

THE LONG TERM EFFECTS OF CONCUSSION ON FINE MOTOR
CONTROL

BY

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THESIS

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ABSTRACT

Concussion has been shown to cause immediate impairment in neurocognitive function and motor control. A widely accepted recovery time from concussion is 7-10 days. However, late life health issues and evidence of chronic inflammatory scar tissue buildup in the brain have been linked to repeated head trauma. In recent electrophysiological studies, concussed individuals have shown differences in EEG profile up to 30 years post-injury. Few studies have examined the long term effects of concussion on persistent brain function. **PURPOSE:** To examine the persisting effects of concussion history on fine motor control. **METHODS:** Twenty individuals (age 20.4 +/-1.7) with no history of concussion and twenty individuals (age 20 +/-1.6) with at least 1 previous episode of concussion, were recruited from a NCAA Division-I football team. Participants each completed a Purdue pegboard task, finger tapping task and a submaximal isometric force control task. Group differences in test performance were assessed using a t-test and a 3-way ANOVA with repeated measures. Correlation analysis was used to assess relationships between test performance and concussion history. **RESULTS:** Data analysis showed no significant group differences between concussed and non-concussed individuals (p 's >0.05) on all measures. No significant relationships were seen between test performance and concussion history. **CONCLUSION:** There was no relation between concussion history and fine motor control performance as evaluated by these tasks. These results contrast the postural control and gait studies which indicated long term changes in concussed individuals. It is unclear why these changes occur, but the presence of sub-concussive blows or greater cortical allocation to fine motor movements are possible explanations. Further study is needed to further understand the long term effects of concussion on fine motor control.

Keywords concussion, long term effects, fine motor control, manual dexterity, football

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CHAPTER 1: INTRODUCTION

BACKGROUND

Concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Concussion results in the rapid onset of short-lived impairment of neurologic symptoms which resolve spontaneously. These acute changes are reflection of a functional injury rather than a structural injury (McCrory, 2009). At the cellular level, head impact causes a cascade of physiological changes in the brain, which result in a severe imbalance between glucose supply and the energy demands needed to restore homeostasis. Metabolic resources are allocated to the inflammatory response to injury and ultimately normal brain functioning is disrupted. As a result, brain function is adversely affected and the brain is unable to respond to further injury (Giza, 2001). Head injury in younger athletes must be treated more conservatively because secondary trauma to a maturing brain can be catastrophic (Guskiewicz, 2004).

Health professionals must realize the significance of concussive brain injury due to its prevalence throughout sport. Concussion rate in the 15 NCAA sports has doubled from 0.17 to 0.34 (per 1000 athlete exposures) from 1988-1989 to 2003-2004 respectively and account for 5% of total injuries during the same period. In high risk contact sports, football and ice hockey concussions accounted for 6% and 7.9% of total concussions respectively (McCrea, 2003). In contact sports such as football, the actual injury rate could be even higher when considering the number of contact exposures (Powell, 1999). More importantly, most of epidemiological figures have only included concussions assessed by a physician or athletic trainer. In reality, the actual incidence of concussion would likely be much higher if unreported concussions were accounted for (McCrea, 2004).

STATEMENT OF THE PROBLEM

There is a large body of research documenting acute deficits associated with concussion. Several clinical neurocognitive tests have been developed that are sensitive to clinical symptoms, memory loss, attention deficits, cognitive processing and executive function (Collins, 1999). Computerized tests allow for further quantitative measurement of neurocognitive function and dysfunction following concussion. These tests have provided evidence that neurocognitive deficits and clinical symptoms typically persist up to and resolve 7 days after initial injury

(Lovell, 2004). In addition, there is also substantial evidence for acute deficits in motor function post-concussion. Quantitative measurements of postural sway using force plate technology have been useful in objectively identifying deficits in postural control in concussed individuals (Guskiewicz, 1996). Concussed subjects have showed an impaired ability to adapt to more challenging balance tasks on an unstable surface or with varied visual stimuli. These findings suggest that brain injury may result in an interruption in sensorimotor integration lasting 3-5 days post-concussion (Guskiewicz, 2001). Concussion subjects also showed impairment in dynamic motor tasks. Concussed individuals displayed more conservative gait pattern when performing simple walking tasks (Parker, 2006). They also showed increased mediolateral sway when neurocognitive symptoms had recovered to baseline after 28 days post-injury (Parker, 2007). This is alarming because this data suggests that the 7-day symptom free recovery period recommended in the NATA Position Statement may not be adequate recovery time before return to play (Guskiewicz, 2004). Some of the current research suggests that residual changes in brain function still persist beyond clinical symptom recovery and that concussion may require a longer recovery time than previously accepted 7-10 days (McCrea, 2003). Therefore, return-to-play guidelines may need to be reconsidered.

Multiple concussive injuries have been linked to late life health issues in retired professional football players. Surveys completed by retired NFL players have indicated a higher prevalence of mild cognitive impairment (Guskiewicz, 2005) and clinical depression (Guskiewicz, 2007) than the general American population. Brain autopsy studies in former NFL players revealed scar tissue (tau protein) deposits indicative of chronic brain inflammation from repeated head trauma (Omalu, 2005) (Omalu, 2007). These papers suggest that residual functional changes in the brain may be related to chronic structural changes in brain tissue.

Electroencephalogram (EEG) testing has provided further insight into the persistent effects of concussion. EEG has provided objective data, documenting changes in brain function in concussed subjects. EEG studies have indicated that resolution of clinical symptoms do not correlate to full brain function recovery (Gosselin, 2006). Concussed subjects between 3 years post-injury (De Beaumont, 2007b) and 30 years post-injury (De Beaumont, 2009) have shown decreased brain activation. Direct analysis of motor cortex function has been also shown to be impaired up to a year post-concussion (De Beaumont, 2007a). Most importantly, EEG profiles seem to be similar between symptomatic and asymptomatic concussed individuals (Gosselin,

2006) and changes are still present 3 years post-concussion when all other clinical tests are normal (Broglia, 2009). However, it is still unclear how these changes in brain function affect behavioral variables; motor function in particular.

HYPOTHESIS

There has been some indication that persisting motor changes exist. Deficits in posture (Thompson, 2005) and fine motor control changes (Slobounov, 2002) have been shown. However further study is needed. Therefore the purpose of this study is to further investigate the long term effects of concussion, focusing on fine motor control changes after concussion. We hypothesize that individuals with a history of concussion will display differences in the fine motor force control tasks. We hypothesize that there will be differences in functional dexterity and tapping speed. We also hypothesize that concussed subjects will display differences in force output variability, indicating an impaired ability to control finger isometric force with concurrent visual feedback.

CHAPTER 2: LITERATURE REVIEW

DEFINITION AND EPIDEMIOLOGY OF CONCUSSION

The most recent definition of concussion has been agreed as “a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces” (McCory, 2009). Concussion results in the rapid onset of neurologic symptoms which resolve spontaneously and reflect a functional disturbance rather than a structural injury. The consensus statement proposed an abandonment of the classification of simple and complex concussions because of the complex nature and high variability of the condition. McCory et al. suggested that evaluation of concussion should be based on a combination of symptom assessment, neurocognitive tests and motor tests (McCory, 2009).

Concussion is important due to its prevalence throughout competitive sports at all levels. Powell et al. (1999) reported that the highest incidence rate of concussion in high school sports was in football, followed by wrestling and soccer. In the injury report data that was collected from 246 high school athletic trainers, concussion accounted for 5.5% of total reported injuries over the 3 year study-period. This study reported 0.59 concussions per 1000 exposures in football. However, this only accounted for reported concussions identified by an athletic trainer so the actual number of concussions may likely have been higher had unreported concussions been considered (Powell, 1999).

