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Prevalence of Neurocognitive Test Failure Following Exertion in Athletes Recovering From  
Concussion  
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## Abstract

**INTRODUCTION:** The current return-to-play (RTP) protocol in place for an athlete recovering from a concussion is based on the subjective measure of self-report rather than more objective tools such as computerized neurocognitive testing. Because of this, it is possible that athletes downplay or lie about symptoms they are experiencing in order to expedite the RTP process.

**OBJECTIVE:** The purpose of the current study was to examine the frequency of post-exertional neurocognitive test failure in a sample of high school athletes recovering from concussion that underwent a standardized exertional RTP protocol. **METHODS:** This research project used a de-identified medical records review of neurocognitive data prospectively gathered from a sample of high school athletes with concussion that sought medical care at a large sport concussion clinic located in the southeastern United States. **RESULTS:** Thirty-nine athletes met inclusion criteria and participated in this study. Upon returning to baseline levels of neurocognitive performance at rest, athletes completed the standardized RTP protocol while reporting to be symptom-free, but 28% (11/39) showed cognitive deficits following this physical exertion. **DISCUSSION:** The findings from this study demonstrated that one-third of concussed athletes that successfully complete the RTP process (i.e., symptom-free) present with neurocognitive deficits. In order to protect these athletes, it is important that clinicians utilize objective CNT to complement symptom reports for determining RTP.

### **Introduction**

Approximately 1.6 to 3.8 million sport-related concussions (SRC) occur every year in the United States (Langlois, Rutland-Brown, & Wald, 2006). Concussion is defined as a complex pathophysiological process induced by traumatic biomechanical forces that result in a variable constellation of physical, emotional, sleep, and cognitive symptoms and impairments that may take days to weeks for recovery (McCrorry et al., 2013). The assessment and management of SRC has evolved from the sole use of symptom reporting to more objective measures in an effort to mitigate the high prevalence of unreported injuries (McCrea, Hammeke, Olsen, Leo, & Guskiewicz, 2004). When injuries go unreported or mismanaged the athlete is put at risk for experiencing a second, worse injury that can, in some cases, result in permanent brain damage or even death. This shift toward a more evidence-based method of assessing the recovery status of the concussed athlete stems from the inherent limitations that come with relying on athletes to be forthright and honest about self-reporting their symptoms.

Several studies demonstrate a discrepancy between subjective symptom reports and performance on objective measures of neurocognitive assessment following concussion (Lovell et al., 2003; Van Kampen, Lovell, Pardini, Collins, & Fu, 2006). Lovell et al. (2003) examined recovery patterns using self-reported symptom reports and objective neurocognitive measures in a sample of high school athletes with concussion. These researchers indicated that athletes self-reported to be asymptomatic as soon as four days following concussion, however memory impairment persisted longer than this four-day recovery period. These results suggest that self-reported symptoms may not be an accurate measure of recovery from concussion and warrant using a multi-faceted approach to concussion assessment. Specifically, using symptom reports as a sole measure of concussion impairment is reported to accurately identify 64% of concussions,

whereas using both symptom reports and objective computerized neurocognitive testing (CNT) increased the diagnostic yield to 93% (Van Kampen et al., 2006). The documented discrepancy between the temporal resolution of post-concussion symptom reports and neurocognitive impairment may be due to either the injured athlete minimizing their injury with the hopes of expediting the return-to-play (RTP) process or reflect a true difference in recovery rates between these two important pieces of clinical information. Nonetheless, the clinical assessment of SRC should include an objective measure of performance that complements subjective symptom reporting.

CNT is referred to as the “cornerstone” of concussion assessment and management and is one part of the recommended multi-faceted approach to concussion management (McCrary et al., 2013). These computerized neurocognitive batteries are a cost-effective, reliable, and valid assessment measure that can objectively quantify the amount of cognitive impairment resulting from concussion (Iverson, Lovell, & Collins, 2005; Nakayama, Covassin, Schatz, Nogle, & Kovan, 2014; Schatz, 2010; Van Kampen et al., 2006). These tools, rooted in traditional neuropsychological testing (e.g., memory, processing speed, reaction time), are ideal for assessing cognitive effects of concussion, as they can be administered prospectively (i.e., comparing pre-injury to post-injury scores) (Lovell, 2006). The use of CNT in combination with symptom reports is the recommended practice to ensure that the athlete returns to their baseline cognitive function and experiences a full resolution of symptoms without engaging in any strenuous physical or cognitive exertion (Van Kampen et al., 2006). Upon becoming asymptomatic and regaining baseline levels of cognitive function, international consensus statements recommend that the concussed athlete complete a graduated step-wise return-to-play

protocol (GRTP) to ensure that the athlete is ready to RTP (Harmon et al., 2013; McCrory et al., 2013).

