VISUAL AND AMBULATORY FUNCTION OF INDIVIDUALS WITH A HISTORY OF CONCUSSION

by

THOMAS PRATO

(Under the Direction of ROBERT LYNALL)

ABSTRACT

Concussions are a growing public health concern, with rising incidence across all levels of sport and age groups. Among the many consequences of concussion, visual dysfunction is both highly prevalent and underexamined despite over half of the brain being dedicated to visual processing. Most clinical assessments emphasize oculomotor control, often neglecting sensory and perceptual components that may be more sensitive to lingering post-concussion deficits. Additionally, few studies have explored how visual disturbances affect gait performance, particularly under real-world visual stressors like glare. This dissertation aimed to evaluate visual performance in individuals with and without a history of concussion using a novel psychophysical battery, and to examine how glare exposure influences gait performance across simple and complex gait tasks. Thirty-one participants (16 with a concussion history, 15 matched controls) completed assessments of macular pigment optical density, temporal contrast sensitivity, critical flicker fusion threshold, and glare sensitivity/discomfort. Gait was assessed during straight path walking and planned/unplanned gait termination, with and without glare exposure administered via mobile glare goggles. There were no significant group differences in

visual or gait outcomes. However, glare significantly altered gait in all participants, reducing stride length and velocity and increasing gait termination time specifically during unplanned gait termination. These findings suggest that even in asymptomatic individuals, visual stressors like glare can affect motor control. Although group differences were not detected, this study introduces novel tools that may have the ability to uncover subtle impairments overlooked by standard clinical measures. The integration of gait analysis under glare conditions offers a promising framework for advancing concussion assessment within everyday scenarios. These findings emphasize the importance of considering visual disturbances in post-concussion care and highlight the need for more comprehensive, real-world evaluations to inform recovery and return-to-activity decisions.

INDEX WORDS: Concussion, Macular Pigment Optical Density, Temporal Contrast Sensitivity Function, Glare, Gait

VISUAL AND AMBULATORY FUNCTION OF INDIVIDUALS WITH A HISTORY OF CONCUSSION

by

THOMAS PRATO

BS, Immaculata University, 2019

MS, Temple University, 2021

A Dissertation Submitted to the Graduate Faculty of The University of Georgia in Partial Fulfillment of the Requirements for the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2025

VISUAL AND AMBULATORY FUNCTION OF INDIVIDUALS WITH A HISTORY OF CONCUSSION

by

THOMAS PRATO

Major Professor:	Robert Lynall
Committee:	Julianne Schmidt
	Lisa Renzi-Hammond
	Randy Hammond

Electronic Version Approved:

Ron Walcott
Vice Provost for Graduate Education and Dean of the Graduate School
The University of Georgia
August 2025

ACKNOWLEDGEMENTS

I would first like to start by thanking Madi Prato for her support throughout my time during this process. From living long distance during the COVID pandemic to taking a leap of faith to move from New Jersey to Georgia with me was more than I could ever have hope for. You have always been there for me, and I can never repay you for that.

To Rob Lynall and Julianne Schmidt, I want to thank you for taking a chance on me during a time when the world created a lot of uncertainty. Rob, your guidance throughout my time at UGA was more than I could ever have imagined when I moved down here. I always look forward to our weekly meetings and you being supportive no matter what crazy ideas I threw at you. Julianne, I will always appreciate your help and guidance especially during my early time in the program. The willingness to answer any question or problem I had was greatly appreciated. I will also miss the fun you brought to team meetings even if it meant Rob had to get everyone on track at some point. The mentorship I received from both of you with forever play an important role for the rest of my career.

Finally, Lisa and Randy Hammond, I would like to thank you for agreeing to the crazy idea of bringing in someone who was not exposed to your research field and teaching me along the way. I had a crazy idea of wanting to incorporate vision into what we do in the concussion and biomechanics labs, and you both were able to make that dream a reality. From Lisa giving individualized lectures to Randy building (and taking selfies with) equipment to be able to answer the questions I had I cannot thank you both enough. You both have made this learning curve as smooth as it possible could have been in my eyes.

TABLE OF CONTENTS

Page

ACKNOWLEDGEMENTS iv

LIST OF TABLES viii

LIST OF FIGURES ix

CHAPTER

1 INTRODUCTION 1

2 LITERATURE REVIEW 7

Concussion 7

Epidemiology 7

Pathophysiology 8

Visual Performance Post-Concussion 10

Visual Psychophysics 13

Macular Pigment Optical Density 13

Glare 17

Temporal Contrast Sensitivity Function 21

Increased Injury Risk Post-Concussion 26

Gait 26

Vision and Gait 28

3 METHODS 30

Participants 30

Demographics 31
Sport Concussion Assessment Tool 6 Symptom Inventory 31
Temporal Contrast Sensitivity 31
Macular Pigment Optical Density 33
Glare Discomfort 35
Glare Sensitivity Questionnaire 36
Straight Path Gait with Glare Stimulus 36
Planned/Unplanned Gait Termination With and Without Glare 37
Statistical Analysis 38
Power Analysis 39
4 A NOVEL VISUAL ASSESSMENT BATTERY IN INDIVIDUALS WITH A HISTORY OF CONCUSSION 40
Abstract 41
Introduction 42
Methods 44
Results 51
Discussion 51
5 THE IMPACT OF GLARE ON GAIT PERFORMANCE IN INDIVIDUALS WITH A HISTORY OF CONCUSSION 57
Abstract 58
Introduction 59
Methods 62
Results 65

Discussion 74

6 FINDINGS SUMMARY 80

REFERENCES 83

APPENDICES

A National Institutes of Health common data element form 103

B SCAT6 Symptom Inventory 104

LIST OF TABLES

Page

Table 2.1: Visual terms definitions 11

Table 2.2: Visual psychophysics outcome variable descriptions 35

Table 3.1: Gait outcome variable descriptions 34

Table 3.2: Gait outcome variable descriptions 37

Table 4.1: Group means, mean differences, and statistical findings from vision assessments 50

Table 4.2: Variability group means and statistical findings from vision assessments 51

Table 5.1: Straight path gait means and statistical findings 67

Table 5.2: Straight path gait mean variability and statistical findings 68

Table 5.3: Planned gait termination means and statistical findings 69

Table 5.4: Planned gait termination task mean variability and statistical findings 70

Table 5.5: Unplanned gait termination means and statistical findings 71

Table 5.6: Unplanned gait termination task mean variability and statistical findings 72

LIST OF FIGURES

Page

Figure 2.1: Typical spectrum of LED light used in glare assessment 19

Figure 2.2: Temporal contrast sensitivity function curve from Renzi and Hammond, 2010 22

Figure 4.1: Example of squint response captured for glare discomfort analysis 49

Figure 5.1: Example of goggles with and without glare stimulus 62

Figure 5.2: Image of the mobile glare goggles during a trial.....63

Figure 5.3: Plot of stride velocity and stride length during the straight path gait task 73

Figure 5.4: Plot of stride velocity, stride length, and gait termination time during the planned gait termination task.....73

Figure 5.5: Plot of stride velocity, stride length, and gait termination time during the unplanned gait termination task.....74

CHAPTER 1

INTRODUCTION

Concussions are a rising epidemic that affect millions of people every year.[1–5] Concussion rates have risen from 1.7 per 10,000 athletic exposures in 1988-1989 to 4.47 per 10,000 in athletic exposures from 2009-2014 within collegiate sports.[6–8] A rise in concussion incidence is also seen in high school and adolescent children.[7,9,10] Elite level sports, where concussions are more common, like the National Football League, have even demonstrated a rate of 66.1 per 10,000 athletic exposures.[7,11] While concussion education and identification have improved, leading to more diagnoses, 35-62% of concussions still go unreported demonstrating that these rates underestimate what may be truly experienced.[7,12–15] Concussions result in clinical symptoms, physical signs, cognitive and neurobehavioral impairments, sleep/wake disturbances, and visual dysfunction.[16] Of the resultant impairments, the extent of visual dysfunction may be the least understood despite its prevalence.

Although the entire visual system is not typically assessed post-concussion, up to 69% of adolescents and 90% of adults report oculomotor dysfunction following concussion.[17–19] This is not surprising given that over 50% of the brain is devoted to vision. Oculomotor dysfunction may result in diplopia, abnormal pupils, and an increase in abnormal eye-tracking movements.[16–19] Despite being classified as a mild traumatic brain injury, concussions can lead to lasting neurological and functional effects. Increasing evidence suggests that concussions are not a transient injury but rather a condition with potential chronic effects, including lasting impacts to

vision. Understanding visual disruptions post-concussion is the first step in understanding many of the subsequent deficits following concussion.

