10.1001/jama.290.19.2549

Guskiewicz, K., Mihalik, J., Shankar, V., Marshall, S., Crowell, D., Oliaro, S., Ciocca, M., & Hooker, D. (2007). Measurement of head impacts in collegiate football players: Relationship between head impact biomechanics and acute outcome after concussion. *Neurosurgery, 61*, 1244-1253. doi: 10.1227/01.NEU.0000280146.79

Hazrati, L., Tartaglia, M., Diamandis, P., Davis, K., Green, R., Wennberg, R., Wong, J., Ezerins, L., & Tator, C. (2013). Absence of chronic traumatic encephalopathy in retired football players with multiple concussions and neurological symptomatology. *Frontiers in Human Neuroscience, 7*, 1-9. doi: 10.3389/fnhum.2013.00222

Hecht, A. N. (2002). Legal and ethical aspects of sports-related concussions: The Merril Hoge story [Special section]. *Seton Hall Journal of Sport Law, 12*, 17-64.

Heitger, M., Jones, R., Dalrymple-Alford, J., Frampton, C., Ardagh, M., & Anderson, T. (2007). Mild head injury-a close relationship between motor function at 1 week post-injury and overall recovery at 3 and 6 months. *Journal of Neurological Sciences* 253, 34-47. doi: 10.1016/j.jns.2006.11.007

Henry, L., Tremblay, S., Boulanger, Y., Ellemberg, D., & Lassonde, M. (2010). Neurometabolic changes in the acute phase after sports concussions correlate with symptom severity. *Journal of Neurotrauma 27,* 65-76. doi: 10.1089/neu.2009.0962

Hinton-Bayre, A., & Geffen, G. (2002). Severity of sports-related concussion and neuropsychological test performance. *Neurology, 59*, 1068-1070. doi: 10.1212/WNL.59.7.1068

Hunt, T., & Asplund, C. (2010). Concussion assessment and management. *Clinic of Sports Medicine* 29, 5-17. doi: 10.1016/j.csm.2009.09.002

Iverson, G., Brooks, B., Lovell, M., & Collins, M. (2006). No cumulative effects for one or two

previous concussions. *British Journal of Sports Medicine 40,* 72-75. doi: 10.1136/bjsm.2005.020651

Joseph, M., Ying, Z., Zhuang, Y., Zhong, H., Wu, A., Bhatia, H., Cruz, R., Tillakaratne, N., Roy, R., Edgerton, V., & Gomez-Pinilla, F. (2012). Effects of diet and/or exercise in enhancing spinal cord sensorimotor learning. *PLoS ONE 7*(7): e41288. doi: 10.1371/journal.pone.0041288

Kain, D. J. (2009). "It's just a concussion;" The National Football League's denial of a causal link between multiple concussions and later-life cognitive decline [Special section]. *Rutgers Law Journal, 4,* 697-736.

Livingston, S., Saliba, E., Goodkin, H., Barth, J., Hertel, J., & Ingersoll, D. (2010). A preliminary investigation of motor evoked potential abnormalities following sport-related concussion. *Brain Injury, 24*(6), 904-913. doi: 10.3109/02699051003789245

Lovell, M., Collins, M., & Bradley, J. (2004). Return to play following sports-related concussion. *Clinics in Sports Medicine, 23,* 421-441. doi: 10.1016/j.csm.2004.04.001

McClincy, M., Lovell, M., Pardini, J., Collins., M., & Spore, M. (2006). Recovery from sports concussion in high school and collegiate athletes. *Brain Injury 20*(1), 33-39. dio: 10.1080/2699050500309817

McCrea, M., Guskiewicz, K., Marshall, S., Barr, W., Randolph, C., Cantu, R., Onate, J., Yang, J., & Kelly, J. (2003). Acute effects and recovery time following concussion in collegiate football players. *Journal of American Medical Association, 19*(290), 2556-2563.

Parker, T., Osternig, L., van Donkelaar, P., & Chou, L. (2007). Recovery of cognitive and dynamic motor function following concussion. *British Journal of Sports Medicine, 41,*

868-873. doi: 10.1136/bjsm.2006.033761

Post, A., Hoshizaki, B., & Gilchrist, M. (2011). Finite element analysis of the effect of loading curve shape on brain injury predictors. *Journal of Biomechanics 45,* 679-683. doi: 10.1016/j.jbiomech.2011.12.005

Schatz, Ph., Pardini, J., Loveall, M., Collins, W., & Podell, K. (2006). Sensitivity and specificity of the ImPACT test battery for concussion in athletes. *Archives of Clinical Neuropsychology, 21,* 91-99. doi: 10.1016/j.acn.2005.08.001

Schneiders, A., Sullivan, A., Gray, A., Hammond-Tooke, G., & McCrory, P. (2010) Normative values for three clinical measures of motor performance used in the neurological assessment of sports concussion. *Journal of Science and Medicine in Sport 13,* 196-201. doi: 10.1016/j.jsama.2009.05.004

Shaw, N. (2002). The neurophysiology of concussion. *Progress in Neurobiology, 67,* 281-344.