The progression of an injured athlete through the steps of the GRTP protocol is a critical piece of the clinical decision-making process when determining readiness for RTP. When the athlete can fully participate in school and social activities without experiencing concussion symptoms, they are then cleared to begin the GRTP process (McCrory et al., 2013). The GRTP protocol follows a step-wise progression of increased physical exertion that includes the following six stages: Stage 1) requires that the athlete is neurocognitively back to their baseline levels of cognitive functioning and asymptomatic at rest; Stage 2) involves light aerobic exercise that increases heart rate like walking, swimming, or riding a stationary bike; Stage 3) involves sport-specific exercise such as completing simple non-head-impact drills; Stage 4) involves non-contact training drills; Stage 5) allows the athlete to participate in full-contact practice; and Stage 6) allows the athlete to fully RTP (McCrory et al., 2013). At each stage, if the athlete self-reports any increase in symptoms, they are required to stop the protocol, wait at least 24 hours or until symptoms resolve, then resume that stage's exertion (McCrory et al., 2013). Once the athlete can participate in a full-contact practice and reports no post-concussion symptoms, he or she will be medically cleared for full RTP (McCrory et al., 2013).

As previously mentioned, solely relying on symptoms to drive clinical decisions in diagnosing and managing concussion is not best practice as many athletes minimize their symptoms and hide their injury. Athletes may withhold their symptoms for reasons including, but not limited to, underplaying the severity of the injury, desire to stay in the game, lack of understanding of what had occurred, or the fear of letting down their teammates (McCrea et al., 2004). However, the current recommendation for determining progression through the GRTP

protocol is to only assess self-reported symptoms, which is not recommended for the sole diagnosis and management of the injury. Therefore, the exclusive reliance on symptom reporting should not be the only determinant on progression through the RTP process. In a recent study addressing this question, McGrath and colleagues (2013) reported that 28% (15/54) of concussed athletes successfully (i.e., asymptotically) completing the GRTP protocol demonstrated a significant decline in neurocognitive performance on CNT. While this was the first study to document the need for more objective assessment during GRTP, McGrath et al. (2013) used a small sample size and used a retrospective medical records review of data gathered from several clinical sites that did not use a standardized exertion protocol. This threat to internal validity could influence results, and additional research that uses a more standardized and controlled exertional RTP protocol is needed. The data used in the current study have been collected using a standardized exertional protocol, which means that all concussed athletes completed the same general protocol during the first two stages of the GRTP process, followed by standardized sport-specific drills throughout the remaining stages. The purpose of the current study was to examine the frequency of post-exertional neurocognitive test failure in a sample of high school athletes recovering from concussion that underwent a standardized exertional GRTP protocol. The hypothesis of this study was that the frequency of post-concussion computerized neurocognitive test failure will be higher than 28% when using a uniform method of exertion.

## Literature Review

### Definition and Prevalence of Sport-Related Concussion

SRC is a popular and widely sought after topic in sports medicine today. Approximately 1.6 to 3.8 million SRCs occur annually (Langlois et al., 2006) and the Center for Disease Control has deemed the injury an “epidemic” (Faul, Xu, Wald, Coronado, & Dellinger, 2010). This number, though, is thought to be a gross underestimate of the true prevalence of SRC due to injuries that go undiagnosed or unreported. The number of SRCs has increased over the years, as earlier studies report an annual incidence of only 300,000 (Thurman, Branche, & Sniezek, 1998). However, this has likely been due to a past lack of education and knowledge about SRC by athletes, coaches, or parents, leading to the injury going undiagnosed, rather than an actual dramatic increase in frequency. Across sports, the highest prevalence of SRC results from participation in football, followed by wrestling, girls soccer, and boys soccer (Lincoln et al., 2011). Overall, collegiate sports have a higher rate of concussions than high school sports, and female athletes show a higher prevalence of concussion than their male counterparts (Gessel, Fields, Collins, Dick, & Comstock, 2007; Lincoln et al., 2011).

SRC is a subset of a traumatic brain injury (TBI) and is sometimes referred to as a minor TBI (mTBI) (McCrory et al., 2013). It is defined as a “change in mental status” caused by a biomechanical disturbance to the brain (Kontos, Collins, & Russo, 2004). Because SRC results in a functional change in the brain as opposed to a structural change, neuroimaging techniques are unable to pick up on the injury (McCrory et al., 2013). SRC can be caused by a direct blow to the body that results in force being transferred to the head (McCrory et al., 2013). This blow may or may not lead to a loss of consciousness, and it can result in a sudden onset of symptoms that

will diminish spontaneously over a period usually lasting between seven and ten days (McCrorry et al., 2013).