Our daily lives rely heavily on the ability to accurately and efficiently process visual stimuli, whether reacting to headlight glare while driving or scanning a complex game environment in real time. The ability to react swiftly and accurately to different stimuli is critical for ensuring safety in various scenarios. Concussions disrupt this processing,[20–22] yet current assessments do not fully capture the sensory-perceptual changes that follow injury. Most visual assessments focus on oculomotor control, including saccadic movements, smooth pursuits, fixations, and the vestibular-ocular reflex.[20,23,24] Clinical assessments, such as King-Devick and the Vestibular Oculomotor Screening assessment, were developed with the intention to isolate these movements.[20,24] Additionally, modern visual tracking technology is used with a focus on these movements.[19] While important, these assessments do not assess true visual deficits experienced post-concussion. The inability to measure perception and sensation of visual stimuli limits our understanding of how concussions impact vision and cognition. For example, critical visual components such as sensory and receptor pathways connecting to the brain's visual centers are ignored.[21,22] The brain has the ability compensate for injury without human comprehension in real time in order to maintain adequate function on a singular task at hand, although it may be less efficient in doing so.[25,26] Therefore, measuring visual function and the magnitude of variability on different tasks at a sensory level could provide a detailed profile of the visual system and its current recovery status post-concussion which is not currently understood.[25,26]

Visual psychophysics is a field of study that seeks to quantify the relationship between physical stimuli commonly encountered in everyday life, such as light and motion, and the

perceptions and sensations they evoke. Despite its importance in understanding visual and neurological function, this line of research has not been explored in individuals with a concussion history. However, it has been used in other clinical populations such as aging populations, patients with dementia, and those with tumors or lesions, proving to be sensitive to these conditions.[27–31] Integrating visual psychophysics into a concussed population could reveal novel insights into visual sensory and receptor damage that is currently overlooked. The brain can compensate for injury in ways not felt or experienced by humans in real time, so measuring visual function at a sensory level following concussion would provide a detailed profile of the visual system and its current recovery status, which is not currently understood.[25,26] This would potentially lead to improved management strategies and outcomes.

Psychophysics assessments such as macular pigment optical density (MPOD), glare sensitivity measurements, and temporal contrast sensitivity offer detailed information on visual acuity, neurocognitive processing, and retinal health. These measures can provide a more complete picture of the visual and cognitive impact of concussions, ensuring that the true sensory aspects of the visual system are not overlooked.[32–37] For example, assessing macular pigment, composed of lutein and zeaxanthin, is particularly relevant in the context of concussion as it plays a critical anti-inflammatory and antioxidant role in the brain and retina.[38,39] Low MPOD has been associated with impaired neurocognitive processing and increased susceptibility to oxidative stress.[40] Given the inflammatory nature of concussion,[41] assessing MPOD may serve not only as an indicator of retinal health but also as a window into the broader inflammatory state of the brain.[25–27]

Beyond simply understanding the extent of post-concussion visual impairments, it is important to recognize that everyday life requires the complex integration of visual information

during movement. Functional movement patterns are important to identify and understand because of the ability to potentially identify injury concerns.[42–45] Following a concussion, there is an increased rate of musculoskeletal injury, and more research is needed to understand the underlying causal mechanisms.[45,46] Prior research has identified that gait impairments can lead to an increase in musculoskeletal injury risk even in non-concussed individuals.[47] Acutely concussed individuals demonstrate more conservative gait strategies that can be detrimental to performance and may make someone more vulnerable to injury.[48–50] Importantly, alterations in gait patterns can linger beyond when a concussed individual is deemed healthy.[51,52]

Previous research has established links between eye movements and performance on simple gait and balance tasks.[53,54] However, as mentioned, these eye movements do not reflect the entire visual system and its current state during movement.[21,22,25,26] Although concussions alter gait performance and cause visual impairments little is known on the complex relationship between the two. No previous work has attempted to measure the influence of visual stimuli on simple gait. This is crucial to identify because common everyday visual stimuli may play an unknown role in the reason an increased risk of additional injury is identified post-concussion.[45,46] For example, light sensitivity is a common symptom following concussion, but it is not known if glare stimuli, which causes light sensitivity, can alter gait movement strategies in ways that may increase risk of further injury.[55,56]

In addition to identifying the relationship between visual function and gait, it is critical to understand how increasing the complexity of gait may alter performance. Higher difficulty tasks, like dual-task and complex gait tasks, can detect motor deficits subacutely after concussion.[50] For example, dual-task tandem gait has shown sensitivity to lingering deficits post-concussion that were not identified during just single-task conditions.[50,57] A gait termination task can

effectively evaluate complex gait and has been used to assess gait acutely and subacutely post-concussion.[58,59] However, it is unclear whether a history of concussion affects performance on this task and how visual stimuli influence performance on this task. Gait and motor control deficits might persist for months or even years after the initial injury, making the evaluation of these potential deficits valuable.[51,52] If a history of concussion is linked to impaired gait termination and impairments increase in the presence of visual stimuli, it may provide valuable information on the factors that influence injury risk post-concussion. Therefore, the purpose of this dissertation was to investigate performance on visual psychophysics measures among individuals with and without a concussion history, and to identify the visual influence on simple and complex gait assessments.

Aim 1: To determine whether individuals with a history of concussion exhibit poorer visual psychophysical performance compared to individuals with no history. Since vision is primarily processed in the brain, disruptions to visual function can reflect broader neurological deficits. We assessed macular pigment optical density (MPOD), glare sensitivity, temporal contrast sensitivity (TCSF), and the variability during each task to evaluate visual function. We hypothesized that individuals with a concussion history would demonstrate worse performance across all measures.

Aim 2: To investigate gait performance between individuals with and without a concussion history and examine whether a glare stimulus further influenced gait outcomes such as stride velocity, stride length, stride width, single leg support percent, gait termination time, and the within trial variability on each outcome. We used a gait walkway and portable glare goggles to

assess spatiotemporal gait outcomes during simple walking and planned and unplanned gait termination tasks, both with and without glare exposure. We hypothesized that individuals with a concussion history would demonstrate more conservative gait patterns, worse performance across all gait tasks, and increased variability during trials, and that glare would exacerbate these effects.

CHAPTER 2

LITERATURE REVIEW

Concussion

Epidemiology

Concussions are a rising epidemic that affect millions of people every year.[1–5] Within the military, traumatic brain injury, especially concussions, are the most common traumatic injury Service members experience.[60] One of the most common causes is sport participation. Athletic concussion rates have risen from 1.7 per 10,000 athletic exposures in 1988-1989 to 4.47 per 10,000 in athletic exposures from 2009-2014 within collegiate sports alone.[6–8] A similar rise in high school and adolescent children is also noted.[7,9,10] Elite level contact sports, like American football in the National Football League, have demonstrated a rate of 66.1 per 10,000 athletic exposures.[7,11] Also in the National Football League, one study found that there were 61.7 concussions per 100 regular season games between 2015-2019.[61] Other high contact leagues, like the men's National Hockey League, have seen concussion rates continually rise from 0.42 concussions per 100 games in 1986 to 4.88 concussions per 100 games in 2012.[62]

Other common injury mechanism includes motor vehicle accidents and military-related activity (e.g., blasts, falls, training injurie). Between 2006-2012, the rate of concussions seen in emergency departments in the United States rose from 569.4 per 100,000 visits to 807.9.[3] Motor vehicle accidents often result in various injuries, with 1 in 61 individuals sustaining a concussion during an accident.[63] Of all brain injuries in the military, 83.1% are classified as concussions.[64] Broken down by military branch, 84.03% in the Army, 81.04% in the Navy, 84.13% in the Air

Force, and 80.6% in the Marines of all traumatic brain injuries reported between 2000-2023 were concussions.[64] Since it is very difficult to track during deployment, 80% of injuries are diagnosed before or after deployment.[60] One study found that undergraduate students who get treated at a student health clinic has a concussion prevalence of 4.3% of all conditions treated, with an incidence rate was 4.5 per 1,000 person months.[65] In line with athletics, female undergraduate students experienced higher rates than males.[65] The rise and prevalence rate of concussion increasing could be due to better education and reporting behaviors. While concussion education and identification have improved, leading to more diagnoses, 35-62% of concussions go unreported, demonstrating these rates still underestimate what may be truly experienced.[7,12–15]

Pathophysiology

In the most recent consensus statement on concussion in sport a concussion can be defined by a traumatic brain injury caused by a direct blow to the head, neck or body resulting in an impulsive force being transmitted to the brain that occurs in sports and exercise-related activities.[16] Symptoms and signs may present immediately, or evolve over minutes or hours, and commonly resolve within days, but may be prolonged.[16]