McCrea et al. (2004) investigated the significance of unreported concussions in high school football. Concussion history data was collected directly from high school football players as opposed to athletic trainers. Concussion history questionnaires were distributed to football players from 20 high schools. They were asked to report history of concussion prior to the current season and any concussions sustained from the current season. The results of the study showed that 15.3% of respondents sustained a concussion during the football season. However, only 47.3% of these individuals actually reported the injury to an athletic trainer, coach or parents. This data suggests that the actual prevalence of concussion was higher than originally reported by Powell et al. (1999), who only included concussions assessed by a health professional (McCrea, 2004).

Guskiewicz et al. (2000) compared the incidence of concussion in NCAA and high school football. Athletic trainers from 242 schools completed a concussion report after each concussion sustained during the season. The results were compiled and an average concussion

rate was further broken down into number of contact exposures. Contact exposures included full contact practices or competitions, excluding walk-through practices where there was little to no risk of head injury. The concussion incidence rate in NCAA division I, II and III was 0.94, 1.34 and 1.31 injuries per 1000 contact exposures respectively. The most notable finding was concussion incidence at the high school level was 1.63 per 1000 contact exposures, which was the highest among all levels. This is important to note because the high school football population includes the largest number of athletes at the highest risk of concussion. Another finding of this study was that football players were at 3 times higher risk of sustaining recurrent concussions in the same season (Guskiewicz, 2000).

Pellman et al. (2004) conducted a retrospective study using data collected from the National Football League Injury Surveillance System to determine the incidence of concussion from NFL games. Concussion data from NFL games from 1996 – 2001 showed 787 reported cases. The average incidence of concussion was 131.2 concussions per year and 0.41 concussions per game. The players that were most at risk of concussion were quarterbacks, wide receivers and defensive backs who were likely to sustain high-velocity impacts, especially from tackling or being tackled (Pellman, 2004).

NEUROMETABOLIC CASCADE

Giza et al. (2001) outlined the neurometabolic cascade of concussive brain injury. The shearing forces on neural tissue in the brain cause damage and an immediate inflammatory response. Ion channels indiscriminately open following trauma, causing an immediate ion imbalance and an unstable membrane potential. The sodium-potassium pump is activated to restore the neural membrane potential. ATP is required for the active transport of these ions, thus increasing metabolic demand. In addition, vasoconstriction in the brain as part of the inflammatory response causes a severe energy supply-demand imbalance. The metabolic effects of concussion at the cellular level likely persist beyond the accepted ‘recovery of symptoms’ and return to play guidelines. Giza et al. suggested that this mechanism is the cause for increased vulnerability to further concussions (Giza, 2001).

SYMPTOMS

Concussion results in symptoms that fall into a several domains: clinical symptoms,

physical signs, behavior, balance, sleep and cognition. (McCrory, 2009). Clinicians rely on self-reported subjective symptoms to assess impairment and severity of injury. The Head Injury Scale is a clinical evaluation tool used to rate the severity of symptoms post-concussion. Piland et al. evaluated the efficacy of the Head Injury Scale (HIS) in identifying neuropsychological, cognitive and somatic deficits. By grouping interrelated symptoms in to this 3-domain model, Piland et al. hypothesized that impairment in specific brain processes may be identified. When the HIS was given to concussed college athletes, a moderate correlation was found between symptom scores and the 3-domain model. This suggested that it was a useful tool for identifying neurocognitive changes post-concussion. In addition, content validity analysis allowed for redundant items on the HIS to be removed so that the 16-item checklist was reduced to just 9-items (Piland, 2003). Piland et al. conducted a further study to evaluate the efficacy of another concussion symptom assessment tool, the Graded Symptom Checklist (GSC). The same 3-domain model was applied to the GSC results from concussed high school athletes. A similar reduced 9-item GSC checklist was derived from the original 16-items on the GSC by removing redundant items from the list. These 9-items provided a good representation of the 3-domain model. The 9-item GSC identified somatic (headache, nausea, balance impairment), neurobehavioral (abnormal sleep, drowsiness, fatigue) and cognitive (feeling slowed down, in a fog and difficulty concentrating) changes post-concussion (Piland, 2006).

ACUTE NEUROCOGNITIVE EFFECTS

In addition to symptoms, specific assessment tools are used to assess neurocognitive changes after concussion. The acute effects of concussion have been well documented throughout the literature. Both clinical neurocognitive test protocols (Collins, 1999) and computerized testing programs (Lovell, 2004) have been used to identify specific areas of neurocognitive impairment.

Collins et al. (1999) used the NFL neurocognitive test battery to investigate the acute effects of concussion on neurocognitive performance in college football players. The testing protocol was designed to identify deficits in verbal learning, memory, executive functioning, word fluency, visual processing, fine motor skills and concussion symptoms. Four NCAA division I football teams were baseline tested and players who sustained a concussion during the season were re-evaluated using the same test protocol within 24 hours and days 3, 5 and 7 post-

injury. The most pronounced deficits in concussion subjects were verbal learning and memory, especially during the first 24 hours post-injury. In the subsequent recovery days, concussion subjects displayed a much slower learning effect across all tests compared to controls. Another finding was that subjects with learning disabilities and history of 2 or more concussions displayed similar baseline scores, which were significantly lower than the group average. This may have indicated perhaps some link between learning changes and multiple concussions (Collins, 1999).

Lovell et al. (2004) used ImPACT, a computer based neurocognitive test, to assess acute cognitive symptoms and recovery post-concussion in high school athletes. Forty three high school athletes were baseline tested using ImPACT and re-assessed at 36 hours and 6 days post-concussion. The mean composite scores from ImPACT were significantly lower for all subjects at 36 hours post-concussion, indicating neurocognitive impairment. Memory deficits and increased symptom scores were the most pronounced acute changes post-concussion. All scores returned to baseline by day 6. Lovell et al. (2004) questioned the same day return to play guidelines from the American Academy of Neurology (AAN) and American Orthopedic Society for Sports Medicine (AOSSM), which recommend same day return to play if clinical concussion symptoms resolve within 15 min. All subjects in this study experienced symptom resolution within 15 min and still showed cognitive impairment when assessed using ImPACT. This indicates that athletes could be at risk of more severe brain injury by returning to activity before full recovery. The current accepted recovery time for acute neurocognitive changes is 7 days (Lovell, 2004).

ACUTE POSTURAL EFFECTS

Recent literature has indicated that concussion results in acute postural control changes (Guskiewicz, 1996) (Guskiewicz, 2001). Postural control assessment is included in sideline clinical tools such as the Standardized Assessment of Concussion (SAC) and the NATA position statement recommends that balance tests should normal before athletes are cleared to return to play (Guskiewicz, 2004).

Guskiewicz et al. (1996) used force platforms to quantitatively measure acute changes in postural sway and center of balance in concussed subjects. Ten high school and college athletes were baseline tested and returned for follow up testing at 1, 3, 5 and 10 days post-injury and

follow up 1 month postseason. Subjects completed a balance protocol using the Chattecx Balance System with varied visual (eyes open/closed) and surface (stable/unstable) conditions. The results from this study provided quantitative evidence for postural control impairment up to 3 to 5 days post-concussion. Impairment was more pronounced in more difficult balance tasks where visual and proprioceptive sensory stimuli were altered (Guskiewicz, 1996).

In a further study, Guskiewicz et al. (2001) showed similar acute postural changes post-concussion using the sensory organization test (SOT) and balance error scoring system (BESS). A similar test schedule was used where subjects were baseline tested and re-evaluated 1, 3 and 5 days post-concussion. Subjects performed the SOT using the NeuroCom Smart Balance Master System where fluctuations in center of gravity were measured during different surface and visual conditions. The BESS is a clinical sideline balance test that was performed under different stance, surface and visual conditions. Higher the number of errors during the BESS indicated more balance impairment. Both the SOT and BESS were sensitive to acute changes in postural control during day 1 post-concussion and returned to baseline by day 3. Despite return to baseline, concussion subjects still performed significantly worse in both tests compared to healthy controls even after day 5. In clinical practice, return to baseline is sufficient recovery for return to play (Guskiewicz, 2001).