Tegner, Y., & Lorentzon, R. (1996). Concussion among Swedish elite ice hockey players. *British Journal of Sports Medicine, 30,* 251-255. doi: 10.1136/bjsm.30.3.251

Tremblay, S., Beaumont, L., Henry, L., Boulanger, Y., Evans, A., Bourgouin, P., Poirier, J., Theoret, H., & Lassonde, M. (2012). Sports concussions and aging: A neuroimaging investigation. *Cerberal Cortex.* doi: 10.1093/cercor/bhs102. Downloaded from http://cercor.oxfordjournals.org/

Wall, S., Williams, W., Cartwright-Hatton, S., Kelly, T., Murray, J., Murray, M., Owen, A., & Turner, M. (2006). Neuropsychological dysfunction following repeat concussions in jockeys. *Journal of Neural Neurosurgery and Psychiatry 77,* 518-520. doi: 10.1136/jnnp.2004.061044

Weber, J. (2007). Experimental models of repetitive brain injury. *Progress in Brain Research, 161,* 253-259. doi: 10.1016/S0079-6123(06)61018-2

Yang, J. & Winter, D. (1983). Electromyography reliability in maximal and submaximal isometric contraction. *Archives of Physical Medicine and Rehabilitation, 64*(9), 417-420.

Table 1
Participant History

Age	# years in sport	medically diagnosed TBI	age(s) of prior TBI
19	8	2	16, 17
19	13	3	14, 15, 16
20	13	2	18, 19
20	10	3	15, 20, 20
19	7	1	18
21	8	0	x
21*	12	0	x
19*	13	0	x
20*	12	0	x
19	11	0	x
21*	11	0	x
21*	12	0	x
20	5	0	x

Note. *Indicates participants believed to have sustained prior TBI(s) not medically diagnosed.

Table 2

EMG Data

mV	mean	SD	max value	min value	RMS	RMS-Average
Baseline						
Vastus Lateralis						0.7767
S1	-0.056	0.41	1.863	-1.843	0.419	
S2	-0.100	1.13	2.356	-4.331	1.134	
Vastus Medialis						0.4321
S1	-0.068	0.55	2.372	-2.656	0.557	
S2	-0.060	0.30	1.279	-1.643	0.307	
24hr post						
Vastus Lateralis						0.2774
S1	-0.012	0.19	1.306	-1.011	0.191	
S2	-0.017	0.36	1.519	-1.801	0.364	
Vastus Medialis						0.1564
S1	-0.011	0.12	0.606	-0.994	0.123	
S2	0.016	0.19	0.739	-1.241	0.190	
7day post						
Vastus Lateralis						0.6936
S1	-0.032	0.38	1.580	-1.419	0.379	
S2	-0.041	0.63	2.029	-2.738	0.630	
Vastus Medialis						0.2522
S1	-0.017	0.18	1.279	-1.229	0.178	
S2	0.007	0.33	1.403	-1.700	0.326	

Note. mV = millivolts; SD = standard deviation; RMS = Root Mean Square; S1 = Set one; S2 = Set two.

Table 3

HUMAC Norm Isokinetic Dynamometer Data

Ft-lbs	Peak Torque		Total Work		Average Power/rep	
	(E)	(F)	(E)	(F)	(E)	(F)
Baseline						
S1	127	67	737	455	111	65
S2	152	90	809	510	130	82
Average	139	78.5	773	482.5	120.5	73.5
24hr post						
S1	69	61	241	247	57	89
S2	95	74	384	361	84	73
Average	82	67.5	312.5	304	70.5	66
7day post						
S1	107	86	393	330	98	82
S2	130	85	485	368	109	81
Average	118.5	85.5	493	349	103.5	81.5

Note. (E) = Extensors; (F) = Flexors; S1 = first set; S2 = second set.

