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Changes in Gait Characteristics Following a Concussion

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CHANGES IN GAIT CHARACTERISTICS FOLLOWING A CONCUSSION

by

KELLY PREVIS

(Under the Direction of Barry Munkasy)

ABSTRACT

Introduction: Recent literature has suggested that gait could potentially provide clinicians with a reliable way to determine if an athlete has sufficiently recovered from a concussion in order to return to participation. The Balance Error Scoring System has typically been used to determine if static and postural control have returned to baseline measurements. It has yet to be determined the level of gait recovery within 24 hours of sustaining a concussion (CD1), on the day the concussed athlete returns to BESS baseline (BBD), or the day they finally return to play (RTPD). Objective: The purpose of this study was to compare a concussed athlete's gait to non-concussed athletes and normal controls, on specific days of their recovery process. Methods: In this study, 45 subjects were divided into groups; 15 concussed intercollegiate athletes (CONCs), 15 non-concussed teammates (NONCs), and 15 normal controls (NORMs) who did not participate in an intercollegiate sport. The NONCs were matched according to sport and gender, and the NORMs were matched to gender of the CONCs. The subjects walked on the GAITRite® walkway, where gait velocity, cadence, step length, step width, and double leg support times were calculated. The NONCs and CONCs walked the same number of days as their CONC match, until the CONC returned to participation. Results: Gait velocity showed significant group differences at CD1 (F= 3.670, p=.034), whereby CONCs had a mean gait velocity significantly less than the NORMs (1.21 ± 0.16 m/s and 1.34 ± 0.09 m/s respectively, p=.036). There were significant main effects for gait velocity, step length, step width, and double leg support times. Most of the subjects increased their gait velocity and step length (F=18.940 and p<.001, F=16.542 and p<.001), respectively) and decreased their double leg support times (F=14.395 and p<.001) between CD1 and BBD. Subjects from CD1 to RTPD showed increases in gait velocity and step length (F= 11.901 and p=.001, F=10.553 and p=.002, respectively), and decreases in step width and double leg support times (F=11.976 and p=.001, and F=10.583, p=.002, respectively). However, there were no significant differences for gait velocity, cadence, step length, step width, or double leg support times between CONC and NONC, CONC and NORM, or NONC and NORM from CD1 to BBD, BBD to RTPD, or from CD1 to RTPD. Conclusion: These findings indicate that a concussed athlete shows slower gait velocity initially after a concussion at CD1, possibly indicating a conservative gait strategy that develops into normal gait patterns as BBD and RTPD occur. However, cadence, step length, step width, and double leg support times during single task walking may not be good indicators on whether an athlete has recovered their postural control. Therefore, it is recommended that a variety of concussion testing tools, including postural control measurements such as gait, be used to make a proper assessment of when the athlete may return to participation.

INDEX WORDS: Concussion, Gait, Single Task, Return to participation, Velocity, Uninjured athlete controls, Postural control

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by

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DEDICATION

This is dedicated to my thesis committee, Dr. Munkasy, Dr. Buckley, and Dr. Joyner, who pushed me beyond my limits to this thesis document, which I never thought I could attain. Thanks for never giving up on me.

This is also lovingly dedicated to my fiancée, Josh Reedy, who stayed with me through this whole process, as well as my parents, Charles and Rose, who supported me as well. The words "gait" and "concussion" will, from now on, never be allowed in the same sentence again.

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CHAPTER 1

INTRODUCTION

Concussions affect between 1.6 to 3.8 million athletes participating in sport every year in the United States.[1] A concussion is defined as a multifaceted injury that affects the brain, involving various biochemical and biomechanical processes that can result in multiple physical, behavioral, and cognitive changes.[2] A concussion, or mild traumatic brain injury, involves a direct or indirect blow to the head that impairs mental status and motor control.[2-5] During this period of recovery, the individual has a greater risk of sustaining a concussion, which leads to increased mental impairment, delayed recovery time, and poorer presentation of future concussions.[6-9]

Athletes who sustain multiple concussions can have slower recovery both neurocognitively and symptomatically.[7, 10] Those with a history of recurrent concussions, or sub-concussive blows, have a five-fold increased risk of later-life memory impairment, as well as early onset of Alzheimer's disease.[11] Those with a history of three or more concussions have a three-fold risk of developing depression.[12] Potentially, if an athlete receives a concussion and then sustains a second blow to the head before fully recovering, the athlete could experience brain hemorrhaging and death within minutes, a condition termed Second Impact Syndrome.[13]

All these harmful and potentially deadly effects make it important for clinicians to determine when an athlete is ready to return to participation. Clinicians face a few difficulties, one being that concussions do not present a standard set of signs or symptoms, and another is there is no standard severity, duration, cognitive, behavioral, or physical deficits involved in an individual athlete's presentation of a concussion.[4, 14] Therefore, it has been recommended that each athlete be assessed and managed on an individual basis as to when they should return to

participation after a concussion.[4, 9, 14-15] Individual management and assessment involves utilizing a variety of concussion testing tools in several domains, including neuropsychological and cognitive testing, postural stability, and symptom resolution to determine whether the athlete has recovered. These standardized tests have been created to assess an individual's unique set of symptoms and deficits in a testing format that athletic trainers can use.[4, 9, 14-15]

Concussion assessments commonly used among athletic trainers may return an athlete prematurely to their sport, because commonly used testing methods may not be as sensitive in detecting lingering impairments from the concussion.[16-19] For example, postural stability assessments, such as the Balance Error Scoring System (BESS) typically return to baseline three to five days postconcussion.[20-21] Cognitive tests such as Standardized Assessment of Concussion (SAC) usually return to baseline within five to seven days.[20] These concussion assessments, however, are not always reliable; for example, it has been shown that both BESS and SAC suffer from practice effects.[21-22] For example, concussed athletes had lower scores Day 7 after a concussion, which means they had improved their score on the test; the concussed did better compared to their non-concussed peers, who had no improvements between baseline or Day 7 scores.[22] Therefore, there is a need for reliable assessment tools that can help identify when a concussed athlete has recovered.

The BESS test assesses postural stability, which is the body being able to withstand internal and external perturbations in the environment.[18] The athlete tries to stand as still as possible, maintaining their center of gravity within their base of support, which are their feet.[18] Postural control is defined as an individual's amount of sway; with greater postural control associated with less sway.[23] If an athlete displays healthy postural stability, it is assumed they have a healthy postural control system.[23] Postural stability changes after a concussion; however,

it has been suggested that traditional methods of assessing postural control, like BESS, may not fully detect changes in postural control.[18] BESS involves static postural control, where the individual stands as still as possible within their base of support.[51] Gait involves dynamic postural control, where the individual changes their base of support throughout the movement.[17] Most athletes, with few exceptions, participate in dynamic postural control on a daily basis. Dynamic postural control measurements, such as virtual time of contact, found residual deficits and postural abnormalities 30 days post-concussion.[19]

Gait involves dynamic postural control, and has been studied in several populations, including older adults, osteoarthritis, post-menopausal women, diabetics and individuals with claudication, as well as Parkinson's disease, and stroke.[24-35] These aforementioned groups, as well as traumatic brain injury patients, have shown a conservative gait strategy after their injury/illness. Conservative gait strategies have been demonstrated by decreases in gait velocity and step/stride length, wider step width, reduced cadence, and increased stance and double leg support time compared to normal, uninjured subjects.[24-27]

Those with mild traumatic brain injuries, or concussions, have recently demonstrated similar tendencies. Concussed individuals demonstrated shorter stride length while walking, on post-concussion days 2 and 14, as well as slower gait velocity than normal controls within the first 24 hours.[17, 36-41] Concussed athletes demonstrated the slowest gait velocity compared to non-concussed athletes, concussed non-athletes, and non-concussed non-athletes Day 2 post-concussion. Furthermore, concussed athletes had the slowest gait velocity out of all the groups Days 5, 14, and 28 post-concussion, and the concussed and non-concussed athletes walked slower than non-concussed non-athletes.[32] (None of these aforementioned results were found to be significant.[17,40]) None of these studies tested specific days of the athlete's recovery process

compared to healthy controls; examples include the day the concussion occurred, the day the athlete recovered their static postural control using the BESS test, and the day the athlete returned to participation in their sport. These studies also did not mention examining cadence, step length, step width, or double leg support times over the athlete's recovery process. Finally, these studies have not used GAITRite[®], a walkway that measures gait, to calculate the gait velocity (they used the Vicon512).

These studies seem to indicate a possible "conservative gait strategy" adopted by concussed individuals.[42-43] Although these previous studies examined both concussed and non-concussed athletes and non-athletes, these studies focused mainly on gait velocity, stride length, stride time, and step width, comparing single and dual task conditions.[17, 28-29, 36-37, 40] However, no studies were found that examined concussed athletes on specific days of the recovery from a concussion, such as Day 1 post-concussion, return to baseline on BESS, and return to participation. These days are important, because the athlete passes neurocognitive, postural stability, and symptom testing (as part of a return to play participation protocol) that eventually returns them to participation.

During the first 24 hours after an individual sustains a concussion (Day 1), it has been shown that concussed individuals display postural instabilities, including swaying completing a dual task, such as walking and spelling a simple word backwards.[17, 40] The individual's postural stability is sensitive to internal perturbations after a concussion, that must be stabilized through visual-vestibular feedback and sensory-motor control.[5,17,40] When a disruption occurs to an area of the brain (cerebellum) post- concussion, the nervous symptom can generally only rely on one sense at a time to orient information, and therefore postural instabilities result from visual-vestibular and sensory-motor conflict.[5] During this time, postural sway occurs both in the

medial-lateral directions, as well as in the anterior-posterior direction during these dual tasks.[17, 40] All concussed athletes face their own brain and body disruptions and conflicts that happen in each athlete during Day 1, but each to a different extent; therefore it is important to examine this specific day in the recovery process.

BESS is a postural stability test that is used to test concussed athletes, to determine stability. As a widely used concussion testing tool, athletes establish their baseline during their pre-season.[4, 9, 14] Concussed athletes must pass BESS, among other concussion tests, in order to start return to participation protocol for a concussion. BESS was found to have a significant practice affect after repeated administrations.[21] It is important to consider if another type of postural control assessment could potentially lessen practice effects and be more accurate in determining if an athlete has recovered from a concussion.

When a concussed athlete is finally cleared to return to participation, they should have passed postural stability/control tests, as well as neurocognitive and neuropsychological tests, and be symptom-free. However, recent studies have found concussed athletes do not recover their postural control within one-month post-concussion[19], even though most concussed athletes are returned to participation in 7 to 10 days.[14,44] Parker et al. found that sub-concussive blows an athlete faces from participation in their sport may delay recovery of postural control. Parker et al. found that concussed athletes had the slowest gait velocity during dual tasks, even slower than non-concussed athletes or concussed non-athletes.[17,36-37, 40-41] It could be hypothesized that these sub-concussive blows produce clinical impairments seen during single task walking, so that concussed and non-concussed athletes alike suffer greater gait impairments than their non-concussed and concussed non-athlete counterparts. [17,36-37,40-41] (Non-athletes suffer concussions;

however, they do not receive subconcussive blows like an athlete would experience on a daily basis due to the nature of the sport.)

The purpose of this study was to identify variations in gait patterns on specific days of the recovery process following a concussion, as compared to athletes who were concussion free and non-athletes. These days include when the athlete reaches: Day 1, return to BESS baseline, and returning to participation. It was hypothesized that CONC subjects would adopt a conservative gait strategy, demonstrating slower gait velocity and cadence, smaller step length and wider step width, and longer time in double leg support during the gait cycle. It was also expected that the non-concussed athletes would have a different gait velocity, cadence, step length, step width, and double leg support times than the normal controls. In other words, concussed athletes would show a more conservative gait strategy than the other two groups.

CHAPTER 2

METHODS

Subjects

There were 45 subjects (27 female) recruited from the Georgia Southern University

student body, divided into three groups: 1)15 NCAA Division I intercollegiate student athletes

who had sustained a concussion (CONC); 2)15 non-concussed NCAA Division I intercollegiate

student athlete teammates (NONC); and 3)15 normal non-intercollegiate student athlete control

subjects (NORM). The NORM subjects were matched to the concussed intercollegiate student

athletes according to gender. The number of CONC subjects from each sport was as follows: 5

women's soccer, 4 football, 2 women's basketball, 1 men's basketball, 1 men's soccer, 1

women's track, 1 female cheerleader. Only the CONC football subjects were matched with a

teammate according to a similar position, due to the potential head impacts that are position-

based in the sport.[12, 45] One subject, a female soccer NONC withdrew from the study due to

injury and was not tested on RTPD. Complete subject demographics are provided in Table 1

(Appendix C).

Recruitment of CONC subjects was upon referral from the athletic training staff of

Georgia Southern University. The CONCs were enrolled within 24 hours of sustaining a

concussion. The NONCs were also referred by the athletic training staff of Georgia Southern

University. If the subject chose to participate in the study, they received the consent form

(Appendix D), and a concussion medical history form (Appendix D), and then provided written

informed consent prior to participation.

The inclusion criteria for the NONC and NORM included no history of a concussion

within the past 6 months, so both groups were healthy controls for CONCs.[46] Exclusion criteria

was obtained through a concussion medical history form for the NONC and NORM subjects. Participants were excluded from the study if they had a history of vestibular or balance disorders; cardiac or respiratory disease; history of neurophysiological disorders; recent ongoing injury to the lower extremity; lower limb dyskinesia; any gait ambulatory aid; and pain with walking.

Instrumentation

The GAITRite[®] Platinum mat (CIR Systems INC., Havertown, PA 19083) was used to collect the gait data. The GAITRite[®] is an instrumented carpet 4.9 m long, 0.902 m wide, and 0.635 cm thick, with approximately 18,432 pressure sensors placed within 1.27 cm of each other, arranged in a 48 x 384 grid.[47] The GAITRite[®] has been shown to have strong concurrent validity and test-retest reliability,[48] with intra-class correlations greater than 0.95 for spatial measurements, and greater than 0.93 for temporal measures.[49] Walking velocity, cadence, and step length were found to have excellent reliability, with intra-class correlations between 0.82 and 0.92.[50]

The sensors are imbedded in the mat and provide immediate calculation of the dependent variables of interest, including velocity, cadence, step length, step width, and double leg support time.