The pathophysiology of concussion is often referred to as the neurometabolic cascade.[41] This cascade is broken into seven parts: ionic flux and glutamate release, energy crisis, cytoskeletal damage, axonal dysfunction, altered neurotransmission, inflammation, and finally, cell death.[41]

Beginning with ionic flux and the release of glutamate, there is a resultant efflux of potassium and influx of sodium and calcium which can trigger voltage- or ligand-gates ion channels.[41,66,67] This leads to a depression-like state that will cause post-concussion impairments.[41] In order to restore homeostasis, ionic pumps shift into overdrive, causing

hyperglycolysis, and depleting energy reserves and increasing ADP.[41,68] An increased energy demand results in an uncoupling between energy supply and demand.[41] The intracellular calcium flux also leads to the sequestration of calcium into the mitochondria worsening the cellular energy crisis.[41] After this period of hyperglycolysis and metabolic uncoupling, glucose metabolic rates are impaired, lasting up to 7-10 days, leading to behavioral impairments in spatial learning.[41,68,69] The biomechanical forces placed on the neurons and glia result in damage to components such as the dendritic arbors, axons, and astrocytic processes.[41] Axons can lose structural integrity following intra-axonal calcium flux, causing neurofilament sidearms to become phosphorylated and collapse.[41,70] This damage, along with axonal stretching, can disrupt axonal transport and increase axolemmal permeability, leading to dysfunction and potential axonal disconnection.[41,70,71] Even if axonal disconnection occurs and does not lead to cell death, the neuron will not be able to function normally. After axonal dysfunction, altered neurotransmission results due to changes in ligand-gated excitatory and inhibitory neurotransmission.[41] Glutamate receptor subunit composition and functional alterations occur with the potential interference of normal developmental plasticity, electrophysiology, and memory.[41,72–75] The post-injury excitatory inhibitory balance can be upset by the disruption of inhibitory neurotransmission involving GABA and its receptors.[41] While studies were performed in rat models this dropout of GABAergic interneurons can lead to the development of anxiety disorders and affects the amygdala which is critical in fear-based learning.[41,76–80] Although previously overlooked in concussive injuries, an inflammatory response is triggered.[41] This response, with the upregulation of cytokine and inflammatory genes, has been associated with damage to the substantia nigra and can increase risk for Parkinson's disease.[41,81–83] It is worth noting that this inflammatory response and the resultant damage has been mostly focused on

more severe traumatic brain injuries, but it is also reported in mild injuries, like concussion.[84] Finally, while rare, complete cell death is possible.[41]

In relation to this project, ionic flux is associated with photophobia and migraine headaches highlighting a connection to the visual system and symptoms that also result from visual dysfunction.[41,85,86] Axonal injury and impaired neurotransmission lead to cognitive impairments, slower processing speeds, and slower reaction times, all of which are linked to the visual system.[41,87–89]

Visual Performance Post-Concussion

Over 50% of the brain is devoted to vision, explaining why oculomotor dysfunction is one of the most common complaints following a concussion.[90] Oculomotor dysfunction can result in specific impairments like diplopia, abnormal pupils, and an increase in abnormal eye-tracking movements. Visual impairments can occur following injury to both afferent and efferent systems.[21,22] Afferent dysfunctions are associated with optic nerve, white matter tract, and cortical damage which causes impairments to visual acuity, color vision, contrast sensitivity, visual field deficits, spatial neglects, and visual processing.[21,22] Efferent damage may result in impairments to accommodation, pursuits, saccades, stereopsis, convergence, and pupillary reaction time.[21,22] Definitions for these terms are seen in Table 1.[21,22,91–98] One study attempting to identify the frequency of oculomotor dysfunction in adolescents found that 70% of individuals who had sustained a concussion experienced oculomotor dysfunction.[18] The most common dysfunctions were vergence disorders (60%) and accommodative disorders (57%).[18] Another study identified similar rates with 69% of adolescents experiencing a vision diagnosis, with accommodative disorders (51%) and convergence insufficiency (49%) again being the most common.[99] In adults with a concussion, as high as 90% of individuals experience oculomotor

dysfunction.[99,100] The current state of post-concussion oculomotor function assessment focuses on the efferent visual system and saccadic movements, accommodations, smooth pursuits, fixations, vergence and the vestibular-ocular reflex.[17,19,23,90,101,102] Definitions for these measures are seen in Table 2.1.[21,22,91,92,94–98]

Table 2.1. Visual terms definitions

Term	Definition
Visual acuity	The clarity or sharpness of vision
Color vision	The ability to discriminate based only on spectral differences between stimuli
Contrast sensitivity	The ability to perceive sharp and clear outlines of objects and identify differences in shadings and patterns
Visual field	The portion of space in which objects are visible at the same moments during a steady gaze in one direction
Spatial neglects	The reduction or loss of spatial awareness
Visual processing	The detection of movement, pattern, and color and integrating all these inputs into a coherent picture
Stereopsis	Perception of depth due to the slight positional differences in images received by each eye
Pupillary reaction time	Time to contract pupil in response to light
Saccades	Rapid eye movements that enable quick and accurate scanning from one object to another
Accommodations	The ability to maintain or change focus by adjusting focal length using ciliary muscle control
Fixations	The ability to steadily gaze at an object
Pursuits	Eye movements to smoothly and accurately track a moving object
Vergence	The ability to aim eyes in opposite directions to follow an object as it moves towards or away oneself
Vestibular-ocular reflex	The use of eye muscles to create an eye movement opposite to that of head movement at the same speed to stabilize the retinal image by keeping focus on an object despite head motion.

One of the most common vision-based assessments on the sideline or in the clinic is the King-Devick test. Performance on this test is based on rapid number naming, while capturing eye movements, during a trial of reading numbers arranged in variable horizontal spacing requiring concentration, attention, and language skills.[90,103] Impairments in any of these skills could result from a concussion with a common performance deficit being slower reading time.[90,103] The Vestibular Ocular Motor Screening (VOMS) assessment is another very common clinical measure to assess vestibular and visual-related impairments following a concussion.[20,104] It consists of measurements in five domains: smooth pursuits, horizontal and vertical saccades, convergence, horizontal vestibular ocular reflex, and vertical motion sensitivity.[20] Following

each measurement, the individual is also to rate any change in headache, dizziness, nausea, and fogginess on a scale of 0 (none) to 10 (severe).[20] An increase in symptom provocation and convergence distance greater than 5 cm are findings that can identify concussed individuals.[20] However, similar to King-Devick, this assessment has a basis in having an individual perform saccadic, convergence, or fixation movements. The main difference is that VOMS includes the assessment of the vestibular ocular reflex, and King-Devick includes rapid number reading. Both King-Devick and VOMS are clinically valuable as they are sensitive to concussion.[24,105–108] Performance on these tests have also been linked to recovery time from concussion.[107,108] However, even with a link to recovery they still are not able to assess the state of the sensory and receptor components of the visual system the way psychophysical assessments can. This limitation means the results of King-Devick and VOMS may be confounded by the brain's adaptations instead of the true extent of visual system deficits post-concussion.[25,26]

Attempts have been made to incorporate technology and more objective measurements of post-concussion visual performance. Camera-based eye tracking technologies are becoming more common, as they are accurate, objective, and non-intrusive.[19,109–111] However, a systematic review and meta-analysis on the use of eye tracking technology post-concussion identified that the predominant focus of this technology was on smooth pursuit or saccadic eye movements.[19] As previously stated, these movements are important to identify as the provide information on brain areas involved in attention and executive function and with the use of camera-based systems it allows for more objective and accurate measures.[19,21,22] One study has even focused on micro-saccadic movements showing the depth and preciseness at which these camera systems can measure,[112] but are still used for the same style of movements that have been previously

studied. While providing more accurate measures of these movements, there has been very minimal literature focusing on any other visual performance outcomes.

With efferent parts of the visual system being heavily addressed in the current literature,[21,22] more work is needed to address the afferent visual system to identify if impairments exist for the sensory aspect of vision instead of just oculomotor. Typical concussion recovery takes place within six months, but one study found visual symptoms in military individuals persist greater than five years post-concussion.[113] Another study, this time college-aged individuals, also reported visual dysfunction over one year following concussion.[114] Identifying the extent to which the entire visual system is impaired could provide valuable information to clinicians to limit these lingering effects.