### **Biomechanics of Sport-Related Concussion**

There are two broad categories of forces that can impact the brain: contact and inertial (Meaney & Smith, 2011). Both can be caused by the head striking an object, but contact forces usually result in skull fracture, while inertial involve impulsive head motion (Meaney & Smith, 2011). The majority of SRCs are results of inertial forces and are considered diffuse brain injuries (Gennarelli, 1993). Within the realm of inertial forces, are acceleration-deceleration, and rotational forces (Bailes & Cantu, 2001). Acceleration-deceleration forces occur when the body experiences rapid deceleration from the impact of a surface or opposing force, such as a soccer player running into a goalpost, or a gymnast being dropped and hitting their head (Guskiewicz & Mihalik, 2006). The free-moving brain resting in the cranium cannot keep up with such deceleration which leads to it bumping the inside of the skull. Another type of biomechanical force that can cause SRCs is rotational force. This can be experienced when the cranium suddenly moves along its axis of rotation while the brain stays relatively still, such as a quarterback sack (Bailes & Cantu, 2001). The reason that such blows are so detrimental to the brain is because of the fact that essentially, the brain is floating in the skull, surrounded by cerebral spinal fluid. When the body is moved by a sudden force, it may cause the brain to bump up against the skull, which is filled with small bony protrusions. When the brain hits these, axonal damage occurs, which triggers a “neurometabolic cascade” (Giza & Hovda, 2001).

### **Pathophysiology of Sport-Related Concussion**

This cascade of events begins by diffuse axonal stretching, which prompts the release of the neurotransmitter glutamate (Barkhoudarian, Hovda, & Giza, 2011; Giza & Hovda, 2001). As

the glutamate is released, it binds to the N-methyl-D-aspartate (NMDA) receptor which leads to further depolarization and an influx of calcium and efflux of potassium; this imbalance of ions leads to detrimental changes in cellular physiology of the neurons (Barkhoudarian et al., 2011; Giza & Hovda, 2001). Because there is so much extracellular potassium, the sodium-potassium pump in the cell must work much harder than normal in an attempt to restore homeostasis (Barkhoudarian et al., 2011; Giza & Hovda, 2001). The pump uses adenosine triphosphate (ATP) in order to function, so the demand for the energy molecule increases, causing cells to hypermetabolize (Barkhoudarian et al., 2011; Giza & Hovda, 2001). In normal conditions, cells use mitochondrial oxidation in order to make ATP because it is most efficient (Barkhoudarian et al., 2011; Giza & Hovda, 2001; Shrey, Griesbach, & Giza, 2011). However, with the initial influx of calcium, the neuronal mitochondria become impaired, thus forcing the brain to use glycolysis as its primary means of producing ATP (Barkhoudarian et al., 2011; Giza & Hovda, 2001). This increases lactic acid production which can have harmful effects such as altered permeability of the blood brain barrier, acidosis, membrane damage, and cerebral edema (Giza & Hovda, 2001; Kalimo, Rehncrona, & Söderfeldt, 1981). The energy crisis experienced by the brain, added to the fact that cerebral blood flow may decrease up to 50% in a concussed brain, are grounds for a high level of vulnerability, and the concussed individual can be at a higher risk of sustaining a second, more traumatic concussion if returned to play prematurely (Giza & Hovda, 2001).

### **Symptoms and Impairments of Sport-Related Concussion**

The symptoms of SRC are different among individual injuries, but can include physical, cognitive, and emotional symptoms, and sleep disturbances (McCrory et al., 2013). Physical symptoms include headache, loss of consciousness, or amnesia; cognitive impairments can

include things such as slowed reaction times or difficulty concentrating; behavioral symptoms include irritability or sadness; finally, sleep disturbances such as insomnia have been observed (McCroory et al., 2013). One commonly used assessment of SRC symptoms is the Post-Concussion Symptom Scale (PCSS). This Likert scale covers the four previously mentioned types of symptoms by asking the injured athlete 22 questions (M. R. Lovell & Collins, 1998). These symptoms result from the physiological changes in the brain, which also lead to certain impairments to the injured athlete. In a study performed by McCrea et al., (2003) it was demonstrated that on average, an athlete's cognitive function took 5-7 days to return to baseline, balance problems took 3-5 days, and reaction time and processing speed took a full week. Complete symptom recovery took an average of 7 days, though 10% of the sample in the study needed more time than average to fully recover (McCrea et al., 2003). Beyond these direct effects, things such as difficulty concentrating or remembering, or chronic headaches, can lead to decreased ability to perform in a school or work setting. They can also indirectly be a cause of depression due to the fact that the injury takes away the athlete's ability and allowance to participate in sports and other neurocognitively stimulating activities such as watching a movie with friends, or going to a concert (Chrisman & Richardson, 2014). This interruption in an injured athlete's social life has the potential to be quite detrimental to their mental health. None of these symptoms or impairments of SRC should be taken lightly, as the brain is in such a vulnerable state, so monitoring the symptoms until they return to baseline is crucial.