Procedure

After informed consent was obtained (Appendix D), the subject completed a concussion medical history questionnaire (Appendix D), to ensure inclusion and exclusion criteria were met. The subjects height, weight, leg length and shoe size were measured and recorded. Leg length (cm) was defined as the distance from the anterior superior iliac spine of the hip to the center of the medial malleolus of the same limb; leg length was measured bilaterally to note any leg length discrepancies.

Subjects began the trials barefoot and standing on two force plates as their "starting mark" located 1.2 m before the GAITRite® walkway (Appendix E). In response to a verbal cue, the subject initiated a normal walking pace and traversed the 4.9 m length of the GAITRite® to a target located approximately 5 m past the end of the walkway. The subject took approximately one step before reaching the GAITRite® walkway. A "normal walking pace" was operationally defined as how the subject would walk at a comfortable pace on a daily basis. The subject was instructed that the "normal walking pace" be how they would walk if they were not in a rush to class and not talking with friends during walking. If the subject appeared to be walking too slow or too fast, the researcher redefined "normal pace" again, and demonstrate what a "normal pace" walking looked like. The trial would then be redone. The subject then turned around, stood at a line 3.6 m from the end of the walkway, in response to a verbal cue, traversed the GAITRite® mat and terminated their gait on the force plates located at the immediate end of the walkway. A total of ten trials were performed; five trials walking away from the starting mark, and five trials walking towards the starting mark. All subjects were tested daily until the matched concussed athlete was cleared to participate by the athletic training staff and the team physician.

Three specific time points were measured for all subjects, which included day one (CD1), when BESS baseline is reached by the concussed athlete (BBD), and the day the concussed athlete returns to participation day (RTPD). These specific days were picked as significant days in a CONC's recovery, where the CONC might show important changes in gait.

CD1 is within 24 hours of the individual sustaining the concussion. BBD is defined as the day the individual score on the BESS test reaches or exceeds their baseline measurement, which was taken during physicals. When an individual returns to baseline on BESS, it is assumed their postural stability returns to normal.[51] The BESS assessment was performed by two

individuals who have had significant expertise and experience successfully administering the test over a number of years. RTPD is when the subject has been cleared to return to participation, because they have returned to baseline on BESS, as well as cognitive and neuropsychological testing. The subject must also have successfully passed a six day return to play protocol that gradually increases exercise, in order to ensure the athlete has no residual symptoms.

Data Analysis

The GAITRite® software triggers data collection to start once the individual steps on the active area of the walkway. The GAITRite® system calculates standard spatiotemporal gait variables including; velocity, cadence, step length, step width, and double leg support time. The gait variables were defined by GAITRite® as follows. Gait velocity (m/s) is determined by GAITRite® as the displacement traveled by the individual divided by ambulation time, or the time between the first and last footfalls.[52] Cadence (steps/min) is the number of steps taken over a period of time.[27] Step length (cm) is the position of initial contact of a footfall to the position of the initial contact of next footfall of the opposite foot.[52-53] Step width (cm) is measured from the midline of the midpoint of a footprint to the midline of the midpoint of the opposite foot.[53] Double leg support time (s) is when both feet are on the floor, from heel contact of one footfall to toe-off of the opposite footfall.[52]

Statistical Analysis

Five 3x3 way Repeated-Measure ANOVAs (group x day) were utilized to determine if there were any differences between groups on gait performance for velocity, cadence, step length, step width, and double leg support time on a specific day . Tukey post-hocs were run with contrasts to identify significance between days of the recovery process.

Fifteen One-Way ANOVAs were run as a post hoc follow-up to determine if there are any differences between groups on a specific day. Tukey post-hoc testing was administered to determine if there were any significant differences. The alpha level was set at .05.

CHAPTER 3

RESULTS

CONCs returned to their BESS baseline an average of 1.8 ± 1.2 days after suffering a concussion, and started return to play protocol after an average of 5.2 ± 2.0 days. CONCs returned to play in practice or competition an average of 10.8 ± 2.9 days after sustaining a concussion.

Gait Velocity

Gait velocity at CD1 for CONC, NONC, and NORM were 1.21 ± 0.16 m/s, 1.31 ± 0.14 m/s, and 1.34 ± 0.08 m/s, respectively. CONC, NONC, and NORM gait velocity at BBD were 1.28 ± 0.19 m/s, 1.34 ± 0.14 m/s, and 1.39 ± 0.09 m/s, respectively. Finally, at RTPD, CONC, NONC, and NORM had gait velocities of 1.34 ± 0.17 m/s, 1.34 ± 0.10 m/s, and 1.38 ± 0.10 m/s, respectively (Appendix C, Figure 1).

There was no significant interactions for gait velocity between groups and days ($F=1.395$, $p=.254$). Therefore, there were no significant differences for gait velocity between CONC and NONC, CONC and NORM, or NONC and NORM from CD1 to BBD, BBD to RTPD, or from CD1 to RTPD. However, there was a significant main effect for day for subjects ($F=8.111$, $p=.002$), whereby all subjects increased their gait velocity from CD1 to BBD ($F=18.940$, $p<.001$), and from CD1 to RTPD ($F= 11.901$, $p=.001$). On average the groups did not increase gait velocity from BBD to RTPD ($F=1.353$, $p=.252$) There was no significant effect between the groups during these specific days ($F= 2.223$, $p=.121$).

At CD1, there were significant differences between the groups for velocity ($F= 3.670$, $p=.034$). Tukey post-hoc showed that CONCs had a mean gait velocity statistically significantly less than the NORMs (1.21 ± 0.16 m/s and 1.34 ± 0.08 m/s respectively, $p=.036$). However,

there were no differences for gait velocity between CONCs and NONCs (1.31 $\pm$ 0.14 m/s, p=.117), or NONCs and NORMs (p=.854). At BBD and RTPD, there were no differences in gait velocity amongst CONCs, NONCs, or NORMs (F= 1.951, p=.115 and F=.468, p=.630, respectively).

Cadence

Cadence at CD1 for CONC, NONC, and NORM were 110.95 $\pm$10.35 steps/min, 113.3 $\pm$ 7.88 steps/min, and 117.31 $\pm$ 6.15 steps/min, respectively. CONC, NONC, and NORM cadence at BBD were 113.07 $\pm$ 10.14 steps/min, 115.03 $\pm$ 7.35 steps/min, and 119.09 $\pm$ 6.11steps/min, respectively. Finally, at RTPD, CONC, NONC, and NORM had cadences of 116.11 $\pm$ 9.14 steps/min, 116.02 $\pm$ 7.19 steps/min, and 117.69 $\pm$ 5.81 steps/min, respectively. (Appendix C, Figure 2).

There was no significant interaction for cadence between groups (F=1.788, p=.159). Therefore, there were no significant differences for cadence between CONC and NONC, CONC and NORM, or NONC and NORM from CD1 to BBD, BBD to RTPD, or from CD1 to RTPD. There was not a significant day effect for subjects (F =2.020, p=.152). There was no significant effect between the groups during these specific days (F=1.769, p=.183).

At CD1, there were no significant differences among CONCs, NONCs, or NORMs for cadence (110.95 $\pm$ 10.35 steps/min, 113.30 $\pm$ 7.88 steps/min, 117.31 $\pm$ 6.15 steps/min, respectively, F=2.242, p=.119). At BBD, there were no differences between groups (113.07 $\pm$ 10.14 steps/min, 115.03 $\pm$ 7.35 steps/min, 119.09 $\pm$ 6.11 steps/min, respectively, F = 2.182, p=.125). Finally, at RTPD, there were no significant differences between groups (116.11 $\pm$ 9.14 steps/min, 116.02 steps/s $\pm$ 7.19 steps/min, 117.69 $\pm$ 5.81 steps/s, respectively, F=.232, p=.794).

Step length

Step length at CD1 for CONC, NONC, and NORM were 65.6 ± 5.1 cm, 69.6 ± 5.0 cm, and 68.6 ± 4.0 cm, respectively. CONC, NONC, and NORM step lengths at BBD were 67.9 ± 6.4 cm, 70.4 ± 5.5 cm, and 70.0 ± 3.8 cm, respectively. Finally, at RTPD, CONC, NONC, and NORM had step lengths of 69.4 ± 6.1 cm, 70.7 ± 4.4 cm, and 70.2 ± 4.7 cm, respectively. (Appendix C, Figure 3).

There was no significant interaction for step length between groups (F=.928, p=.431). Therefore, there were no significant differences for step length between CONC and NONC, CONC and NORM, or NONC and NORM from CD1 to BBD, BBD to RTPD, or from CD1 to RTPD. However, there was a significant main effect for day for subjects (F=7.513, p=.003), whereby subjects increased their step length from CD1 to BBD (F=16.542, p<.001), and from CD1 to RTPD (F=10.553, p=.002). There were no significant differences from BBD to RTPD (F= 1.243, p=.271). There was no significant effect between the groups during these specific days (F=1.175, p=.319).

At CD1, there were statistical trends in step length (65.6 ± 5.1 cm, 69.6 ± 5.0 cm, 68.6 ± 4.0 cm respectively, F=2.924, p=.065). However, Tukey post hoc showed that there were no differences between CONCs and NONCs (p=.063), and CONCs and NORMs (p=.201), and NONCs and NORMs (p=.832). At BBD, there were no significant differences in step length between CONCs, NONCs, and NORMs (67.9 ± 6.4 cm, 70.4 ± 5.5 cm, 70.0 ± 3.8 cm respectively, F =.921, p=.406). At RTPD, there were no significant differences in step length between CONCs, NONCs, and NORMs (69.4 ± 6.1 cm, 70.7 ± 4.4 cm, 70.2 ± 4.7 cm respectively, F=.248, p=.781).

Step width

Step width at CD1 for CONC, NONC, and NORM was 12.3 $\pm$ 3.9 cm, 11.9 $\pm$ 4.0 cm, and 11.5 $\pm$ 3.2 cm, respectively. CONC, NONC, and NORM step width at BBD was 12.2 $\pm$ 3.8 cm, 11.9 $\pm$ 3.7 cm, and 11.5 $\pm$ 3.2 cm, respectively. Finally, at RTPD, CONC, NONC, and NORM had step widths of 11.8 $\pm$ 4.0 cm, 10.5 $\pm$ 3.1 cm, and 10.5 $\pm$ 2.8 cm, respectively. (Appendix C, Figure 4).

There was no significant interaction for step width between groups (F=1.116, p=.342). Therefore, there were no significant differences for step width between CONC and NONC, CONC and NORM, or NONC and NORM from CD1 to BBD, BBD to RTPD, or from CD1 to RTPD. There was a significant main effect for day for subjects (F=12.894, p<.001), whereby subjects had a significant decrease from both BBD to RTPD (F=15.773, p<.001), and from CD1 to RTPD (F=11.976, p=.001). However, there were no significant decreases from CD1 to BBD (F=.004, p=.948). There was no significant effect between the groups during these specific days (F=.315, p=.731).

At CD1, there were no significant differences between groups for step width (12.3 $\pm$ 3.9 cm, 11.9 $\pm$ 4.0 cm, 11.5 $\pm$ 3.2 cm respectively, F=.155, p=.857). At BBD, there were no significant differences between groups for step width (12.2 $\pm$ 3.8 cm, 11.9 $\pm$ 3.7 cm, 11.5 $\pm$ 3.2 cm, respectively, F = .177, p=.839). At RTPD, there were no significant differences between groups for step width (11.8 $\pm$ 4.0 cm, 10.5 $\pm$ 3.1 cm, 10.5 $\pm$ 2.8 cm, respectively, F=.826, p=.445).

Double leg support time

Double leg support times at CD1 for CONC, NONC, and NORM were 0.26 $\pm$ 0.052 s, 0.24 $\pm$ 0.047 s, and 0.23 $\pm$ 0.028 s, respectively. CONC, NONC, and NORM double leg support

times at BBD were 0.25 $\pm$ 0.051 s, 0.23 $\pm$ 0.043 s, and 0.22 $\pm$ 0.028 s, respectively. Finally, at

RTPD, CONC, NONC, and NORM had double leg support times of 0.24 $\pm$ 0.042 s,

0.23 $\pm$ 0.027 s, and 0.22 $\pm$ 0.030 s, respectively. (Appendix C, Figure 5).

There was no significant interaction for double leg support times between all groups

(F=.628, p=.584). Therefore, there were no significant differences for double leg support time

between CONC and NONC, CONC and NORM, or NONC and NORM from CD1 to BBD, BBD

to RTPD, or from CD1 to RTPD. There was a significant main effect for day for all subjects

(F=7.995, p=.003), whereby all subjects decreased their double leg support times from CD1 to

BBD (F=14.395, p<.001), and from CD1 to RTPD (F=10.583, p=.002). There were not

significant increases from BBD to RTPD (F=2.769, p=.104). There was no significant effect

between the groups during these specific days (F=1.552, p=.224).

At CD1, there were no significant differences between groups for double leg support time

(0.26 $\pm$ 0.052 s, 0.24 $\pm$ 0.047 s, 0.234 $\pm$ 0.029 s respectively, F=1.696, p=.196). At BBD, there

were no significant differences between groups for double leg support time (0.25 $\pm$ 0.052 s, 0.23

$\pm$ 0.043 s, and 0.22 $\pm$ 0.028 s, respectively, F = 1.613, p=.211). At RTPD, there were no

significant differences between groups (0.24 $\pm$ 0.042 s, 0.23 $\pm$ 0.027 s, 0.22 $\pm$ 0.030 s,

respectively, F=.648, p=.528).