McCrea et al. (2003) confirmed the 3 to 5 day recovery in postural control post-concussion. College football players were assessed for balance changes using the BESS during the first 3 hours post-injury and again at 1, 2, 3, 5, 7 and 90 days post-concussion. All subjects recovered to baseline within 3 to 5 days, which was consistent with the findings from Guskiewicz et al. (2001). This study confirmed that the BESS is a useful sideline evaluation tool and has shown to accurately identify postural control deficits consistent with force plate assessment techniques performed in the lab. The current accepted recovery time for postural control is 3 to 5 days (Guskiewicz, 1996) (Guskiewicz, 2001) (McCrea, 2003).

ACUTE GAIT EFFECTS

Acute changes in gait have been documented throughout the literature. However, current return-to-play guidelines do not include specific assessment of gait. The graduated return to play protocol is more for athletic trainers to assess any deterioration in clinical symptoms rather than assess motor changes (McCrary, 2009).

Parker et al. (2006) showed acute changes in dynamic motor tasks in concussed subjects. Fifteen college athletes who sustained a grade-2 concussion, completed a walking gait test along a straight walkway under single and dual task conditions. The single task condition involved just walking, whereas the dual task condition involved walking and simultaneously performing simple arithmetic, spelling words and reciting months backward. The differentiation between the 2 conditions was to show how divided attention affected gait. Gait patterns were observed and recorded using a motion tracking camera system. Subjects were tested at 2, 5, 14 and 28 days post-concussion and were compared to matched healthy controls. The results showed that the concussion group adopted a more conservative gait pattern than the control group. A shorter stride length and slower walking speed was evident especially in the more challenging dual task at 14 and 28 days respectively. The concussion group consistently performed more conservatively in the dual task, whereas the control group showed a learning effect to the dual task by day 5. Mediolateral sway was significantly increased at 2 and 5 days and returned to normal 14 days before increasing again at 28 days. Parker et al. attributed this finding increased exertion from return to play after day 14, causing a possibly deterioration in symptoms. The increased sway at day 28 may have been an indication that recovery was not complete. This is alarming because subjects showed increased instability after being cleared to return to activity (Parker, 2006).

In a further study, Parker et al. (2007) investigated dynamic motor changes post-concussion while concurrently documenting neurocognitive recovery. A sample of 29 college athletes who sustained a grade-2 concussion performed the same single task and dual task gait walking protocol as the previously mentioned study (Parker, 2006). In addition, subjects also completed ImPACT testing to evaluate acute neurocognitive changes post-concussion. Subjects were assessed at intervals 2, 5, 14 and 28 days post-concussion. All neurocognitive domains (reaction time, visual processing, memory and symptoms) returned to normal after day 5. The most notable finding from the gait analysis was that mediolateral sway was increased in the concussion group even after day 28. No deterioration in ImPACT scores were seen at day 28. This was consistent with the previous study by Parker et al. and indicates that cognitive and motor function recovery may resolve differently (Parker, 2007).

Catena et al. further investigated the effects of concussion dynamic motor tasks by using a more complex dual task gait protocol. The gait testing protocol used included a similar gait

testing protocol to Parker et al. (2007) with level walking and a cognitive dual task condition. The addition of an obstacle condition required subjects to cross obstacles along the walkway. Testing was conducted at similar intervals to Parker et al. at 2, 6, 14 and 28 days post-concussion. The most notable finding was that conservative gait patterns were different at different stages of recovery in the concussion group. During the initial recovery phase at day 2, the cognitive task elicited a conservative reduction in anterior-posterior motion. However, during the later stages of recovery at day 28, the obstacle task instead elicited conservative medio-lateral motion. These results indicated that cognitive attention tasks are more sensitive to impairment during the acute phase of concussion recovery and obstacle crossing tasks are sensitive to impairment during the later stages of concussion recovery (Catena, 2009).

LONG-TERM EFFECTS OF CONCUSSION ON HEALTH

Recent literature has linked concussion has been to late life deterioration in cognitive functioning. Guskiewicz et al. (2005) conducted a retrospective study, which linked mild cognitive impairment (MCI) to concussion in retired football players. A sample of retired NFL players completed a general concussion history survey and a MCI assessment instrument. The results of the study revealed a significant correlation between history of recurrent concussions and MCI. The most common issue was a high rate of memory problems. Severity ranged from mild memory loss to more severe symptoms pertaining to Alzheimer's disease. This sample of retired football players included in this study had a higher prevalence of Alzheimer's disease compared to the general American population. These results indicated that the memory impairment seen acutely after concussion may have cumulative effects causing late life changes (Guskiewicz, 2005).

In a further study, Guskiewicz et al. (2007) investigated the prevalence of clinical depression in a similar sample of retired NFL players. Eleven percent of all respondents to the survey reported at least one episode of depression, which was consistent with the general prevalence in the general American population. However, individuals with a history of 3 or more of concussions displayed a three-fold increased prevalence of depression. Guskiewicz et al. (2007) suggested that there was some link between cognitive changes from multiple concussions that caused an increased risk of clinical depression (Guskiewicz, 2007).

Although concussion has been agreed to be a functional injury rather than a structural

injury (McCrary, 2009), some studies have shown evidence for chronic structural changes in the brain. Omalu et al. (2005 & 2006) performed two brain autopsy examination case studies on retired NFL players. Both subjects were offensive lineman who played extensively in college and at the professional level. At the time of autopsy, subjects were several years retired from playing football and had long concussion free playing careers. In both cases, cause of death was not directly related to traumatic brain injury. The most notable finding consistent in both cases was the presence of tau-positive neurofibrillary tangles and neuropil threads. These were protein scar tissue deposits that were indicative Chronic Traumatic Encephalopathy (CTE), possibly from repeated brain microtrauma during football career. One subject was diagnosed with clinical depression following retirement, whereas the other subject experienced no neuropsychological pathology. Yet, autopsy revealed similar findings. With no significant concussion history in both subjects, these findings suggest that repeated sub-concussive head trauma could be a factor in causing these structural changes (Omalu, 2005 & 2006).

LONG TERM EFFECTS OF CONCUSSION ON NEUROCOGNITIVE FUNCTION

Current literature and return to play guidelines suggest that concussed individuals recover from neurocognitive deficits within 7 days. However, this is dependent on the sensitivity of neurocognitive tests. Broglio et al. evaluated the sensitivity of ImPACT concussion assessment software to detect cognitive impairment in athletes while asymptomatic. Athletes were baseline tested before participation and tested twice post-injury; once while symptomatic and once later in recovery phase when they were asymptomatic. ImPACT composite scores revealed that 35% of subjects displayed cognitive impairment during the asymptomatic recovery phase. This suggested that the absence of concussion symptoms was not an indication for full recovery of cognitive function. Broglio et al. concluded that athletes, who are cleared to return to play based solely on symptom based assessment, may put themselves at risk of further injury. In addition, normal ImPACT scores may not necessarily indicate full recovery from concussion (Broglio, 2007).

In a further study, Broglio et al. (2009) evaluated the sensitivity of ImPACT testing in subjects who were 3 years post-injury. No long term impairment was seen in ImPACT scores in concussed subjects and no differences were found when ImPACT performance was compared to a group of non-concussed subjects. These findings indicate that although ImPACT testing may be

useful in detecting acute cognitive impairment, it may not be sensitive to long-term impairment (Broglia, 2009).