Visual Psychophysics

Macular Pigment Optical Density

Structural isomers known as carotenoids are broken into subclasses known as xanthophylls and carotenes.[38] They are synthesized by plants and designed for the coloration and absorption of light energy.[38] Like all carotenoids, since they are not made within the body they must be obtained from dietary items such as dark green, leafy vegetables and supplements.[38] Lutein and zeaxanthin, which are xanthophylls and collective known as macular pigment, are concentrated within the macula more than any other part of the body.[38,115] As a result of their concentration, the macula appears yellow.[38,115] The concentration of both lutein and zeaxanthin is located in the center of the macula and begins to decrease eccentrically within millimeters outside of the center.[38] Focused centrally in the macula, it is designed to reduce and protect against short wavelengths of visible light, with maximum absorption of 460 nm

wavelengths.[38,116] These wavelengths are more dangerous since they are efficient at general reactive oxygen species.[38] The outer retina, which is oxygen rich, is very vulnerable to oxidative damage due to fatty acids that are susceptible to photo-oxidation and exposure to high-energy blue light, like the 460 nm wavelength continuing to demonstrate the importance of macular pigment.[38] Lutein and zeaxanthin are also antioxidants to aid in reducing reactive oxygen species.[38]

Initial attempts to measure macular pigment used serum carotenoid levels or dietary surveys, but they were not good indicators and non-invasive techniques were needed, especially for clinicians.[38] Macular pigment optical density (MPOD) is a non-invasive measure of how well the macular pigment in the eye attenuates blue light.[38] MPOD is associated with the amount of lutein and zeaxanthin in the macula.[38] Due to the decreasing and undetectable levels of macular pigment at 6 to 8 degrees eccentricity it has been recommended to assess MPOD at multiple locations to gain a complete profile of the true MPOD levels.[117] Only measuring MPOD at one central site may not accurately predict the true amount of macular pigment one has.[38,115] The distribution of macular pigment is believed to be based on the distribution of the cone photoreceptors, which also decrease from the center of the fovea outwards.[116] Cone photoreceptors are critical for detecting color and visual acuity.[118]

Multiple techniques are available to measure MPOD, but one of the most commonly used is heterochromatic flicker photometry.[38,115,117] This procedure uses a 460 nm blue test wavelength, which is absorbed by macular pigment, and a green wavelength around 550 nm, but can vary from 550 to 570 nm.[115] Measurements are made at the center of the fovea and a location where there is minimal macular pigment.[115] Both wavelengths are presented in a square wave alternation pattern with the test stimuli at the foveal location presented at 12 to 15 Hz and 6 to 7

Hz for the parafoveal condition.[115] The stimulus appears flickering to the subject, and they are to adjust the intensity of the blue light stimulus until the flickering ceases.[115] Subjects with higher MPOD values indicate better protection from harmful wavelengths.[115] While this method is a good non-invasive approach, methodological concerns were raised regarding temporal sensitivity, light transmission, and perceptual differences at the foveal and parafovea location.[119]

To ensure the best results and highest accuracy, customized heterochromatic flicker photometry was developed. This involves two additions to the traditional procedures. The first procedural addition is to determine the best flicker frequency for each subject.[119] If this is not performed there is a chance that the subject will experience a large zone of no flicker as the settings are adjusted, or they will not be able to eliminate flicker at all.[119] The use of a quick critical flicker fusion (CFF) task will reduce measurement error and ensure good performance on the MPOD task.[119] For example, older subjects and those with age-related macular degeneration (AMD) have significantly slower temporal vision, so optimizing the flicker rate is crucial for them.[119] Secondly, the short-wave test stimulus is to be inversely yoked with the mid-wave reference component.[119] This means when the short-wave is increased the mid-wave is also decreased, and vice versa. This was created to keep the stimulus brightness the same and to not confuse brightness changes as changes in flicker.[119]

No research has assessed macular pigment within individuals with a concussion. However, multiple studies show the importance of macular pigment in protecting against AMD. It is estimated that 54% of visual impairment and 22.9% of blindness are a result of AMD.[120] It is also worth noting that AMD is irreversible, so it is crucial to identify all that influences AMD, especially early onset.[38] Rates of early onset AMD cases are estimated to continue to rise and increase to 17.8 million cases by the year 2050.[121] There is adequate evidence supporting the

consumption of lutein and zeaxanthin reducing the risk and prolonging the effects of AMD,[38,122–124] but more information is needed to develop a causal relationship. In addition to visual impairments, MPOD is associated with worsening cognitive performance.[125] Using the Montreal cognitive assessment and mini-mental state examination, one study found those with lower MPOD values has poorer prospective memory, took longer to complete a trail-making task, and had slower reaction times compared to individuals with higher MPOD values.[125] Within aging and mildly cognitively impaired groups a similar trend was identified with worsening/aging participants associated with lower macular pigment levels compared to healthy control groups.[126]

Within brain tissue, specifically in the cerebellum, pons and frontal and occipital cortices, lutein and zeaxanthin have been discovered.[127,128] Importantly, macular pigment levels are found to significantly correlate with the concentrations found within the brain.[128] Similar to how the retina has an important protective role within the eye, it is possible that these carotenoids within the brain can provide functions like antioxidant, anti-inflammatory, structural and functional enhancement of synaptic membranes and gap junction communication.[39] These functions are critical to deter potential cognitive impairments caused by disease or injury. One research group found that patients with Alzheimer's disease exhibit lower macular pigment, worse vision, and higher rates of AMD compared to controls.[129] Supplementing lutein and zeaxanthin, and increasing macular pigment, has resulted in improvements to visual function in patients with Alzheimer's.[130] Other recent work has identified macular pigment levels are related to better global cognition, verbal learning and fluency, recall, and processing and perceptual speed.[40] These relationships have been crucial to identify as they demonstrate that in addition to macular pigment being an important factor for visual performance, it may also demonstrate use as a biomarker for cognitive function. In relation to this project, aging, Alzheimer's and cognitive

performance have all been linked to concussion. In individuals over the age of 65, one study found those with a concussion performed worse on composite neuropsychological and computerized tasks, with age being a direct predictor of cognitive performance compared to a control group.[131] Based on the evaluation of event related potentials, a subset of electroencephalogram (EEG), older concussion history populations may be unable to compensate for their neurophysiological deficits, especially on complex tasks, as well as younger concussion history groups.[132] One study that focused on the early onset of Alzheimer's in retired football players noted that although there was no association between the number of recurrent concussions and Alzheimer's, there was an earlier onset of the disease compared to the general American male population.[133] Other contradictory work has found no early onset dementia in retired contact sports athletes.[134] Cognitive performance impairment is one of the trademark aspects of a concussion leading to a lower health-related quality of life.[135] The link between aging, Alzheimer's, cognitive performance and concussion, as well as macular pigment, is crucial to understand as it demonstrates a potential link between a concussion and visual psychophysical measures. The ability to prolong and aid in minimizing visual decline using supplementation within these populations related to concussed groups suggest these same methodologies may be able to be focused on improving individuals visual and cognitive performance who had sustained a concussion.[38,122–124]

Glare

Glare is the result of light entering the eye in excess of its adaptive state that does not aid vision.[136,137] Humans experience glare in everyday life from sources such as the sun, car headlights, and indoor lighting. Many factors can influence how uncomfortable a light is including the adaptive state, spectral composition of the glare source, luminance of the

background and glare source, spatial properties of the glare source, angle of the glare source with respect to the line of sight and the time of day.[138,139] Macular pigment also influences outcomes as individuals with more macular pigment have faster photostress recovery times, lower disability glare contrast thresholds, and lower visual discomfort.[136,140–142] Different categories of glare exist with two of the most common being disability glare (glare that impairs vision) and discomfort glare (glare that causes annoyance).[137] They are usually concurrent, but may be independent, and can range from insignificant to incapacitating for humans.[137] Disability glare is caused by intraocular light scattering (straylight) that reduces the contrast of retinal images by spreading a veiling luminance across them.[137] The point spread function (PSF) describes how light from a point stimulus is focused on the retina.[137] Straylight is the light energy that spreads beyond one degree from the center of the PSF.[137] Photostress is the exposure of very short, intense light that also results in loss of vision.[136] This loss of sight is cause by a combination of photopigment bleaching and adaptation and requires photopigments to regenerate slowly over many seconds.[136,137] Discomfort glare is caused by light that is deemed too intense or variable for a human.[137] Discomfort can be manifested as subjective annoyance measures, squinting, distraction, blinking, tearing, pain, and light aversion.[137] An abnormal response to discomfort glare is referred to as photophobia.[137]