An athlete should never, under any circumstances, be returned to play directly after sustaining a head injury because the brain is in its most vulnerable state in the first 30 minutes following a SRC (Giza & Hovda, 2001). During this time, the brain is experiencing hypermetabolism in order to restore ionic balance, so returning an athlete to play and creating

more energy demands can promote cellular death (Giza & Hovda, 2001). In addition, the accumulation of mitochondrial-inhibiting calcium in the cell can lead to further apoptosis when coupled with an increase in stimulation (Giza & Hovda, 2001). Also, the NMDA receptor that becomes activated in response to sustaining a SRC can become altered for up to one week, according to animal studies (Giza & Hovda, 2001). This disturbance in normal functioning can equate to impaired neurotransmission, thus leading to cognitive dysfunction in the athlete (Giza & Hovda, 2001). This dysfunction increases the risk of the athlete sustaining a second, worse SRC because it affects important functions such as reaction time, attention, and concentration (Giza & Hovda, 2001). It should be noted that the findings from these animal studies may not precisely parallel the timeline of physiological changes in the human brain following an mTBI, but are still indicative of certain risks and precautions that should be taken when dealing with an injured athlete.

### **Second Impact Syndrome**

The dangers of returning an athlete to play immediately after sustaining a SRC are just as concerning as those of returning a symptomatic athlete to play, no matter how long ago their injury was sustained. By not waiting the appropriate amount of time before making the RTP call, the athlete is put at high risk of sustaining a second concussion, or developing second impact syndrome (SIS). It has been shown that athletes that sustain a second SRC before they have fully recovered from the first one can have more severe and longer lasting symptoms (Barkhoudarian et al., 2011; Vagnozzi et al., 2013). Not only this, but the risk of developing SIS is present, which has a mortality rate of nearly 50% (Cantu, 1998). SIS, as the name implies, can occur when a second head injury, even if it is seemingly minor, is sustained by someone before they have fully recovered from a previous one (Cantu, 1998). The syndrome is characterized by 15-

60 seconds of the conscious athlete feeling dazed, followed by a collapse into a semi-comatose state with dilating pupils, respiratory failure, and a lack of eye movement (Cantu, 1998).

Physiologically, this occurs because of an inability of the brain to regulate its blood supply, which leads to increased intracranial pressure and compromises the functionality of the brainstem (Cantu, 1998). This syndrome is, however, extremely preventable with proper monitoring of head injury, and education of parents, coaches, and athletes. Simply stated, if an athlete is at all symptomatic from a previous head injury, steps should be taken to ensure that they are not placed in a position of risk for experiencing another such injury. The main issue with this though, is the determination of when an athlete is truly asymptomatic.

### **Computerized Neurocognitive Testing**

SRC is one of the most difficult sports-related injuries to diagnose because it provides no biological markers that show up on clinical imaging, there are no perfect tests to identify or diagnose it, and athletes may not always report the injury (McCrea et al., 2004). However, the implementation of CNT is a valuable, objective tool and has been referred to as the cornerstone of concussion management (McCrory et al., 2013). CNT tools, specifically, Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT), are extremely important because of the objectivity, validity, and reliability that they provide (Elbin, Schatz, & Covassin, 2011; Iverson et al., 2005; Nakayama et al., 2014; Schatz, 2010). The ImPACT tool collects demographic information, concussion history, symptom inventory both before and after testing administration, and uses seven different computerized neurocognitive testing modules (Lovell, 2006). Each of these modules tests different aspects of neurocognitive health and ability, including memory, learning, cognitive speed, and impulsivity (Lovell, 2006). After the athlete completes the 20-25 minute test, results are immediately accessible and are summarized in the

form of five composite scores: verbal memory, visual memory, processing speed, reaction time, and impulse control (Lovell, 2006). Although the test includes a validity indicator, there will always be the question of motivation and effort put forth by the athlete in determining the validity of the assessment at an individual level. However, this issue is not absent from traditional pencil-and-paper testing methods either.

The use of CNT is very advantageous because of its practicality, objectivity, and the variables that it is able to assess, such as reaction time (Collie, Darby, & Maruff, 2001). In a 1998 study, it was reported that significant differences in reaction time can be as small as 100 milliseconds – the same accuracy would not be able to be reached by using a stopwatch (Bleiberg, Halpern, Reeves, & Daniel). In addition to this, the implementation of CNT is an effective way to control for underreported symptoms from athletes. One study found that while only 64% of a sample of concussed athletes reported symptoms, 83% of the sample were identified as having abnormal test results following CNT, with that number increasing to 93% when symptom reports and CNT were combined (Van Kampen et al., 2006). Whether due to intentional dishonesty, or the athlete's unawareness of their neurocognitive deficit, this 29% discrepancy shows that the underreporting of symptoms is a real concern, and given that concussion management has traditionally heavily relied on subjective self-reports, it is important to recognize its inefficiency and inaccuracy (Cantu, 1992; Kelly & Rosenberg, 1997). In another study, it was found that only 47% of athletes reported a concussion they had sustained during the previous season for reasons including, but not limited to, underplaying the severity of the injury, desire to stay in the game, lack of understanding of what had occurred, or the fear of letting down their teammates (McCrea et al., 2004). This study also shows the severity of the issue with

relying on an athlete's subjective self-report of such a complex and potentially debilitating injury.

