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Baseline Postural Control Measures: An Indicator for Increased Injury Frequency Following Sports Related Concussion

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BASELINE POSTURAL CONTROL MEASURES: AN INDICATOR FOR INCREASED INJURY FREQUENCY FOLLOWING SPORTS RELATED CONCUSSION

by

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ABSTRACT

INTRODUCTION: An increase in acute lower extremity (LE) injuries have been observed in athletes following sports-related concussion.[1, 2, 3] It has been suggested that lingering postural control deficits as a result of concussive injury, may play a role in the increased prevalence of injury.[1] **PURPOSE:** To investigate the relationship between baseline postural control metrics (Root Mean Square; Peak Excursion Velocity; Sample Entropy) and acute LE injury frequency in NCAA Division I student athletes (SA) with a previous history of concussion. **METHODS:** Eighty-four NCAA Division I SA were selected from a single university, 42 SA with a previous history of concussion (CONC) and 42 without as the control group (CTRL). Baseline postural control assessment, measured via force platform (Sample frequency 1000Hz), and medical charts were retrospectively reviewed. Postural control assessment consisted of three trials of eyes-open (EO) and eyes-closed (EC) quiet stance for duration of 30 sec. Center of pressure data was used to quantify peak excursion velocity (PEV), root mean square (RMS) and sample entropy (SampEn) in the anterior-posterior (AP) and medial-lateral (ML) direction. Medical records were assessed for all acute LE injuries sustained one-year following baseline postural control assessments. **RESULTS:** Chi squared analysis revealed a significant increase in frequency ($p = 0.025$) of acute LE injuries within

CONC (22/49 = 44.9%) in comparison to CTRL (10/44 = 22.7%). Paired sample t-test demonstrated a significant decreased in EC PEV AP (p = 0.006) of the CONC group (0.063 ± 0.025) compared to CTRL (0.078 ± 0.038) with moderate effect (*Cohen's d* = 0.487), but lacked significance in all other conditions. While the logistic regression model lacked overall significance (p = 0.379), participant group (B = 1.302, P = 0.033) and EO SampEn AP (B = -6.086, P = 0.062) were significant predictors for acute LE injury. **CONCLUSION:** The results of this study suggest that SA with a previous history of concussion do have a higher acute LE injury frequency than those without a history of concussion. And variations in baseline postural control assessments may help to identify this increase in frequency

INDEX WORDS: Postural control, Sports related concussion, Acute lower extremity injury

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DEDICATION

I dedicate this project to my family and friends, near and far. For without their constant encouragement, reassurance, and much needed mental breaks, this project would not have been completed. To my mom, Kathy, thank you for listening to me on the phone for hours and being the kick in the butt that I often needed. To my dad, Mike, thank you for somehow knowing exactly when I was in need of a caffeine boost and for reminding me what an amazing opportunity I have been given here. To my sister, Abby, for encouraging me to take to the outdoors when I was feeling stressed and reminding me to have fun every once in a while. To Jesse, I don't thank you often enough for your unbelievable support and I am so appreciative of how patient and understanding you have been throughout this process. To Maggie and Brett, your friendship means the world to me and I beyond grateful for the influence you have had in my life. Finally, to Katelyn, Megan, Nichole, and Colleen, I can never thank you enough for your love, support, and laughter. You made me feel at home at a time when I was very far away from my own, and for that I am forever grateful.

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CHAPTER 1: INTRODUCTION

CHAPTER 1.1 EPIDEMIOLOGY

Understanding the pathology and lasting effects behind concussive injuries have moved to the forefront of research within the sports medicine community. Concussions are defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces[4]. Approximately 1.6 - 3.8 million concussions occur annually within the United States.[5] A multitude of transient symptoms occur following concussive injury including neurological deficits, cognitive impairments, and postural control alterations[6]. While assessing and managing these acute symptoms is a primary concern of clinicians, research suggests the presence of long term alterations that could potentially affect the athletic population once they have been returned to play.

CHAPTER 1.2 POSTURAL CONTROL

Several studies have identified lingering postural control deficits within collegiate athletes following sports related concussions. Postural control has been defined as the act of maintaining a state of balance during any posture or activity.[7] The ability to maintain postural control requires the integration of afferent information from the visual, vestibular, and somatosensory system to the central nervous system.[8] This information is processed within the cerebellum to produce modulated signals to descending neurological pathways, which stimulate skeletal muscle contractions. This system of communication between afferent sensory input from the extremities and modulation of efferent signal to skeletal muscles allows for the maintenance of upright human posture.[9]

Communication between these two systems is maintained by feed-forward and feedback control mechanisms. In order to make adjustments in response to external stimuli the brain relies on a feedback control mechanism. This often results in an oscillating motion as the brain either overestimates or underestimates the desired response. In comparison, a feed-forward control mechanism makes anticipatory adjustments from incoming sensory information in order to reach a desires outcome.[9] The cerebellum utilizes both feed-forward and feedback control mechanisms in order to maintain upright human posture.

Diminished or compromised feedback from any of these systems demands more reliance on the remaining systems to provide more sensory feedback. For example, closing one's eyes while maintaining a static upright posture places more demand on the vestibular and somatosensory systems. This results in increased postural sway (i.e., movement while performing an upright stance) to compensate for the loss of the visual system and to provide more somatosensory input. Following a concussive injury there is a functional damage to the brain and its neurological pathways that cause a disruption in the central integration of afferent information from the visual, vestibular, and somatosensory systems.[10] Lack of afferent sensory information from proprioceptors or inappropriate feedback responses form the cerebellum my lead to over compensatory or less fluid skeletal muscle responses.[11] Therefore, it is common to observe variations in postural control and a lack of balance once an athlete has sustained a concussion.[12]

A variety of methods are used in order to assess postural control deficits following sports related concussions. Within the clinical setting, postural control deficits have traditionally been measured using the Balance Error Scoring System (BESS).[13] The

BESS provides a cost efficient and portable method of balance assessment that is readily available to clinicians for sideline use[13]. However, this assessment relies heavily on the clinician's interpretations of errors resulting in potential inconsistencies. Education and training of clinicians performing and scoring the BESS also plays into effect the reliability of scores. In addition, the BESS has demonstrated to only be reliable for detecting changes in postural control up to 7 days following injury. The BESS is designed for the clinician to detect large postural responses (i.e. touching a foot down, falling out of a stance, large abduction of the hip) in comparison to laboratory assessments, which identify minute postural changes. Therefore, the BESS may not be the most appropriate and sensitive measures for which to base return to play decisions [14]

In comparison to clinical assessments, laboratory measures provide a more sensitive assessment of balance. Typically, laboratory assessments utilize force plate metrics (e.g. position, displacement, acceleration and velocity) to identify variations in static posture thus providing clinicians with an objective measure of postural control.[15] Force plate assessments typically evaluate the center of pressure (COP) variable, which has been identified as a valid and objective measure for postural control.[16] Center of pressure, a linear metric, is a measure of the vertical ground reaction forces and moves in relation to alterations in postural stability to account for changes in the center of gravity.[16,17] Following a concussive injury there is an increase in both the excursion and excursion velocity of COP in comparison to healthy controls.[12, 18] This increase in COP velocity occurs as a reflection of the anticipatory adjustments that are being made in order to account for the increase in COP displacement. Furthermore, the individual’s speed of movement increases as they attempt to correct for their inability to maintain

postural stability. Whereas excursion is a first-order variable controlled primarily by somatosensory feedback, excursion velocity requires the integration of both the visual and vestibular system. Following a concussion, variations in COP excursion between concussed and control populations tend to resolve faster than variations in excursion velocity.[12] This suggests that while the somatosensory system is recovering, the integration of the visual and vestibular system may still be impaired.

The use of linear force plate metrics have demonstrated to be successful in identifying postural control alterations following concussive injury. However, the use of non-linear metrics may provide a more robust and sensitive measure for detecting postural deficits long after a concussive injury has occurred.[19] Sample entropy (SampEn) is a non-linear force plate metric that quantifies the amount of randomness, or irregularity, contained in a time series. SampEn is particularity suited for short, noisy biological output signals associated with human movement therefore making it a valid measure for postural control assessment.[20] Entropy values are represented on a scale of zero to infinity, ranging from high regularity to high randomness, respectively. Following a concussive injury, there is an increase in postural control regularity, suggesting that the individual is attempting to limit their degrees of freedom in order to maintain control over their balance.[20] Healthy individuals tend to demonstrate a certain amount of irregularity within their time series and maintain entropy values around one. Cavanaugh et al. were able to identify increased regularity within the concussed population using entropy measures when no changes in COP measures observed.[20] This observation suggests that the use of nonlinear metrics such as entropy, provide an improved method for

longitudinally detecting alterations in postural control within the concussed population in comparison to linear metrics.[19]

CHAPTER 1.3 LONG TERM EFFECTS

Previous studies conducted using force plate analysis have identified lingering postural control deficits in student athletes that had sustained a sports related concussion more than nine months prior to testing.[19, 21] The athletic environment requires significant neuromuscular control and coordination; therefore the presence of lingering postural control changes may be detrimental to the student athlete's performance. These findings have led to more recent research evaluating whether or not lingering postural control deficits may have an effect on the rate of acute lower extremity (LE) injuries within a previously concussed population.