Like many visual functions, alterations in glare discomfort are noticed with aging populations. With an increase in aging there is an increase in straylight which is due to large particle light scattering by multilamellar bodies in the crystalline lenses.[137] Cataracts, which is common in older adults, also increases glare disability.[143] This is crucial because older drivers in conditions where glare is present, i.e., nighttime driving with headlights, put themselves at greater risk of crashing and sustaining an injury if they cannot see properly.[144] A study focused

on glare while driving found that subjects who experienced increased susceptibility to glare and diminished vision reported more real-life accidents over a five year span.[144] Photostress recovery time, which is the time from when a photostressor is experienced until when vision returns to normal, is linked to AMD with prolonged recovery times being associated with both early and late cases of AMD.[145,146]

When evaluating glare discomfort in experimental settings, the spectral qualities of the light source are important to consider. The custom-built stationary glare apparatus and portable glare goggles used in this study each consist of a circular ring of bright white LEDs, with the lights in the portable version encased in an adjustable goggle frame and powered by a battery pack. These LEDs are considered broad-spectrum and produce a strong spike in blue light at around 460 nm which is the same wavelength as bright blue sky light.[147,148] This type of LED is commonly used and is also a well-documented contributor to glare discomfort.[147] A typical spectrum of this kind of LED light and its intensity is seen in Figure 2.1.[149] The discomfort induced by such lighting is especially relevant in ecologically valid environments, as blue-rich white light has been shown to exacerbate subjective annoyance and visual strain.[147]

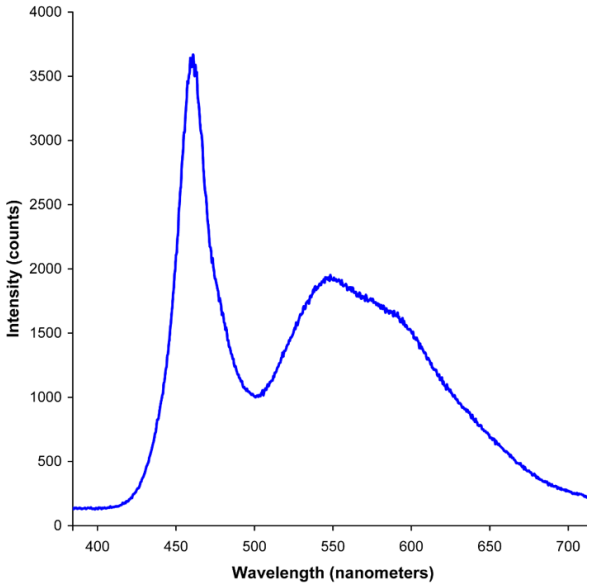

Figure 2.1. Typical spectrum of LED light used in glare assessments[149]

While objective measures of post-concussion glare may be limited, photophobia is commonly associated with concussion.[150] It has been considered the second most common symptom of concussion, and post-concussion syndrome, with 60-75% of Service members experiencing photophobia after blast-related concussion.[56] In a separate study not limited to military-specific injuries, it was estimated that 43% of individuals after a concussion experience photophobia with the majority of these patients rating it as severe.[55,56] While it is present acutely after concussion, patients with photosensitivity were also found to have an increase in sensitivity to light up to six months following concussion.[151,152] It is thought that this may be due to a variety of causes such as magnocellular damage, cortical hyperexcitability, and binocular vision disorders.[152] Recent research has also implied that intrinsic photosensitive retinal ganglion cells, which project to both trigeminovascular and trigeminoautonomic pathways, contribute to photophobia which may play a role in post-concussion light sensitivity.[153] Given its potential to interfere with daily activities such as mobility, reading, and driving, photophobia should be thoroughly evaluated as part of post-concussion assessments.

Only one subjective question on common clinical concussion assessments addresses sensitivity to light. On the Sport Concussion Assessment Tool 6 (SCAT6) symptom evaluation individuals are asked to rate their sensitivity to light on a scale from 0 (no sensitivity) to 6 (extreme sensitivity).[154] This question is similar to the De Boer index which quantifies glare discomfort on a scale from 9 (unnoticeable) to 1(unbearable).[155] Little work has focused specifically on the SCAT sensitivity to light symptom on its own. However, in adolescents, light sensitivity on the SCAT was a significant predictor for distinguishing between concussed and non-concussed individuals, highlighting the importance of assessing this symptom.[156] No objective measures for glare disability or discomfort are common post-concussion. This is an

important gap in the literature to identify as light sensitivity may last up to months or years following injury.[55] More is needed to assess the impact of glare following concussion.

Temporal Contrast Sensitivity Function

Human's visual contrast sensitivity function characterizes its frequency response, and it can be described as a bandpass filter, increasing and decreasing sensitivity at various frequencies.[157] The contrast sensitivity function can be broken into temporal and spatial vision.[158] Temporal vision is characterized by the sensitivity to contrast as a function of time, known as the temporal contrast sensitivity function (TCSF).[158] This is different from spatial vision which is sensitivity to contrast as a function of spatial frequency.[158] In order to obtain this function, one must present a stimulus that varies sinusoidally over time.[158] The visibility of the modulated stimuli is dependent on the rate of presentation and the depth of modulation which refers to the difference between the maximum and minimum luminance of the stimuli.[158] For example, a higher modulation depth means that there is a greater difference between the maximum and minimum luminance of the stimuli. The TCSF can be depicted by an inverted U-shaped function with high frequencies decreasing sharply, and less sharply at low frequencies (Figure 2.2).[159] While the specific anatomical properties of the TCSF may not be fully understood, it is accepted to view TCSF as an envelope of more narrowly defined sub-channels, as it is unlikely temporal channels operate in isolation.[158,159] These temporal channels also do not operate in isolation with interactions between spatial, temporal, and chromatic channels.[158]

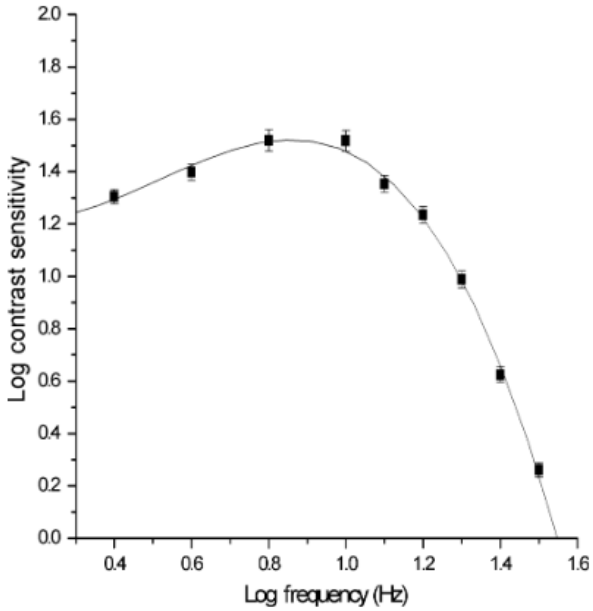

Figure 2.2. Temporal contrast sensitivity function curve from Renzi and Hammond, 2010.[159]

Critical Flicker Fusion (CFF) does not fully characterize temporal vision, but it has been used as an index of temporal vision.[28,159] CFF is the highest flicker frequency someone can detect at 100% modulation.[159] Prior research has stated that this function of temporal vision is determined post-receptorally, and even after someone believes the stimulus is no longer flickering the retina is still responding to high-frequency flicker.[159,160] CFF is correlated with cortical arousal as measured by the alpha component of an EEG.[161,162]

Many factors, like any visual function, are important to capture and interpret with TCSF and CFF. These factors include stimulus size (Granit-Harper law),[163] luminance (Ferry-Porter law),[164,165] and retinal location.[158] The Granit-Harper law describes how stimulus size influences visual function.[163] For example, in relation to CFF, the Granit-Harper law states that CFF values increase as the size of the stimulus increases. The Ferry-Porter law states that CFF increases

directly with the logarithm of stimulus illuminance.[164,165] For example, increases in the luminance of the stimuli result in increases in CFF.