### **Step-wise Return-to-play Exertional Protocol**

The current RTP protocol is shown in Table 1 and involves a step-wise progression of increasing physical exertion (McCrorry et al., 2013). Once an athlete becomes reportedly asymptomatic at rest, they begin the GRTP process. At each stage, the athlete is asked to report if they experience a recurrence of symptoms, and if symptoms return the athlete is required stop the protocol, wait at least 24 hours or until symptoms resolve, then resume that stage's exertion (McCrorry et al., 2013). Once the athlete can participate in a full-contact practice and reports no post-concussion symptoms, he or she will be cleared for full return-to-play (F RTP) (McCrorry et al., 2013).

Although this RTP protocol is widely accepted and used, one major problem within the method is the fact that the entire thing relies on an athlete's subjective self-report of symptoms.

Table 1

#### *Current Return-to-Play Protocol*

<u>Stage</u>	<u>Types of Exercise Performed</u>	<u>Objectives</u>
1) No activity	Physical and cognitive rest	Recovery
2) Light aerobic exercise	Walking, swimming, stationary bike	Increase in heart rate
3) Sport-specific exercise	Simple and non-head-impact drills	Add movement
4) Non-contact training drills	Complex training drills, resistance training	Exercise, coordination, and cognitive stimulation
5) Full contact practice	Normal training activities with medical clearance	Restore athlete's confidence while coaching staff assesses athlete's ability to return-to-play
6) Full return-to-play	Normal game play	

As stated above, in order to begin the RTP process, the athlete must be asymptomatic at rest, so it stands to reason that the asymptomatic athlete should show CNT results indicating they have returned to baseline, or are consistent with norms. Furthermore, as the athlete is brought through each stage of the RTP protocol and reports no symptoms, there is no reason their neurocognitive performance should decrease, especially because of the fact that studies have shown that bouts of moderate physical activity lend to increases in cognitive functions (Lambourne & Tomporowski, 2010). However, in a preliminary study, it was found that 28% of reportedly asymptomatic athletes showed significant decline in a CNT evaluation right before F RTP (McGrath et al., 2013). This indicates that those individuals had not fully recovered from their SRC before being cleared for RTP, despite the fact that they completed the protocol put in place to protect athletes from returning to play prematurely. This decline in CNT results was not only due to overall performance, rather, more specifically was reflective of slowed reaction times and effects on memory and cognitive functioning – things that should be intact during contact sport participation in order to best avoid injury (McGrath et al., 2013). Based on these findings, it can be concluded that an injured athlete that is motivated to RTP may underplay symptoms and do whatever necessary to be cleared for play. The lack of reporting symptoms can lead to premature RTP and put the athlete at unnecessary risk.

As it stands, CNT is not currently recognized as part of the official RTP protocol (McCrory et al., 2013). However, due to the fact that CNT offers a sensitive, objective evaluation of neurocognitive functioning of a concussed athlete, this tool should be implemented. It would allow health care professionals to use more than just the physical, self-reported symptoms of athletes recovering from concussions to make RTP decisions; it would allow cognitive symptoms such as reaction time and memory to be monitored as well. When these cognitive functions

remain compromised in an athlete recovering from concussion, it is an indication of lingering physiological changes and imbalances in the brain; this shows that the athlete has not yet fully recovered from their injury (Giza & Hovda, 2001).

## Methods

### Research Design

This research project used a de-identified medical records review of neurocognitive data prospectively gathered from a sample of high school athletes with concussion that sought medical care at a large sport concussion clinic located in the southeastern United States.

### Participants

Sixty concussed high school-aged athletes with a valid pre-injury (i.e., baseline), acute (1 – 5 day post-concussion), return-to-baseline (RTB), and post-exertion (PE) CNT assessment were included in the study. Participants competed in various sports including football, basketball, soccer, volleyball, and wrestling.

### Measures/Instrumentation

**Demographics.** Demographic data including age, sex, grade level, migraine/headache history, ADHD, learning disorders, concussion history, and current symptoms were gathered via a demographic portion of the CNT battery.

**Neurocognitive Performance.** Neurocognitive performance was measured using ImPACT. The ImPACT battery takes approximately 20-25 minutes to complete, has five different test versions to minimize practice effects, and produces outcome scores for the cognitive domains of verbal memory, visual memory, processing speed, and reaction time. The ImPACT battery has demonstrated acceptable validity and reliability over 8 days across 4 administrations, yielding correlation coefficients ranging from .62 to .88 for outcome scores (verbal memory, visual memory, processing speed, and reaction time) (Iverson, Lovell, & Collins, 2005). ImPACT also assesses current symptom reports via the Post-Concussion Symptoms Scale (PCSS), which is a 22-item 7-point Likert symptom inventory and yields a total

reported symptom score which was also used as an outcome variable for this study. The reliability and validity of the PCSS has been well documented in previous studies (Lovell & Collins, 1998; Pardini et al., 2004).