Electroencephalogram (EEG) testing has shown to be useful in identifying residual changes in neurocognitive functioning. Gosselin et al. (2006) used EEG together with clinical tests to assess attention changes in asymptomatic concussed athletes. Professional and university level hockey, football and soccer athletes who had sustained concussion were divided into 2 groups based on presence of concussion symptoms assessed using the Post Concussion Symptom (PCS) scale. A control group was comprised of tennis and volleyball athletes for comparison. Subjects performed a modified auditory Oddball paradigm where they were asked to respond to certain auditory stimuli by pressing a key while EEG activity was monitored. The results showed no group differences in task performance but differences in EEG profile. The 2 concussion groups displayed a similar EEG profile, which were significantly lower EEG amplitudes compared to the control group. The EEG data from this study suggest that functional changes were present in concussed subjects despite no impairment in clinical testing. In addition, both symptomatic and asymptomatic concussed subjects displayed similar EEG profile, indicating that functional changes may still be present upon recovery of symptoms (Gosselin, 2006).

De Beaumont et al. further compared event-related potential (ERP) measurements in concussed subjects who were 3 years post concussion. An Oddball paradigm similar to Gosselin et al. (2006) was performed while EEG activity was monitored and neurocognitive impairment was concurrently assessed. All concussed subjects were asymptomatic and divided into a multiple concussion and single concussion group. Subjects with a minimum of 9 months since their last concussion were included and the average time since last concussion was about 3 years. The control group and both concussion groups displayed no differences in neurocognitive testing. However, subjects with multiple concussions displayed suppressed ERP responses during the Oddball task. This indicates some functional deficits in information processing exist in individuals with multiple concussions (De Beaumont, 2007b).

Broglia et al. (2009) confirmed the findings from De Beaumont et al. (2007b) in a similar study measuring ERP during the visual Oddball task while also evaluating neurocognitive function using ImPACT. Concussed subjects who were an average of 3 years post-concussion were compared to control subjects. No group differences were seen in neurocognitive symptoms from ImPACT scores. However ERP amplitude was significantly lower in the concussed group.

Although clinical assessments were normal, ERP decrements revealed some residual functional differences in concussed subjects. Broglio et al. (2009) concluded that long term deficits may exist even only after one previous episode of concussion.

LONG TERM EFFECTS OF CONCUSSION ON MOTOR FUNCTION

While several studies indicating that concussion causes long term changes in brain information processing, few studies have investigated the long term effects of concussion on motor function. De Beaumont et al. (2007a) performed a study investigating the effects of concussion on the motor cortex. Subjects were divided into 3 groups; multiple concussions, single concussion and control. Concussed subjects were no less than 9 months from their last concussion. The motor cortex was stimulated using Transcranial Magnetic Stimulation (TMS) to elicit involuntary muscle contraction of the dorsal interosseous muscle in the index finger. The Cortical Silent Period (CSP), the time between stimulation of the cortex and resulting muscle EMG activity, was compared between groups. The results showed a prolonged CSP in concussed subjects, which meant a longer time from stimulus to muscle contraction. The difference in CSP was more pronounced in individuals with a greater number of concussions, supporting previous literature on the cumulative effects of concussion (Guskiewicz, 2003). The findings indicate a functional impairment of the motor cortex causing a delay in muscle activation (De Beaumont, 2007a).

De Beaumont et al. (2009) repeated their previous measurements of cognitive processing and motor activation in older adults who were former university hockey and football players. The average time from concussion was more than 30 years for all subjects. Previous findings were confirmed in reduced ERP amplitude during the Oddball task and increased CSP during motor cortex stimulation. These findings confirm that there may be chronic brain function changes following concussion (De Beaumont, 2009).

Despite the fact that chronic changes in brain function have been documented, most studies show normal performance in neurocognitive clinical tests. However, some studies have indicated that some residual motor changes may persist. Slobounov et al. (2002) investigated the effects of concussion using a fine motor finger task. A group of concussed college athletes, 10-20 months post-injury, performed a fine motor task while concurrent ERPs were measured. The task involved pushing a load cell with their dominant index finger. Visual feedback was given on a

computer screen and subjects were asked to accurately match the force trace to a target line. The concussion group showed significantly more variability in force production when asked to perform a more demanding, higher-force output task. This impairment was concurrently seen with lower amplitude in Movement-Related Potentials (MRP). These findings indicate concussed subjects display reduction in brain activation and impaired fine motor control (Slobounov, 2002).

Thompson et al. (2005) studied EEG patterns in asymptomatic concussed subjects during postural tasks. Subjects were an average of 3 months post-concussion and were college athletes who were cleared for sport participation. EEG activity was recorded while subjects performed open and closed eyed, standing and sitting tasks. The results showed overall lower EEG amplitude across the spectrum in the concussed group, especially during standing tasks, indicating decreased brain activation. In addition, the concussed group showed a greater fluctuation in center of pressure during the more difficult closed eyed, standing condition. These findings suggest there may be increased postural instability in concussed individuals that persist beyond the acute recovery phase. EEG data suggest some impairment in processing of visual, vestibular and somatosensory information in the brain (Thompson, 2005).

Cavanaugh et al. (2006) investigated a technique that could be used to show persisting differences in postural sway from otherwise normal scores from the sensory organization test (SOT). Concussed collegiate athletes completed the SOT during the day 1 and day 3 post-injury. All subjects returned to normal by day 3 according to SOT scores. Cavanaugh et al. performed further analysis using approximate entropy (ApEn) to quantify center of pressure (COP) oscillations from the SOT data. ApEn analysis revealed that COP oscillations did not return to baseline within the same time frame, indicating residual changes in postural control. These results indicate that normal SOT scores are not an absolute indication of postural control recovery and that impairment may persist beyond the accepted 3-5 day recovery period (Cavanaugh, 2006).

CHAPTER 3: METHODOLOGY

SUBJECTS

A total of 40 male subjects were recruited from the University of Illinois varsity football team during the 2009-2010 football season. Subjects were divided in half, into a concussion group and a control group. The concussion group consisted of 20 subjects with at least one self-reported concussion. The control group consisted of 20 subjects with no self-reported history of concussion. All subjects were free from any acute upper body injury or acute head injury that would have affected motor performance. Approval was obtained from the University of Illinois Institutional Review Board prior to testing. Support from the head football coach and head football athletic trainer had been obtained prior to recruitment of subjects. A formal letter was sent to both parties explaining the non-invasive nature of the study and benefits of contributing to new knowledge in the study of concussion (see APPENDIX A). Participants were recruited by the Graduate Assistant Athletic Trainer (co-investigator).

PROTOCOL

Each of the 40 participants in the study followed the same protocol. The investigator tested each subject in a 1 on 1 basis in a quiet, distraction free environment. Subjects were seated comfortably at a desk during testing. At the beginning of each test session, subjects completed a health history questionnaire. The fine motor tasks were performed in the following order: peg board trial 1, tapping test trial 1, peg board trial 2, tapping test trial 2, peg board trial 3, tapping test trial 3, index finger abduction MVC (3 trials), isometric force control test (36 trials).

Health history questionnaire: A custom screening instrument written by the investigators was completed by each subject. Subjects were asked about history of previous head injury, the nature of each injury and how it was managed. The definition of concussion for the purpose of this study was a traumatic head injury assessed by a physician or athletic trainer which caused the athlete to be removed from activity. This criteria was explained to each subject as they completed the questionnaire.

Purdue peg board: This was a test to assess manual dexterity. The Purdue peg board is a wooden board with 2 lines of holes aligned down the middle of the board. Twenty metal pegs were distributed evenly in 5 pockets on the top side of the board. At the beginning of the test,

subjects were instructed to start with their right hand face down on a marked space on the board. On command from the investigator, subjects were instructed to replace the metal pegs one at a time into the holes going down the left side of the board. Subjects were given 30 seconds to place as many pegs in the holes as they could and the total number of pegs correctly placed was recorded. This task was repeated 3 times and subjects were encouraged to beat their previous score each time.