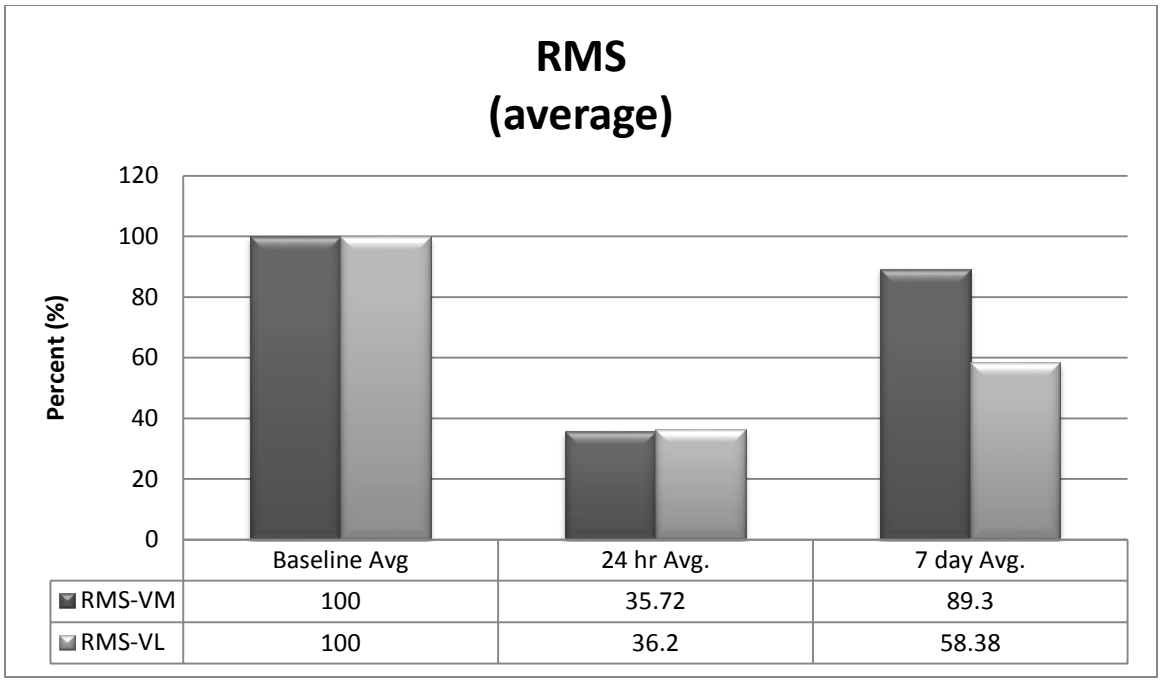


Figure 1. A bar graph that represents the percent difference of average RMS for the vastus lateralus and vastus medialias (calculated in mV) at baseline, 24 hours post-TBI, and 7 days post-TBI. Subject had not returned to baseline by the normal 7-day return-to- play guideline.

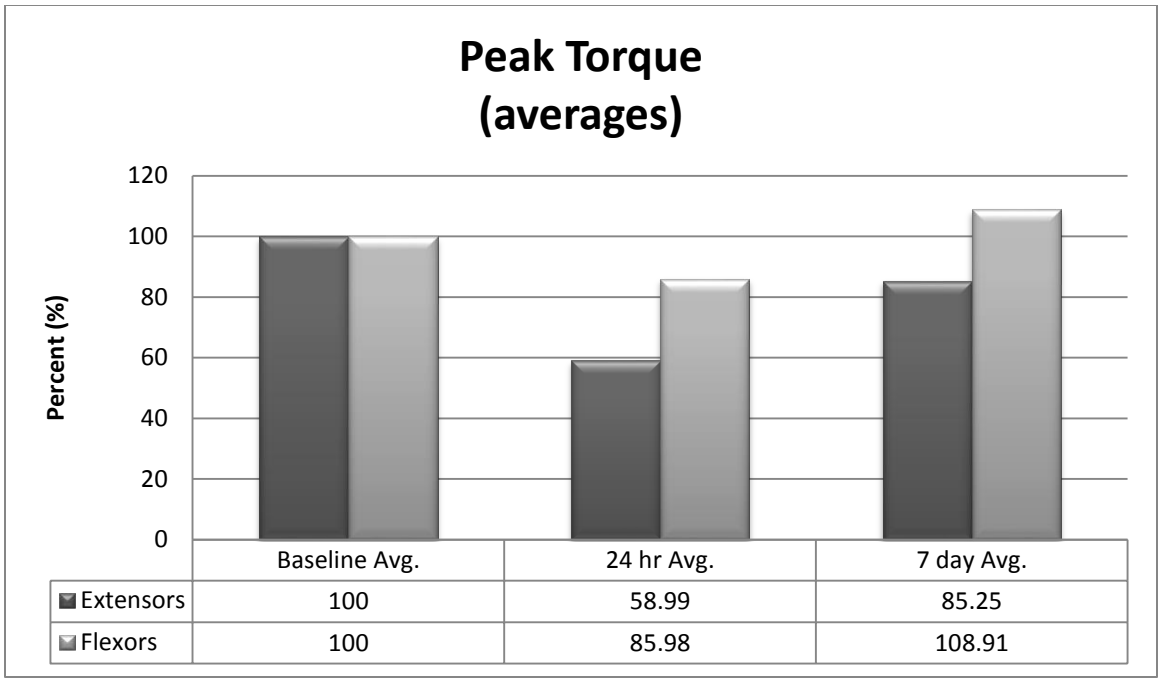


Figure 2. A bar graph of the percent differences in average peak torque for extensors and flexors from baseline, 24 hour post-TBI, and 7 days post-TBI. Subject had not returned to baseline in extensors peak torque by normal return-to-play guideline time period.