**Procedures.** Upon receiving University IRB approval, researchers gathered de-identified, prospective clinical data from the participating sports medicine clinic from the years 2011-2014. These data were de-identified by the treating clinician and a number coding system was used in place of patient names and other identifying information. There were no records linking these subject codes to the original medical charts. Researchers were responsible for cleaning and identifying patient data that met study inclusion criteria for the designated retest intervals (valid baseline, 1 – 5 day post-concussion CNT assessment, RTB CNT assessment, and a PE CNT assessment.)

Each athlete in the study underwent a standardized RTP protocol, which included 10-15 minutes on a stationary bike at low intensity (50% maximum effort), or walking, as the first stage of the process, followed by 10-20 minutes on a stationary bike at moderate intensity (60%-75% maximum effort), or jogging, as the second stage of recovery. Stages 3-6 included sport-specific training drills at increasing difficulties. Though these drills varied between athletes of different sports or positions, the difficulty level of the drills at each stage was controlled and relatively consistent across all sports. The effort put forth by the athletes was determined and monitored by clinicians, though these data were not available to the researchers of the current study. There were nine specific protocols based on the concussed athlete's sport or position: baseball, basketball, cheerleading, football (specified by position of linebacker, quarterback, or defensive back), soccer, volleyball, and wrestling.

**Data Analysis.** Statistical analyses were conducted with SPSS version 20. A series of repeated measures ANOVAs were conducted to examine changes in CNT performance and symptom reports across the testing occasions for the entire concussed sample to document a neurocognitive and symptom decline indicative of concussion. Independent variables were time (baseline, acute, RTB, PE) and the dependent variables were the ImPACT outcome scores (verbal memory, visual memory, processing speed, and reaction time) and total reported symptoms. In order to examine the frequency of PE CNT test failure, reliable change estimate (RCI) cutoffs were applied to the change scores between the RTB and PE time points. The frequency of PE CNT failure was expressed as a percentage.

To examine the course of neurocognitive recovery among the PE-Pass and PE-Fail groups, a second series of repeated measures ANOVAs were conducted to examine changes in CNT performance and symptom reports across the testing occasions among the groups. Independent variables were time (baseline, acute, RTB, and PE) and group (PE-Pass, PE-Fail). Dependent variables were the ImPACT outcome scores (verbal memory, visual memory, processing speed, and reaction time) and total reported symptoms. A bonferroni-corrected level of significance was used to control for multiple comparisons ( $p \leq .05$ ) for all analyses to control inflation of Type I error rate.

## Results

### Demographic Information

Thirty-nine athletes met inclusion criteria to participate yielding a response rate of 65% (39/60). There were 36 males and three females with the average age of the participants being  $16.27 \pm 1.32$  years. Four participants had had one previous concussion, while the other 35 had no history of the injury. Participants came from 18 different high schools. There were no statistical differences among the groups on any demographic variables (See Table 2).

Table 2

*Mean and Standard Deviations for Demographic Information among the PE-Fail, PE-Pass, and Total for Overall Sample.*

	<u>PE-Fail</u>	<u>PE-Pass</u>	<u>Total</u>
Number	11	28	39
Age (years)	$16.19 \pm 0.96$	$16.29 \pm 1.44$	$16.27 \pm 1.32$
Number of Concussions	$0.13 \pm 0.35$	$0.09 \pm 0.29$	$0.10 \pm 0.31$
Days to Acute Evaluation	$3.45 \pm 2.62$	$4.43 \pm 2.04$	$4.15 \pm 2.23$
Days to RTB	$15.73 \pm 10.15$	$14.57 \pm 7.39$	$14.90 \pm 8.12$
Days to PE	$23.00 \pm 11.36$	$22.79 \pm 9.05$	$22.85 \pm 9.60$

\* $p \leq .05$ ; RTB = return to baseline; PE = post-exertion

### Pre- to Post-Concussion Changes in Neurocognitive Performance and Symptom Reports for the Entire Sample

The results of a series of one-way repeated measures ANOVAs revealed significant within-subjects main effects for time on verbal memory (*Wilks*  $\lambda = .57$ ,  $F [3,36] = 9.16$ ,  $p \leq .001$ ,  $\eta^2 = .43$ ), visual memory (*Wilks*  $\lambda = .42$ ,  $F [3,36] = 8.57$ ,  $p \leq .001$ ,  $\eta^2 = .42$ ), processing speed (*Wilks*  $\lambda = .48$ ,  $F [3,36] = 12.96$ ,  $p \leq .001$ ,  $\eta^2 = .52$ ), reaction time (*Wilks*  $\lambda = .53$ ,  $F [3,36] = 10.86$ ,  $p \leq .001$ ,  $\eta^2 = .48$ ), and total symptoms (*Wilks*  $\lambda = .41$ ,  $F [3,36] = 12.90$ ,  $p \leq .001$ ,  $\eta^2 = .59$ ). Post-hoc analyses revealed significant decreases in performance at the acute time period compared to baseline for verbal memory ( $p \leq .001$ ), visual memory ( $p = .004$ ), processing speed ( $p \leq .001$ ), reaction time ( $p = .002$ ), and total symptoms ( $p = .004$ ). The means and standard deviations for these outcome variables can be found in Table 3.