CHAPTER 4

DISCUSSION

Research Hypothesis

The purpose of this study was to identify variations in gait patterns on specific days of the recovery process following a concussion, as compared to athletes who were concussion free and non-athletes. It was hypothesized that CONC subjects would adopt a conservative gait strategy, demonstrating slower gait velocity and cadence, smaller step length and wider step width, and longer time in double leg support during the gait cycle. However, few significant differences were identified in walking characteristics between groups on the specific days investigated. Although there was not a significant interaction for any of the groups on the days tested, there was a significant day effect for gait velocity, step length, step width, and double leg support time. Subjects increased their gait velocity and step length from CD1 to BBD, and from CD1 to RTPD, and on average, subjects increased their step width and decreased their double leg support time from CD1 to RTPD. Increases in gait velocity, step length, step width, and decreases in double leg support times over the days tested possibly indicates that the CONC, NONC, and NORM subjects adopted a conservative gait strategy at the beginning of the study, then improved their gait characteristics until the day the specific CONC subject returned to participation.

Gait Velocity

It was hypothesized that CONC subjects would initially have the slowest gait velocity, gradually increasing their gait velocity to that of NORMs and NONCs by the time they reached RTPD. The results indicated that CONCs had a mean gait velocity significantly less than that of

NORMs at CD1, and that all subjects increased their gait velocity from CD1 to BBD, and from CD1 to RTPD.

Gait velocity in individuals who suffered a concussion has been shown to be slower than that of their non-concussed counterparts.[17, 36-37, 40-41] The CONC subjects in this study had similar gait velocities (1.28 m/s) to a series of previous studies (1.22 m/s - 1.25 m/s) investigating changes in gait characteristics following a concussion.[17, 36-37, 40-41] Parker et al. tested gait velocity for athletes on day 14 post-concussion, which by this time, the majority of the CONCs had their RTPD (average of 10.7 days for RTPD). Average walking velocity for Parker et al.'s concussed athletes during day 14 was 1.32 ± 0.09 m/s[17, 40-41]; comparably, mean gait velocity for CONCs in this study was 1.34 ± 0.17 m/s at RTPD.

Conversely, substantial differences were noted between this study and Parker et al.'s study for the non-concussed athlete groups (1.31 m/s – 1.34 m/s compared to 1.22 m/s - 1.28 m/s, respectively). [17, 36-37, 40-41] Non-concussed athletes in Parker's et al.'s studies had an average velocity of 1.22 ± 0.134 m/s at day 2, compared with NONCs in this study at 1.34 ± 0.14 m/s during BBD.[17, 40-41] As well, non-concussed athlete in Parker's et al.'s studies at day 14 had a mean gait velocity of 1.27 ± 0.14 m/s, compared with NONCs in this study at 1.34 ± 0.10 m/s during RTPD.[17, 40-41]

NONCs in this study at BBD, as well as RTPD walked faster than non-concussed athletes at day 2 in Parker et al.'s study.[40] It may be that Parker et al.'s non-concussed athletes had a greater number of sub-concussive blows, as well as multiple concussions, potentially leading to slower gait velocity. [17, 36-37, 40-41] The majority of athletes in Parker et al.'s study were from contact sports such as football, rugby, and lacrosse; whereas in this current study, the majority of the concussed were women's soccer players (n = 5) and football athletes (n = 4). Parker et al.'s

studies saw greater differences between non-concussed athletes and non-concussed controls possibly due to the athletic population tested. For instance, 13 out of 15 athletes in this study were involved in Division I NCAA contact sports (except 1 track, 1 cheerleader). Parker et al.'s 14 concussed athletes consisted of Division I NCAA or University Club sport athletes.[17, 40-41] Sub-concussive blows and concussion could differ from club sports and NCAA sports; this study did not include sports such as lacrosse, rugby, or hockey, which could all have been represented in Parker et al.'s studies. The number of concussions and sub-concussive blows could be dependent on the athlete's sport; those requiring more contact would seem to increase sub-concussive blows as well as resulting in a higher rate of concussions. The higher rate of sub-concussive blows and concussions for a sport could result in the majority of differences from Parker et al.'s study to this study. Although sub-concussive blows and incidence rates of concussion are higher in those of football, women's soccer has the highest rate of concussions amongst female sports.[54] The 4 CONC football players in this study were matched NONCs of similar position because sub-concussive impacts are similar in magnitude within a similar position.[55] For instance, a skills player, such as a quarterback, will receive a different number and magnitude of sub-concussive impacts than an offensive lineman. Sports distribution of the athletes enrolled in the study could potentially have an effect on gait characteristics, possibly due to the number of sub-concussive blows and concussions in which heavier contact sports are more prone.

Impairments the athlete suffers are not noticeable enough to be acutely diagnosed, although with multiple concussions and sub-concussive blows over a period of time, the athlete may have a decrease in postural control.[17, 40-41] Athletes not reporting every concussion, as well as sub-concussive blows during an athlete's career are hard to objectively measure, which could

mean decreases in postural control and gait velocity for every athlete may be different. Some athletes may not even demonstrate slower gait velocity. Therefore, it is only speculation that sub-concussive blows and multiple concussions could potentially lead to slower gait velocity and changes in gait characteristics that lead to decreased postural control.

It is possible that concussion severity could have differed greatly in this study compared with previous studies. For example, Parker et al. used the AAN grading scale, whereby all concussed subjects had a Grade 2 concussion.[17, 40-41] This indicates that those athletes had transient confusion, no loss of consciousness, and signs and symptoms lasting greater than 15 minutes.[14, 56] The CONCs in this study were not limited to Grade 2 concussions according to the AAN; this study used the Cantu Revised Scale. Therefore, the CONC subjects in this study likely experienced some transient confusion, whereas others did not; and some had symptoms less than or greater than 15 minutes. This could potentially be why NONCs did not have slower gait velocity than NORMs in this study compared with Parker et al.'s study. [17, 40-41]

Motivation and setting are other factors that could have influenced the gait variables. For example, most subjects in this study increased their gait velocity, step length, step width, and double leg support times in this study. Once the subjects were comfortable with the testing environment, they seemed to gain confidence in walking down the mat. Even though the researcher would instruct the subject if they started to walk faster, minor gait velocity changes could not be detected visually by the researcher.

This study found differences for gait velocity between CONC and NORM subjects at CD1 and BBD, showing that postural instability issues were greatest for these groups between these days, an average of 1.8 days post-concussion. The NONCs and NORMs in this study and previous studies had similar mean gait velocity (1.39 m/s as compared to

1.32 m/s - 1.38 m/s).[17, 40-41] Normal gait velocity in healthy adults is 1.37 m/s, men averaging closer to 1.43 m/s, and women averaging closer to 1.28 m/s.[46-48] Murray et al. found mean values of 1.33 m/s – 1.52 m/s for healthy men, and 1.22 m/s – 1.35 m/s for women.[46-48] Non-concussed non-athletes in Parker et al.'s studies had a mean gait velocity of 1.42 ± 0.11 m/s at day 14, comparable with the NORMs in this study at 1.37 ± 0.10 m/s during RTPD.[17, 40-41]

This study found that at RTPD, CONC subjects had gait velocity comparable with NONCs and NORMs, possibly indicating at RTPD CONC subjects have recovered, and their gait velocity returned to that of the non-concussed groups. When the CONC subjects returned to their baseline on BESS, their gait velocity was only 1.28 m/s, compared to NONCs at 1.33 m/s and NORMs at 1.39 m/s.

Gait velocity was only significantly slower for the CONCs when compared with NORMs. The CONCs at CD1 had an average velocity of 1.21 ± 0.16 m/s, increasing their velocity to 1.34 ± 0.17 m/s at RTPD. These velocities were not significantly slower than NORMs from CD1 until RTPD, with an average velocity of 1.34 ± 0.09 m/s and 1.37 ± 0.10 m/s, respectively. This current research study suggests that single-task gait might not be as good an indicator of postural deficits during recovery from a concussion compared with dual-task gait. It might not be as a specific a measure to determine when an athlete has recovered, because the only significant difference found in the study was that CONCs increased their gait velocity compared to NORMs from CD1 to BBD. Dual task gait, where the individual completes a verbal task while walking, as well as obstacle avoidance, have been shown in previous studies to be good predictors of athletes who suffer from their concussion and their recovery.[17, 36-41]

Single task walking alone may not be the best predictor on a concussed athlete's recovery. The supraspinal centers associated with gait may not be adversely affected or

sufficient compensatory mechanisms may exist.[28, 42] When adding additional tasks or increasing

task difficulty, gait becomes more challenging. Dual tasks are more challenging because they

require additional attention, interfering with active sensory feedback; it has been indicated that

balance control, when attention is divided, can be impaired for up to one month after a

concussion.[17, 40-41]

CONCs in this study increased their average normal walking velocity from 1.23 ± 0.18

m/s at CD1 to 1.34 ± 0.17 m/s at RTPD, indicating that subjects had a slower gait velocity than a

normal population of healthy adults at CD1, but finally reached normal gait velocity at RTPD.

Measuring velocity on the GAITRite® could possibly determine when a concussed athlete is

able to return to participation, just as a variety of neuropsychological, cognitive, postural

stability, and symptom resolution indicate.

Cadence

It was hypothesized that CONCs would have slower candence than that of NONCs or

NORMs until they returned to participation. However, the results indicated that CONCs,

NONCs, and NORMs had similar cadences throughout the time points.

Cadence was measured indirectly as stride time in Parker et al.'s studies, and was not

found in the literature for concussed athletic individuals.[17, 40-41] Cadence showed no significant

differences between the groups at any time point. These results were possible due to several

reasons. Cadence is the rate at which steps are taken, and is related to velocity and stride

length.[53] Increasing velocity is due to increasing step length, step rate, or step length and step

rate. Velocity among the CONCs and NORMs showed significant differences at CD1, meaning

CONCs decreased their velocity compared the NORMs. However, in Parker et al.'s study,

although not significant, step length decreased due to a conservative gait strategy adopted by the

concussed athletes.[17, 40-41] If step length or step rate decreased, stride length would decrease, and therefore one would expect cadence to decrease. This was not found to be the case. It might be that CONCs took more steps that were shorter, increasing gait velocity from CD1 to BBD, compared with NORMs.

Mean cadences for healthy adults is approximately 113 steps/min,[53] comparable with NORMs who had a cadence of 117.31 $\pm$ 6.15 steps/min at CD1. At CD1, CONCs and NONCs had cadences of 110.95 $\pm$10.35 steps/min and 113.3 $\pm$ 7.88 steps/min. Several studies have examined traumatic brain injuries in individuals that have been impaired for several years, both physically and cognitively. Traumatic brain injured individuals showed mean cadences of 67 $\pm$ 30 steps/min for males, and 73 $\pm$ 43 step/min for females.[27] Those with traumatic brain injuries have much slower cadences comparative to CONCs at CD1, the slowest cadences recorded among the groups. Concussed athletes, who suffer from a mild traumatic brain injury would likely not suffer the same gait impairments as those with a traumatic brain injuries. CONCs seemed to have cadences comparable to healthy adults, even at CD1, BBD, and RTPD. Healthy adults (113 steps/min) [53] had cadences comparable with NONCs and NORMs as well on CD1, BBD (115.03 $\pm$ 7.35 steps/min and 119.08 $\pm$ 6.11steps/min, respectively), and RTPD (116.02 $\pm$ 7.19 steps/min and 117.69 $\pm$ 5.81 steps/min, respectively).

The data suggested that the CONCs did not increase their cadence from CD1 to BBD (110.95 $\pm$10.35 steps/min to 113.07 $\pm$ 10.14 steps/min) and from BBD to RTPD (116.11 $\pm$ 9.14 steps/min), as well as from CD1 to RTPD. This has been shown to be statistically insignificant. NONCs and CONCs showed relatively the same cadence over CD1, BBD, and RTPD, and it was not found to be significant.

Step length

It was hypothesized that step length would be shorter for CONCs, and NONCs and NORMs would have longer step lengths, and then CONCs would increase their step length to that of NONCs and NORMs by RTPD. However, there were no significant differences between the groups over the time points. There was a significant day effect for the groups, whereby the groups increased their step length from CD1 to BBD, and from CD1 to RTPD.

Step length was not found to be a variable previously studied in concussion literature; stride length is more commonly used, as it was in Parker et al.'s studies.[17, 40-41] Step length, if leg length and stride is normal, should be half that of a stride length.[53] Step length at CD1 for CONC, NONC, and NORM were 65.6 $\pm$ 5.1 cm, 69.6 $\pm$ 5.0 cm, and 68.6 $\pm$ 4.0 cm, respectively. CONC, NONC, and NORM step lengths at BBD were 67.9 $\pm$ 6.4 cm, 70.4 $\pm$ 5.5 cm, and 70.0 $\pm$ 3.8 cm, respectively. Finally, at RTPD, CONC, NONC, and NORM had step lengths of 69.4 $\pm$ 6.1 cm, 70.7 $\pm$ 4.4 cm, and 70.2 $\pm$ 4.7 cm, respectively. There were no significant differences between any of the groups on any of the time points studied. However, there was a significant main effect for day for subjects, whereby subjects increased their step length from CD1 to BBD, and from CD1 to RTPD.

Parker et al. found that stride length was longer for non-athlete, non-concussed controls on Days 2 and 14, compared to concussed athletes.[17, 41] Likewise, it would be predicted that step length would be longer for NORMs than CONCs on CD1 or BBD or RTPD. However, step length showed no significance between CONCs, NONCs, or NORMs at CD1, BBD, or RTPD.

This could be due to several reasons. Gait velocity in this study was significant at CD1 between CONCs and NORMs, whereby NORMs had greater gait velocity than CONCs. It would seem that NORMs would have greater step lengths to have a greater gait velocity than CONCs. However, this is not true. NORMs had similar step lengths to CONCs, but greater gait

velocity at CD1, possibly because their stride time was shorter covering the distance of the walkway. In patients with traumatic brain injuries, mean step lengths have been found to be 62 $\pm$ 15 cm, compared with CONCs having a step length of 65.6 $\pm$ 5.1 cm at CD1.[27] Even though those with traumatic brain injuries have step length comparable to CONCs, there were no significant differences at CD1 between groups.