Finger Tapping test: This was a test of finger speed. A laptop computer keyboard and an open word processing document was used. Subjects were instructed to start with their hand face down flat on the computer keyboard with their right index finger on the spacebar. On command from the investigator, subjects were instructed to tap the spacebar as fast as possible. Subjects performed 30 second trials and the total number of spacebar taps was recorded for each trial. This task was repeated 3 times and subjects were encouraged to beat their previous score each time.

Force control test: This was a test of isometric force control in the finger. A custom apparatus was built to assess finger abduction. A load cell was setup to measure and record force production. The load cell was connected to a Coulbourn Instruments analog to digital converter box, which was connected to a laptop computer. Data was collected using a custom protocol in LabView software. Prior to testing subjects were instructed to place their right hand into the appropriate finger slots and the concept of the load cell was explained. The investigator explained to subjects that the tasks would involve application of pressure on the load cell by index finger abduction. They were instructed to make contact with the load cell using the distal interphalangeal joint of their index finger. Finger abduction was used to isolate the dorsal interossei muscle - a seldomly used muscle. This was to avoid variability in fine motor skill due to experience playing musical instruments or computer keyboard use. On the force control board, subjects performed 2 tasks:

- Maximum voluntary contraction (MVC): Subjects were instructed on cue to apply maximum isometric pressure on the load cell by performing right index finger abduction. Subjects were instructed to apply and maintain maximum pressure for 6 seconds. Online visual feedback in the form of a force trace was displayed on the computer screen at gain 4 pixels/N. The MVC test was repeated 3 times and the highest value was used for subsequent testing.

- Submaximal force control task: Subjects were instructed on cue to apply constant pressure on the load cell for 20 seconds. Online visual feedback was given on a computer screen in the form of a force trace and a target line. Subjects were instructed to adjust the amount of force during each trial so that the force trace accurately stayed as close to the target line as possible. The target line was set at either 5% or 30% of the subject's highest MVC. The investigator randomly adjusted the gain and % MVC for each trial. The gain was varied between 4, 16, 64, 128, 256 and 512 pixels/N. Two levels of MVC, 6 levels of gain each repeated 3 times made a total of 36 randomly assigned conditions.

DATA ANALYSIS

The total scores for each Purdue peg board and tapping trial were recorded. Each of the 3 MVC trials was recorded in newtons (N). The isometric force data was sampled and coefficient of variance was calculated. A t-test analysis was used to compare group differences in Purdue peg board and tapping test performance. A 3-way (group x MVC x gain) ANOVA was used to compare group differences between isometric force control performance. Correlation analysis was used to analyze relationships between isometric force control performance and concussion history. Descriptive statistics were used to analyze all other data collected from the health history questionnaire. All data analysis was completed using SPSS statistical package version 17 (SPSS, Inc; Chicago, IL). Analyses were considered significant when $p < 0.05$.

CHAPTER 4: MANUSCRIPT

ABSTRACT

Concussion has been shown to cause immediate impairment in neurocognitive function and motor control. A widely accepted recovery time from concussion is 7-10 days. However, late life health issues and evidence of chronic inflammatory scar tissue buildup in the brain have been linked to repeated head trauma. In recent electrophysiological studies, concussed individuals have shown differences in EEG profile up to 30 years post-injury. Few studies have examined the long term effects of concussion on persistent brain function. **PURPOSE:** To examine the persisting effects of concussion history on fine motor control. **METHODS:** Twenty individuals (age 20.4 +/-1.7) with history of concussion and twenty individuals (age 20 +/-1.6) with at least 1 previous episode of concussion, were recruited from a NCAA Division-I football team. Participants each completed a Purdue pegboard task, finger tapping task and a submaximal force control task. Group differences in test performance were assessed using a t-test and a 3-way ANOVA with repeated measures. Correlation analysis was used to assess relationships between test performance and concussion history. **RESULTS:** Data analysis showed no significant group differences between concussed and non-concussed individuals (p 's >0.05) on all measures. No significant relationships were seen between test performance and concussion history. **CONCLUSION:** There was no relation between concussion history and fine motor control performance as evaluated by these tasks. These results contrast the postural control and gait studies which indicated long term changes in concussed individuals. It is unclear why these changes occur, but the presence of sub-concussive blows or greater cortical allocation to fine motor movements are possible explanations. Further study is needed to further understand the long term effects of concussion on fine motor control.

Keywords concussion, fine motor control, dexterity, football

INTRODUCTION

Concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Concussion results in the rapid onset of short-lived neurologic impairment that resolves spontaneously. These acute changes are a disruption of brain function, rather than structural injury (McCory, 2009). At the cellular level, head trauma causes a cascade of physiological changes in the brain, which result in a severe imbalance between

glucose supply and the energy demand needed to restore homeostasis. Metabolic resources are allocated to the inflammatory response to injury and ultimately normal brain functioning is disrupted. As a result, brain function is adversely affected and is not able to respond to further injury (Giza, 2001).

Health professionals must realize the significance of concussive brain injury due to its prevalence. Concussion rate in the 15 NCAA sports has doubled from 0.17 to 0.34 (per 1000 athlete exposures) from 1988-1989 to 2003-2004 respectively and account for 5% of total injuries during the same period. In the high risk contact sports football and ice hockey, concussions accounted for 6% and 7.9% of total injuries respectively (McCrea, 2003). In contact sports such as football, the actual injury rate could be even higher when considering the number of contact exposures (Powell, 1999). Most importantly, most of epidemiological figures have only included concussions reported to physician or athletic trainer. In reality, the actual incidence of concussion is likely much higher if unreported concussions were accounted for (McCrea, 2004).

There is a large body of research documenting acute deficits associated with concussion. Several clinical neurocognitive tests have been developed that are sensitive to clinical symptoms, memory loss, attention deficits, cognitive processing and executive function (Collins, 1999). Computerized tests allow for further quantitative measurement of neurocognitive function and dysfunction following concussion. These tests have provided evidence that neurocognitive deficits and clinical symptoms typically persist up to 7 days after initial injury (Lovell, 2004). In addition, there is also substantial evidence for deficits in motor function. Quantitative measures of postural sway using force plate technology have been useful in objectively identifying deficits in postural control in concussed individuals (Guskiewicz, 1996). Concussed subjects have shown an impaired ability to adapt to more challenging balance tasks on an unstable surface or with varied visual stimuli. These findings suggest that brain injury may result in an interruption in sensorimotor integration utilized in postural control 3-5 days post-concussion (Guskiewicz, 2001). Concussion subjects also showed impairment in simple dynamic motor tasks. Concussed individuals display a more conservative gait pattern when performing simple walking tasks (Parker, 2006). They also continue to show increased mediolateral sway following neurocognitive and symptoms resolution 28 days post-injury (Parker, 2007). This is alarming because current NATA Position Statement return-to-play guidelines suggest a 7 day symptom

free recovery period, followed by a gradual exercise progression (Guskiewicz, 2004). The current research suggests that concussion recovery may require a longer recovery time than previously accepted 7-10 days (McCrea, 2003). Therefore, return-to-play guidelines may need to be reconsidered.

Further evidence suggest persistent effects of concussion can be seen with electroencephalogram (EEG) testing that has provided objective data of differences in brain function. EEG studies have indicated that recovery of symptoms does not correlate to recovery of brain function (Gosselin, 2006). Concussed subjects between 1 year post-injury (De Beaumont, 2007b) and 30 years post-injury (De Beaumont, 2009) have shown differences in EEG profile and motor cortex function (De Beaumont, 2007a) compared to non-concussed controls. The study of EEG profiles in concussed individuals shows that there may be residual long lasting functional deficits. However, current neurocognitive and motor function testing has shown a lack of sensitivity to these long lasting deficits. There has been some indication to persisting functional deficits in posture (Thompson, 2005) and fine motor control (Slobounov, 2002), however further study is needed. Therefore the purpose of this study is to further investigate the long term effects of concussion on fine motor control.