Control of these factors when measuring TCSF can be challenging. A practical method for measuring TCSF has used a device that was customized to successfully measure TCSF while controlling for potential confounding factors.[158] To minimize absorption by the anterior media and macular pigment, a red LED stimulus consists of a one-degree, 660 nm target with a 5.5-degree 660 nm surround.[158] A knob is presented to the participant to allow for fine changes in modulation depth from 0% to 100% at different frequencies.[158] A 3mm artificial pupil is used to control pupil size variation.[158] This size was determined to be the smallest consistent size for humans regardless of individual differences.[158] Similar to MPOD, a foveal and parafoveal measurement is needed.[158] Participants are asked to view a point and fixate at a point in the center of the vision for the foveal measurement, and a fixation point 7-degrees laterally for the parafoveal measurement.[158] Starting at 0% modulation the participant is to use the dial and adjust the depth of the modulation until they see a fused stimulus.[158] There are also trials where the participant begins with a fused stimulus and adjusts the modulation until a flicker is just noticeable.[158] Previous research has used multiple frequencies to capture the entire TCSF and confirm the recreation of the U-shaped curve.[158] Multiple studies performed by Wooten et. al. and Renzi and Hammond have used the frequencies of 2.5, 4, 6.3, 10, 12.6, 15.8, 20, 25, and 32 Hz.[158,159] All frequencies are assessed in the fovea and parafovea. For the purposes of this project, not all frequencies will be assessed. This is due to consistent replication of the TCSF which allows for the calculation of the curve without all frequencies measured, if low, medium, and high frequencies are measured to encompass the extremes of the curve. A previous study using four frequencies (0, 5, 10, 15 Hz) was able to recreate the same temporal curve seen in

Figure 1.[166] This study also noted that the differences between the 5 and 10 Hz frequencies were minimal and considered within measurement variation demonstrating that potentially one frequency within the 5-10 Hz range will suffice for measurement.[166] CFF is measured on the same device with the same one-degree stimulus at the center of a 5.5-degree, 660 nm surround.[167] Only focused on the center of the target for this measure, light flickers at 100% modulation and the participant identifies when flicker ceases or is just viewable as the frequency is adjusted.[167]

TCSF, and CFF, provides valuable information into the functioning of the visual system, but also cognitive processing and neural integrity.[167] Specifically, many studies have focused on the impact of aging and disease on the TCSF and have identified the measure as prognostic.[167,168] As is the case with other aspects of neural processing, aging and various disease cause significant processing slowing which is due to anatomical and physiological alterations.[158] Due to age-related breakdown of myelin the conduction rate of neural axons is decreased.[169] It was previously found these temporal-based measurements are sensitive indicators of optic nerve damage in patients with open angle glaucoma.[170] TCSF is sensitive to the visual processing ability, and any potential deficits.[168] Differing frequencies during the TCSF measurement are sensitive to different conditions as well.[171] Deficits at lower to moderate frequences are correlated with drusen (yellow deposits under the retina made up of lipids and protein) and/or retinal pigment epithelium atrophy.[171] These are known to be very accurate predictors of AMD development. One study found that 84% of people with early AMD had flicker abnormalities in their central foveal region.[172] TCSF at high frequencies is strongly related to normal aging and disease.[171] CFF is negatively affected by tumors and lesions in all four lobes of the brain,

demonstrating that it is an adequate measure of overall neural integrity.[173,174] Multiple studies have shown that Alzheimer's disease results in a decreased CFF performance.[158,159,174]

The full TCSF has not been assessed following a concussion, or in individuals with a history of concussion. CFF specifically has been minimally assessed in individuals with a concussion, but differing methods were used for these studies.[175–177] One study used a participant group ranging from 19-83 years of age with the concussion group ranging from 0.25-15 years post-concussion.[175] They also used a different stimulus and apparatus which used white LED's with a spectrum on 460-555 nm and a frequency range of 30-60 Hz instead of the method described above.[175] This study found no differences in CFF values between the concussed group and healthy group but did mention the concussion group had higher variability in their performance and trended toward lower CFF values.[175] This study did record whether motion or light sensitivities were present in the concussed individuals and found that CFF among the concussed group was higher for those with light and motion sensitivity compared to those not experiencing these symptoms at the time of testing. Another study, using the same methodology and similarly large age and time since injury ranges, also found no significant differences between a concussed and control group but also reported trends towards worsening performing in individuals with a previous concussion.[176] One other study using a Pocket CFF tester among other visuomotor and visual perception tasks found no differences in individuals with a previous concussion.[177] While not measured in concussed individuals, a study conducted using younger and older-aged groups used CNS Vital Signs, a common concussion neurocognitive assessment to understand the relationship between CFF and higher cognitive function.[167,178] The CFF methods from this study are similar to what will performed for this project.[167] The authors found, as they expected, that age was inversely related to CFF and all CNS cognitive measures except

for visual memory with higher age resulting in lower scores.[167] They also found that CFF predicted executive function across both groups and accounted for variance in performance.[167]

Psychophysics measures, i.e., MPOD, glare sensitivity, and TCSF, may provide critical information into sensory and perceptual visual functions that have not been measured in concussed individuals. Current common visual assessments post-concussion do not capture this information as they are limited to just oculomotor function which could be altered due to protective mechanisms by the brain.[21,22,25,26] Deficits on psychophysics tasks are linked to aging, cognitive decline, disease, tumors, and lesions.[27–31] Treatment strategies using supplementation has prolonged and aided in minimizing visual decline in these clinical populations.[38,122–124,130] These strategies could also be performed in people with a concussion, however, only a very small amount of work has focused on any of these assessments.[175–177] There has also been no work done on a link of visual psychophysics methods and gait in concussed individuals. With an increased risk of injury post-concussion,[47] visual sensation and perception may be a role in gait alterations which can lead to injury that has not been explored.

Increased Injury Risk Post-Concussion

Gait

Gait, the pattern of walking, is a fundamental part of human movement.[179] Within gait there is the stance phase, when the foot is on the ground, and the swing phase, when the foot is moving forward.[180] In one gait cycle, 60% is spent in stance phase while 40% is spent in the swing phase.[180] Disruptions to normal gait patterns can signal underlying health defects and various medical conditions.[179] In older adults, changes in gait patterns are associated with the likelihood of a being recurrent faller.[181,182] Aging populations exhibit lesser knee extension at

heel-strike and knee flexion during the swing phase.[183,184] Lower extremity injuries can also alter gait, causing antalgic gait (limping) when a person shortens their stance on the effected side and have a rapid swing phase with the contralateral leg to minimize loading.[180] A neurological disorder like Parkinson's can cause decreased gait speed, step height, and length.[185,186]

Gait is often impaired following concussion.[57,187,188] Gait analysis after a concussion has revealed neuromuscular control deficits that persist longer than what standard postural control assessments, such as the Balance Error Scoring System, can detect.[44,189] Gait is normally assessed through various measures including tandem gait and normal walking gait with single and dual task condition.[57] A single-task condition is when the individual performs the gait task and only focuses on the gait task, and a dual-task condition is when a cognitive task is performed while performing a gait trial.[57] Various dual-task conditions exist, ranging from counting backwards during the trial to terminating walking when a buzzer is heard, known as planned or unplanned gait termination.[57–59]

Currently there is little existing literature on planned or unplanned gait termination/initiation in individuals with a history of concussion. Healthy individuals without a history of concussion exhibit shorter stride and step lengths, slower gait velocity, wider stride width, and more time in double leg support during unplanned compared to planned gait termination.[58] Gait termination has been used in elderly and neurodegenerative clinical populations as a single- and dual-task gait measurement.[190,191] Few studies exist using gait termination in acutely concussed individuals. During gait initiation, acutely concussed individuals exhibit significantly slower step velocity and shorter step length compared to those with a concussion history and control groups.[59] Also acutely post-concussion, it was found that concussed individuals exhibit alterations in braking and propulsion patterns.[59] These alterations

were found to linger into the subacute phase, showing that this task is sensitive enough to pick up on changes in gait even post-recovery on other clinical concussion tests.[59] While it seems to be useful immediately following a concussion, no work has been done to identify gait termination deficits in individuals with a history of a concussion and adding visual stressors to mimic everyday life while performing this task. This could provide valuable information on the true recovery time for performance on the gait termination task following a concussion, and how real-world influence may alter performance potentially adding to why there is an increased risk of injury following a concussion.

Vision and Gait

Vision during gait is critical as it allows for us to collect crucial information about the environment and remain safe.[54,192] Previous work has described the importance of saccadic movements on efficient locomotion.[192] For example, the visual information gathered during the latter half of the preceding step influences the step length of the following step.[193] On uneven terrain it was found that fixations increased to where individuals eventually stepped.[194] The timing of footfalls is altered based on the amount of visual information present, e.g., slower footfalls in conditions with less visual information.[195] During turning tasks, the initiation of saccadic movement preceded the head, trunk, or leg, continuing to support that movement is influenced by vision.[196,197] In an elderly population participants with a fall history focused on the more imminent footfall target instead of two steps ahead of them which is common for healthy participants.[198,199] A study on individuals with Parkinson's disease, who are known to have gait and saccadic abnormalities, found that patients who would freeze during gait were slower in initiating saccadic movements and had increase saccadic variability.[200] In concussion, gait and vision have been dually assessed rarely.[54] It was found that people with a concussion had

reduced saccadic frequency, duration, and velocity which was correlated with gait velocity.[54] However, like previous visual studies focused on concussed individuals, the visual tasks of interests in these studies are focused on saccadic and fixation movements which ignores the sensory and receptor pathways connecting to the brain's visual centers. The current study aims to provide an understanding of the visual sensory and perceptive systems which has not been done in people with a concussion.[21,22] In addition, it will add to the literature on how gait is influenced by visual stimuli. Instead of a focus on how saccadic movements are linked to gait, we will identify how visual stimuli common in everyday environments may alter gait. This will provide valuable insights into the interaction between visual processing and motor control post-concussion which can lead to improved management strategies.