Healthy young adults have step lengths of 77.61 $\pm$ 5.73 cm and 77.24 $\pm$ 6.16 cm for right and left legs, respectively; [50] another study showed the same with subjects having step length of 76.0 $\pm$ 12.8 cm for males, and 69.5 + 6.0 cm for females at a "preferred walking speed".[57] It seems that this study has relatively smaller step lengths for NORMs compared with the healthy, young adults of the other studies.[50, 57] The average height in these studies was 171.8 cm for both males and females, and in the other study the average heights were 172.8 cm for males and 157.0 cm for females.[50, 57] Average height in this study was 171.7 $\pm$ 10.6 cm, which is comparable to the other studies, indicating that height should not have made any differences in step length. Leg length was not available for either study.[50, 57] Both of these previous studies only examined healthy individuals without impairments or injury.   It could be that the aforementioned study recruited twice as many normal subjects as this study (30 individuals comparative with 15 non-concussed non-athletes).[50] More subjects could translate into more significant findings, not only in step length, but for velocity, cadence, step width, and double leg stance times.

Step width

It was hypothesized that CONCs would have longer step widths than NONCs or NORMS, and NONCs and NORMs would have shorter step widths over the time points until they reached RTPD. However, the groups had no significant differences for step width at the

time points studies. There was a significant day effect for the groups, whereby the groups' step width decreased from both BDD to RTPD, and from CD1 to RTPD.

Step width has been studied before in concussion gait studies, such as Parker et al.'s, but has not yet been studied over this study's specific time points in the course of a concussed athlete's recovery.[17] In this study, NORMS had a step width of 11.5 ± 3.2 cm at BBD, comparable to Parker et al.'s study with healthy controls having a step width of 11.4 ± 3.5 cm.[17] Step width for healthy males at normal walking speeds in one study was found to be 9.0 ± 3.5 cm, and for females 7.0 ± 2.6.[57] The researches stated that "preferred" walking speeds in healthy individuals, average age of 25.9 years for males and 20.8 years for females, would have this step width.[57] The age groups are comparable to this study, subjects having an average age of 20.2 years between all groups. Height measurements were likewise similar, with 172.8 cm for males and 157.0 cm for females in the previous study, and this study had an average of 179.3 for males and 151.8 cm for females between all subjects.[57] Likewise, Murray et al. reports a healthy step width to be 8.6 ± 3.4 cm for males, and 6.9 ± 2.9 cm for females.[58-60] Murray et al. studied various adults who were able to walk without an ambulation device and had no history or vestibular or balance disorders.[62-63] They did not just focus on young, healthy adults as the control group, which could be the reason why the NORMs had greater step width than previous studies.

This study had normal walking speeds that were faster than speeds of the previous study, which subjects walked at their "fastest" pace. This could be due to several reasons. Even though the previous study had a walkway, it measured ink dots from the felt-applied squares the subjects wore.[57] This is another way to measure step width, although not as good a method as the GAITRite®, which has high test-retest reliability and strong concurrent validity.[48-49] Subjects

from this current study, even as they were instructed on the term "normal" walking speed for the first few sessions, were not instructed every time down the GAITRite® walkway. This could make the subjects have different step width than the previous study.

Those in the NORM group theoretically should have not increased or decreased their step width over time, because they are the controls. Step width for NORMs showed no significance from CD1 (11.5 ± 3.6 cm) to BBD (11.5 ± 3.2 cm) or RTPD (10.5 ± 2.8 cm). Step width also showed no significant differences for the NORMs between CONCs or NONCs on any of the following days studied. This data confirms that the NORMs showed no differences for step width between any of the other groups on any of the days studied.

Likewise, healthy controls during day 14 had a step width of 0.117 ± 0.036 m, comparable to NORMs at RTPD had a step width of 10.5 ± 2.8 cm (0.105 + 0.028 m).[17] Concussed subjects in Parker et al.'s study at day 2 had a step width of 0.125 ± 0.062 m, comparable with CONCs at BBD with a step width of 12.2 ± 3.8 cm (0.122 ± 0.038 m).[17] Likewise, concussed subjects at day 14 had a step width of 0.118 ± 0.042 m, comparable with CONCs at RTPD with a step width of 11.8 ± 4.0 cm (0.118 ± 0.004 m).[17] Therefore, the step widths found in this study are like Parker et al.'s results. Not only did this study find no significant differences in step width, but Parker et al. did not find any either, which suggests that step width may not be a gait variable involved in determining gait variations during a concussion.

As for NONCs and CONCs in this study, at CD1 their step width was 12.3 ± 3.9 cm and 11.9 ± 4.0 cm, respectively. Traumatic brain injured subjects are noted to have a step width of 9 ± 6 cm for males and 10 ± 6 cm for females.[27] Logically, one would expect NONCs and CONCs to have smaller step widths than those suffering from a traumatic brain injury. In fact, the

NONCs and CONCs showed no differences between themselves or NORMs at any of the time points studied. This could potentially mean that step width is a good variable for testing traumatic brain injury patients, but not specific enough to measure those athletes who have sustained concussions. Likewise, step width might be a better variable to test athletes in the future who have suffered multiple sub-concussive or concussive blows.

Double leg support time

It was hypothesized that CONCs would have longer double leg support time than NONCs or NORMs throughout the time points until the CONC reaches RTPD. However, there were no significant differences for double leg support time between the groups at the time points. There was a significant day effect for the groups, whereby the groups decreased their double leg support times from CD1 to BBD, and from CD1 to RTPD.

Healthy controls should have a double leg support time of 0.21 s during a normal walking speed, according to one study.[57] Double leg support times for NORMs at CD1 were 0.26 ± 0.052 s, at BBD were 0.22 ± 0.028 s, and at RTPD were 0.22 ± 0.030 s. The NORMs in this study are comparable to the healthy controls in the other study. Likewise, NORMs did not show significant differences between test sessions. This indicates that NORMs did not change their double leg support times over time, which they would not as the normal controls.

Double leg support times did not significantly increase or decrease between test sessions. This could indicate several things. Firstly, double leg support times of healthy individuals were similar to those of NONCs and CONCs at CD1 (0.26 ± 0.052 s and 0.24 ± 0.047 s, respectively), as well as BBD (0.25 ± 0.052 s and 0.24 ± 0.043 s, respectively), and finally RTPD (0.24 ± 0.042 s and 0.23 ± 0.027 s, respectively). Double leg support times may not be a good measure in determining whether an athlete is able to return to participation after a concussion.

Although double leg support time measurements are uncommon, and to date have only been used in traumatic brain injury studies, the CONC and NONC groups may not show deficits in this area of gait. Double leg support times of traumatic brain individuals compared to CONCs was 0.18 ± 0.08 s and 0.262 ± 0.052 s, respectively.[26] It seems that in the traumatic brain injury study, those injured, as well as healthy controls, had less double stance time than that CONCs and NORMs in this study.

Limitations

As stated previously, the sample was taken for convenience, and the demographic of CONC might differ from a randomized sample. It should be noted that a female soccer NONC was unable to complete the entirety of the study due to a lower extremity injury. This individual was only missing pertinent data for this study on RTPD of the matched female soccer CONC; however, missing this one time point could have affected the data. This occurred only minimally during the testing period. Lastly, there were two different researchers in this study who tested subjects; even though subject testing was uniform, interaction with the participants could have changed between examiners.

Future Studies

A Hawthorne effect could have taken place, which is when a subject knows they are being tested and reacts to the environment as such. For example, most of the subjects were recruited by the researcher, who initially was unfamiliar with being in a kinesiology laboratory setting. All the subjects could walk; however, their normal walking speeds increased throughout the study. This could indicate that as the subjects became more comfortable with the environment and the researcher, as well as the instructions for the test, they increased their speeds. Although the Hawthorne effect is difficult to control in such studies, it might be ideal to

have all athletes tested so that they become comfortable with the testing situation and the researcher; then when one sustains a concussion, they will be more familiar with their environment. The same applies to the paired non-athlete controls.

Future studies need to examine the impact sub-concussive blows or multiple concussions affect an athlete's gait patterns, particularly several years down the road when the athlete is no longer participating in inter-collegiate sports. This would give insight to whether NONCs and CONCs will eventually show the same gait abnormalities compared to healthy subjects, and whether multiple sub-concussive blows create more deviate gait abnormalities than multiple concussed individuals, compared to normal controls. Same sport studies could determine if the specific nature of certain contact sports have more gait abnormalities due to sub-concussive blows or concussions. Finally, it would be interesting to determine if these sub-concussive blows or concussions can lead to gait abnormalities in the prime of an athlete's life, possibly comparable in an athlete's elderly years like that of a TBI patient.

Conclusion

Gait velocity showed significant differences between CONCs and NORMs on CD1. Previous studies on concussed individuals compared with normal controls have not found significant differences on Day 2, Day 5, or Day 14 for cadence, step length, or step width between concussed and normal controls. Postural stability recovered in this study well before the CONC had their RTPD. In theory, if the BESS score has returned to baseline than the individual's postural control mechanisms have recovered which would likely be associated with a normal and healthy gait pattern. The CONCs in this study passed BESS in an average of 1.8 days. By BBD, CONCs returned to normal gait velocity, indicating that although BESS may not be as sensitive a test as gait measurements when determining where an athlete has recovered

their postural stability and eventually can return to participation, cadence, step length, step width, and double leg support times may not as well. Hence, it is recommended that concussion assessments include neuropsychological and symptom scoring in addition to postural stability testing, such as gait, where the athlete must pass all these tests in order to return to participation.

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APPENDIX A

RESEARCH HYPOTHESIS, DELIMITATIONS, LIMITATIONS, ASSUMPTIONS

RESEARCH HYPOTHESES

Nulls:

- H_{NV1}: Gait velocity will show no significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or CD1 to RTPD.

- H_{NV2}: Gait velocity will show no significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NV3}: Gait velocity will show no significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NV4}: Gait velocity will show no significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{NV5}: Gait velocity will show no significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{NV6}: Gait velocity will show no significant differences between CONCs, NONCs, or NORMs at RTPD.

- H_{NC1}: Cadence will show no significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NC2}: Cadence will show no significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NC3}: Cadence will show no significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NC4}: Cadence will show no significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{NC5}: Cadence will show no significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{NC6}: Cadence will show no significant differences between CONCs, NONCs, or NORMs at RTPD.

- H_{NL1}: Step length will show no significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NL2}: Step length will show no significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NL3}: Step length will show no significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NL4}: Step length will show no significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{NL5}: Step length will show no significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{NL6}: Step length will show no significant differences between CONCs, NONCs, or NORMs at RTPD.

- H_{NW1}: Step width will show no significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NW2}: Step width will show no significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NW3}: Step width will show no significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{NW4}: Step width will show no significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{NW5}: Step width will show no significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{NW6}: Step width will show no significant differences between CONCs, NONCs, or NORMs at RTPD.

- H_{ND1}: Double leg stance time will show no significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{ND2}: Double leg stance time will show no significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{ND3}: Double leg stance time will show no significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{ND4}: Double leg stance time will show no significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{ND5}: Double leg stance time will show no significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{ND6}: Double leg stance time will show no significant differences between CONCs, NONCs, or NORMs at RTPD.

Alternatives:

- H_{AV1}: Gait velocity will show significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AV2}: Gait velocity will show significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AV3}: Gait velocity will show significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AV4}: Gait velocity will show significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{AV5}: Gait velocity will show significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{AV6}: Gait velocity will show significant differences between CONCs, NONCs, or NORMs at RTPD.

- H_{AC1}: Cadence will show significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AC2}: Cadence will show significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AC3}: Cadence will show significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AC4}: Cadence will show significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{AC5}: Cadence will show significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{AC6}: Cadence will show significant differences between CONCs, NONCs, or NORMs at RTPD.

- H_{AL1}: Step length will show significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AL2}: Step length will show significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AL3}: Step length will show significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AL4}: Step length will show significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{AL5}: Step length will show significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{AL6}: Step length will show significant differences between CONCs, NONCs, or NORMs at RTPD.

- H_{AW1}: Step width will show significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AW2}: Step width will show significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AW3}: Step width will show significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AW4}: Step width will show significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{AW5}: Step width will show significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{AW6}: Step width will show significant differences between CONCs, NONCs, or NORMs at RTPD.

- H_{AD1}: Double leg stance time will show significant differences between CONCs and NONCs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AD1}: Double leg stance time will show significant differences between CONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{AD1}: Double leg stance time will show significant differences between NONCs and NORMs from CD1 to BBD, from BBD to RTPD, or from CD1 to RTPD.

- H_{ND4}: Double leg stance time will show significant differences between CONCs, NONCs, or NORMs at CD1.

- H_{ND5}: Double leg stance time will show significant differences between CONCs, NONCs, or NORMs at BBD.

- H_{ND6}: Double leg stance time will show significant differences between CONCs, NONCs, or NORMs at RTPD.

DELIMITATIONS

The sample included Georgia Southern University students and student- athletes (ages 18-25) as a convenience sample.

LIMITATIONS

As stated previously, the sample was taken for convenience, and the demographic of CONC might differ from a randomized sample. It should be noted that a female soccer NONC was unable to complete the entirety of the study due to a lower extremity injury. This individual was only missing pertinent data for this study on RTPD of the matched female soccer CONC; however, missing this one time point could have affected the data. This occurred only minimally

during the testing period. Lastly, there were two different researchers in this study who tested

subjects; even though subject testing was uniform, interaction with the participants could have

changed between examiners.

ASSUMPTIONS

It was assumed that the athletic training staff accurately reported if an athlete sustained a

concussion, and accurately reported when the athlete BBD and RTPD occurred. It is also

assumed that the subjects stated an accurate medical history that would allow them to participate.