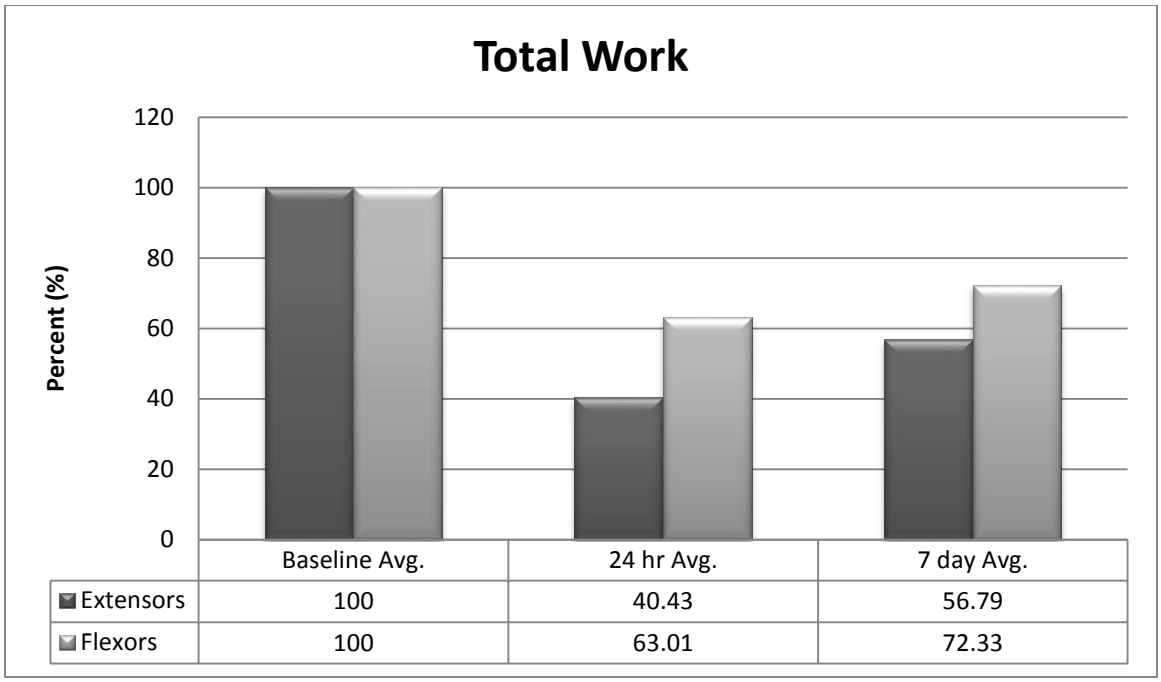
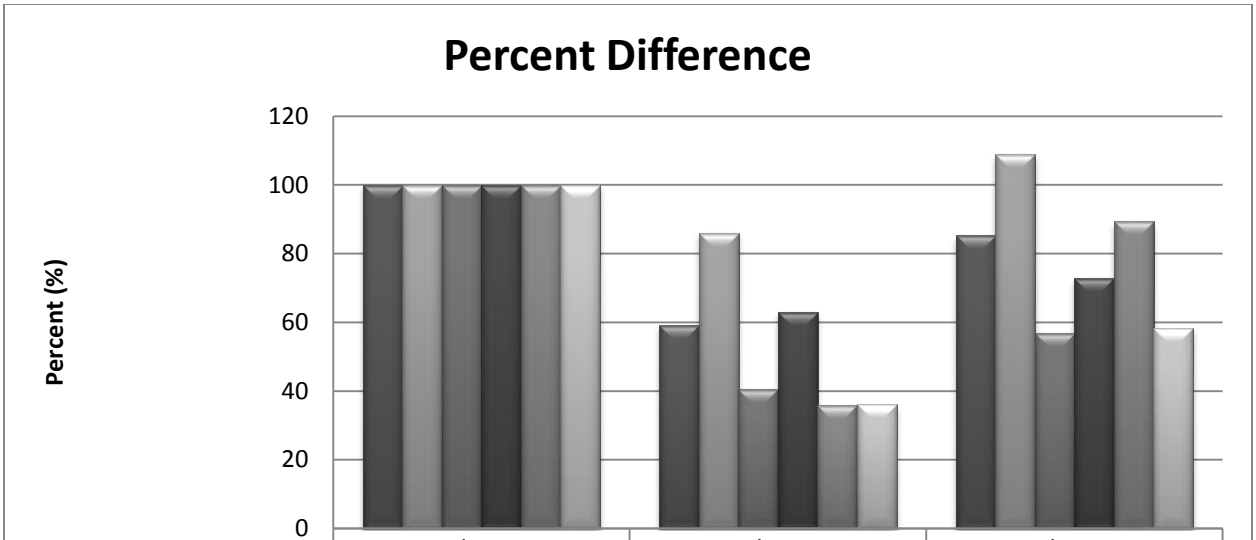


Figure 3. A bar graph of the percent difference in average total work performed in extensors and flexors at baseline, 24 hours post-TBI, and 7 days post-TBI. Subject had not returned to 75% percent of baseline data at the general guideline period for return-to-play.



	Baseline Avg.	24 hr Avg.	7 day Avg
Peak Torque Extensors	100	58.99	85.25
Peak Torque Flexors	100	85.98	108.91
Total Work Extensors	100	40.43	56.79
Total Work Flexors	100	63.01	72.79
RMS-VM	100	35.72	89.3
RMS-VL	100	36.2	58.38

Figure 4. A bar graph that displays the percent difference of all variables measured at baseline, 24 hours post-TBI, and 7 days post-TBI.