CHAPTER 1.4 ACUTE LOWER EXTREMITY INJURIES

Within a year following a concussive injury, athletes are twice as likely to sustain an acute (LE) injury in comparison to matched controls.[2, 3] Increased injury rates have been associated with acute injuries (i.e., ligament sprains and muscle strains) but not gradual onset injuries (i.e., tendiopathies, stress fractures).[3] It has been suggested by Nordstrom and Lynall that this increase in acute (LE) injuries may be the result of lingering postural control deficits that are affecting the neuromuscular control of student athletes who have previously sustained a concussion.[2, 3] However, at this time no current research exists to directly assess the impact that postural control deficits have on student athletes once they have been returned to play.

STATEMENT OF PURPOSE

While an increase in (LE) injury rates have been identified in a previously concussed population, the mechanism behind this phenomenon has yet to be thoroughly assessed. Therefore, the purpose of this study was to: (1) Identify variations in baseline postural control measures between collegiate athletes who have a previous history of concussion and those who do not, and (2) determine if baseline postural control measures may be used as an indicator for increase acute LE injury frequency within a previously concussed population. We hypothesized that student athletes with a previous history of concussion would have an increased acute LE injury frequency in comparison to those without. In addition, we hypothesized that significant differences in baseline postural control assessments would be observed between student athletes with a previous history of concussion and those without. And lastly, that there would be a significant relationship between acute LE injury frequency and baseline postural control measures. This may allow clinicians in the future to use baseline postural control assessments to identify student athletes who are at higher risk for sustaining an LE injury as a result of previous concussion.

CHAPTER 2: METHODS

CHAPTER 2.1 PARTICIPANTS

Student athletes (SA) from a single National Collegiate Athletic Association (NCAA) Division I university were used as participants for this study. Student athletes were defined as a participant, aged 18-25 years, within a university sanctioned competitive sport, which is sponsored by the educational institution in which he or she is enrolled. The participants were divided into two groups (1) previous history of concussion, and (2) no previous history of concussion. Previous history of concussion was determined from the SA's pre-participation exam form (Figure 1).

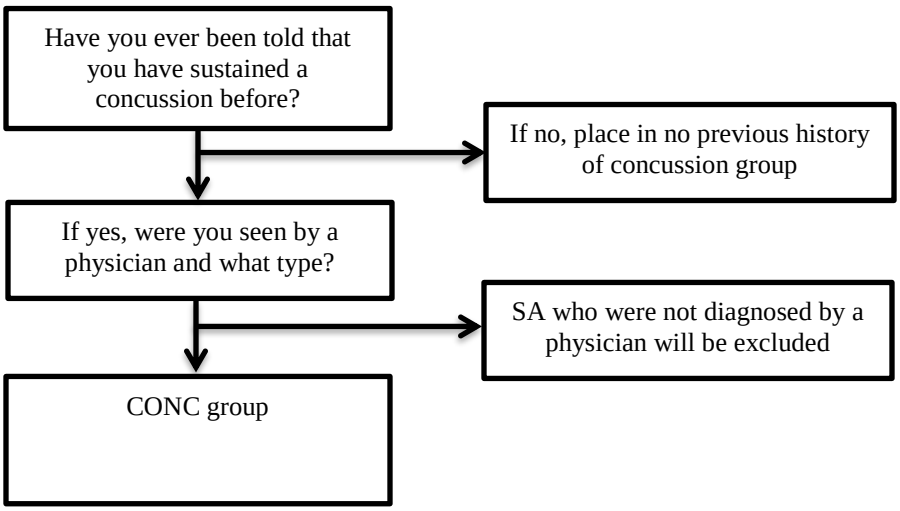

Figure 1. Inclusion criteria progression based on patient pre-participation exams; CONC = previous history of concussion

SA who reported no history of concussion on their pre-participation exam but later sustained a concussion while at Georgia Southern University were included within the previous history of concussion (CONC) group if their medical chart included full documentation of the injury by the supervising Athletic Trainer (ATC) and the concussion was diagnosed by the team physician.

The no previous history of concussion (CTRL) group must have had no documented history of concussive injury on their pre-participation exam form. If the SA later sustained a concussion during the injury surveillance period following their baseline assessment, they were either excluded from the study or moved to the CONC group if they fulfill the inclusion criteria stated above. Control participants were matched to participants within the CONC group based on age, gender, sport, and skill set. Subjects for which a matched control could not be found were excluded.

Baseline postural control data that has been previously collected for ongoing research was used in this study. Both SA within the CONC and CTRL groups must have had baseline postural control data to be included within this study. Furthermore, SA who sustained a concussion while at Georgia Southern, were placed within the CONC group so long as they performed a re-baseline for postural control assessment following return to play.

Table 1. Criteria for Inclusion and Exclusion for Study Participants

Inclusion	Exclusion
- Complete and available paper medical chart - Baseline postural control data - CONC: Previous history of concussion documented by MD - CTRL: No previous history of concussion	- Incomplete or unavailable medical chart - Missing baseline postural control data - Documented vestibular, metabolic, or neurologic pathology or severe lower extremity injury that has permanently effected the patient's ability to perform a static upright stance. - Documented chronic injury

One-hundred and sixteen athletes met the initial criteria to be included in this study. Of the 106 subject, 22 subjects (9 CONC; 13 CTRL) were identified as outliers

based on their postural control data; new matched controls were found for those controls excluded. Outliers were excluded in order to account for any errors that may have occurred during data collection (i.e. stepping off the plate, not zeroing out the force plate, or administration errors). An additional seven participants were removed for chronic injuries. Sport, gender, injury history, and time from last concussion to baseline testing was reported (Table 2).

Table 2. Participant demographics of CONC and CTRL following exclusion of outliers and participants with chronic injuries[a]

Group	Sport	Injury	Time from CONC to BL
Concussion	Football (n = 19)	Acute Injury (n = 15)	< 6 mo (n = 7)
	Cheerleading (n= 5)	No Injury (n = 27)	6 – 12 mo (n = 9)
	Women's Soccer (n = 5)		> 12 mo (n = 26)
	Baseball (n = 4)		
	Men's Soccer (n = 4)		
	Men's Basketball (n = 4)		
	Volleyball (n = 1)		
Control	Male (n = 31)	Acute Injury (n = 8)	
	Female (n = 11)	No Injury (n = 34)	

[a] Forty-two student athletes with a previous history of concussion were matched to 42 healthy control based on gender, sport, and skill set; CONC = concussion; BL = baseline; mo = months; Injury count is representative of the number of student athletes who did or did not sustain an injury, not total number of injuries sustained

CHAPTER 2.1 INSTRUMENTATION

Postural Control

Ground reaction forces were collected using a 40 cm (length) x 60 cm (width) in-ground AMTI force platform (1000 Hz, AMTI OR-6 Series, Watertown, MA, USA). Center of Pressure (COP) data was calculated from the ground reaction forces recorded from the force platform.

Medical Chart Documentation

All medical charts were retrospectively reviewed for the use of this study. University standardized pre-participation exams were reviewed for patient inclusion criteria (Figure 1). A coded identifier (Appendix C) list was utilized in order to de-identify all personal health information collected by the researcher. A standardized data collection form (Appendix C) was used in order to record all information from the participant's medical charts to be utilized for analysis.

CHAPTER 2.3 VARIABLES

All variables examined in this study were derived from the raw COP data obtained from the force platform. A custom MATLAB code was used to further analyze all variables. Previously, linear measures (e.g., Root Mean Square and Peak Excursion Velocity) have been used to detect postural control deficits in a CONC population.[12] Recently, it has been suggested that non-linear measures (e.g., SampEn) may provide a more robust and time sensitive assessment to detect these deficits.[20, 22] Therefore both measures were assessed in this study.

Root Mean Square

Root mean square (RMS) is defined as the standard deviation of the displacement of the COP.[23] RMS measures the average absolute displacement around the mean COP. The algorithm used to calculate RMS_{AP} is shown in Eq. (1):

RMS_{AP} =

$$\frac{1}{\mathrm{N}}\sum[\,AP(n)^2]^{1/2}$$

Peak Excursion Velocity

Peak Excursion Velocity (PEV) represents the average of the instantaneous velocity. The algorithm used to calculate PEV_{AP} is shown in Eq. (2):

PEV_{AP}=

$$\int_{n}^{n+1}\frac{1}{fs}(AP[n+1]-AP[n])$$

The maintenance of excursion velocity requires the integration of both the visual and vestibular system, therefore providing a more accurate representation of the connection between the motor and neural mechanism of postural control[8]. The measure of velocity is comprised of both the direction and magnitude of the COP characterizing it as a second order variable. This measure may be further utilized as an indicator for the anticipatory movements made by the neurological system in order to control muscular contractions.