APPENDIX B

LITERATURE REVIEW

Concussions

Concussions are defined by the U.S. Center for Disease Control and Prevention as a "complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces secondary to direct or indirect forces to the head".[13, 62] Concussions involve impairment of neurologic function from the result of functional disturbances in the brain, resulting in a different set of symptoms and/or behavior changes for each athlete.[1, 13] No two concussions are alike; in other words, "the degree of metabolic dysfunction, the tissue damage and duration of time need to recover, the number of previous concussions, and the time between injuries" are all very different, and should all be considered when managing an athlete with a concussion.[13]

There are approximately anywhere between 5.5% to 9.2% of high school and collegiate players that sustain a concussion each year.[16, 44, 63-64] There is a higher frequency of concussion occurrence in high school athletes, representing 8.9% of all athletic injuries.[44, 63] However, concussions represented a higher rate among collegiate athletes, even though concussions account for only 5.8% of all athletic injuries.[44, 63] Gessel at el. stated that higher incidence rates of concussions may be due to the intensity of collegiate sports, as well as the increased skill levels and size of players.[16,44, 63] Greater intensity of play can lead to greater impacts to the head, especially in football.[16, 44]

Covassin et al. estimate a 10-fold increase in football concussions for games compared with practices.[64] Approximately 5.1% of football players sustain at least one concussion, and 14.7% sustain a second concussion within the same season.[16] Football players who sustained one

concussion during their season were three times more likely to sustain a second concussion within the same season.[11] Concussions account for 96% of all head injuries.[63]   More concussions occur during completion than practice, 65.4% occurring in competition compared with 34.6% in practice.[44, 63] There is a concussion rate of 9.2% of all practice athletic injuries, and 12.0% of all competition injuries.[63]

The majority, 40.5%, of all concussions occur in football, followed by 21.5% in girls' soccer, then boys soccer comprising 15.4% of all concussions.[44] Girls have a higher rate of concussions, at 0.21 concussions per athletic exposures, comparative with boys at 0.007 concussions per athletic exposure.[44, 64] (An athletic exposure is defined as one athlete's participation in a practice or competition).[44] There are several reasons why girls sustain a higher rate of concussions than boys. Biomechanical differences, such as females having a "smaller head to ball ratios or weaker necks", which can lead to "greater angular acceleration and displacement of the head and neck", leading to an increased rate of concussions in female soccer players.[44] Boys are also less inclined to report concussions than females, maybe because they want to be tough and not removed from participation.[65]

During football, the majority of concussions happened during a running play, whereas in soccer the majority happened during heading the ball in both boys and girls soccer (55.4%, 40.5%, and 436.7%, respectively).[44] Girls soccer has approximately a 10-fold rate of sustaining a concussion from heading a soccer ball than boys soccer.[44] The majority of concussions in men's basketball occurred during rebounding, whereas in women's basketball most occurred during defending (30.5% and 22.2%, respectively).[44] In other sports, such as wrestling, the majority of concussions occurred during takedown (42.6%); in baseball, most concussions occurred during batting (50.6%), whereas in softball they occurred during catching (29.7%).[44]

During a concussion, a neurometabolic cascade of events occur in the brain.[66] First, the impact to the head creates a delpolarization of the brain and action potentials allow for a release of excitatory amino acids.[66] An influx of potassium ions enter the depolarized channels, and the ionic membrane pumps try to restore homeostasis.[66] Hyperglycolysis attempts to restore this, and adenosine triphosphate (ATP) is produced for energy.[66] Lactate accumulates as a byproduct of the ATP useage.[66] An influx of calcium enters the channels and the mitochondria is unable to make glucose, because its oxidative metabolism is impaired.[66] Therefore, there is a decreased energy supply of ATP, and cerebral glucose is decreased, because it is not receiving ATP from the mitochondria as usual.[66] This lack of blood flow to the cerebrum is meant for healing to occur. However, calpain is activated due to the influx of calcium, which starts apoptosis of the neuron.[66] This is followed by further axolemmal disruption and calcium influx, further impairing glycolysis.[66] Neurotransmission between neurons is disrupted, as the axon falls apart during axotomy, or axonal disconnection.[66]

Athletes will suffer several symptoms from this process, most commonly including headache, dizziness, and confusion (40.1%-93.6%, 15.3%-85.1%, 8.6%-83.0%, respectively).[7, 16, 44, 62, 67] Other symptoms that are less common include loss of consciousness, amnesia, disorientation, depression, nervousness, nausea, vomiting, fatigue, irritability, sleep problems, sensitivity to light, and blurred vision.[7, 17, 44, 62, 67] Athletes may experience delayed onset of symptoms, or deny the symptoms so they will not be withheld from play.[22, 67] Loss of conscious (LOC) is thought to occur for a concussion to happen, although this is not true.[6, 68] Loss of consciousness only is involved in 6.3%-25% of all concussions, with 80% of these cases lasting less than 30 seconds.[7, 16, 67] There is no relationship between an athlete's concussion history and the presence of LOC.[7] Lovell et al. found no significant differences during neuropsychological

testing between athletes who suffered LOC and those who did not after sustaining a concussion.[67,68] An athlete who suffers LOC does not represent poorer outcome, nor is it associated with the number or duration of symptoms present during follow-up evaluations.[6, 13, 67, 68] Essentially, it is recommended that LOC not be used as a return to play decision, for other symptoms such as amnesia and confusion represent longer last effects.[9, 13, 68] Cognitive impairment is the most important predictor for the duration of post-concussive symptoms.[67] Amnesia is more predictive of post-concussion difficulties, seen three days post-injury, rather than LOC.[6] Athletes who had cognitive deficits and greater symptoms three days post-concussion were 10 times more likely to exhibit some type of amnesia, compared to concussed athletes who had good presentation 3-days post-injury.[6]

Girl athletes who sustain a concussion have been found 1.7 times more likely to be cognitively impaired than males.[69] Females seemed to experience greater symptomology.[69] This could be due to the fact males were helmets for the sports they play (i.e., football, lacrosse), whereas a female would not.[69] Estrogen may have an impact on females to increase their symptoms.[69] Females also may have greater metabolic demands in the brain, which increase or prolong symptoms due to a decrease in cerebral blood blow with increased need for glucose.[66,69]

One study estimated 30.8% of athletes returned to play the same day that they sustained a concussion, only being held out for an average of 13 minutes.[16] It was found that 33% of athletes who return to play the same day they suffered a concussion experienced delayed onset of symptoms 3 hours post-injury, indicating an athlete may be asymptomatic upon immediate evaluation, but onset of symptoms may be delayed or caused by exertional activities.[20] In fact, neuropsychological deficits may be apparent post-injury that will not be seen on the sideline after an athlete sustains a concussion.[4, 7, 13] This is why it is stated that the athlete should not return to

play the same day they sustain the concussion, even if their symptoms decrease, or neuropsychological and postural stability testing are back to baseline.[4, 13]

If the athlete is withheld from participation, it is likely that they will return to play within 7 to 10 days, which may not be enough time for the athlete to fully recover from their concussion.[13, 44] It was found in one study that 75% of athletes with a concussion who returned to participation prematurely suffered another within 7 days, and 91.7% of those concussed who returned to play too soon suffered another within 10 days.[7] Likewise, another study stated that more than 50% of the athletes in one study returned to play in 9 days or less.[44] Athletes have been shown to return to participation before they have fully recovered, which is why athletes who sustain one concussion in a season are three times more likely to sustain a second concussion in the same season.[16]

That is why it is imperative for each individual should be managed objectively on an individual basis, using a variety of measurements that include neuropsychological evaluation, symptoms checklist, and postural stability testing.[4, 13, 15, 20, 69-70] No single concussion assessment tool should be used to determine an athlete's ability to participate, and the athlete should only be returned when they are asymptomatic on all concussion tests.[4, 13, 15, 71] Younger athletes should be treated more conservatively, not only due to "different physiological responses and longer recovery after a concussion", but because adolescents can have diffuse cerebral swelling that increases risk of secondary injury through second impact syndrome.[4, 15, 62] Athletes with a history of multiple concussions should also be treated conservatively, for they experience longer recoveries than athletes with no concussive history.[7] It was found that neurological testing in addition to self-reported symptoms aided in identifying concussed athletes rather than relying on symptoms alone.[7, 13, 15]

Self-reporting is not accurate or reliable, because it depends on the athlete.[14, 72] Even though it has been reported 62% of football players self-reported the same number of concussions they had actually sustained, players were still as likely to underreport and they were to over-report their concussions.[72] Rather, neuropsychological testing is based on empirical data that had been validated.[9] "Practice effects" which occur when the athlete is re-administered the standardized test several times, is limited by the use of computer randomized testing.[9] The athlete must return to their baseline scores on all these testing methods in order to be assured they have recovered from the concussion.[9, 14, 20, 73] Baseline testing allows for post-injury concussion testing scores to be compared with what considered "normal" for the individual athlete.[8]

If no baseline measures are available, post-injury results are compared with population normative values which are based off a "large sample of the representative population".[14] Balance testing has been shown to return to baseline scores between days 3 and 5, whereas cognitive functioning returns to baseline between days 5 and 7, and symptoms take approximately 7 days to resolve.[20, 43, 51] Guskiewicz stated that, "It appears that although symptom severity, neurocognitive function, and postural stability are often affected initially following concussion, they are not necessarily related or even affected to the same degree."[5]

If the clinician relies on one concussion testing tool alone, the athlete may be return to participation prematurely before symptoms, neurocognitive function, and postural stability have all returned to normal.[4] The Balance Error Scoring System, or BESS, which assesses postural stability, and requires the athlete to stand on a flat surface, as well as a piece of foam, with eyes closed for 20 seconds.[21, 43, 46, 51] On the two surfaces, athletes stand as still as they can in a double leg, single leg, and tandem stances, trying not to commit an error.[21, 43, 46, 51] An error is

counted as lifting hands off the iliac crests; opening eyes; stepping, stumbling, or falling; remaining out of the test position for more than 5 seconds; moving hip into more than 30 degrees of flexion or abduction; and lifting forefoot or heel.[21,43, 46, 51] This test is used by athletic trainers, usually in the "absence of sophisticated computerized balance systems".[46] It is easy to administer, and does not require extensive knowledge about postural stability; however, BESS has its faults.[46]

BESS showed subjects reaching their baseline scores or better by day one post-concussion.[5] BESS was found to have a significant practice affect after repeated administrations.[21] It has been shown that over a period of testing, the control group had a decrease in BESS errors over time.[74] This improvement in control subjects scores occurred with the second testing session, indicating a practice effect over time.[74] In fact, the study showed that the athletes had lower scores by day 7 of testing than during their baseline measurements.[21] As well, if an athlete is tested on the sidelines with BESS and is experiencing fatigue, they will commit more errors due to lack of postural control.[5, 51] Therefore, it is recommended that BESS be used after 20 minutes of the athlete suffering the concussion.[5, 51]

SAC, or Standardized Assessment of Concussion, a cognitive test, requires the athlete to remember a numbers and words and recall them after a short period of time.[21] Controls in one study improved from their SAC baseline scores within 48 hours of testing, indicating possible practice effects during administration.[21] The Standardized Assessment of Concussion, or SAC, which tests neurocognitive functioning, was shown to have low test-retest reliability.[75] Lastly, athletes can lie about their symptoms and deny they exist.[9]

When an athlete obtains their baseline results, a graduated return to participation protocol is put into effect.[9, 43] This means that the athlete has returned to baseline on neuropsychological

and postural stability testing, and is asymptomatic.[9, 62, 70] The athlete is able to attempt light

aerobic exercise at first; if the athlete is still asymptomatic after 24 hours, then they can progress

to the next step.[4,9,62] However, if during the light aerobic exercise the athlete becomes

symptomatic, they must rest until they are asymptomatic again, and can attempt the next

step.[4,9,62] Once the athlete is able to participate in game play, the athlete is considered fully

recovered and has completed the return to participation protocol.[4,9,62] There is no standardized

time period for the athlete to return to participation; rather, the protocol is individualized so that

the athlete will return when they are clinically asymptomatic.[4, 9, 14, 62, 70]

Mayers suggests athletes might require at least four to six weeks to adequately recover

from a concussion, to ensure the athlete does not suffer from secondary concussion after the first,

mentioned also by Guskiewicz et al.[7, 76] An athlete may experience post-concussion syndrome,

which occurs in approximately 10-20% of athletes who sustain a concussion.[9] The athlete will

experience concussion symptoms, such as headache, fatigue, and cognitive impairment for more

than one month after their concussion.[9] If an athlete is properly managed after they sustain a

concussion, the likelihood of this condition happening is rare.[9] It is also important to withhold an

athlete for an adequate period of time due to a condition called "second impact syndrome".[9]

Second impact syndrome occurs after an athlete sustains a first concussion, returns to activity too

soon, then receives a secondary blow to the head.[9] It is hypothesized this condition is a result of

metabolic dysfunction in the brain, which leads to a period of "neurological vulnerability".[9]

This condition is often fatal, that typically occurs in children as a result of brain swelling.[9]

Recent studies have focused an individualized approach, to avoid fatalities like post-concussion

and second impact syndrome, where concussed athletes are tested on more difficult tasks, like

dynamic postural control.[9,17, 36-41, 77]

Postural Stability and Control

Sports activity requires postural stability and control, which at the highest level involves the brain, the lowest level the brainstem and spinal cord.[74,78] The highest level of the brain is used for "attention, concentration, memory, and emotion", as well as associating with other brain structures.[5, 78] The middle level, which involves the sensorimotor cortex, cerebellum, and parts of the basal ganglia, is responsible for controlling posture and balance by incorporating muscle, joint, skin, eyes, and ear sensory information to coordinate and learn movments.[5, 78] Afferent pathways come from the eyes, vestibular area (ears), and proprioceptors, which are known as "postural reflexes".[5, 78] Efferent pathways are alpha motor neurons that lead to skeletal muscles, and incorporate neural networks from the brain to the spinal cord.[5, 78] The brainstem and spinal cord are the lowest level, where the motor neurons exit.[5, 78] The motor neurons carry muscle contractions, as well as joint angles from the muscle to the spinal cord and brainstem, onto the cerebellum.[5, 78]

During a concussion, the brain centers that integrate vestibular, visual, and somatosensory information from the muscles and joints are impaired, leading to central and peripheral deficits.[5, 78-79] The vestibular, visual, and somatosensory centers allow equilibrium to be maintained through processing and integrating afferent pathways to the central nervous system (CNS).[79] The CNS can only focus on one center at a time when the athlete suffers a concussion.[5, 78-79] Feedback from the sensory centers cannot generate the appropriate responses between the muscles to maintain postural stability, and the athlete's balance is adversely affected.[5, 78-79] Therefore, because studies have determine postural stability/balance is affected after a concussion, studies have looked at dynamic postural control during gait, to see if gait is also affected.