Appendix A

IRB Approval

March 11, 2014 IRB Application #: 13145

Proposal Title: *Acute Effect of TBI on Motor Power Output*

Type of Review: Amendment-Expedited

Investigators:

Ms. Andrea Gilliland
Dr. Gregory Farnell
Department of Kinesiology and Health Studies
College of Education and Professional Studies
Campus Box 189
University of Central Oklahoma
Edmond, OK 73034

Dear Ms. Gilliland and Dr. Farnell:

Re: IRB Amendment Application

We have received and reviewed your request for an amendment to your approved IRB application and supporting materials. The UCO IRB approves the following amendments to your application:

Changes to approved research site; changes in procedures; change in Informed Consent Form; change in recruitment material

Original Approval Date: 9/25/2013

Approval Expiration: 9/24/2015

This project is approved for a one year period from the original approval date and any further modification to the procedures and/or consent form must be approved prior to its incorporation into the study. A written request is needed to initiate the amendment process. You will be notified in writing prior to the expiration of this approval to determine if a continuing review is needed.

We wish you continued success with your project. If our office can be of further assistance, please do not hesitate to contact us.

Sincerely,

Jill A. Devenport, Ph.D.
Chair, Institutional Review Board
Director of Research Compliance, Academic Affairs
Campus Box 159
University of Central Oklahoma
Edmond, OK 73034
405-974-5479
jdevenport@uco.edu

Appendix B

Informed Consent Form

UNIVERSITY OF CENTRAL OKLAHOMA

INFORMED CONSENT FORM

Does head trauma cause an acute decrease in motor power output and sensory response time?

Researcher (s): Andrea (Andy) Gilliland

A. Purpose of this research: To determine if head trauma (i.e. concussion) causes an acute decrease in motor power output and sensory response time.

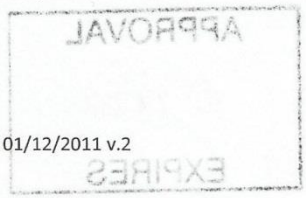
B. Procedures/treatments involved: You will receive and complete background history information upon arrival for baseline testing in the Wantland Hall Computer Lab. You will then be placed and properly fitted on the Humac Norm isokinetic dynamometer for testing. Noninvasive, surface electrodes will be placed on the vastus medialis and vastus lateralis muscles on the upper leg. Electrical activity will be recorded using an electromyography (EMG). You will then perform two sets of six repetitions at a set speed of 60 degrees per second. This will consist of 12 knee extensions rotating 60 degrees per second with a rest period after the 6th repetition. This initial testing will constitute your "baseline" measure.
After the initial participants receive a concussion, as a diagnosed by the team's medical staff, and then you will undergo the same testing within 24-36 hours and again 7 days after the acute TBI.

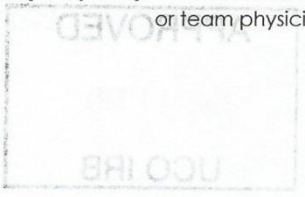
C. Expected length of participation: maximum of 10 minutes for each testing session

D. Potential benefits: You may know when they return to their baseline level of strength after acute TBI, and may give insight on premature return-to-play procedures for future athletes and sports medical professionals.

E. Potential risks or discomforts: There is minimal risk associated with the device and testing procedures. The Isokinetic Dynamometer is a machine that provides computerized measurements for movements. This allows for the device arm to move at a set speed and measures the force applied, and not require a large amount of force exertion. Principal investigators have been trained to properly use the IDK.

F. Medical/mental health contact information (if required): You should contact your personal or team physician.





G. Contact information for researchers: Andrea (Andy) Gilliland
(c) 918.346.9350
agilliland7@uco.edu
andy.gilliland@okstate.edu

Dr. Gregory Farnell
(o) 405.974.5304
gfarnell@uco.edu

H. Contact information for UCO IRB: Dr. Jill A. Devenport
Chair, UCO Institutional Review Board
Director, Office of Research Compliance
Academic Affairs
ADM 216
Edmond, OK 73034
405.974.5497
405.974.3825 (fax)

I. Explanation of confidentiality and privacy: You will receive a blank notecard at the time of recruitment, and will return notecard with contact information if you desire to participate. You will receive randomized numbers at baseline measurements, and no data will be seen by any persons except the PI and Co-PI that can be identifiable. Data will only be identifiable by assigned number and reported as group means.

J. Assurance of voluntary participation: No person is required to participate in the research study. Subjects may withdraw from participation in the study at any time without penalty.

AFFIRMATION BY RESEARCH SUBJECT

I hereby voluntarily agree to participate in the above listed research project and further understand the above listed explanations and descriptions of the research project. I also understand that there is no penalty for refusal to participate, and that I am free to withdraw my consent and participation in this project at any time without penalty. I acknowledge that I am at least 18 years of age. I have read and fully understand this Informed Consent Form. I sign it freely and voluntarily. I acknowledge that a copy of this Informed Consent Form has been given to me to keep.