METHODS

A total of 40 male subjects were recruited from the University of Illinois varsity football team during the 2009-2010 football season. Subjects were divided into a concussion group and a control group. The concussion group consisted of 20 subjects with at least one self-reported concussion. The control group consisted of 20 subjects with no self-reported history of concussion. All subjects were free from any acute upper body injury or acute head injury that would have affected motor performance. Approval was obtained from the University of Illinois Institutional Review Board prior to testing and all participants provided written informed consent.

Each of the 40 participants in the study followed the same protocol. The investigator tested each subject in a 1-on-1 basis in a quiet, distraction free environment. Subjects were seated comfortably at a desk during testing. At the beginning of each test session, subjects completed a health history questionnaire. The fine motor tasks were performed in the following order: peg board trial 1, tapping test trial 1, peg board trial 2, tapping test trial 2, peg board trial

3, tapping test trial 3, force control MVC (3 trials), isometric force control test (36 trials).

Health history questionnaire: A custom screening instrument written by the investigators was administered to the subjects asking about history of head injury, the nature of each injury and how it was managed. The definition of concussion for the purpose of this study was a traumatic head injury diagnosed by a physician or athletic trainer which caused an athlete to be removed from activity. This criteria was explained to each subject as they complete the questionnaire.

Purdue peg board: To assess manual dexterity the Purdue peg board was administered. This test uses a wooden board with 2 lines of holes aligned down the middle of the board. Twenty metal pegs were distributed evenly in 5 pockets on the top side of the board. At the beginning of the test, subjects were instructed to start with their right hand face down on a marked space on the board. On command from the investigator, subjects were instructed to replace the metal pegs one at a time into the holes going down the left side of the board. Subjects were given 30 seconds to place as many pegs in the holes as they could and the total number of pegs correctly placed was recorded. This task was repeated 3 times and subjects were encouraged to beat their previous score each time.

Finger Tapping test: This was a test of finger speed. A laptop computer keyboard and an open word processing document was used. Subjects were instructed to start with their hand face down flat on the computer keyboard with their right index finger on the spacebar. On command from the investigator, subjects were instructed to tap the spacebar as fast as they could. Subjects were given 30 seconds and the total number of spacebar taps was recorded. This task was repeated 3 times and subjects were encouraged to beat their previous score each time. spacebar was tapped in each trial will be recorded.

Force control test: This was a test of isometric force control in the finger. A custom apparatus was built for finger abduction testing. A load cell was setup to measure and record force production. The load cell was connected to a Coulbourn Instruments analog to digital converter box, which was connected to a laptop computer. Data was collected using a custom LabView software protocol. Prior to testing subjects were instructed to place their right hand into the finger slots and the concept of the load cell was explained. The investigator explained to subjects that the tasks would involve application of pressure on the load cell by index finger abduction. They instructed to make contact with the load cell using the distal interphalangeal

joint in their index finger. Finger abduction was used to isolate the dorsal interossei muscle, a seldomly used muscle. This was to avoid influence variability in fine motor skill due to experience playing musical instruments or computer keyboard use. On the force control board, subjects performed 2 tasks:

Maximum voluntary contraction (MVC): Subjects were instructed on cue to apply maximum isometric pressure on the load cell by performing right index finger abduction. Subjects were instructed to apply and maintain maximum pressure for 6 seconds. Online visual feedback in the form of a force trace was displayed on the screen at gain 4 pixels/N. MVC was performed 3 times.

Submaximal force control task: Subjects were instructed on cue to apply constant pressure on the load cell for 20 seconds. Online visual feedback was given on a computer screen in the form of a force trace and a target line. Subjects were instructed to adjust the amount of force during each trial so that the force trace accurately stayed as close to the target line as possible. The target line was set at either 5% or 30% of the subject's highest MVC. The investigator randomly adjusted the gain and % MVC for each trial. The gain was varied between 4, 16, 64, 128, 256 and 512 pixels/N. Two levels of MVC, 6 levels of gain each repeated 3 times made a total of 36 randomly assigned conditions.

DATA ANALYSIS

The total scores for each Purdue peg board and tapping trial were recorded. Force generated during each of the 3 MVC trials was recorded in newtons (N). The isometric force data was sampled and coefficient of variance was calculated. Separate independent samples t-test analyses were used to compare group differences in Purdue peg board and tapping test performance. A 3-way (group x MVC x gain) ANOVA was used to compare group differences between isometric force control performance. Correlation analysis was used to analyze relationships between isometric force control performance and concussion history. Descriptive statistics were used to analyze all other data collected from the health history questionnaire. All data analysis was completed using SPSS statistical package version 17 (SPSS, Inc; Chicago, IL). Analyses were considered significant when $p < 0.05$.

RESULTS

A total of 41 subjects were recruited, 1 subject was excluded from final data due to a previous finger injury which prevented him from performing the force control task. Data from 40 subjects was collected and used for final analysis. Subjects were 20.2 (+/-1.5) years of age with an average of 9.7 (+/-3.4) years of football experience. The average height and weight for the sample was 187.5 (+/-5.8) cm and 107.9 (+/-20.1) kg. The concussion group sustained an average of 1.5 (+/-0.7) previous concussions and were 20.4 (+/-24.3) months post the most recent injury. A summary of demographic information can be found in table I in APPENDIX B.

T-test analysis of Purdue peg board [$t(38)=1.15$, $p=0.26$] and finger tapping task [$t(38)=0.37$, $p=0.71$] performance revealed no significant group differences. Three-way ANOVA (group x MVC x gain) with repeated measures on MVC and gain showed no differences in the force control task between groups for mean force output, standard deviation and coefficient of variance (see chart I and II in APPENDIX B). No significant correlation was found between coefficient of variance and time from last concussion across all conditions (see tables II-VII in APPENDIX B). The strongest correlations were seen in the 5%MVC/4pixels/N and 30%MVC/512pixels/N conditions with r-values 0.44 ($p=0.06$) and -0.42 ($p=0.07$) respectively. Although these r-values indicate a weak-moderate correlation, these were not statistically significant at the 95% confidence interval. No significant correlations were found between coefficient of variance and number of concussions. The strongest correlation was seen at the 5% MVC/512 pixels/s condition with an r-value -0.26 ($p=0.27$). No significant correlations were found between coefficient of variance and football playing experience. The strongest correlation was at the 5% MVC/256 pixels/s condition with an r-value of -0.25 ($p=0.12$).

DISCUSSION

The findings from this study failed to support our hypothesis that subjects with history of concussion would perform differently in fine motor tasks compared to subjects with no history of concussion. No differences were found between groups in the maximum number of pegs replaced in the Purdue peg board or the maximum number of spacebar taps in 30 seconds. There were no differences between groups in standard deviation or coefficient of variability in index finger force control across all MVC and gain conditions. No significant correlation was found between finger force control variability and both time post-concussion and football playing

experience.

These findings indicate one of several possibilities: First, the tasks performed in this study may not have been sensitive to fine motor control changes in concussed individuals. Second, if fine motor control deficits do result from blows to the head, multiple sub-concussive impacts may have a similar effect on motor control as diagnosed concussions. Third, concussions and potentially sub-concussive blows to the head may not manifest deficits in fine motor control.

Although not statistically significant, there does appear to be some relationship between time from injury and isometric force control variability. The 5% MVC/4 pixels/N condition displayed a positive correlation and the 30% MVC/512 pixels/N condition displayed a negative correlation. These results suggest that concussed individuals may display acute changes in fine motor control after injury, which gradually recover over time. However, further study is needed to document the recovery curve for the finger control task used in this study.