A conservative gait strategy may occur due to changes in central pattern generators (CPGs), which are neural networks within the spinal cord that produce rhythmic movement such as walking. [28, 42] CPGs are thought to be autonomic in producing movement, in which walking involves many muscles that need to be coordinated during each phase of the gait cycle.[28,42] CPGs consist of neurons located in different areas of the CNS.[28, 42] After a brain injury the CPGs may be impaired, due to CNS interruption. [28,42] A concussed individual may require more neural recruitment to walk normally, whereas it would be strictly autonomic in a non-concussed individual.

Gait, or dynamic postural stability following concussions has involved divided attention tasks and obstacle avoidances, mainly focusing on body center of mass motion and sway.[36-41] Subjects were comprised of athletes, non-athletes, and concussed athletes and concussed non-athletes.[36-41] Gait was tested over a 28 day period on specific days. [36-41] Data analysis suggested that controlling and maintaining stability during walking was affected in the concussed groups. [36-41]

In Parker et al.'s 2005 study, as well as Parker et al.'s 2006 and Parker et al.'s 2008 study, the concussed athletes were comprised of intercollegiate, club, or intermural athletes, not specifically intercollegiate athletes.[17, 40-41] What is unique about Parker et al.'s 2008 study is that concussed athletes and non-athletes were paired with matched controls, more specifically the concussed athletes were paired with other athletes of the same sport and similar positions.[40] The concussed in all studies were matched according to gender, age, height, and weight of the matched non-concussed controls, and also matched according to "physical activity profile".[40] However, the subject numbers varied from 10 concussed and 10 control subjects,[41] 28 concussed and 28 controls[40] to 15 concussed and 15 nonconcussed subjects.[17]

In both of Cantena et al.'s 2009 studies the researchers matched the concussed subjects, who were involved in intermural, club, or intercollegiate sports, with those who matched a similar activity level profile.[38-39] However, in Cantena et al.'s 2009 study, the concussed "athlete" subjects had matched controls who played a similar position.[39] Control subjects for the concussed were not matched according to age, height, or weight.[38-39] There were 17 concussed subjects with matched controls in Cantena et al.'s 2009 study[39], and 30 concussed subjects with matched controls in Cantena et al.'s other 2009 study.[38] In Cantena et al.'s 2007 studies, subjects were non-athletes who suffered a concussion, and matched to controls according to gender, age, mass, and height.[36-37] In both studies, 14 concussed and 14 controls were used.

Parker et al.'s 2005 study only required one testing session within 48 hours of the concussion.[41] Parker et al.'s 2006 study, concussed were assessed on days within 48 hours as well, but also on days 5, 14, and 28 post-injury.[17] Controls were only assessed during one testing session. In the 2008 study, Parker et al. had both the concussed and control group tested within 48 hours, and days 5, 14, and 28.[40] Both Cantena et al.'s 2007 studies only had one testing session for all subjects.[36-37] The concussed and control group in both of Cantena et al.'s 2009 studies had testing sessions at 48 hours, and days 6, 14, and 18.[38-39]

Single and dual divided attention tasks have been used during gait in Parker et al.'s 2005 and 2008 studies, as well as Cantena et al.'s 2007 study, during level walking without obstructions.[36-37, 40-41] Single attention tasks on a level walkway were attempted by the subjects by Parker et al.'s 2006 study.[17] However, Cantena et al.'s 2007 and Cantena et al.'s 2009 studies the subjects performed both single and dual tasks walking, as well as obstacle avoidance.[36, 38-39] Obstacle crossing alone was attempted by Cantena et al.'s 2009 study.[39]

Concussion groups had shorter stride length, and slower, although insignificant, gait velocity with the single and dual task conditions.[36-37, 41] Gait velocity was significantly slower for the concussed subjects within the first 48 hours, approaching normal gait velocity at day 5 for both single and dual task conditions.[17] Cantena et al.'s studies in 2007 found that concussed subjects had slower gait velocity in general during single and dual tasks, as well as obstacle avoidance.[36-37] Concussed athletes had the slowest gait velocity, and non-concussed athletes had slower gait velocity than concussed non-athletes.[40]

Stride length was also significantly shorter during single and dual tasks for the concussed subjects through day 14.[17] It took concussed subjects longer to finish a stride during dual tasks and obstacle avoidance.[36-37] Stride width was also wider for the concussed during single tasks and obstacle crossing tasks.[36-37]

Concussed subject adopt a slower, more conservative gait strategy and whole body center of mass to maintain dynamic stability during walking with single and dual divided attention tasks.[17, 36-41] A conservative gait strategy is considered slower gait velocity, greater step width, and shorter stride lengths to maintain that stability. Secondary tasks might challenge the brain more in less severe brain injuries.[38-40,] Sub-concussive blows may have an effect on an athlete's gait over time, for non-concussed athletes had slower gait velocities during single and dual tasks than concussed non-athletes.[40] There are long-term objective effects on the control of gait stability with concussed individuals, and therefore there needs to be more sufficient concussion testing methods to determine whether an athlete has recovered.[17]

Gait After Traumatic Brain Injuries

Gait has been used in the aforementioned studies to measure velocity, stride length and stride width during single and dual obstacle avoidance tasks. However, gait has been examined

in osteoarthritis, postmenopausal women with low bone density, those with fibromyalgia and claudication, diabetics, those with vestibular disorders, as well as older adults who have a greater risk of falling.[38-39, 58-59, 80-81] Parkinson's disease, individuals who suffered a stroke, traumatic brain injuries, and also concussion studies have used gait to examine mental impairment causing motor dysfunction.[17, 25, 37, 40-41, 82]

Traumatic brain injuries (TBIs) have looked at specific features of gait over the duration of injury. Traumatic brain injuries occur due to a traumatic, high velocity force, usually a car accident, fall from a height, etc.[27] TBIs result in corticospinal system damage that impair the CNS.[25,27] One study stated that the CNS might be impaired in its control of repetitive movements, such as gait.[25] This impairment causes decreased ability to make every stride the same, because the CNS loses central control of these repetitive movements.[25] As well, the body's center of mass and base of support might be compromised during this injury process, increasing gait variability as well.[25] Although concussions are not as severe, impairment of the CNS still occurs.[5, 78]

Upper motor neuron lesions may interfere with normal gait patterns.[27] TBI adult subjects were found to have slower velocity, or in one study, half the normal velocity as healthy controls.[26,27] This is because reduced gait velocity following a TBI may be due to a "compensatory strategy to improve postural control and stability."[26] Because stride time and cadence correlate with walking velocity, stride time increased, and cadence decreased.[24,27] Decreased cadence can result in decreased postural stability and control.[26] Double leg support time was also increased.[26] Step length was shorter, and step width wider for those with TBIs as well.[24,26-27] In a study of children with TBIs, there was an increase in step length variability.[25] It

was found that increased in brain damage are associated with greater step length variabilities.[24-25] Therefore, it is important to recognize normal gait versus variations during a gait cycle.

<center>Gait</center>

Gait is the body moving forward, where one limb supports the body as the other advances it.[53] The limbs then switch roles.[53] A gait cycle (GC) is a support and advancing phase of one limb.[53] More commonly, the start of a gait cycle is when the individual makes contact with the floor.[53]

In normal gait with a healthy individual, the person will strike with their heel first, which is the initial contact (IC).[53] Normal gait is made of two periods, termed the stance and swing phases.[53] Stance is the beginning of gait, comprised of three intervals, where at least one foot is in contact with the surface.[53]

The first interval of stance is 'initial double stance', in which both feet are originally in contact with the floor.[53] 'Single limb support' is the second interval, which involves one foot in contact with the surface while the other foot is swinging.[53] The body's total weight rests on the foot in contact with the ground.[53] Lastly, the third interval, or 'terminal double stance' starts with the contralateral limb in initial contact with the floor (also known as 'contralateral initial contact').[53] Terminal double stance ends with the beginning of the first interval, as the contralateral limb touches the floor, and the ipsilateral limb starts to rise for swing.[53] The ipsilateral limb rising for swing is termed as 'ipsilateral toe-off'.[53]

Normal gait patterns show that stance is 60% of gait, and swinging comprises 40%.[53] Breaking stance into the three intervals, both initial double stance and terminal double stance make up 20%, or 10% each, of the gait cycle; single limb support consists of the remaining

40%.[53] Non-injured individuals have a standard gait cycle of 62% stance and 38% swing, with an average a gait velocity of 80 meters/minute during walking.[53]

As an individual begins to increase their walking speed or transition to running, the stance and swing phases become shorter.[53] Walking at a greater velocity will increase the single stance interval and decrease the double stance intervals. Therefore, during running, the double stance interval is almost non-existent.[53]

Strides are the same as a gait cycle, where the individual's limb has contacted the floor twice. [53] For example, IC (initial contact) with the right foot, then IC again with the right foot. [53] A step, however, is between the right and left limbs.[53] One stride consists of two steps; an individual step is defined as the time between IC of the left limb and IC of the right limb.[53]

It is important to note that in individuals with paralysis, arthritis, or amputees, a heel strike may not occur until later in the gait cycle, or may never occur in one limb.[53] Likewise, IC may use the whole foot, termed 'foot flat', in which case there would be no forefoot contact occurring later in gait after a heel strike.[53] Because of these problems, Rancho Los Amigos gait analysis committee created generalized terms for the different phases in gait.[53]

Each stride or gait cycle consists of 8 subphases.[53] Three basic tasks allow the limb to move: weight acceptance (WA), single limb support (SLS), and limb advancement (LA).[53] Weight acceptance (WA) begins stance and consists of initial contact and loading response.[53] The limb during this phase needs stability and absorbs the shock, because the limb has just finished its swing and is making IC with the ground.[53] Initial contact, which is the first subphase, is when the ipsilateral foot initially touches the ground, which establishes how the limb will situate itself for the next step.[53] Initial contact comprises the first 0-2% of the gait cycle.[53] Loading response, which is the first 0-10% of the gait cycle, begins with the initial foot

contact of the ipsilateral foot with the ground, and proceeds with the contralateral foot lifted for swing.[53]

Single limb support (SLS), which consists of mid stance and terminal stance, occurs when one limb supports all the body's mass.[53] Mid stance occurs at 10-30% through the gait cycle, as the contralateral foot is lifting, and body mass over the ipsilateral forefoot.[53] Terminal stance progresses the single limb support, as the ipsilateral heel rises off the ground, and contralateral foot makes contact with the ground.[53] Body mass during this subphase is positioned in front of the ipsilateral forefoot.[53] This represents an interval of 30-50% of the gait cycle.[53]

Limb advancement (LA) being the last phase of stance, continuing through with the phases of swing: pre-swing, initial swing, mid swing, and finally terminal swing.[53] Pre-swing starts with ipsilateral toe-off and contralateral heel strike.[53] Consisting of interval 50-60% of the gait cycle, it is the last phase of double stance.[53] The ipsilateral limb prepares to swing at this time.[53] Initial swing is when the ipsilateral foot is lifted off the floor and the limb moves forward, while the contralateral limb is in mid stance.[53] The phase has an interval of 60-73% in the gait cycle.[53] Consisting of 73-87% of the entire gait cycle, mid swing advances the ipsilateral limb in swing, while the contralateral limb is in stance.[53] Lastly, terminal swing allows for the ipsilateral limb's final advancement as the foot hits the ground.[53] This subphase is the last 87-100% of the gait cycle.[53] Once the shank progresses ahead of the thigh, the gait cycle is finally complete.[53]

The center of gravity in normal humans is usually the tenth thoracic vertebrae.[83] Stability during locomotion is balance between the body's muscles and it alignment.[53] Everybody segment strives to maintain alignment to the joint centers, which is pertinent to create stability.[53]

However, the human body is more mobile than stable, as the joints are round and bones are long.[53]

In theory, standing still and balancing with both feet does not require muscle activation.[53] In order for this to happen, the head, arms, and thorax must be aligned from the eleventh thoracic vertebrae over joints in the hip, knee, ankle, and subtalus.[53] Because the joints are not locked, the body is not stable, and therefore balance can be upset by any rocking of the individual.[53] During swinging of a limb during gait, there needs to be a lateral transfer of body mass and muscle activation of abductors at the hip to keep the body stable.[53]

During walking, the body's support changes from ipsilateral heel strike to foot on ground to forefoot; then the ipsilateral leg swings, so the support is on the contralateral leg.[53] Because the support changes feet, posture is only stable during midstance when both feet are touching the ground.[53] During walking, as the body loads the ipsilateral leg, the mass is alignment in front of the hip and behind the knee and ankle.[53] As the body moves to midstance, the body mass is in front of the ankle and behind the knee, and then shifts ahead of the ankle and knee.[53] Lastly, during terminal stance, body mass is in front of the knee, with most of the mass over the ankle.[53]

During initial contact of the heel to the floor, the heel rocks as the calcaneus touches the ground.[53] Muscles like the anterior tibialis help to slow foot drop, yet at the same time pull the tibia forward.[53] At the same time, the quadricep muscles resist the knee from flexing.[53]