CHAPTER 3

METHODOLOGY

Participants

Participants were included if they were 18 years of age or older. Concussion history was collected and self-reported using the National Institutes of Health common data element form (Supplementary Figure 1),[201] and participants were categorized into either a concussion history or no concussion history group. Per the 6th international consensus statement, a concussion was defined as a "traumatic brain injury caused by a direct blow to the head, neck or body resulting in an impulsive force being transmitted to the brain."[16] Participants in the concussion history group must have sustained a concussion within two years of study enrollment and be asymptomatic, defined as having a total severity score ≤ 13 on the symptom inventory (Supplementary Figure 2).[202] This cut off was based on normative data that, at baseline, males have a 2.7 total symptom score, and females have 2.9, which was rounded up to 3.[202] The published reliable change score of 10 was then applied, resulting in a final cutoff score of 13.[202]

Participants were excluded if they reported a significant lower extremity injury (e.g., fractured leg, lower extremity surgery) within a year of testing, visual impairment (measured by the test administrator to be greater than 20/50 even if corrected), neurological disorder, or ocular disease. Those in the no concussion history group must have never had a diagnosed concussion.

Participants without a self-reported concussion were matched to those with a history based on age (±2 year), sex, and height (±10cm). Participants were recruited from across the university through online university postings, flyers, and in-class verbal recruitment. These

methods have previously been used successfully.[203,204] Participants were also paid an honorarium of $20 in total for participation.

Demographics

Demographic information included age, sex, height, mass, dominant eye, activity level, and injury history. Participants were also given the National Institutes of Health concussion history common data element form.[201] Concussion mechanism, diagnosed/undiagnosed concussion, date of concussion, age at time of injury, loss of consciousness, loss of consciousness duration, difficulty remembering before or after concussion, minutes with memory loss, and symptom duration were obtained from this form.[201]

Sport Concussion Assessment Tool 6 Symptom Inventory

The Post-Concussion Symptom Scale is a 22-item symptom checklist designed to assess and track symptoms following a concussion.[205,206] Each symptom was scored on a Likert scale from 0 to 6, with 0 indicating the symptom is not present, and 6 indicating the symptom is present and severe. The total number of symptoms was calculated by adding the symptoms that are present together, with a possible score of 0 to 22.[205,206] Total symptom severity was calculated by adding the total severity values from each symptom with a possible score of 0 to 132.[205,206] This scale was administered upon enrollment, and after the completion of each assessment to identify if, and by how much, the visual psychophysics and gait tasks increase total symptom burden as compared to the participants' baseline, which was the first one completed upon enrollment.

Temporal Contrast Sensitivity

The assessment order of temporal contrast sensitivity and macular pigment optical density were randomized. There were two parts to the measurement of temporal contrast

sensitivity: temporal contrast sensitivity function (TCSF) and critical flicker fusion threshold (CFF).[167] Although CFF is a component of the TCSF, slight modifications to the test stimulus were necessary to accurately measure CFF.[167] Therefore, it was treated and described as a standalone assessment. The order in which the TCSF and CFF are measured was randomized. Participants were measured using their dominant eye. Dominant eye was assessed by instructing the participant to focus with both eyes on an object smaller in size (e.g., doorknob, letter on a Snellen eye chart).[207] The participant then brought their hands together in front of them to form a triangle-shaped hole with the focal point in the center of that hole.[207] They were then instructed to close one eye at a time and verbally indicate when the target remains visible through their hands and when it does not remain in focus.[207] The eye that was open when the target was still visible through the hole is deemed the dominant eye.[207] Participants were then seated in front of a custom apparatus designed to accurately measure TCSF and CFF.[158,167,208] They were instructed to look through a 3 mm artificial pupil designed to control the variation in the luminance that is caused by individual differences in pupil size.[158,167,208]

When measuring CFF a circular flickering stimulus, a one-degree, 660 nm target on a 5.5-degree 660 nm surround, was presented at 100% modulation, and participants were instructed to focus on the one-degree target in the center of the surround.[167] Using a method of ascending (increasing flicker frequency until the stimulus appears solid) and descending (decreasing the flicker frequency until a flicker is detected) trials, a final CFF threshold value was determined after six trials of this task were completed. Following these trials, the stimulus was changed to create the TCSF curve seen in previous literature.[158,208] Instead of adjusting the frequency, like CFF, the next trials were completed by adjusting the modulation while keeping set frequencies at low (2.5 Hz), optimal (10 Hz), and high (25 Hz) frequencies. These

frequencies were selected due to previous work showing the use of them being able to provide enough data in order to create the temporal contrast sensitivity function curve.[158] At each frequency the participant viewed a flickering stimulus through the same artificial pupil.[158,208] Just as the previous assessment, the participant tried to identify when the flicker ceases and the stimulus seems to have fused.[158,208] The test administrator used the same style of ascending and descending trials, but instead of increasing and decreasing frequency, the modulation was adjusted from 0-100%.[158,208] Six trials at each frequency were completed for a total of 24 trials total to measure the entire TCSF and CFF. Practice of this task was completed on a small scale by the main investigator with results similar to that of experts in the field when testing the same individual.

Macular Pigment Optical Density

To accurately measure MPOD and account for individual differences at different loci in the fovea, customized heterochromatic flicker photometry was used.[115,119,126,209] The use of customized heterochromatic flicker photometry requires the assessment of CFF before measuring MPOD. CFF must be obtained to reduce measurement error and facilitate good participant performance.[119,126,209] This customization helped to ensure the participant is correctly seeing and identifying what the assessment is designed for. Since the flicker stimulus presented by the macular densitometer differed slightly from that used in the full CFF assessment described below, a separate CFF value was calculated specifically to customize the MPOD measurement. This CFF value was not included in data analysis. It was used solely to tailor the flicker stimulus to each participant's threshold and ensure accurate MPOD assessment. The participants' CFF value was then input into the densitometer to customize the stimulus to their flicker threshold. Participants were seated in a chair and positioned themselves in front of the macular

densitometer so they could view the initial stimulus centrally in their vision using just their dominant eye.[207] They viewed the stimulus through a small hole while their chin rested on an adjustable chin rest. The non-dominant eye remained closed throughout the task. If the participant could not close their non-dominant eye for the duration of the task, an eye patch was provided.

When measuring MPOD the participant viewed a stimulus wavelength, centrally, at 30' (distance from the center of the fovea in arcminutes) eccentricity because that is where macular pigment is the densest.[119,126,209] The stimulus was the combination of two wavelengths, 460 and 570 nm light sources. These were presented in square-wave counter-phase orientation which gave the appearance of a uniform flicker.[119,126,209] Like the measurement of CFF, the participant perceived this flickering stimulus while the test administrator used a rotary knob to adjust the stimulus until the flicker ceased. Once the flicker ceased a red button linked to the densitometer was pressed to mark that moment.[119,126,209] This was done five times. After that was completed, similar procedures were performed in the participants' peripheral vision to capture a value where there is no macular pigment present to provide a baseline comparison. The peripheral (also known as parafoveal) location was at 7° (degrees away from the center of vision) eccentrically.[119,126,209] The participant remained looking through the same small hole, but were presented with a small red focal light in their peripheral vision. Without moving their head, the participant focused their eyes on this red dot for the duration of the trials, and was instructed to perform a similar task, which was to identify when the flickering of the stimulus ceases, but the stimulus was now in their peripheral vision.[119,126,209] This was also performed five times and the difference between locations allowed for a final MPOD calculation.[119,126,209] Practice of this task

has been completed on a small scale by the main investigator with results similar to that of experts in the field when testing the same individual.