Sample Entropy

Sample Entropy (SampEn) was used to measure the regularity of the COP movements observed during the trial periods. SampEn represents the negative natural

logarithm of the time series and estimates the probability of the COP excursion to remain in a similar spot from one time point to the next. Measures of SampEn range from zero to infinity, where higher values represent increased randomness within the time series and values approaching zero reflect a highly regular, predictable time series. The algorithm for SampEn is denoted in Eq. (3)

$SampEn_{AP}$ (m,r,n)=

$$-ln \sum_{i=1}^{n-m\cdot r} Ai \,/\, \sum_{i=1}^{n-m\cdot r} Bi$$

Where *m* was the length of compared runs (m = 2), r is the tolerance window length between 2 adjacent points in a time series (*r* = 0.2*SD), and *n* was the number of samples during the CoP time series (i.e. number of data points). A lag of 100 was applied in order to smooth the data, therefore reducing the sampling frequency to 10 Hz.[24] SampEn was selected over other forms of entropy measures (i.e. approximate entropy) because it has demonstrated to be a more reliable and consistent method for the analysis of biological time series.[19]

CHAPTER 2.4 PROCEDURES

Baseline Postural Control Assessment

Baseline postural control data was obtained from an ongoing investigation performed at Georgia Southern University. Baseline assessments are performed on all SA entering the university (i.e., freshman or transfer student-athletes). Participants performed three trials of both an eyes-open (EO) and eyes-closed (EC) quiet stance for a duration of 30 seconds per trial. The International Society for Posture and Gait Research has suggested that a 25-40 seconds collection period has demonstrated to be a reliable

duration for postural control measures without the effects of fatigue on the subject.[25] The participants were instructed to place their feet together with their hands on their hips during both conditions. Participants perform the quiet stance while standing on a Wii Balance Board (WBB) (Nintendo Corporation, Redmond, WA, USA) that has been place atop of the force platform. While the Wii balance board was placed on the force platform during data collection it was not used for the purposes of this study. The height of the board was factored into the equation used to calculate center of pressure. During the EO trials, the participants were instructed to look at a fixed point within the center of a projection screen 1.4 m away from the force platform.

Medical Chart Review

Paper medical charts stored on Georgia Southern University's campus were cross referenced with baseline postural control data. All SA medical charts with baseline postural control data were retrospectively reviewed for concussion history and acute LE injuries. Charts were assessed for all recorded injuries (i.e acute and chronic) that were sustained within 1-year following baseline assessment in order to capture all four seasons (i.e., pre-season, in-season, post-season, off-season). Previous literature has utilized a similar surveillance period for recording injury frequency within this population.[2] Charts were sorted into either the CONC or CTRL groups based on the inclusion criteria stated above. Demographic information for all charts were recorded. In addition, the date of the SA's most recent concussion was recorded in order to calculate the time span between injury and baseline assessment. SA within the CONC group were further categorized into three groups based on time span (i.e., <6mo, 6-12mo, >12mo form last concussion to

baseline assessment) for later evaluation of the influence of time to baseline assessment on postural control measures.

Injury records were obtained from Georgia Southern University sports medicine paper medical charts. Both acute and chronic LE injuries were recorded for this study. Acute injuries were defined as 'a muscle strain, ligament sprain, or noncontact fracture or dislocation to the foot, ankle, lower leg, knee, thigh, or hip complex' that occurred during sports-related activities. Chronic injuries were defined as 'stress fractures, bursitis, or tendonitis of the foot, ankle, lower leg, knee, thigh, or hip complex. Other injuries that did not fit either category such as contusions, abrasions, and lacerations were not recorded within the injury surveillance period. Only acute injuries were statistically analyzed within this study in order to demonstrate the effects of altered postural control on motor coordination in an athletic environment.[2] Chronic injuries were noted solely for exclusion criteria, and therefore were not statistically analyzed. Overall acute LE injury frequency was recorded for each SA in both the CONC and the CTRL groups. In addition, all injury data was grouped according to location (e.g., hip, groin, thigh, knee, lower leg, ankle, foot) and type (e.g., acute fracture, muscle strain/tear, ligament sprain/rupture) in order to provide demographic information regarding the injuries sustained.

CHAPTER 2.5 DATA ANALYSIS

Force Plate Measures

Raw analog data collected from the AMTI force platform was amplified by the AMTI MiniAMP A-6 (Watertown, MA) and converted to digital using the Vicon Motion

Capture System LTD v. 1.8.5 (Edgewood, MA, USA). Raw COP data was filtered using a fourth order Butterworth low-pass filter with a cutoff frequency of 10 Hz. The COP data was further analyzed using a custom MATLAB software code in order to obtain RMS, PEV, and SampEn values for both the EO and EC conditions in the AP and ML directions (MathWorks Inc., USA).

CHAPTER 2.6 STATISTICAL ANALYSIS

Several statistical analyses were performed in order to assess the data collected. All analyses were performed using SPSS v 23.0. All postural control measures were analyzed using paired samples t-test to compare between the CONC and CTRL groups. A Holm-Bonferroni correction factor was utilized in order to correct for performing multiple analyses and avoid type-1 error.[26]

Comparison of overall injury frequency between groups was performed using a chi square analysis. A chi square test allows for analysis of significant difference between the injury frequencies in each group. Lastly, a logistic regression was conducted to determine the use of baseline postural control measures as a potential indicator for resultant injury frequency. The alpha level for all analyses was set at $p < 0.1$ *a priori.*

CHAPTER 3 RESULTS

CHAPTER 3.1 DEOMOGRAPHICS

A paired samples t-test was used to assess the age, height, and weight of the subjects between each group. There was no statistically significant difference found between age ($p = 0.07$), height ($p = 0.76$), and weight ($p = 0.97$) of the subjects (Table 3).

Table 3: Demographics of CONC and CTRL subjects[a]

	CONC (n = 42)	CTRL (n = 42)
Age (yrs)	18.80 ± 1.36	18.34 ± 0.75
Height (cm)	178.58 ± 10.73	177.82 ± 11.29
Weight (kg)	81.86 ± 18.06	82.02 ± 20.99

[a]Demographic information was recorded at time of baseline concussion testing; height and weight may have changed within the 1-year injury observation period.

CHPATER 3.2 INJURY FREQUENCY

A total of 15 CONC participants sustained 22 acute LE injuries in comparison to the eight CTRL participants who sustained ten acute LE injuries within the one-year injury observation period. Sprains and strains to the ankle and thigh were the most common injury type and location, respectively. Full description of the injury demographics are displayed in Table 4.

Table 4: Demographics of injuries sustained by CONC and CTRL

		CONC (n = 22)	CTRL (n = 10)
Injury Location	Foot	0	2
	Ankle	7	3
	Knee	6	3
	Thigh	7	0
	Hip	2	2
Injury Type	Strain	9	2
	Sprain	11	8
	Fracture	1	0
	Cartilage Tear	1	0

In order to assess the overall frequency of injuries sustained between groups each injury incident was treated as an independent observation (i.e. if SA 'A' sustained three injuries in the one year time span, they were treated as three different SA). Therefore the group sizes were represented as CONC (n = 49) and CTRL (n = 44). A chi squared analysis revealed a significant association (p = 0.025) of acute LE injury within CONC (22/49 = 44.9%) in comparison to CTRL (10/44 = 22.7%) (Figure 2).

An odds ratio was calculated based on the number of participants that either sustained an injury or did not sustain an injury in each group. Student athletes that had a previous history of concussion were 2.36 times more likely to sustain an acute lower extremity injury in comparison to those without a history of concussion.

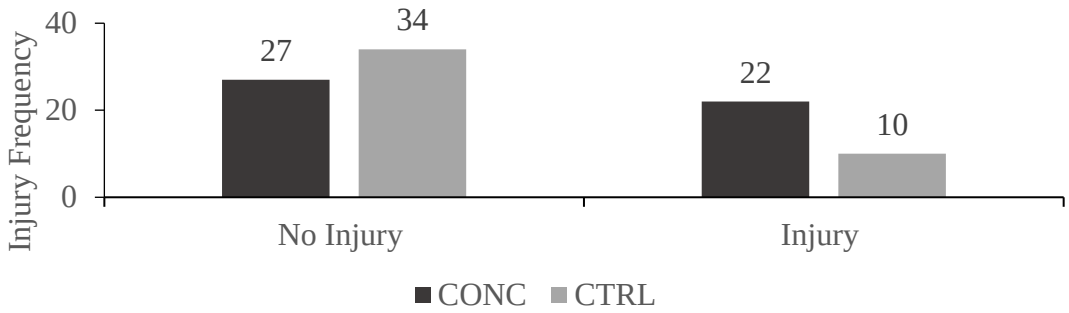

Figure 2. Chi square analysis of acute lower extremity injury frequency

CHAPTER 3.3 POSTURAL CONTROL MEASURES

The primary purpose of this study was to assess the differences in baseline postural control measures between groups. Therefore, paired samples t-tests were used to compare each postural control measure under each condition (i.e. EC, EO, ML, and AP). A Holm-Bonferroni sequential correction was run following initial t-tests to account for multiple comparisons.