After the heel strikes the floor, the ankle 'rocks' to continue the momentum.[53] As the ankle dorsiflexes and weight is transferred onto the metatarsal heads, and the soleus contracts to limit knee extension.[53] Likewise, the soleus and gastrocnemius both assist in moving the tibia forward.[29]

Next, the heel lifts off the floor as part of the forefoot 'rocking', and the metatarsal heads are the center of pressure.[53] Ankle plantar flexion and knee flexion occur, and the adductor muscles flex the hip.[53] Body mass is ahead of the foot support, and this allows a driving force during swing; the mass then unloads to the contralateral limb.[53] During swing, the knee and foot are unstable.[53] Knee extension, as well as the advancement of the high and tibia help to drive the limb, and the body mass falls and is caught on the same foot as the next gait cycle begins.[53]

In a normal population, gait velocity is 80 m/min and cadence is 117 steps/min for both males and females.[53, 60-61] Normal gait velocity for males is 1.54 m/sec, and in females is 1.32 m/sec.[60-61] Males have a normal step length of 79 cm, females 66 cm; normal step width is 8.1 cm for males, and 7.1 cm for females.[60-61] Normal gait cycles have 40% spent on single leg stance time, 20% spent on double leg stance time, and 20% spent on swing time of leg.[53]

One instrument that measures gait, called the GAITRite® walkway system, is a portable, simple clinical tool.[30-33] When compared with video-based and paper/pencil instruments to measure gait, GAITRite® has excellent correlations between the two for temporal-based measures.[32] The GAITRite® walkway has good to excellent test-retest reliability when tested over a one week timespan.[30] This instrument has also been found to have strong concurrent validity, with moderate to high correlations for single limb support time.[31, 33] In addition, cadence, stride length, single limb support time, and double limb support time was highly reliable when the subject walked at a fast pace or preferred pace.[31]

TABLES and FIGURES of RESULTS

Group	Age (years)	Gender	Height (cm)	Leg length – right (cm)	Leg length – left (cm)	Mass (kg)	BMI (kg/m²)
CONC	20.0 ± 1.4	9 F, 6 M	168.0 ± 8.7	91.1 ± 6.9	91.2 ± 6.9	80.2 ± 19.4	26.2 ± 5.6
NONC	19.9 ± 1.2	9 F, 6 M	173.8 ± 13	92.6 ± 5.8	91.3 ± 5.8	75.6 ± 16.9	26.9 ± 4.2
NORM	20.6 ± 1.2	9 F, 6 M	173.5 ± 8.9	89.1 ± 5.0	89.1 ± 4.9	80.3 ± 21.6	26.5 ± 4.7

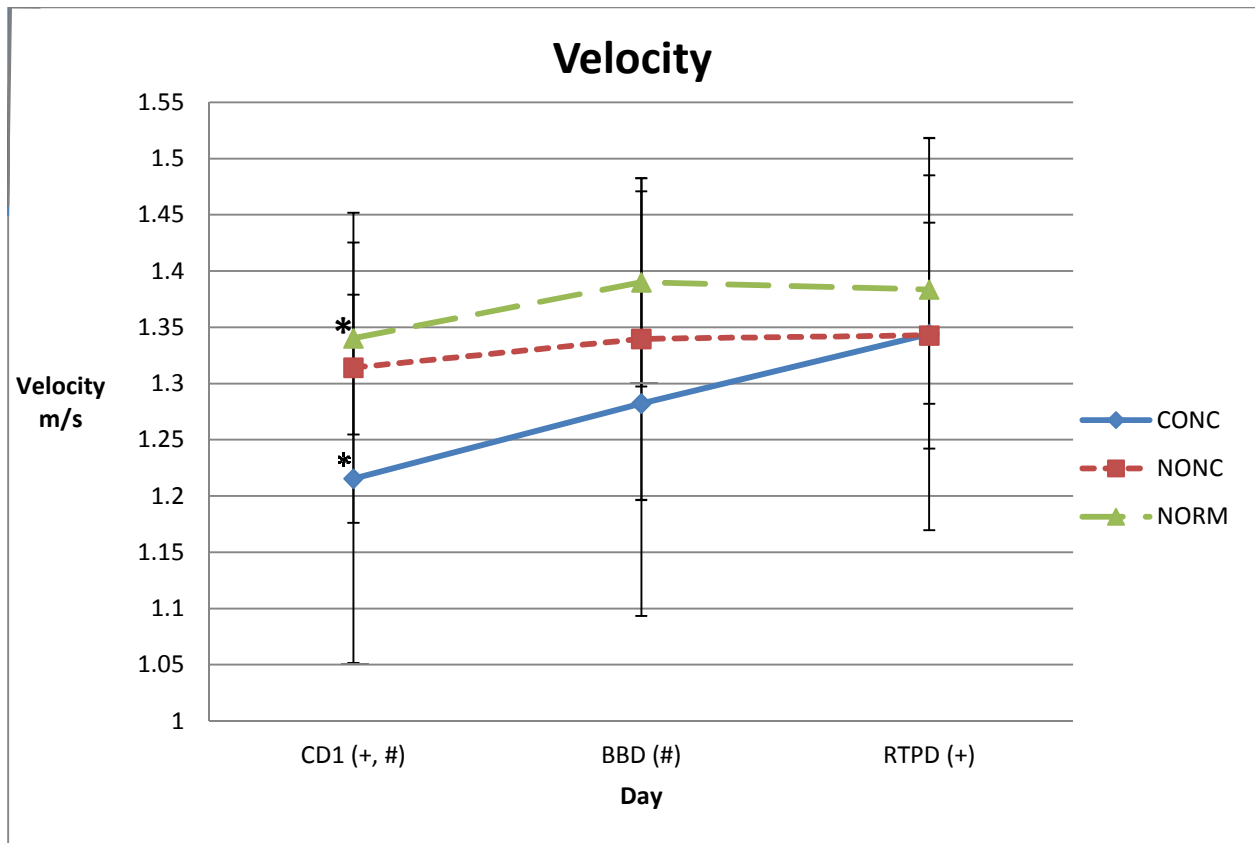*Table 1. Subject demographics; group mean ± standard deviation.*

Figure 1. Gait Velocity of CONCs, NONCs, and NORMs over CD1, BBD, and RTPD. Group means and standard deviation for gait velocity in single task, level walking condition. *At CD1, CONC < NORM. Groups increased their gait velocity from CD1 to BBD (#), and from CD1 to RTPD(+).
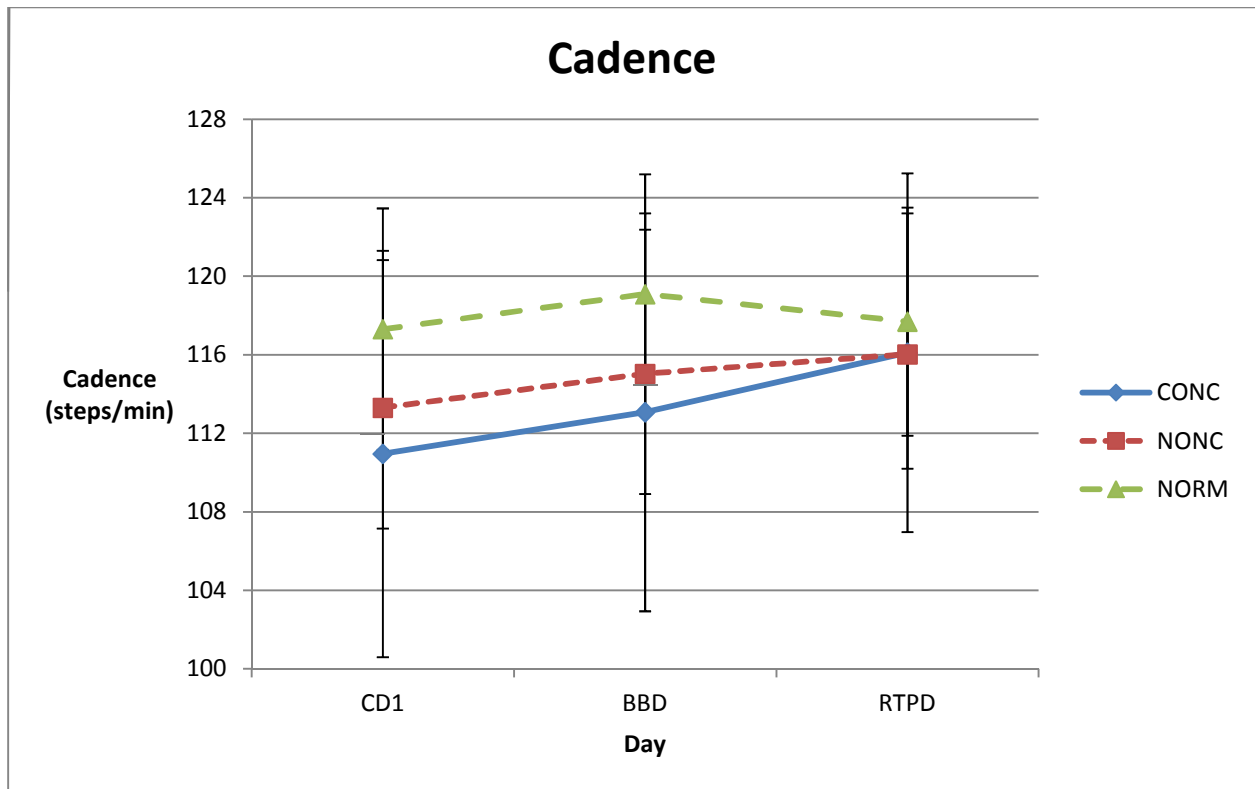
Figure 2. Cadence of CONCs, NONCs, and NORMs over CD1, BBD, and RTPD. Group means and standard deviation for cadence in single task, level walking condition.
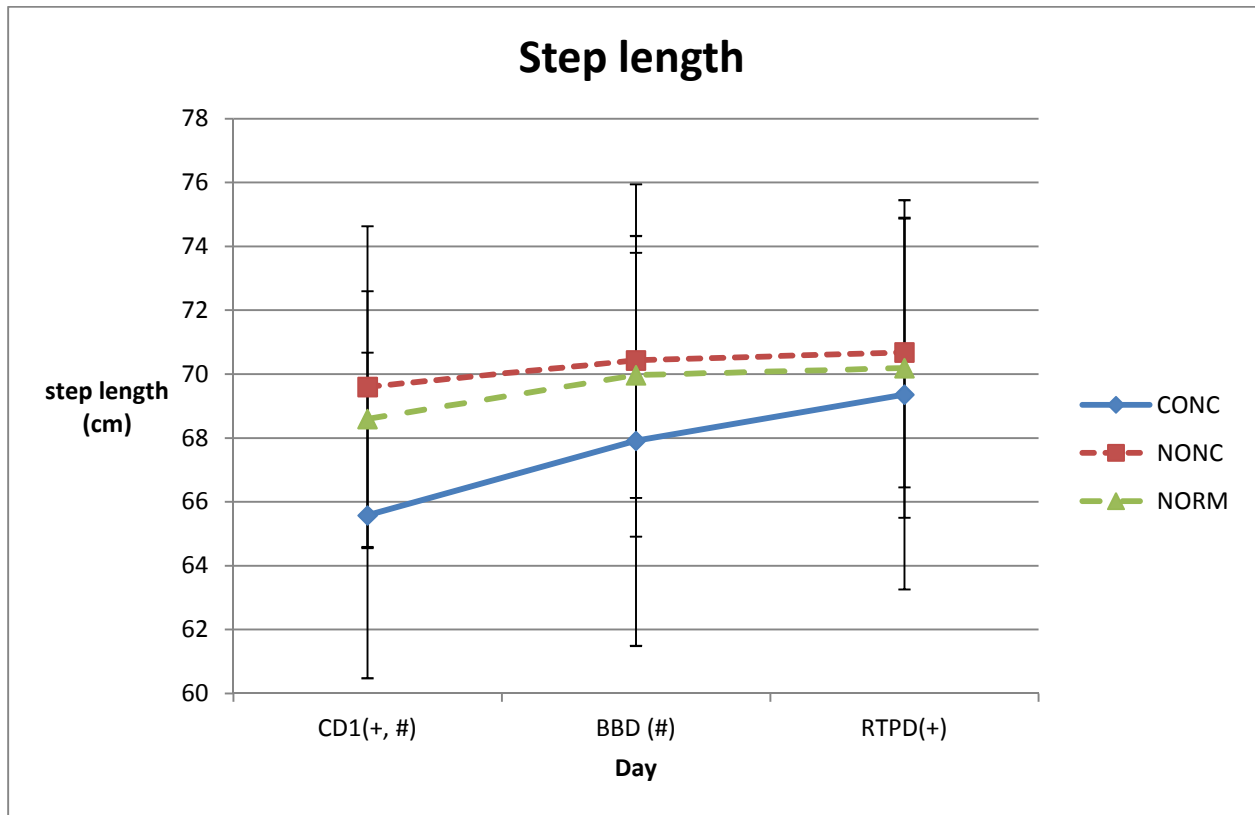
Figure 3. Step length of CONCs, NONCs, and NORMs over CD1, BBD, and RTPD. Group means and standard deviation for step length in single task, level walking condition. Groups increased their step length from CD1 to BBD (#), and from CD1 to RTPD (+).
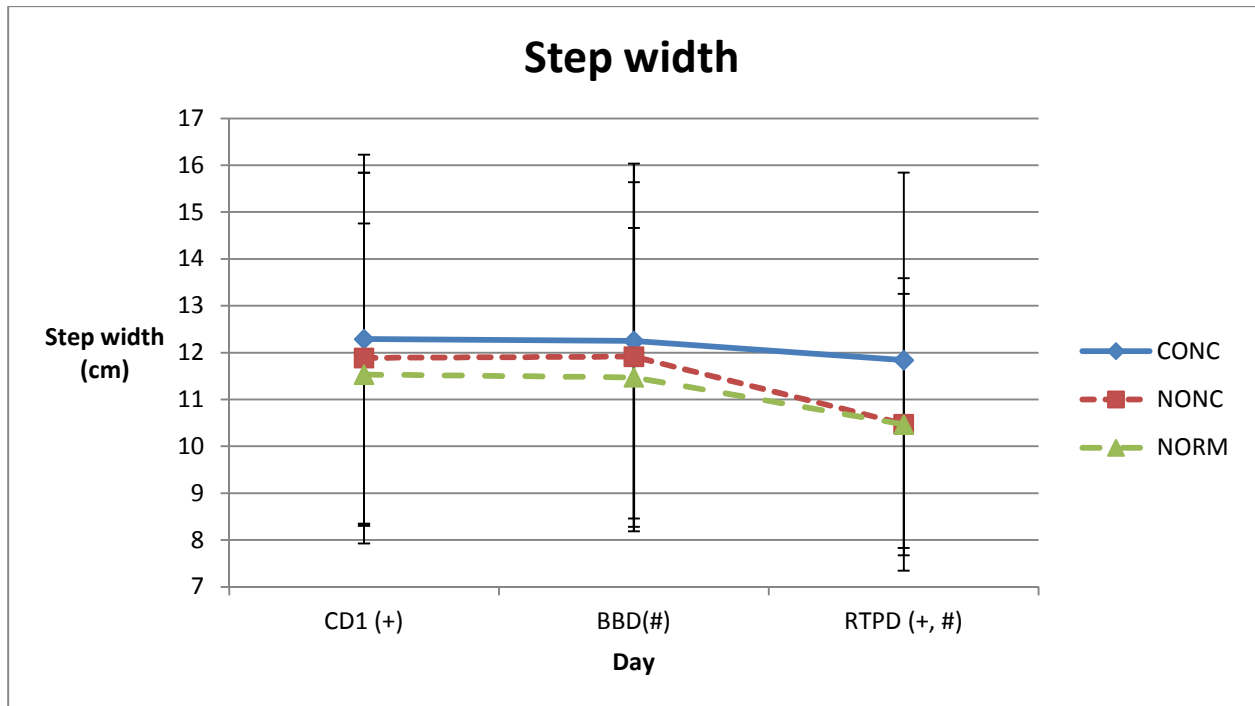
Figure 4. Step width of CONCs, NONCs, and NORMs over CD1, BBD, and RTPD. Group means and standard deviation for step width in single task, level walking condition. Groups increased their step width from BBD to RTPD (#), and from CD1 to RTPD (+).
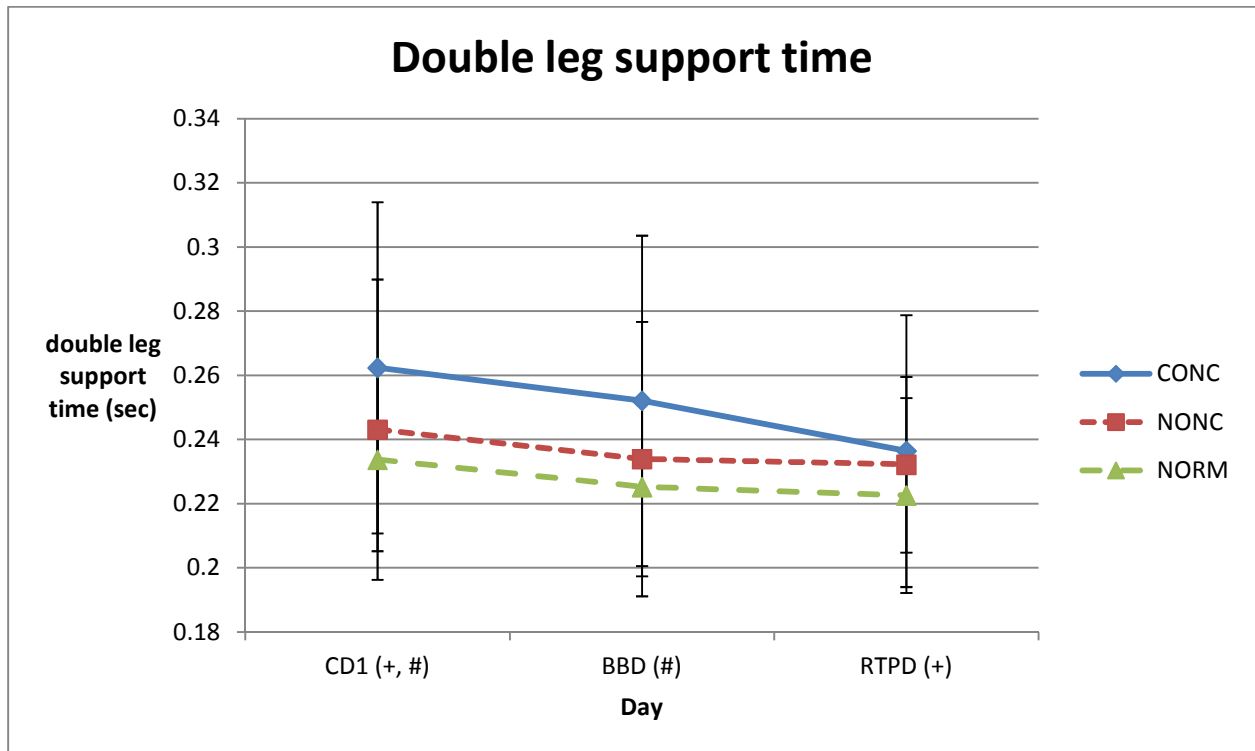
Figure 5. Double leg support times of CONCs, NONCs, and NORMs over CD1, BBD, and RTPD. Group means and standard deviation double leg support times in single task, level walking condition. Groups increased their step length from CD1 to BBD (#), and from CD1 to RTPD (+).