The findings from our study do not match those reported by Slobounov et al. (2002) who used a similar finger force control protocol evaluate differences in motor performance in concussed individuals 10-20 months post-injury. Subjects were instructed to apply gradual pressure on the load cell until the force trace reached a target line. Similar to our study, Slobounov et al. (2002) used 2 force level; 25% and 50% MVC, but reported that the concussed group showed significant impairment in the accuracy of force development during the ramp phase in the more challenging 50% MVC condition. In our investigation, we placed emphasis on constant isometric force output, whereas Slobounov et al. focused on the ramp phase, which involved concentric muscle contraction as opposed to an isometric contraction. In addition, Slobounov et al. found significantly reduced amplitude in movement-related potentials (MRP) during the 50% MVC task. This indicated that motor performance differences were related to central nervous system (CNS) impairment (Slobounov, 2002). No EEG data was collected in our study, so it was unknown whether our subjects had similar CNS impairment despite no differences in motor performance.

Our findings also do not support the findings from Sosnoff et al. (2010) who showed persisting differences in postural control in concussion subjects who were at least 6 months post-injury. Concussed and non-concussed athletes were tested using the Sensory Organization Test (SOT) and performance was evaluated using approximate entropy analysis. Sosnoff et al. found

differences in postural dynamics (sway) although functional postural performance (SOT score) was normal (Sosnoff, 2010). Most notably, concussed individuals displayed differences in postural sway irregularity in the anterior-posterior direction (Sosnoff, 2010). The lack of findings seen in our study may have been because the motor tasks used were only sensitive to functional differences as opposed to similar subtle differences seen by Sosnoff et al.

Martini et al. reported gait differences in previously concussed individuals. Individuals with history of concussion displayed a more conservative gait pattern, particularly during a more difficult dual-task condition (Martini, 2010). The gait protocol used by Martini et al. incorporated a challenging cognitive task sensitive to subtle differences between concussed and non-concussed individuals. The difficulty level in the motor tasks used in this study may not have been challenging enough to display subtle group differences. Incorporation of a cognitive dual task or decision making component would have increased difficulty and perhaps increased sensitivity to group differences.

The lack of fine motor differences in this study could possibly be explained by the relationship between motor cortex activation and motor output. The motor cortex has traditionally been divided into 4 divisions: the primary motor cortex, the supplementary motor area, the premotor cortex and the cingulate motor area. Although their specific functions overlap, it is the interaction of these different parts of the motor cortex that allow movement to take place (Roland, 1996). Both fine motor and gross motor movements involve the same parts of the motor cortex, but the difference is in the level of activation (Roland, 1996). Gross motor tasks such as postural control and gait involve a large number of muscles and cognitive processing of sensory feedback, therefore greater activation of the motor cortex (Roland, 1996). The scar tissue build up following concussion (Omalu, 2007) could impede normal motor cortex function, causing increased processing time and delayed muscle activation, as shown by De Beaumont et al. (2007a). The lack of fine motor differences noted in this investigation may have been the result of brain activation at levels below the threshold where behavioral differences were evident. Slobounov et al. (2002) showed decreased EEG amplitude while concussed subjects performed a fine motor task. Further, the motor cortex area allocated to the lower extremity is much smaller than that of the hands. Thus, fewer secondary pathways may be available to compensate for scar tissue resulting from concussion or sub-concussive blows. As a result, we see the differences displayed in gross motor tasks, such as postural control (Sosnoff, 2010) and gait

(Martini, 2010), but not fine motor evaluations.

Darling et al. (2009) investigated the effects of surgically induced motor cortex lesions on fine motor recovery using animal models. The most notable finding from this study was the duration of fine motor impairment was strongly correlated to the volume of white matter lesions. This indicates that the degree of white matter damage can be used as a predictor of expected fine motor recovery. Darling et al. (2009) also indicated that injury to the frontal lobe may produce more motor deficits due to proximity to the motor cortex. The nature of concussive injuries are so variable that specific parts of the brain that are affected are different in each individual. The degree of damage to motor cortex white matter in our sample is unclear. The concussed subjects used in this study may not have had any white matter damage from concussion. However, the subjects studied by Slobounov et al. (2002) did show fine motor differences, which may have been related to some white matter damage. However, the involvement of motor cortex white matter remains unclear, as no imaging or autopsy was performed with either group.

A primary limitation to this study is lack of a control group. The subjects used in this study were all NCAA Division-I football players and have had exposure to contact sport. Contribution of unreported concussions and sub-concussive blows may cause this population to differ from other populations who are not involved in contact sport. A control group consisting of normal sedentary individuals or non-contact athletes (e.g. tennis, track & field) would be useful in identifying whether participating in contact sport causes changes in fine motor function. Ultimately, this would provide greater insight into the effects of sub-concussive head trauma over time.

CONCLUSION

In conclusion, we have demonstrated no long lasting fine motor differences between concussed and non-concussed college football players. No differences were shown in Purdue pegboard, tapping or force control performance. These findings indicate that no differences were shown in dexterity, finger speed or isometric finger control. Our study did not reproduce isotonic finger control differences shown by Slobounov et al. (2002) and did not support the postural and gait findings of Sosnoff et al. (2010) and Martini et al. (2010). Regardless, there are clear changes to some aspects of motor control that persist far beyond resolution of all clinical symptoms. An extension of this study would be to increase the difficulty of motor tasks by

introducing a dual-task decision making or cognitive processing condition. This would require a greater amount of brain resource and perhaps cause concussed individuals to show differences in performance. In addition, direct measurement of brain activation during motor tasks would show underlying differences in brain function that may not be evident by observing motor performance. The use of EEG and ERPs would be useful in measuring brain activation to assess changes in motor cortex function consistent with the findings from Slobounov et al. (2002).

APPENDIX A – SUPPLEMENTAL DOCUMENTS

LETTER TO COACH AND HEAD FOOTBALL ATHLETIC TRAINER

I would like to ask your support for a concussion research study using University of Illinois Football players.

This is a master's thesis research study investigating the long term effects of concussion on fine motor skills. A brief medical history will be collected on each subject regarding previous head injury. Privacy of information is assured according to Institutional Review Board standards. Individual names will not be linked to data.

Testing will be non-invasive. Each subject will be asked to perform 3 simple tasks:

- Purdue peg board: Subjects will be tested on manual dexterity. Subjects will be instructed to replace pegs back into their appropriate slot as fast as they can in a given amount of time. The number of correctly replaced pegs will be recorded.
- Tapping: Subjects will be tested on finger tapping speed. Subjects will be instructed to tap a button as many times as they can during each trial. Number of repetitions will be recorded.
- Force production: Subjects will be tested on isometric force control in the index finger. Subjects will be instructed to push on and maintain pressure on a digital pressure sensor. Variability in force output will be calculated from the recorded force output data.

Subjects will be given the choice not to participate or withdraw from testing at any time. The anticipated testing time for each subject will be about 30 min. Data collection will take place during the summer session and 2009 season.

There are few previously published studies which investigate concussion and fine motor control. This study will contribute new knowledge to this subject area and a better understanding of the long term effects of concussion. More importantly this can further improve guidelines for management of concussion and return to activity.

Thank you for your attention.

Sincerely,

Callum Chia, ATC
Graduate Assistant Athletic Trainer

HEALTH HISTORY QUESTIONNAIRE

Age _____ Height (in) _____ Weight (lbs) _____

Dominant Hand (R/L) _____

Primary Position: _____

Secondary Position _____

How many years have you played football?

Are you currently being treated for or experiencing concussion related symptoms?

Yes No

Are you currently being treated for an injury to the hand, arm, or shoulder?