The paired sample t-test revealed a significant difference between CONC and CTRL at baseline assessment within the EC PEV AP condition ($p = 0.006$) (Table 5) with a moderate effect (*Cohen's d* = 0.487). The CONC athletes moved significantly slower (0.063 ± 0.025) than CTRL athletes (0.078 ± 0.038) at baseline assessment.

Within the RMS measure no significant differences between groups were found within any condition. The EO AP condition revealed more total displacement within the CONC group (0.005 ± 0.002) versus the CTRL group (0.004 ± 0.001) however it lacked significance ($p = 0.017$*; Cohen's d* = 0.514) following the application of the correction factor. All other conditions lacked significance: EC RMS AP ($p = 0.386$), EC RMS ML ($p = 0.944$), EO RMS ML ($p = 0.183$) (Table 5).

As stated previously the CONC athletes moved significantly slower ($p = 0.006$; *Cohen's d* = 0.487) than their CTRL counter parts. Peak excursion velocity in all other conditions revealed no significant difference between groups: EO PEV AP ($p = 0.742$), EC PEV ML ($p = 0.314$), EO PEV ML ($p = 0.454$).

Finally, no significant difference was found between CONC and CTRL at baseline assessment for any SampEn conditions: EC SampEn ML ($p = 0.037$), EO SampEn ML ($p = 0.086$), EC SampEn AP ($p = 0.227$), EO SampEn AP ($p = 0.506$).

Within the EC SampEn ML condition the CONC athletes had less variance (0.68 ± 0.18) in comparison to CTRL (0.77 ± 0.16) however it lacked significance (p = 0.037; *d* = 0.534) following the application of the correction factor.

Table 5. Means and standard deviations for baseline postural control measures of CONC and CTRL

			CONC	CTRL
EC	AP	RMS	0.005 ± 0.002	0.005 ± 0.001
		PEV**	0.063 ± 0.025	0.078 ± 0.038
		SampEn	0.668 ± 0.164	0.708 ± 0.118
	ML	RMS	0.005 ± 0.001	0.005 ± 0.001
		PEV	0.073 ± 0.032	0.080 ± 0.028
		SampEn*	0.683 ± 0.178	0.772 ± 0.158
EO	AP	RMS*	0.005 ± 0.002	0.004 ± 0.001
		PEV	0.046 ± 0.020	0.048 ± 0.023
		SampEn	0.612 ± 0.144	0.631 ± 0.136
	ML	RMS	0.004 ± 0.001	0.003 ± 0.001
		PEV	0.049 ± 0.017	0.046 ± 0.016
		SampEn	0.521 ± 0.141	0.581 ± 0.167

*****Indicates a significant observation prior to correction factor; ****** Indicates a significant observation following correction factor

CHPATER 3.4 PREDICTING VARIABLES

Two binary logistic regressions were performed. First, to determine if the categorical groupings of previous history of concussion and the baseline postural control measures could be used to predict LE injury. And secondly, if the baseline postural

control measures and presence of an acute LE injury could be used to correctly predict subject groupings. There were significant regression outcomes for subject grouping (R^2 = 0.364, P = 0.013) (Figure 3) but not for LE injury (R^2 = 0.221, P = 0.379) (Figure 4).

Injury category (B = 1.448, P = 0.029) and EC PEV AP (B = 48.129, P = 0.043) were significant predictors for subject group. The overall prediction success was 76.2% (73.8% CONC and 78.6% CTRL). Participant group (B = 1.302, P = 0.033) and EO SampEn AP (B = -6.086, P = 0.062) were significant predictors for LE injury (p = 0.033). The overall predication success was 75% (91.8% for no injury and 30.4% for injury). The prediction equations for each regression model are listed:

Linear regression model for predicting subject group:

$$\log (p/p\text{-}1) = 2.854 + 1.448*\text{injury}(1) + 48.129*\text{EC PEV AP}$$

Linear regression model for predicting acute lower extremity injury:

$$\log (p/p\text{-}1) = 4.356 + 1.302*\text{Group}(1) - 6.086*\text{EO SampEn AP}$$

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CHAPTER 4: DISCUSSION

CHAPTER 4.1 REVIEW OF PURPOSE

The purpose of this study was to identify variations in baseline postural control measures between collegiate athletes who have a previous history of concussion and those who do not. In addition, our goal was to determine if baseline postural control measures may be used as an indicator for an increase in acute LE injury frequency within a previously concussed population. It was hypothesized that there would be significant differences in baseline postural control assessments between student athletes with a previous history of concussion and those without. In addition, it was hypothesized that student athletes with a previous history of concussion would have an increased acute LE injury frequency in comparison to those without. And lastly, that there would be a significant relationship between acute LE injury frequency and baseline postural control measures.

CHAPTER 4.2 REVIEW OF THE RESULTS

Our hypothesis regarding an increase in acute LE injury frequency in the CONC population in comparison to CTRL was met. The participants within the CONC group sustained twice as many (n = 22) injuries than the CTRL group (n =10) within the one-year observation period. Previous studies of this nature have reported odds ratios and injury rate ratios which were calculated using injury reports and athlete exposure time.[1, 2] Brooks et al (2016) stated that the odds of sustaining an acute LE injury were 2.48 times greater in previously concussed athletes than their controls.[1] Similarly, Lynall noted that CONC were 1.64 times more likely to experience an acute LE injury than healthy participants.[2] All studies arrived at the conclusion that athletes with a previous history of concussion were twice as likely to sustain an acute LE injury. Due to the retrospective nature of this study, we were unable to calculate athlete exposure

time. Regardless, we were able to calculate odds ratios, which demonstrated that similar to previous literature, student athletes with a previous history of concussion were 2.36 times as likely to sustain an acute lower extremity injury in comparison to CTRL.[1, 2, 3] Overall the most common injuries were sprains and strains to the ankle, knee, and hip. This is consistent with the findings observed by Brooks.[1]

It has been suggested that the increase in acute LE injury frequency may be the result of lingering postural control deficits following concussion. Previous studies have demonstrated the continual presence of postural control deficits from the time of return to play, up to nine months following concussion. Our hypothesis that postural control deficits would be present at baseline assessment in previously concussed athletes was partially met. Statistically, only EC PEV AP, was significantly different between CONC and CTRL. Previous literature, conducted by Powers et al, noted CONC participants had a trend towards increased velocity in comparison to CTRL, particularly in the EC condition.[12] However, these observations were made on recently concussed participants rather than baseline assessments for athletes with a history of concussion. Whereas Powers identified an increase in velocity, the CONC participants within this study had a slower PEV in comparison to the CTRL.[12]

Velocity represents a measure of the anticipatory movements made by the neurological system in order to control muscular contractions, particularly within a frequently changing environment such as athletic participation.[8] The maintenance of excursion velocity requires the integration of both the visual and vestibular system, and therefore requires more time to recover following concussion. The increased complexity of velocity maintenance may explain why there were continued deficits within this metric and not RMS, which requires less sensory integration. The decrease in PEV observed within the CONC group suggests that there is a lack of

communication between the neural feedback loops, which has resulted in slower alterations in neuromuscular control to maintain upright posture.

From a clinical standpoint, a decrease in velocity could be detrimental to providing stability to a joint during dynamic movement. A quick inversion mechanism to the ankle while completing a cutting drill requires significant neuromuscular control.[27, 28] The lack of speed and communication between the sensory systems observed in PEV could explain the increase in acute LE strains and sprains, as the patient is unable to respond as quickly to changes in their base of support.

Although no other postural control measures were statistically significant at baseline between CONC and CTRL, clinically the variations in RMS and SampEn should be noted. Based on the hierarchy levels of integration in linear postural control metrics, it was expected to see a more significant difference in PEV than RMS. However, previous literature has suggested that nonlinear metrics provide a more robust measurement to postural control in comparison to linear metrics. Therefore, it was expected that more significant variations in SampEn would be noted within this study.

Consistent with previous literature, the CONC participants had more displacement than the CTRL. Primarily within the EO AP condition, the CONC (RMS = 0.005m) group had more displacement than CTRL (RMS = 0.004m). Similar to our results, Powers et al observed significantly more AP displacement when comparing CONC to CTRL, and no difference in the ML direction.[12] Winter described the maintenance of upright stance through an inverted pendulum model in which the body pivots around the ankle.[16] Upright posture is maintained by continuous correction of sway through the ‘ankle strategy’. Due to the increased degrees of freedom within the AP direction, often more displacement is seen in this condition. The presence

of increased displacement in the CONC group suggests that there is still a deficit in the feedback from the somatosensory system that is altering the reactionary neuromuscular response to changes in posture. This decreased proprioception may contribute to the increased acute LE injury frequency noted within our CONC population.

Across all conditions, the CONC group had a decreased SampEn value in comparison to CTRL. Sample entropy is a unit-less value ranging from 0 to infinity, where smaller values represent more regularity and larger values suggest higher irregularity. In particular, there was a large decrease in SampEn within the EC ML condition for the CONC group (SampEn = 0.68) in comparison to CTRL (SampEn = 0.77). Previous literature has also observed an increase in regularity within CONC patients as they attempt to limit their degrees of freedom in order to maintain an upright stance.[29] This is said to represent a less complex processing system as a result of pathology.