Table 3

*Means and Standard Deviations for Changes in Neurocognitive Performance and Symptoms across Post-Concussion Retest Intervals (N = 39).*

	<u>Baseline</u>	<u>Acute</u>	<u>RTB</u>	<u>PE</u>
Verbal Memory (% correct)	84.77 ± 9.68	79.31 ± 13.66*	88.33 ± 11.72	91.13 ± 8.79
Visual Memory (% correct)	75.10 ± 10.40	66.92 ± 13.96*	75.92 ± 11.74	78.28 ± 11.42
Processing Speed	35.86 ± 6.19	35.00 ± 6.59*	37.84 ± 6.20	39.51 ± 5.34
Reaction Time (s)	0.60 ± 0.07	0.67 ± 0.12*	0.58 ± .08	0.56 ± .08
Total Symptoms	11.13 ± 12.44	24.03 ± 6.28*	4.43 ± 6.28	1.60 ± 2.80

\* $p \leq .05$  – Significantly different than baseline

### **Frequency of Post-Exertional Neurocognitive Test Failure in the Current Sample**

Change scores were calculated for verbal memory, visual memory, processing speed, and reaction time by subtracting the difference between PE and RTB time points for each athlete. If at least one change score exceeded RCI cutoffs as determined by Iverson, Lovell, and Collins (2003), the athlete's post-exertional neurocognitive test performance was classified as fail. In the current sample, 28% (11/39) of athletes demonstrated at least one neurocognitive outcome score outside RCIs. Ten out of the 11 athletes in the PE-Fail group had one neurocognitive score outside of RCI intervals and one athlete had two scores outside of RCI intervals.

### **Comparing Neurocognitive Performance and Symptom Reports between PE-Pass and PE-Fail Groups at Baseline, Acute, RTB, and PE**

A series of 2 group (PE-Pass, PE-Fail) x 4 time (baseline, acute, RTB, PE) repeated measures ANOVAs revealed within-groups main effect for time on verbal memory (*Wilks*  $\lambda = .64$ ,  $F [3,35] = 6.71$ ,  $p \leq .001$ ,  $\eta^2 = .37$ ), visual memory (*Wilks*  $\lambda = .63$ ,  $F [3,35] = 6.95$ ,  $p \leq .001$ ,  $\eta^2 = .37$ ), processing speed (*Wilks*  $\lambda = .50$ ,  $F [3,35] = 11.91$ ,  $p \leq .001$ ,  $\eta^2 = .51$ ), reaction time (*Wilks*  $\lambda = .60$ ,  $F [3,35] = 7.95$ ,  $p \leq .001$ ,  $\eta^2 = .41$ ), and total symptoms (*Wilks*  $\lambda = .42$ ,  $F [3,35] = 16.23$ ,  $p \leq .001$ ,  $\eta^2 = .58$ ). As expected from the previous analyses of the total group ( $N = 39$ ), the RTB time point was significantly higher than the acute time point for verbal ( $p = .001$ ), visual ( $p = .007$ ), processing speed ( $p = .05$ ), reaction time ( $p = .001$ ) and total symptoms ( $p = .001$ ). There were no significant between-subjects effects for group on verbal memory ( $F [1,37] = .36$ ,  $p = .55$ ,  $\eta^2 = .01$ ), visual memory ( $F [1,37] = .57$ ,  $p = .46$ ,  $\eta^2 = .02$ ), processing speed ( $F [1,37] = .40$ ,  $p = .53$ ,  $\eta^2 = .01$ ), reaction time ( $F [1,37] = 1.34$ ,  $p = .25$ ,  $\eta^2 = .04$ ) or total symptoms ( $F [1,37] = .02$ ,  $p = .90$ ,  $\eta^2 = .00$ ). There were no significant group x time interactions

for verbal memory (*Wilks*  $\lambda = .92$ ,  $F [3,35] = 1.01$ ,  $p = .40$ ,  $\eta^2 = .08$ ), visual memory (*Wilks*  $\lambda = .94$ ,  $F [3,35] = .74$ ,  $p = .54$ ,  $\eta^2 = .06$ ), processing speed (*Wilks*  $\lambda = .88$ ,  $F [3,35] = 1.67$ ,  $p = .19$ ,  $\eta^2 = .13$ ), reaction time (*Wilks*  $\lambda = .94$ ,  $F [3,35] = 80$ ,  $p = .50$ ,  $\eta^2 = .06$ ), or total symptoms (*Wilks*  $\lambda = .97$ ,  $F [3,35] = .36$ ,  $p = .78$ ,  $\eta^2 = .03$ ). The means and standard deviations for the PE-Pass and PE-Fail groups can be found in Table 4.