Name: _____

Signature: _____ Date _____

Appendix C

Medical History Survey

Research #:_____
(Research Committee use only)

Background Information
(CONFIDENTIAL)

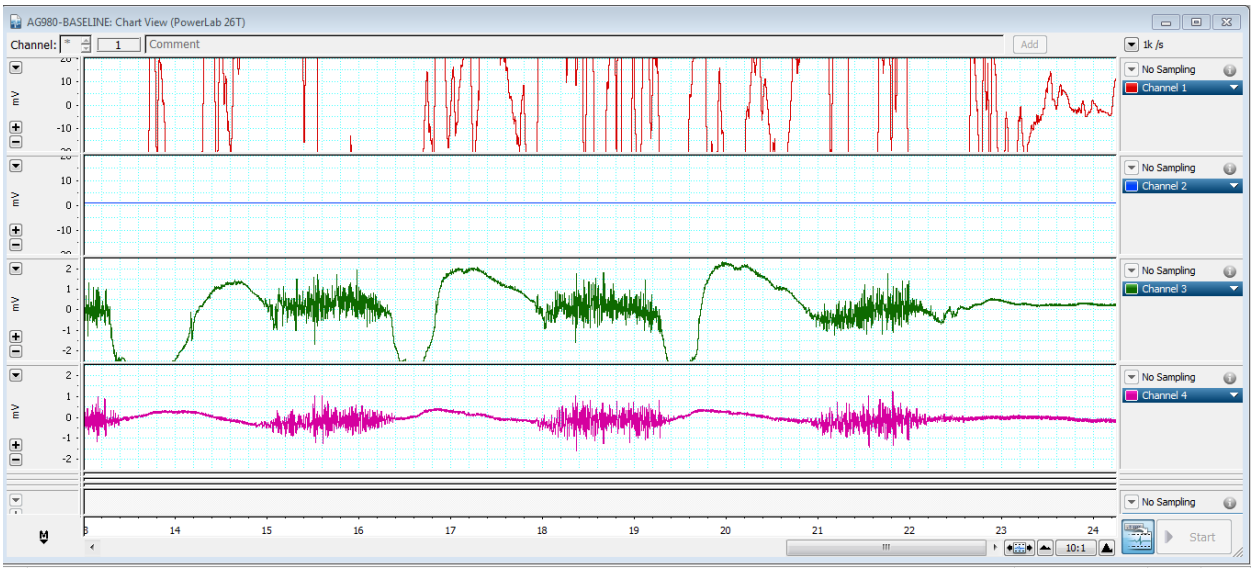
1. What is your age? _____
2. Which contact sport are you listed on the 2013-2014 roster? _____
3. How many years have you been active in this sport? _____
4. Have you ever been medically diagnosed with a concussion? _____
5. If yes, how many concussions have you had? _____
6. At what ages were the concussions acquired? _____
7. Do you think you have had any concussions that were not medically diagnosed? _____
8. If yes, how many? _____
9. What best describes your current weekly workout routine? (Circle One)

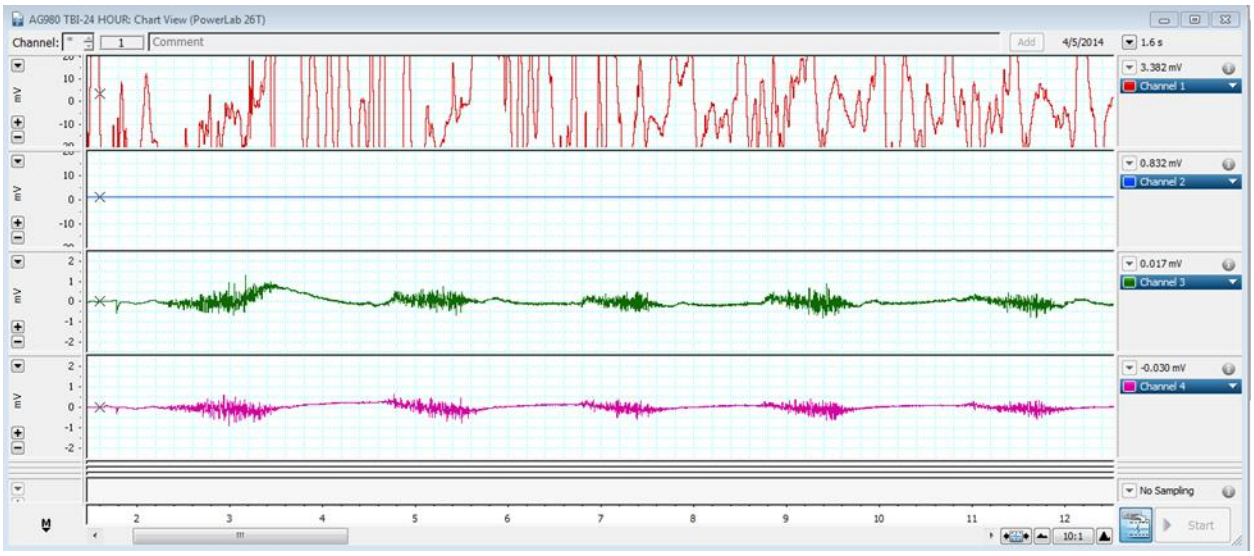
   Light
   Moderate
   Heavy
   Exhaustive