Under this constrained system, CONC subjects are less apt to respond to quick changes in their base of support. The loss-of-complexity theory of disease provides a framework for which to rationalize these findings. Integration of our control systems (i.e. sensory feedback loops) are responsible for our neurological and mechanical output. It has been observed that the compromised feedback system within concussed patients produces alternations in our ability to maintain normal balance. Yet when we add additional sensory and cognitive demand, such as in a dual task environment, the complexity of the systems output decreases even further. Therefore, the additional dual-task demand that athletic participation requires produce further regularity in non-linear time series of CONC patients.[20]

Previous research has speculated that alterations in postural control may be a significant contributor to the observed increase in injury frequency following concussion. Although our

overall regression model lacked significance, EO AP SampEn and group assignments were still significant contributors to predicting LE injury. Interestingly, the non-linear metric was the only postural control measure to be a significant predictor for acute LE injury. Furthermore, assessment of baseline postural control within collegiate athletes should not rely on solely linear metrics, as they may not detect this increased risk. Similar remarks were made by Cavanaugh et al in which initial impairments in postural stability had been resolved, yet values of the nonlinear metric remained depressed.[20] Overall, it should be noted that this model identified the categorization of CONC and CTRL groups to be a significant indicator of injury. In addition, our second regression model indicated that the presence of a LE injury was a significant indicator of previous history of concussion. Overall, 76% of participants were correctly identified as CONC (73.8%) or CTRL (78.6%) based on whether or not they sustained an acute LE injury following their baseline assessment. Therefore, clinicians lacking access to more advanced postural control assessments may consider the presence of a previous history of concussion as a predictor for increased acute LE injury frequency.

This study was not without it's limitations. Overall, the retrospective nature of this study required the dependency of the primary investigator on the medical documentation and baseline postural control assessment of other staff members at the university in which this study was conducted. It must be assumed that all procedures were completed correctly, that the sports medicine staff accurately recorded all acute LE injuries, and that the SA were honest on their pre-participation exams regarding previous history of concussion. In addition this study was limited based on sample size. Previous literature in the area of injury surveillance following concussion typically has large sample populations in comparison to research within

biomechanics. In order to mitigate Type I error a Holm-Bonferroni correction of the alpha value was used.

CHAPTER 4.3 CONCLUSION

In conclusion, athletes with a previous history of concussion have twice the frequency of acute LE injuries in comparison to matched controls. Significant decreases in PEV may contribute to the increase in acute LE injuries as a result of slower reaction time to changes in base of support during athletic participation. Variations within RMS and SampEn were also observed between groups but lacked significance. By utilizing logistic regression, SampEn and a history of previous concussion were identified as significant predictors for acute LE injury. Overall clinicians may utilize both linear and non-linear postural control metrics during baseline assessment in order to identify SA at risk for acute LE injury. Future research may consider a longitudinal study that prospectively follows athletes in order to assess injury frequency both prior to and following concussion. This study provides as basis from which future clinicians may attempt to develop and incorporate lower extremity proprioceptive maintenance protocols for athletes with a previous history of concussion in order to decrease the number of LE injuries sustained.

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APPENDIX A

LIMITATIONS

1. Lack of injury documentation by athletic training staff
2. The investigator had to rely solely on the injury data that had been documented by the university's medical staff.
3. Concussion history data was limited to what the student athlete reported on their pre-participation screening.
4. Only injury frequency was reported due to the inability to calculate injury rate, which would have require the investigator to have knowledge of each individual athlete's exposure rate.

DELIMITATIONS

1. The population that was assessed was limited to Division I athletes from a single university
2. Subjects who had prior history of concussion had to have been diagnosed by a physician
3. Subjects within the concussion group had to have an available matched control based on gender, sport, and skill set

ASSUMPTIONS

The following items are assumed within this study:

1. The university's sports medicine staff has documented all cased of acute lower extremity injuries.
2. Subjects fully disclosed all concussion history on their pre-participation screening.
3. Baseline postural control assessments, conducted by members of the university's biomechanics lab staff, were administered and collected of accurately and consistently across all subjects.

RESEARCH QUESTIONS

It is the aim of the current study to investigate the following research questions:

1. Is there a difference in acute lower extremity injury frequency between student athletes with a previous history of concussion and those who have no previous history of concussion?
2. Is there a difference in baseline postural control measures between student athletes with a previous history of concussion and those who have no previous history of concussion?
3. Can baseline postural control measures be used as an indicator for increased acute lower extremity injury frequency following sports related concussion?

HYPOTHESIS

H_1: Student athletes with a previous history of concussion will have an increased acute lower extremity injury frequency in comparison to those student athletes with no history of concussion

H_2: A significant difference will be observed in baseline postural control measures between a previously concussed student athlete population and those without a previous history of concussion

H_3: There will be a significant relationship between acute lower extremity injury frequency and baseline postural control measures

APPPENDIX B
REVIEW OF LITURATURE

Introduction

As knowledge of concussion has moved to the forefront of the research field, the understanding of concussions and their lasting effects are continuing to evolve. The definition behind concussion has changed significantly since the 13th century term 'commotio cerebri' in which Lanfrancus defined as a transient disruption of cerebral function that was the result of shaking of the brain.[30] The idea that concussions resulted in the presentation of transient symptoms continued on through the 20th century, at which time the Committee to Study Head Injury Nomenclature defined a concussion as a 'transient impairment of neural function'.[31] Presently, the definition of concussion is comprised of some of these fundamental ideas; however, there is growing concern regarding the alterations to neurological function that may last long after the resolution of the immediate symptoms.

The most recent International Conference on Concussion in Sport held in Berlin in 2016 defined a sports related concussion as "a traumatic brain injury induced by biomechanical forces".[32] While the Berlin statement identifies the presence of acute clinical and cognitive symptoms, the possibility of prolonged and long term effects are also noted. This opened the door for further inquiry into the prolonged alterations in neurological function following a concussion, and what role these deficits play in the safety of the athletic population once they have returned to play following a concussive injury.

The National Athletic Trainer's Association has endorsed the use of a multifaceted approach to managing concussion order to ensure the assessment of all components of concussive injuries.[6] Following a sports related concussion a multitude of symptoms may occur. These symptoms are categorized into four different domains: (1) Somatic/Physical (2) Cognitive

(3) Emotional and, (4) Sleep related.[33] Physical symptoms may or may not be present to the clinician, as some symptoms must be reported by the athlete. Physical symptoms may include loss of consciousness, amnesia, and balance/postural deficits. Similarly, cognitive symptoms may or may not be obvious to the clinician (e.g. difficulty remembering, difficulty concentrating, feeling slowed down, feeling in a "fog"). Some student athletes may experience behavioral symptoms, which typically present as increased irritability or sadness or feeling generally more emotional than usual. Lastly, the fourth domain identifies changes within the student athlete's sleep patterns (i.e. sleeping more or less than usual).[33]

The key to the multifaceted approach of concussion is allowing clinicians to assess each domain of symptoms. A key component to the multifaceted approach is the assessment for balance and postural control deficits following injury.[6] Traditional clinical assessment for balance is conducted using the Balance Error Scoring System (BESS), which can identify postural control deficits up to 3-5 days following injury.[14] However, current studies have suggested that these postural deficits may last much longer than this window of time, perhaps well past the athlete's return to play.[12] It has been proposed that these lingering postural control deficits may influence the neuromuscular control and coordination of athletes once they have returned to an athletic environment. Increased LE injury rates have been observed in athletes who have recently sustained a concussion.[2, 3] It has been postulated that this increased injury rate may be the result of lingering postural control deficits, however the use of more sensitive postural control measures to determine the relationship between these two variables has not yet been conducted.

Epidemiology of Injury

Concussion Epidemiology

The Center for Disease Control and Prevention has estimated that approximately 300,000 sports-related concussions occur annually in the United States with varying incidence rates based on sport, participation level, and gender.[34] This number, which was derived from emergency room records, may be grossly underestimated, due to the underreporting of concussions to health care professionals. A more accurate number may be the estimated 1.6 - 3.8 million reported and unreported sports-recreation related mTBIs annually.[5]

The incidence rate of concussions in collegiate athletics has been estimated at 8.9% per athletic exposure by the National Collegiate Athletic Association's Injury Surveillance System (ISS) which collects injury data from 17 different collegiate sports across all divisions.[35] Over the past 16 years of data collection, the injury rate for concussions in the collegiate setting has increased by 7%, which may be the result of better awareness or diagnostic methods.[35] Of the sports under surveillance, football, women's ice hockey, and women's soccer had the highest number of reported concussions annually. Concussion rates were significantly higher during competition (53.2%) when compared to practice (46.8%).[36]

Lower Extremity Epidemiology

Current literature describing the epidemiology of musculoskeletal injures in collegiate athletes varies significantly in methodology and is traditionally categorized by sport or injury. Therefore, it is difficult to report overall injury epidemiology across all athletes and body regions. Typically, an injury is defined as any pathology that requires attention from the team physician or athletic trainer during a school sanctioned activity.[37] Lower extremity injuries are

defined by the NCAA-ISS as injury to the areas of the hip/groin, upper leg/thigh, knee, lower leg/Achilles, ankle, and foot/toes.[37]

According to the NCAA-ISS LE injuries account for over 50% of all reported injuries during both practice (53.7%) and games (53.8%), of which knee and ankle injuries are most common.[38] From 2009-2015 the NCAA conducted a study following 68 NCAA football programs to record LE injury rates during competition. Of the injuries recorded, knee (33.6%) and ankle (28.5%) injuries were most prominent and were a result of player contact (59.2%).[37]

A study conducted by Hunt et al. in 2007 reviewed the epidemiology of lower leg, ankle, and foot injuries sustained at a single Division I NCAA university across 37 sports. Over the course of two years 3861 injuries were recorded, of which 1035 involved the foot, ankle, and lower leg (27%).[39] Ankle injuries were most prominent in this population (44%), of which the majority of injuries were either sustained in football (20%) and soccer (14%).