Table 4

*Means and Standard Deviations for PE-Pass (n = 28) and PE-Fail (n = 11) groups across baseline, acute, RTB, and PE time points.*

	<u>PE-Fail (n=11)</u>	<u>PE-Pass (n=28)</u>
<b>Verbal Memory Composite</b>		
(% correct)		
Baseline	87.55 ± 7.57	83.68 ± 10.31
Acute	79.73 ± 18.59*	79.14 ± 11.59*
RTB	90.45 ± 8.13	87.50 ± 12.90
Post-Exertion	90.55 ± 9.05	91.36 ± 8.84
<b>Visual Memory Composite</b>		
(% correct)		
Baseline	76.73 ± 8.44	74.46 ± 11.15
Acute	66.73 ± 16.85*	67.00 ± 13.00*
RTB	80.73 ± 11.53	74.04 ± 11.47
Post-Exertion	78.64 ± 15.44	78.14 ± 9.75
<b>Processing Speed Composite</b>		
Baseline	35.23 ± 4.66	36.10 ± 6.76
Acute	36.23 ± 7.14*	34.51 ± 6.43*
RTB	39.87 ± 4.76	37.05 ± 6.59
Post-Exertion	40.20 ± 1.94	39.24 ± 6.20
<b>Reaction Time Composite (s)</b>		
Baseline	0.61 ± 0.07	0.59 ± 0.08
Acute	0.70 ± 0.12*	0.66 ± 0.13*
RTB	0.59 ± 0.10	0.57 ± 0.08
Post-Exertion	0.59 ± 0.09	0.55 ± 0.07
<b>Total Symptoms</b>		
Baseline	8.91 ± 9.22	11.79 ± 13.65
Acute	26.64 ± 21.42*	23.68 ± 21.46*
RTB	3.64 ± 5.12	4.71 ± 8.19
Post-Exertion	0.73 ± 1.85	1.54 ± 2.77

\* $p \leq .05$  – Significantly different than baseline

## Discussion

The purpose of this study was to examine the frequency of post-exertional neurocognitive test failure in athletes recovering from concussion. Previous studies have documented a significant decline in CNT performance in approximately 28% of athletes between the time that an athlete begins RTP exertional protocol and the time that they are returned to play (McGrath et al., 2013). The findings of this study were consistent with the literature in that 28% of the sample showed significant declines in one or more areas of CNT performance, which was determined using RCIs (Iverson et al., 2003). The current study's hypothesis, that a more controlled RTP exertional protocol would yield a frequency of neurocognitive test failure higher than 28%, was not supported. Although the frequency of post-exertional test failure was comparable to previously published data, the post-concussion neurocognitive profiles of these two groups did not differ at any time point.

Determining when a concussed athlete is ready to return to play is a critical decision for the sports medicine professional. Although current consensus statements (McCroory et al., 2013) encourage the ongoing monitoring of self-reported symptoms during the recommended return-to-play exertional protocol, increasing objectivity to this process is needed. This need is in direct response to the frequent lack of symptom reporting by athletes in hopes of expediting their return-to-play (McCrea et al., 2004). Post-exertional CNT failure has been documented in the literature; however McGrath and colleagues (2013) did not use a uniform method of exertion. The current study attempted to address this limitation by gathering data from concussed athletes that underwent a standardized exertional protocol (University of Pittsburgh Medical Center) during the RTP process. Variations in methods used to exert concussed athletes during RTP may influence neurocognitive performance and symptom reporting, leading to artificially high or low

rates of post-exertional neurocognitive test failure. These variations may confound the clinical decision making of the sports medicine professional.

All athletes in the current study demonstrated significant neurocognitive and symptom impairment at the acute time point, showing a clear effect of injury. The study also found that 28% of the sample demonstrated CNT failure following exertion, which is consistent with other similar studies (McGrath et al., 2013). However, while trends consistent with previous studies were observed, there was a lack of significant neurocognitive differences between the PE-pass and PE-fail groups across any neurocognitive domain. This may have been due to the small sample size used in this study. While McGrath et al. (2013) reported significant differences between the two groups in verbal memory and visual memory composite scores, they utilized a larger sample size with 54 subjects. Other limitations of this study include the fact that the subjects were from 18 different high schools, and while there was a standardized RTP protocol in place, its administration was not closely monitored. Therefore, variables such as the setting of testing and complete concussion management were not strictly controlled.

Overall, this study confirms the importance of increased objective measures in the management of SRC. Future studies should focus on gathering a larger sample size in order to observe more pronounced findings. There also should be a focus on selecting a sample that is more evenly distributed in terms of sex. Only three out of 39 subjects were female, therefore this study was unable to examine sex differences in post-exertional CNT failure.

Without the use of objective measures in concussion management, athletes are put at risk for being returned to play prematurely. A premature RTP may increase the risk of a second, potentially worse injury. The findings from this study, which are supported by the literature, show that it is possible for nearly one-third of athletes recovering from concussion to complete

the RTP process, reportedly symptom-free, while still having certain neurocognitive deficits. In order to protect these athletes, it is important that clinicians implement more than just subjective symptom reporting as the primary measure to monitor an athlete as they complete RTP protocol.

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