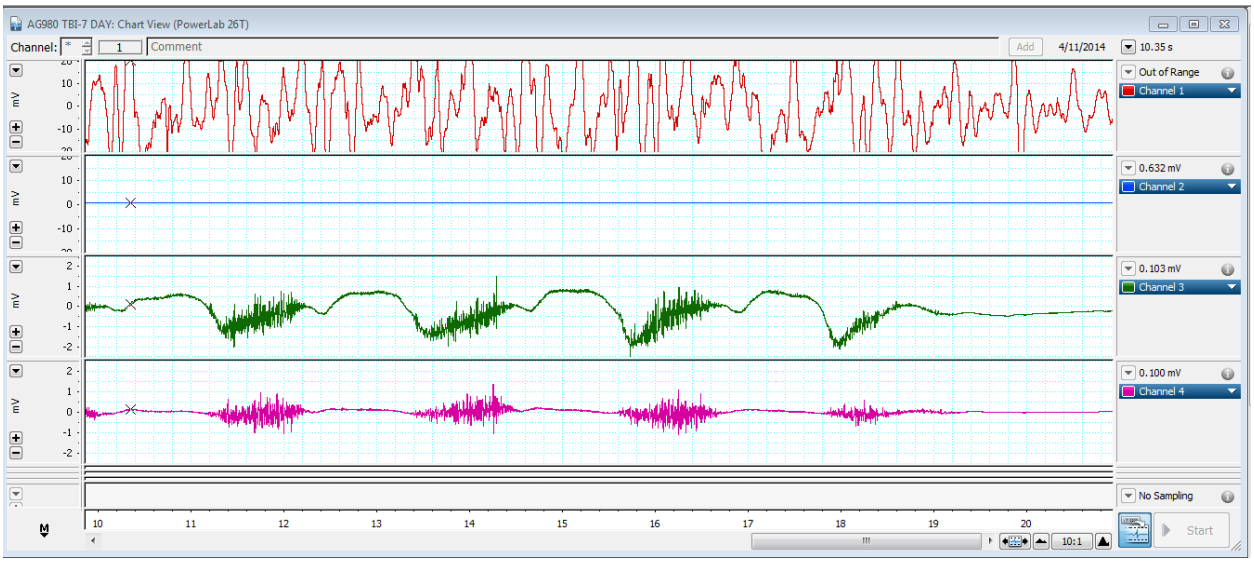
10. What is the normal duration of your workouts? _____
11. Are you currently taking any supplements, please list supplements? (Vitamins, workout supplements, hormones)_____

Appendix D

Example of EMG Raw Data Recordings


AG980-BASELINE: Chart View (PowerLab 26T)
Channel:
Comment
Add
1k /s
No Sampling
Channel 1
Channel 2
Channel 3
Channel 4
mV
10:1
Start


AG980 TBI-24 HOUR: Chart View (PowerLab 26T)
Channel:
Comment
Add
4/5/2014
1.6 s
3.382 mV
Channel 1
0.832 mV
Channel 2
0.017 mV
Channel 3
-0.030 mV
Channel 4
No Sampling
mV
10:1
Start


AG980 TBI-7 DAY: Chart View (PowerLab 26T)
Channel:
Comment
Add
4/11/2014
10.35 s
Out of Range
Channel 1
0.632 mV
Channel 2
0.103 mV
Channel 3
0.100 mV
Channel 4
No Sampling
mV
10:1
Start

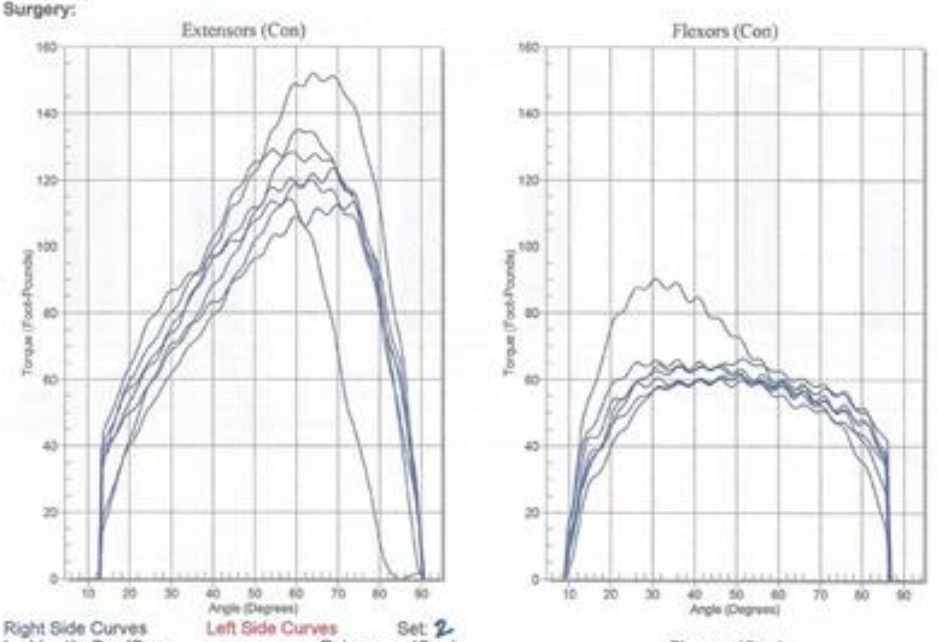
Appendix E

Example of Humac Norm Printout

HUMAC Measurement & Training Systems by CSMi

Short (Trq vs. Pos) - Knee Extension/Flexion

Name:	AG980,	ID:		Right/Left:	3/25/2014 3/25/2014 11:39am
Birth date:			Involved Side:	Right	Group 1:
Height:	Inches		Preferred Side:	Left	Group 2:
Weight:	0 Pounds	Doctor:			
Gender:	Male	Tester:	Andy Gilliland		
Diagnosis:					
Surgery:					