Musculoskeletal Injuries Following Concussions

The odds ratio of sustaining a LE injury is 1.6-2.9 greater among collegiate athletes that have sustained a concussion.[40] Following concussive injury a significant relationship has been observed between lateral ankle sprains (p= 0.012), knee injuries (p = 0.002), and LE muscle strains (p= 0.031).[40] Interestingly, athletes that reported having a concussion only had an increased risk for knee injuries (2.1x greater), whereas unreported concussions had an increased risk for all LE injuries (e.g., ankle sprains, knee injuries, and LE strains).

Mechanism of Injury

The mechanism of a concussion can result from either direct or indirect forces applied to the body that causes rapid acceleration or deceleration of the brain within the skull.[4, 6] Damage to the brain occurs either when it comes into contact with a part of the skull or as a result of the linear and rotational forces. Contact of the brain with the skull can be defined as either a coup or contrecoup mechanism, based on whether the contact occurred on the same or opposite side of the force impact, respectively.[41] These rotational forces create a shearing mechanism which can be defined as the movement of brain matter over other brain tissue and the stretching of axons within it. Shearing forces to the brain as a result of acceleration and deceleration are more damaging to neural tissue when compared to compressive forces that are traditionally associated with skull fractures.[41]

In general, direct contact with another player is the most common mechanism of injury, but is not the only cause of concussions.[42] Injury due to contact with equipment, such as the sticks and balls in lacrosse and field hockey, were the result of 1/3 of concussive injuries in those sports.[42] The use of protective equipment cannot prevent the occurrence of concussions, but are more likely to distribute the force applied to the head. The magnitude of the force applied to the head is not necessarily correlated with the severity or occurrence of concussion.[10] Impacts to the head that are above 80G have typically been linked to the diagnosis of a concussion. However, statistically significant changes in MRI scans have been observed in football athletes that have received several subconcussive blows (i.e. 25G-80G impact).[21] This suggests that athletes that are exposed to repetitive subconcussive impacts may still be at risk for long-term changes in brain functionality. Overall, it is likely that the cause of concussion is a result of a combination

of mechanical factors including impact location and linear or rotational acceleration rather than magnitude of force alone.[43]

In addition, variations in biomechanics such as neck strength and head mass can have an effect on the mechanism of a concussion. The increased mass of a child's head as well as males compared to females has been associated with a decrease in linear accelerations, potentially decreasing the overall concussion rate.[44] Similarly, weaker neck musculature has been associated with increased angular acceleration in female athletes, which would predispose them to higher concussion rates.[45]

Pathophysiology

Concussions have been defined as alterations in mental status caused by a traumatic event, such as a direct or indirect force transmitted to the brain, which result in functional neurological damage or microstructural injury.[4] This damage presents as the transient neurological symptoms that are observed by clinicians. The resulting neurological symptoms that follow a concussion are the result of neural shearing, which causes a neurometabolic cascade that produces changes in metabolism[46] and ion balances.[47] When determining differential diagnoses, it is important to note the lack of macroscopic neural trauma or significant structural damage to the brain. Functional neural damage has been defined as perturbations of either cellular or physiological function.[48] This can include ionic shifts, metabolic changes or altered function of neurotransmitters.[48] Whereas gross structural damage can be identified using imaging, such as magnetic resonance imaging (MRI) or computer tomography (CT) scans, the microstructural damage that results from an mTBI may not be evident. Functional MRIs (fMRI) assess the neuronal activation patterns within the brain, and therefore may be the only

appropriate form up imaging to use in order to assess the functional nature of a concussive injury.[49]

The metabolic and ionic changes that occur following injury are termed the neurometabolic cascade, which begins immediately following injury.[50] Despite the resolution in clinical symptoms, the duration of these neurologic changes are unknown and is still under investigation. Initially following injury, there is a disruption of the neural membranes and axonal stretching causing the regulation of ion exchange to become unchecked followed by the release of excitatory neurotransmitters and further neural depolarization.[47] This depolarization results in the efflux of potassium (K+) and the influx of calcium (Ca2+) within the cell. As a result of this depolarization and ion flux, the sodium-potassium pump (Na+-K+) is required to work in excess in attempts to maintain normal membrane potential. Because of the required energy demands of the sodium-potassium pump, there is a dramatic rise in glucose metabolism. In a post-injury environment, there is decreased cerebral blood flow creating a conflict for obtaining this necessary glucose supply.[47] Following injury cerebral blood flow may be reduced up to 50% of its normal volume.[51] The resulting energy crisis leaves the brain less apt to respond which may explain why a second injury would produce longer lasting deficits.[47]

Following the increased excitement caused by the unregulated potassium flux and neural depolarization, there is a period of time in which the neurons are suppressed, termed spreading depression.[47] Unlike traditional spreading depression, this phenomenon when experienced as a result of post-traumatic brain injury occurs throughout different area of the brain at the same time.[50] The energy crisis and changes in cerebral blood flow may be the cause of this wide spread depression that is specific to post-traumatic depression.[47, 52] This diffuse spreading

depression may be the cause behind loss of consciousness, amnesia, and the acute cognitive decline that we observe immediately following injury.[52]

In attempts to regulate the ion fluxuation and return the cellular metabolism to normal, glycolysis is accelerated further. As a result, there is an increase in lactate production and concurrent decline in lactate metabolism, producing a significant lactate accumulation. Further neural damage (e.g. cerebral edema, acidosis, altered blood brain permeability) is caused by this buildup in addition to the increased vulnerability for ischemic injury.[53] Understanding the pathophysiology behind the injury allows for the development of assessments to correlate with the symptoms that will develop as a result of this injury.

Symptomology

Symptoms following a concussive injury are presented in a variety of ways, including neurological deficits, cognitive impairment, and somatic dysfunction. The presentation of symptoms following concussion vary in quantity and severity based on the individual athlete, averaging 5.29 ± 2.94 symptoms on a 17-item checklist.[36] The most commonly reported symptoms by collegiate athletes are: headache (92.2%), dizziness (68.9%), and difficulty concentrating (58.3%). Within the collegiate population, symptoms typically resolved within one week (60.1%), however 6.2% of athletes experienced symptoms lasting greater than four weeks.[36] Throughout the duration of the NCAA's ISS study, the number of athletes requiring more than one week for symptom resolution increased from 42.7% to 70.2% and those athletes experiencing long term symptoms (> one month) increased to 8.0%.[36]

Symptomology is a key component to the concussion evaluation and is one of the most prevalent aspects of the concussion assessment utilized by clinicians (85).[54] Several variations of the symptom checklists are available including the Graded Symptom Checklist (GSC), Head

Injury Scale (HIS), and Post-Concussion Symptom Checklist (PCSC). While symptom checklists provide clinicians with beneficial information, this is just one component to the multifaceted concussion assessment.

Concussion Assessment

Unlike traditional musculoskeletal injuries, concussions cannot be diagnosed through a single imaging technique or diagnostic test. Clinicians rely on a multifaceted approach of symptoms, balance assessment, and cognitive assessment to determine if patients have sustained a concussion.[6] These assessments do not provide definitive answers as to the severity of the concussion; therefore, use of a grading scale for the purpose of treating these injuries may not be appropriate. The most recent position statement released by the National Athletic Trainer's Association recommends the use of a multifaceted assessment battery, as well as baseline and follow-up neuropsychological testing.[6]

Baseline Assessment

Prior to the beginning of the athletic preseason, all athletes should undergo baseline assessments.[6, 55, 56] These assessments should consist of a clinical history of concussions, symptoms, and any learning disabilities, physical and neurological evaluations, postural stability, and neurocognitive function.[4, 11, 57, 58] Identifying preexisting symptoms and establishing baseline scores sets a foundation for an individualized assessment should the athlete sustain a concussion. While normative values can be utilized, they are not as accurate in comparison to individual baseline assessments. Common measures used during baseline assessment include the Balance Error Scoring System (BESS) for postural stability, Standardized Assessment of Concussion (SAC) and the Immediate Post-Concussion Assessment and Cognitive Test (ImPACT) for

neuropsychological testing. The setting of baseline testing should be replicable, therefore should the athlete sustain a concussion and require follow up assessment they will have as much consistency as possible.[59] Additionally, all athletes who have sustained a concussion should be re-baselined following their competitive season to account for changes in cognitive function and postural stability that may have resulted from the previous injury.[6] Normative data is available for these assessments, however, a more accurate evaluation can be made if completed on an individual basis. Concussion diagnoses nor return to play decisions should be made based on the results of follow up testing from baseline results, but rather should be used as a part of the multifaceted approach.[6]

Upon suspicion of a concussion, the athlete in question should be removed from play and assessed by a medical professional. A sideline assessment tool such as the SAC can be utilized in conjunction with a symptom checklist, motor-control evaluation, and a physical and neurological clinical examination.[6] Symptoms reported by athletes can be very beneficial when diagnosing a concussion. Symptom checklists such as the Graded Symptom Checklist (GSC), Head Injury Scale (HIS), and Post-Concussion Symptom Checklist (PCSC) provide subjective measures to the clinician and tend to have high sensitivity rates for concussive injuries immediately following injury (GSC = 0.89; HIS = 0.77; PCSS = 0.81.[57, 60, 61] The symptom checklist that is used during baseline assessment should be the same for all follow-up assessments, for consistency.