INFORMED CONSENT

GEORGIA SOUTHERN UNIVERSITY

COLLEGE OF Health and Human Sciences

DEPARTMENT OF Health and Kinesiology

INFORMED CONSENT

Title of Investigation: Changes in Gait Characteristics Following a Concussion

Investigator's Name: Kelly Previs, ATC/LAT
        Phone: (412)-874-5684   Email: kelly_m_previs@georgiasouthern.edu   Office: Hanner 1205

Data Collection Location: Biomechanics Laboratory, Hanner Building, Georgia Southern University

Participant's Name _____   Date_____

1. The purpose of this study is to determine how an athlete's gait is affected over the period of concussion recovery. There will be approximately 45 subjects in this study. This study aims to further aid in certified athletic trainer's treatment and evaluation of a concussion, as well as determining when an athlete's concussion has resolved, so that they can safely return to play.

2. This study includes concussed athletes, non-concussed teammates, and normal control subjects. If you are the athlete who suffered a concussion, you will attend one testing session per day until you are cleared to return to participation. Your testing session will take approximately 15 minutes per day. If you are the non-concussed teammate or the normal control subject, you will attend one testing session per day, until the concussed athlete you are paired with returns to participation. This will take approximately 15 minutes.

   During each session you will be asked to walk several times at a normal pace down a walkway. You will begin and end at a specified location. You will then perform 2 trials of static stance on a single limb for 20 seconds each followed by a single trial of dual limb static stance for 2 minutes.

3. The risks associated with this study are similar to what you might experience during walking and standing. If you were to become injured during this study, medical care may be provided on campus by Health Services, who can be reached at 912-681-5641. However, the researcher and thesis advisor are not held responsible for any financial or medical compensation nor free treatment of injury. In this manner, you understand you are not am not waiving my rights against Georgia Southern University for injury resulting from negligence of the investigators.

4. There is no compensation or reward for participating in this study. You are not responsible for any financial costs required to run the study. Your results may be provided to you upon request after the study

is completed, if you so choose. Upon the conclusion of the study, the results may help clinicians better understand how to manage and treat an athlete with a concussion.

5. If you are the non-concussed peer of similar position on the team, or the normal control, you will complete testing sessions over a period of days that will take approximately 15 minutes until you match the concussed athlete. If you are the concussed athlete, you will participate in one testing session daily until you have been cleared to participate. In this case, your testing session will take approximately 15 minutes as well.

6. You understand all the data concerning my participation in this study will be kept confidential by Kelly Previs, ATC/LAT, as well as Dr. Barry Munkasy, Ph.D., and Dr. Thomas Buckley, Ph.D., ATC. In order to receive your data, a written request must be submitted to the principle investigator. The data collected from your participation will be held in confidence like that of a medical record. Your identity will be in the form of a case number, and your name and identity will not be mentioned in any publications of the research results. In unusual circumstances, a court of law with a specific release may have access to the data in the study, or a specific government agency might inspect the data collected. After the ending of this investigation, the research data will be kept for a period of five years where only the advisor and other investigator have access besides the principle investigator.

7. If you have any questions concerning the research, you may contact Kelly Previs, ATC/LAT at (412)-874-5684, or kelly_m_previs@georgiasouthern.edu. If you have any questions or concerns as a research subject, you may contact the IRB Coordinator at the Georgia Southern University Office of Research Services and Sponsored Programs at (912)-478-0843.

8. You understand that participation is voluntary, and you have the right to choose not to participate. You can choose to end your participation at any time by telling the primary investigator, Kelly Previs, ATC/LAT, or any of the co-investigators, Dr. Thomas Buckley, Dr. Barry Munkasy, and Dr. Barry Joyner.

9. If you choose to end your participation, you will not be prejudiced to future care, reimbursement of expenses, compensation, employment status, or course grade except provided herein, and that owing to the scientific nature of the study, the investigators may use his/her absolute discretion to terminate the procedures and/or the investigation at any time. As an athlete, if you choose to end your participation, it will not affect your return to participation status or playing time for your team.

10. You understand that there is no deception involved in this project.

11. You certify that you are at least 18 years of age or older and have read the aforementioned statements as it has been read to me, and you understand my involvement in the study. If you have any questions that pertain to this study and the research data collected, you may contact the principle investigator listed at the top of the consent form and the phone/email information given.

12. You will be given a copy of this consent form to keep for your records. This project has been reviewed and approved by the GSU Institutional Review Board under tracking number H11323.

13. **Title of Investigation: Gait Analysis during a Concussion Using the GAITRite® walkway**

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Other Investigators: Dr. Thomas Buckley, Ph.D., ATC
        Phone: (912)-478-5268   Email: tbuckley@georgiasouthern.edu   Office: Hollis 2121C

_____ _____

Participant Signature   Date

I, the undersigned, verify that the above informed consent procedure has been followed.

_____ _____

Investigator Signature   Date

APPENDIX D

MEDICAL HISTORY FORM

Dynamic Postural Control Assessment During Gait Following a Concussion

A. Demographic Data
1. Date of birth _____
2. Age _____
3. Gender_____
4. Year in School (circle):

 Freshman   Sophomore   Junior   Senior   5th year   Grad student
5. Sport in which you participate – if you are an athlete (circle):

 Basketball   Football   Soccer   Baseball   Softball   Golf
 Cheerleading   Volleyball   Track & Field/XC   Swimming/Diving   Tennis

B. Concussion History
1. When did you suffer your concussion?   _____
2. With THIS concussion, have you suffered:

 A. *Loss of consciousness*?   Yes   No
 B. Amnesia (memory loss)?   Yes   No
3. What symptom do you suffer from most in the last 24 hours?

 _____
4. Have you ever suffered a concussion before?   Yes   No

 A. If you have, how many? _____
 B. If yes, when was your last concussion (not including this
  one)? _____
5. Have you ever been "knocked out" when playing a sport?   Yes   No

 A. If yes, how many times has this happened? _____
6. Have you ever had your "bell rung" or gotten "dinged"

 following a blow or hit to your head?   Yes   No
 A. If yes, has it happened this season?   Yes   No
 B. If yes, how many times this season? _____
 C. Has it happened to you before?   Yes   No

7. Have you ever been hit in the head while playing your
  sport and suffered from 2 or more of the following

(see list on bottom of this page)?   Yes   No
    A. If yes, has it happened this season?   Yes   No
    B. If yes, how many times this season? _____
    C. Has it happened to you in a previous season?   Yes   No
8. Have you ever been diagnosed with:
    A. A balance disorder?   Yes   No
    B. Metabolic disorder?   Yes   No
    C. Neurological disorder?   Yes   No
    D. Vestibular disorder?   Yes   No
9. Are you currently taking any medications?   Yes   No
10. If yes to either questions 8 or 9, describe your condition/medication
    and when you were diagnosed:_____

_____

_____

_____

Question #7 Addendum:

After receiving a hit or blow to the head, have you experienced any of the following:

Loss of consciousness	Yes	No
Disorientation	Yes	No
Feeling dazed/confused	Yes	No
Headache	Yes	No
Dizziness	Yes	No
Balance problems	Yes	No
Memory difficulties	Yes	No
Blurred or abnormal vision	Yes	No
Nausea	Yes	No
Trouble concentrating	Yes	No
Ringing in the ears	Yes	No
Bothered by loud noises	Yes	No
Feeling slowed down or fatigued	Yes	No
Feeling in a "fog"	Yes	No
Becoming emotional or irritable	Yes	No
Blacking out	Yes	No
Trouble returning to activities	Yes	No
Sleep problems	Yes	No
Problems with coordination/skills	Yes	No

Total number of "yes" answers _____

APPENDIX E

GAITRite® walkway

GT Path of Progression

| FP #1 | | GAITRite® | .6 m width | |
| FP #2 | FP # 3 | 4.9 m length | | 5m length |

.4m .6m

Parfion

GT Path of Progression

The GAITRite® walkway. Subjects start with left foot positioned on force plate one (FP #1), and right foot positioned on force plate two (FP #2). Then, subjects will start with right foot, and transverse the GAITRite® walkway until they reach past the end of the walkway, at a target (partition) located 5 m beyond its end. Starting at a mark located 3.6 m beyond the end of the walkway, subjects will then transverse the walkway in the opposite direction, until they reach the forceplates and end on them.