Right Side Curves Left Side Curves Set: 2

Isokinetic Con/Con	Extensors (Con)			Flexors (Con)			
Speed 60/60 deg/sec 7 Reps	Value	Cof Var	%BW	Value	Cof Var	%BW	Ratio
Peak Torque (Foot-Pounds - Best Repetition)							
Right	152	0.11	0	90	0.16	0	59
Left	0	0.00	0	0	0.00	0	0
Deficit	0			0			
Work per Repetition (Foot-Pounds - Best Repetition)							
Right	136	0.11	0	88	0.10	0	65
Left	0	0.00	0	0	0.00	0	0
Deficit	0			0			
Average Power per Repetition (Watts - Best Repetition)							
Right	130	0.16	0	82	0.08	0	63
Left	0	0.00	0	0	0.00	0	0
Deficit	0			0			

Appendix F

Thesis Summary Document

Thesis Summary

Problem Statement

Concussions are the most complex and common form of traumatic brain injuries (TBIs). Those sustained in heavy contact sports have been correlated with a diminished quality of life decades after the occurrence of injury. There are gaps in research that do not allow for medical personal to understand the acute and chronic effects of TBIs, or for a standard method of TBI assessment to be established. These gaps allow for athletes to return-to-play before a TBI has properly healed, and ultimately leads to subsequent and more severe injuries.

Summary of Literature

Previous research has shown TBIs sustained in heavy contact sports to produce negative neurological effects, with a positive linear relationship among the number of received concussions and severity of effects. TBIs, specifically concussions, are a metabolic rather than structural injury. However, multiple metabolic injuries and neural healing mechanisms have shown to induce structural changes and damage. Brains of former professional football players that have been donated to science have all presented some degree of protein plaques, and many have shown ventricle deformation. Chronic traumatic encephalothapy (CTE) is a neural injury specific to contact sports, and has been observed in athlete donor brains. The chronic nature of non-concussive hits athletes are subjected to have shown to produce cumulative effects, and may potentially lead to a TBI. Once an athlete has sustained a TBI, it is crucial for the injured athlete to completely return to their neuro-metabolic state prior to the concussion before returning to competition. Failure to completely heal the injury is a prominent problem, and often leads to the more grave issue, Second Impact Syndrome. Therefore, it is imperative for medical professionals to be able to deliver a proper and full assessment of TBIs.

The assessments of TBIs conducted by sports medical professionals are often limited to visible symptoms displayed by the injured athlete and neurocognitive tests. Neurocognitive testing provides a means to measure cognitive performance and functioning after a TBI and compare them to test scores obtained prior to the sport season. Results of studies have shown TBIs to have not been healed when concussed subjects appeared symptom-free. There are medical devices that possess the capability to assess the metabolic structure of the brain, but are not deemed feasible to assess sport TBIs due to costs, nature of devices, and medical protocol.

Thesis Statement

Post-TBI assessment would show an acute decrease in motor power output of concussed athletes.

Research Methodology

The study was originally designed as a quasi-experimental design with subjects serving as their own controls examining the effects of TBI on motor power output. However, since only one subject experienced a TBI following baseline assessment, descriptive statistics in the form of percentage change were reported.

Summary of Findings

Data obtained from EMG activity showed that there was a decrease in motor power output at seven days post-TBI; reflecting the general time period which previous research has designated to wait until athletes return-to-play. The TBI subject was operating at less than 30% of baseline electrical nerve signaling for both vastus measurements when tested 24 hours after the injury. Although the subject had significant improvement, he was still only operating at 58-89% seven days after the sustained TBI. There are different mechanisms that may be responsible for the continued decrease of motor power output. Frequency, rate coding, and synchronization are all responsible for the strength of a nerve signal conduction. Data from this study suggest that there may be a retardation or issue with one or multiple mechanism. Other instrumentation used in the study allowed for more variables to be measured: total work performed in ft-lbs, responded similarly to the RMS of electrical data and had not returned to baseline at 7 days post-TBI. The decrease in electrical activity (motor power output) may have contributed to the decrease of total work performed (torque). There seemed to be no noticeable pattern in percent improvements of variables.

Confirmation of Thesis

The hypothesis was supported due to the negative acute effect on motor power output at post-TBI assessment on the concussed participant.

Significance Statement

The results of the current study were similar to previous research which investigated the change of internal variables in regards to TBIs. There was a decrease in motor power output of the TBI subject at 24-hours and 7-days post-TBI. The testing procedures used in the current study may offer future methods of TBI assessment, and ultimately a decrease in premature return-to-play and decreased quality of life.

Future Research

Future studies should be conducted that include more participants to allow for a larger sample size, and should also include multiple sports to ensure testing methods produce similar results among different practices. Studies should also include female athletes to determine any gender differences. Future research should continue to try and determine measureable internal

markers that could be used to assess TBIs, and decrease the prevalence of premature return-to-play and diminished quality of life in contact sport athletes.