An alteration in mental status is the key component to defining a concussion. However, less than 10% of athletes experience a loss of consciousness, and only 25% of athletes experience some sort of post traumatic amnesia.[11] Immediately following injury, some form of cognitive assessment should be performed in order to rule out differential diagnoses. The SAC is a brief sideline neurocognitive assessment battery used to evaluate the following domains of

cognitive function: orientation, immediate memory, concentration, and delayed recall.[60] While the SAC is sensitive to concussive injuries immediately following the onset, its sensitivity begins to decline following 24 hours post injury (sensitivity = 0.80, specificity = 0.89-.98 at time of injury), and is best used when combined with other assessments such as the GSC and the BESS.[60]

In-depth neurocognitive testing assesses a variety of domains including: verbal memory, reaction time, visual memory and processing speed. These tests can be administered at baseline, as well as following the resolution of symptoms measured by a self-reported symptom scale. The objective measures obtained through these tests allow clinicians to measure the initial decline in cognitive function followed by the resolution of symptoms as the athlete progresses. Traditional neurocognitive assessments were originally administered through pencil and paper, and required a trained neuropsychologist to decipher the results. However, computerized applications for neurocognitive testing have recently been developed and are becoming more readily used by clinicians. Computerized assessments allow for mass testing of student athletes, which provides the clinician the ability to have individualized baseline test scores. In addition, the use of computerized assessment eliminates the need for a neuropsychologist to interoperate each assessment which not all clinical settings have access to.

Balance Assessments

Motor control deficits are common following concussive injury, and have been observed as alterations in gait pattern and postural control[6]. Postural control has been defined as 'the act of maintaining a state of balance during any posture or activity'.[7] Maintaining postural control requires the integration of the visual, vestibular, and somatosentory system[8]. The terms balance

and postural control are often interchanged, however, balance refers specifically to the ability to maintain control of the position and motion of the body's center of mass (COM) relative to the base of support.[16, 17] Following a concussive injury, it is common to observe deficits in balance as a result of the disruption of the communication between these systems.[12]

Balance Error Scoring System

Sideline assessment for deficits in postural control is most commonly conducted using the BESS. The BESS can be used in order to identify a lack of postural control during static stability 3-5 days following injury.[14] The BESS assesses balance control based on 3 separate static stance trials (double, single, and tandem leg support) on both a firm and soft surface. The participant is assessed based on the number of deviations or errors from the original position within a 20-second observation period. This cost efficient and portable method of postural control assessment is readily available for sideline use and requires little additional knowledge from the clinician. However, this assessment relies on the interpretation of the clinician to detect errors, resulting in the potential for inconsistent interrater reliability (ICC = 0.78-0.96).

At baseline assessment the BESS has a sensitivity of 0.34 and a specificity range from 0.91 to 0.96 up to 7 days following injury.[14] Factors that could alter the reliability of this assessment include variations in testing surfaces, lack of baseline scores, the presence of chronic ankle instability in the athlete, or a LE injury in between the time of baseline measurements and suspected concussion. In addition, this assessment has demonstrated to be subject to practice effects as a significant increase in pre and postseason scores were observed (P = 0.003).[62] Overall, though the BESS is commonly used in clinical settings, it is a rather subjective test that may be unable to detect subtler deficits in the postural control system after injury. Further studies

have noted decreased postural stability during static assessment up to 15-30 days post-injury using more sophisticated measures of postural assessment such as the force platform.[12] Therefore there may be a need for more sensitive measures for postural stability to identify lingering deficits that the BESS cannot assess.

Romberg Test

The Romberg test was designed to identify balance impairments by placing participants in a visually reduced environment, therefore increasing their reliance on their vestibular and somatosensory systems. Participants are asked to stand feet together while performing a static stance with an eyes-open and an eyes-closed trial. Observed sway or inability to maintain the stance indicates a postural deficit. While the validity and reliability of this assessment has not been studied for use with concussed individuals, an evaluation has been conducted with Parkinson's patients who fall within the category of neurological impairments. The reliability of the Romberg test has been found to be excellent in individuals with Parkinson's disease (ICC = 0.84, reduced vision condition; 0.86, normal vision condition).[63] However, when assessing the validity of the Romberg test within a population of individuals with a vestibular dysfunction, sensitivity (0.55) and specificity (0.64) were moderate.[64]

Sensory Organization Test

The Sensory Organization Test (SOT) provides clinicians with a more sophisticated method of measuring postural deficits following concussive injury. This assessment was designed to systematically disrupt feedback from the athlete's senses by altering the orientation and information to the somatosensory and visual system. The athlete is assessed in six different

conditions for three trials of 20-seconds each. Force plates are used to measure the athlete's ability to maintain a quiet stance throughout each trial.

The reliability of this assessment has been tested within a group of healthy individuals, and has demonstrated to have moderate levels of reliability in this population (ICC = 0.26-0.64).[65] Following concussive injury, the SOT has demonstrated the ability to identify balance deficits 3-5 days following injury. Furthermore, application of approximate entropy techniques to SOT data has revealed balance deficits that continue to persist past this timeframe.[10] Therefore, the use of linear force plate data alone may not be sophisticated enough to identify postural deficits at the time of return to play.

Postural Stability

The ability to maintain posture and equilibrium requires feedback to the central nervous system (CNS) from the sensors of the visual, vestibular, and somatosensory systems.[10] Each system provides certain information that is used in order to maintain our upright position. Locomotion and obstacle avoidance is the primary function of the visual sensory system, whereas the vestibular system senses for linear and angular acceleration. Lastly, the somatosensory system provides information on the position and velocity of the body, as well as its contact with objects and the ground.[17] Should one of these systems be diminished or compromised, more reliance is placed on the remaining two systems to provide sensory feedback.[8] For example, closing one's eyes while maintaining upright posture places more demand on the vestibular and somatosensory systems. The individual will compensate for the loss of the visual system by increasing their postural sway in order to provide more feedback though the vestibular and somatosensory systems. This feedback allows for communication from

the brain to the extremities in order to maintain postural stability. Following a concussion there is functional damage to the brain and its neurological pathways causing a disruption in the communication between these systems.[10] Therefore, it is common to observe postural deficits and lack of balance control in either the anterior-posterior direction, medial-lateral direction, or both, when an athlete sustains a concussion.

Postural stability has been traditionally measured clinically through the use of BESS, however the window for reliability of this measure is between 3-5 days following injury.[14] In addition, the validity of the BESS test may decrease due to practice effects and the type of athlete (i.e., soccer player with chronic ankle instability).[10] In order to account for learning effects a 4-point increase from baseline assessments has been established as the clinically meaningful difference.[58] Force plates and biomechanical measurements have been utilized as a more sensitive and objective measure for concussion research in order to identify postural deficits lingering after the resolution of acute symptoms.