Yes No

Have you ever been diagnosed with a concussion by a medical professional (Doctor, Athletic Trainer, EMT)

Yes No

If yes how many? _____

	Date of Injury (approximate)	Length of symptoms (days)	Number of days missed (practices and games)
Injury 1			
Injury 2			
Injury 3			

APPENDIX B – DATA TABLES AND CHARTS

	Concussed		Non-Concussed		All Participants	
		<i>sd</i>		<i>sd</i>		<i>sd</i>
Age (years)	20.4	1.7	20.0	1.3	20.2	1.5
Height (in)	74.2	1.7	73.4	2.5	73.8	2.3
Weight (lb)	240.4	39.6	235.3	49.4	237.8	44.3
Football Experience (years)	11.4	2.6	7.9	2.6	9.7	3.4
# of Concussions	1.5	0.7	n/a		1.5	0.7
# Months From Last Injury	20.4	24.3	n/a		20.4	24.3

Table I: Demographics

	5% mvc Correlation vs. Time (n=20)					
Gain (pixels/N)	4	16	64	128	256	512
Correlation	0.44	0.12	0.18	0.01	-0.01	0.07
p-Value	0.06	0.62	0.46	0.95	0.96	0.78

Table II: Correlation between 5% MVC Co-variance vs. Time from Last Concussion

	30% mvc Correlation vs. Time (n=20)					
Gain (pixels/N)	4	16	64	128	256	512
Correlation	0.14	0.05	-0.13	-0.15	-0.10	-0.42
p-Value	0.56	0.83	0.60	0.52	0.67	0.07

Table III: Correlation between 30% MVC Co-variance vs. Time from Last Concussion

	5% mvc Correlation vs. # of Concussions (n=20)					
Gain (pixels/N)	4	16	64	128	256	512
Correlation	0.00	-0.09	-0.19	-0.01	-0.21	-0.26
p-Value	0.99	0.69	0.42	0.97	0.38	0.27

Table IV: Correlation between 5% MVC Co-variance vs. Number of Previous Concussions

	30% mvc Correlation vs. # of Concussions (n=20)					
Gain (pixels/N)	4	16	64	128	256	512
Correlation	-0.05	0.06	0.13	-0.02	-0.06	-0.08
p-Value	0.84	0.79	0.60	0.95	0.80	0.75

Table V: Correlation between 30% MVC Co-variance vs. Number of Previous Concussions

	5% mvc Correlation vs. Experience (n=40)					
Gain (pixels/N)	4	16	64	128	256	512
Correlation	-0.06	0.21	-0.01	0.03	-0.25	0.07
p-Value	0.69	0.18	0.96	0.85	0.12	0.65

Table VI: Correlation between 5% MVC Co-variance vs. Football Playing Experience

	30% mvc Correlation vs. Experience (n=40)					
Gain (pixels/N)	4	16	64	128	256	512
Correlation	0.19	0.08	0.16	0.11	0.04	0.04
p-Value	0.25	0.60	0.32	0.51	0.79	0.79

Table VII: Correlation between 30% MVC Co-variance vs. Football Playing Experience

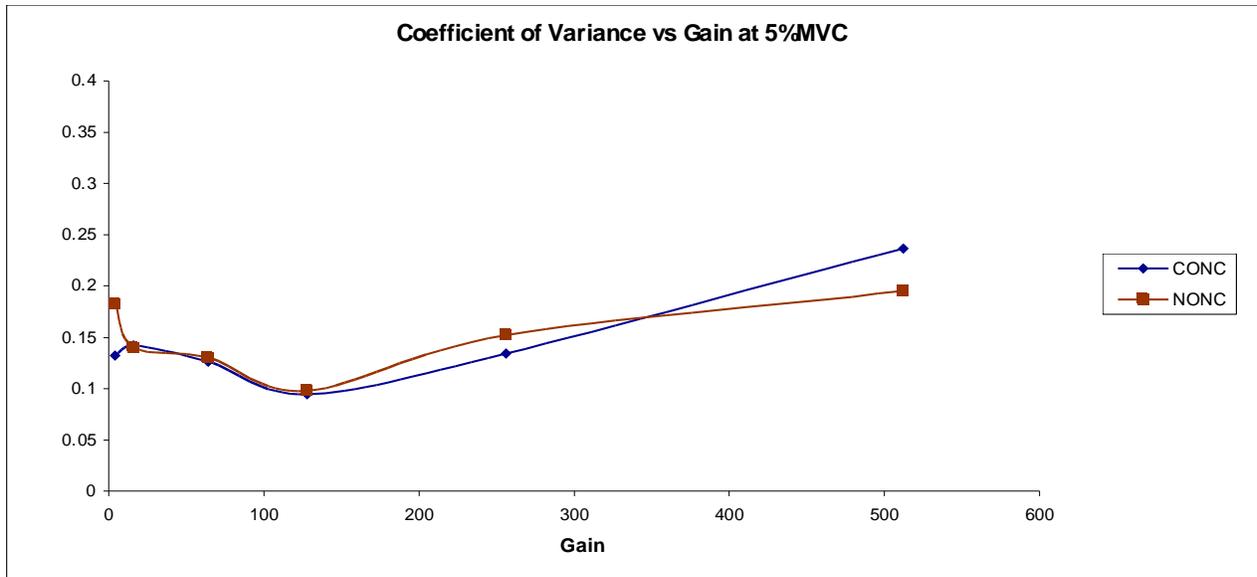


Chart I: Force control task coefficient of variance vs. gain at 5% MVC. CONC = concussed group and NONC = non-concussed group.

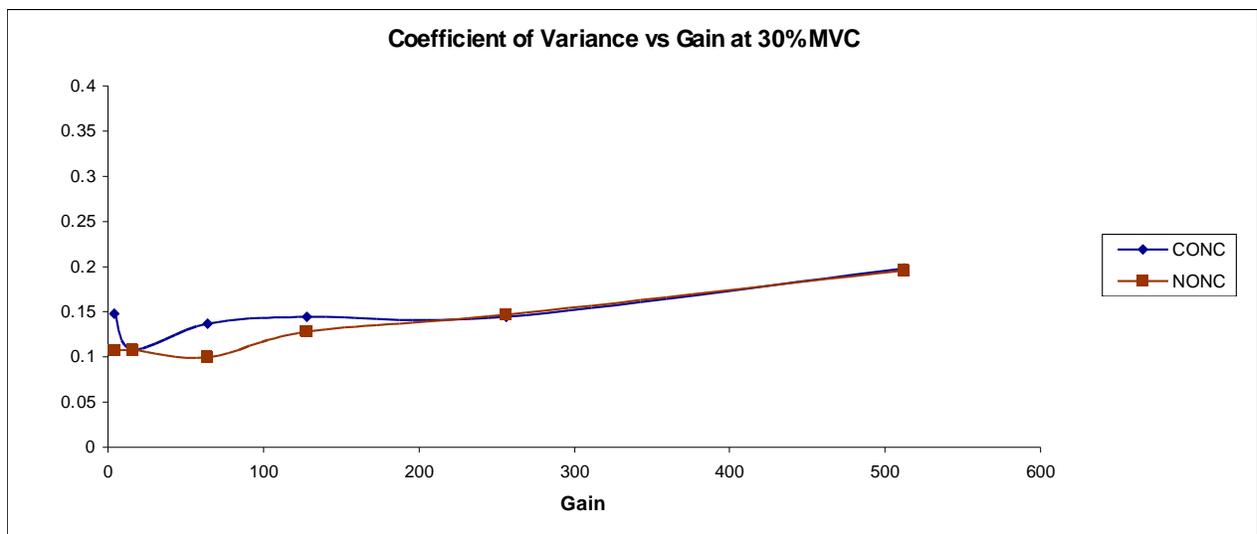


Chart II: Force control task coefficient of variance vs. gain at 30% MVC. CONC = concussed group and NONC = non-concussed group.

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