Linear Metrics for Postural Control

The variable of center of pressure (COP) had been identified as a valid and objective measure for postural control.[16] COP is the vector of the vertical ground reaction forces and moves in relation to alterations in postural stability in order to account for changes in the center of gravity (COG).[16, 17] Guerts et al. utilized the linear measurements of COP displacement and COP displacement velocity in order to identify lingering postural deficits up to two years following traumatic brain injuries in motor vehicle accidents and falls.[66] Based on this principle, Powers et al. indicated that the same variables could be used to identify that concussed athletes were not fully recovered at their time of return to play.[12]

Linear measurements of COP are coded into orders of functioning based on their level of integration of sensory feedback. Displacements is a first-order variable based on it's sole reliance on the feedback from the somatosensory system.[8] In comparison, velocity requires the integration of both the visual and vestibular system, and is therefore coded as a second-order variable.[8] Removal or inhibition of one of these sensory system requires the other systems to increase aggregation of information in order to maintain postural stability. Following a concussive injury the impairment to the visual and vestibular systems results in an increase in both COP displacement and velocity in comparison to healthy controls.[12] The increase in COP velocity is a reflection of the anticipatory adjustments that are being made to account for the increase in COP displacement. Powers et al noted that the velocity of the COP was a more sensitive measurement in comparison to COP displacement, as increases in velocity were still present despite normal COP displacement measures.[12]

The variations in COP displacement and COP velocity between concussed individuals and healthy controls increases as higher demands are placed on the sensory systems. On a basic level, the removal of the visual systems during a static stance requires more input from the vestibular and somatosensory systems. While a change in COP velocity and displacement are observed between eyes-closed (EC) and eyes-open (EO) conditions in a healthy population, the variation between these conditions are much more pronounced in concussed individuals suggesting poor postural stability.[12]

Non-Linear Metrics for Postural Control

While the use of linear metrics have demonstrated to be successful in identifying postural instability in concussed athletes, the use of non-linear metrics may provide a more robust and

sensitive measure to detect lasting deficits.[22] The two most common nonlinear measures used for postural control include approximate entropy (ApEn) and sample entropy (SampEn). Both variables are represented by a unit-less number ranging from 0 to infinity, where in zero represents less order in the time series and increased values represents a completely random and irregular time series. ApEn quantifies the ensemble amount of randomness, or irregularity, contained in a time series and is particularly suited for the short, noisy biological output signals associated with human movements.[20] Within a healthy population, the COP during a quiet, upright position is characterized by a relatively irregular and small amplitude. The ApEn value for the anterior-posterior time series ranged from 0.50-0.84, in comparison to the medial-lateral ApEn range of 0.75-0.93.[20] When assessing an individual with altered sensory input, such as a concussed individual, the COP oscillations become more regular, value becomes closer to zeros, and have a larger amplitude.[20] The relationship between COP amplitude and regularity is not linear, and therefore provides more sensitive information on the state of postural control in comparison to traditional linear measures. Therefore the use of ApEn may be a more reliable tool when determining the return to play for concussed athletes.[22]

Although ApEn has been used as the primary nonlinear measure throughout current postural control studies, SampEn has demonstrated to be an equally as reliable measurement.[22] In an analysis conducted by Richman & Moorman, SampEn statistics were much more agreeable with theory for random numbers with known probabilistic character over a wide range of operating conditions and maintained consistency throughout measures, whereas ApEn did not.[22] The use of SampEn statistics provide an improved method for assessing time series regularity, and therefore may be a useful measure for identifying the regularity in postural control within the concussed population.[19]

Lower Extremity Injuries Following Concussion

The use of more sensitive measures for postural stability have helped to identify lingering postural deficit following concussion that may last well beyond the athlete's time of return to play. Alterations in postural stability and neuromuscular control can leave these athletes susceptible to further injury. Primary assessment of LE injury rates following concussion have identified that athletes who have sustained a concussion within their last year of participation are two times more likely to sustain LE injury compared to matched controls.[2, 3] In the year following concussive injury, collegiate athletes were most likely to sustain an acute LE injury 180-365 days following their return to play.[2] Subsequent studies have looked at the LE injury rates following concussion 90-days post return to play and have found comparable results[1]. Increased injury rates have been associated with acute (i.e., ligament sprains and muscle strains) injuries but not gradual onset (i.e., tendinopathies, stress fractures) injuries.[3] In addition, the time between return to play and LE was recorded, however no significant differences were found between concussed athletes and matched controls.[1]

The goal of the multifaceted approach to concussion assessment is to provide clinicians with the appropriate information in order to be able to safely return their athletes to play. Within these studies, all athletes had successfully passed and progressed through a return to play protocol without the recurrence of symptoms. It has been suggested that the increase in LE injuries following concussion may be the result of several factors. The idea that detraining may play a factor in these increased injury rates is not necessarily likely considering the time from return to play and LE injury does not vary between concussed athletes and matched controls.[3] LE injuries as a result of detraining would be more likely to occur within the first few weeks of

return to play. However, no significant pattern for time occurrence of injury was noted, if not significantly past the window of detraining.[67]

Assessments of static and dynamic postural control following concussion have demonstrated alterations in postural stability[10] and the development of a conservative gait pattern that last beyond the resolution of acute symptoms.[68, 69] In addition, the performance of dual-tasks paradigms (e.g., cognitive assessment while walking, obstacle avoidance) increase the postural deficits in these athletes compared to controls.[70] Similarly, the demanding environment of athletic participation may become problematic for athletes who have sustained a concussion as they continue to display deficits in neuromuscular control. Utilizing the more specific assessments of force plate metrics for postural control may help to identify these lingering deficits following concussion and determine which athletes are predisposed to sustaining a LE injury.

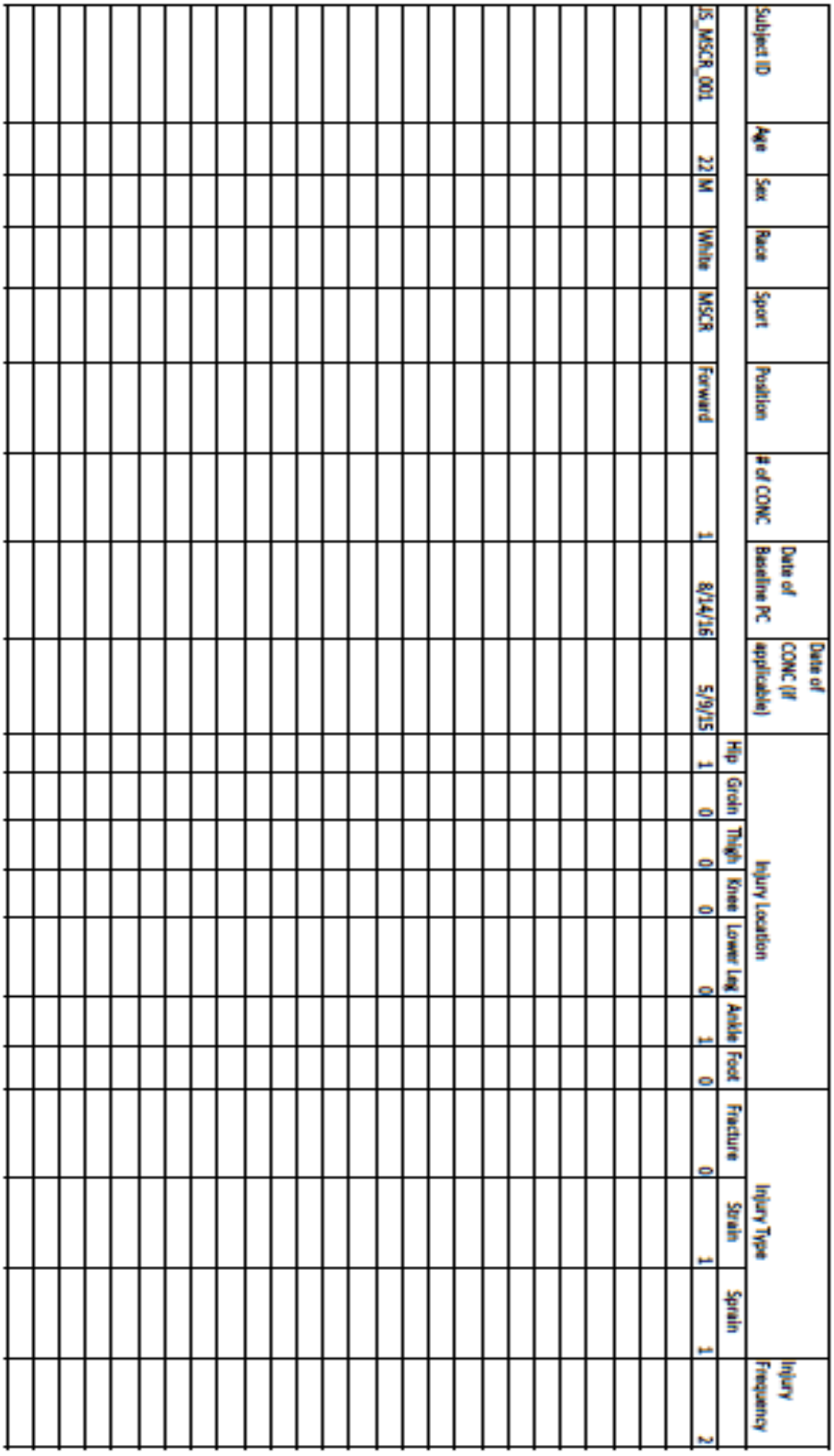
APPENDIX C

INSTRUMENTS

CODED IDENTIFIER LIST

Patient Name	**Sport**	**Study ID**
Ex: John Smith	Men's Soccer	JS_MSCR_001

MEDICAL CHART REVIEW FORM



Subject ID	Age	Sex	Race	Sport	Position	# of CONC	Date of Baseline PC	Date of CONC (if applicable)	Injury Location							Injury Type			Injury Frequency
									Hip	Groin	Thigh	Knee	Lower Leg	Ankle	Foot	Fracture	Strain	Sprain	
JS_MSCR_001	22	M	White	MSCR	Forward	1	8/14/16	5/9/15	1	0	0	0	0	1	0	0	1	1	2