Glare Discomfort

When measuring glare discomfort participants were seated in front of a custom apparatus with a video camera directly in front of their eyes. They rested their chin on an adjustable chin rest in a manner that allows for the video camera to view their left eye in optimal focus for data processing. Once the apparatus and camera were set up to record, the participant was told to look straight ahead at the wall in front of them in the absence of the photostressor and act as they normally would.[138,210] The test administrator then introduced the photostressor (a light stimuli produced by a 1000-Watt xenon arc source) for a period of five seconds.[138,210] This was done once. After the trial, participants were given a glare sensitivity questionnaire to identify how bothersome they believed the stimulus to be. After testing was completed, the video recording of the participants eye was opened in QuickTime by a different rater who was not aware of which group the participant was in.[138,210] Two frames were identified for analysis, and these were a frame when the participant's eye were open before the stimulus and a frame with the maximum level of squint in the presence of the stimulus.[138,210] The distance of the palpebral fissure between the top and bottom lids was measured in millimeters for a final glare discomfort value.[138,210]

Table 3.1 Visual psychophysics outcome variable descriptions

Outcomes	**Description**
Visual Psychophysics	
MPOD	The density of macular pigment (lutein and zeaxanthin) within the retina
TCSF	The sensitivity to contrast as a function of time (0.2, 0.8, 1.4 log Hz)
CFF	The highest average flicker frequency at which light appears to be flickering
Glare discomfort	The difference in distance of the palpebral fissure (in mm) after exposure to glare stimulus
Glare sensitivity	Subjective rating of how bothersome the glare stimulus was on a scale of 1-9

Glare Sensitivity Questionnaire

A glare sensitivity questionnaire was administered after the glare assessment.[210] It was designed to assess how sensitive and bothersome the participant feels the glare exposure during the trial was.[210] It is a Likert scale ranging from 1 to 9, with 1 indicating minimal to no sensitivity to the glare stimulus and 9 indicating severe sensitivity and bother.[210]

Straight Path Gait with Glare Stimulus

As soon as the glare discomfort measurement was completed, participants were fitted with mobile glare goggles and an adjustable belt which housed the battery pack for the goggles. These goggles were a portable version of previously published procedures assessing the effects of photostressors.[138,210] Before testing, participants were given practice trials for all gait tasks and practiced initiating and terminating gait in response to an audible buzzer, which served as the start cue for all tasks and the stop cue for the unplanned gait termination task. All gait trials were completed on a 6.1 x 0.61-m Zeno Walkway (Protokinetics, Havertown, PA). Straight path gait was always completed first, but the order of unplanned and planned gait termination was randomized.

In order to collect baseline straight path gait trials the participant began at a starting point on the walkway and was instructed to walk as they normally would throughout their everyday life while keeping their focus straight ahead of them through the end of the walkway. This was completed three times. For the glare condition, participants were told to get set at the same initial starting point and close their eyes. Once their eyes were closed the glare goggles were turned on by the test administrator. The participant was then instructed to open their eyes and begin walking once they heard the audible buzzer cue.

A member of the research team was there to spot the participant in case they began walking off the walkway to ensure safety. They will continue walking until through the end of the mat as they did with the baseline trials. The administrator then turned off the stimulus, giving a washout time, and instructed the participant to return to the starting point. This was be completed three times. Clinical spatiotemporal gait variables such as stride velocity (cm/s), stride width (cm), stride length (cm), and percent of time in single leg stance (%) were calculated by the ProtoKinetics Movement Analysis Software (PKMAS; Table 3.2).[58]

Table 3.2. Gait outcome variable descriptions

Outcomes	Description
Spatiotemporal	
Stride Velocity	Stride length divided by the stride times (cm/s).[42,43]
Stride Length	Distance from the heel of one foot to the following heel of the same foot (cm).
Stride Width	Distance between a line connecting two ipsilateral foot heel contacts (one stride) and the contralateral foot heel contact between those events and is measured perpendicular to the stride (cm).
Single Leg Support Percent	Single support time presented as a percentage of gait cycle time (percent).

Planned/Unplanned Gait Termination With and Without Glare

All planned and unplanned gait trials were completed on the same Zeno Walkway (Protokinetics, Havertown, PA). Participants were instructed to stand on the walkway and to get set and wait for the audible buzzer to initiate movement. They were instructed to walk at a normal self-selected pace. When performing planned termination trials, participants were instructed to terminate gait once they reached a marked location near the end of the walkway.[58,59] During unplanned termination trials, participants were instructed to begin the walk the same way as the planned trials. They then terminated their gait as soon as they heard a second audible buzzer that was manually triggered upon a right heel strike, and this buzzer occurred at any point in the trial after gait initiation.[58,59] To minimize anticipation, catch trials were randomly inserted

into the testing session, where no signal was used to cue gait termination and participants walked through the end of the walkway.[58,59] Participants were made aware catch trials would occur at least once. A total of three trials per condition (unplanned/planned termination with and without glare exposure) was performed for a total of 12 trials. The glare condition was performed in the same manner as the straight path gait. Gait termination time (GTT) was calculated, in seconds, as the time from the third to last step until the center of pressure velocity matches or falls below the average center of pressure velocity prior to gait initiation.[58] GTT was normalized to the participants' gait velocity by dividing GTT by gait velocity, resulting in a normalized GTT outcome (GTT/velocity = normalized GTT, s^2/m).[58] Gait velocity was calculated as the displacement in the anteroposterior plane between the footfall of the first step and the second to last termination step divided by time (m/s).[58] Clinical spatiotemporal gait variables such as stride velocity (cm/s), stride width (cm), stride length (cm), and percent of time in single stance (%) were calculated by the ProtoKinetics Movement Analysis Software (PKMAS; Table 3.2).[58]

Statistical Analysis

Demographic characteristics were compared between the concussion history and no concussion history group using an independent samples t-test, Mann-Whitney U or chi-square based on distribution.

Aim 1: To determine whether individuals with a history of concussion exhibit poorer visual psychophysical performance compared to individuals with no history.

Statistical Analysis: Visual psychophysical performance (MPOD, CFF, temporal contrast sensitivity function, glare discomfort, and glare sensitivity rating; Table 3.1) were compared between groups using an ANCOVA. Time since concussion was included as a covariate.

Aim 2: To investigate gait performance between individuals with and without a concussion history and examine whether a glare stimulus further influences gait outcomes such as stride velocity, stride length, stride width, single leg support percent, gait termination time, and the within trial variability on each outcome.

Statistical Analysis: For gait performance, a 2 (glare vs no glare) x 2 (concussion history vs no concussion history) mixed model analysis of variance was used. The dependent variables were GTT, stride velocity, stride length, stride width, single leg support percent (Table 3.2). Within trial variability was also compared using a 2 x 2 mixed model analysis of variance. If the omnibus test was significant, post hoc testing was completed using Tukey's HSD.

Power Analysis

We conducted a power analysis to determine an optimal sample size. Based on our alpha level of 0.05 and desired power of 80%, we calculated that 52 participants (26 in each group) would be necessary, and this was the desired sample size. This analysis was informed by a study comparing CFF values in younger and older adults, given the relevance of CFF to our project's focus on younger adults and its association with other visual assessments we determined this to be an adequate sample size.[167]

CHAPTER 4

A NOVEL VISUAL ASSESSMENT BATTERY IN INDIVIDUALS WITH A HISTORY OF CONCUSSION[1]

[1]Prato TA, Renzi-Hammond L, Hammond BR, Schmidt JD, Lynall RC. To be submitted to a peer reviewed journal

Abstract

Purpose: Visual dysfunction is one of the most common post-concussion symptoms and may persist even after other symptoms resolve. However, clinical assessments often focus on oculomotor function while overlooking perceptual and cortical visual processing. This study aimed to assess visual function using a novel battery of macular pigment optical density (MPOD), temporal contrast sensitivity function (TCSF), critical flicker fusion threshold (CFF), glare sensitivity and discomfort, and the between trial variability in individuals with a history of concussion compared to matched healthy controls.

Methods: A cross-sectional design was used to compare 16 participants with a concussion history to 15 matched controls. MPOD was assessed using customized heterochromatic flicker photometry. Temporal vision was measured using temporal contrast sensitivity function at differing frequencies and critical flicker fusion threshold. Glare sensitivity was assessed via a subjective discomfort rating and discomfort via an objective squint response to a light stimulus. Group comparisons were made using independent t-tests or Wilcoxon rank-sum tests.

Results: There were no statistically significant differences between groups in MPOD ($p = 0.62$, Partial $\eta^2 = 0.01$), CFF ($p = 0.40$, Partial $\eta^2 = 0.03$), TCSF at any frequency ($p = 0.50–0.84$ Partial $\eta^2 < 0.01$-0.02), glare sensitivity rating ($p = 0.64$, Partial $\eta^2 = 0.01$), or glare discomfort ($p = 0.83$, Partial $\eta^2 = 0.05$). Variability for MPOD ($p = 0.43$, Partial $\eta^2 = 0.02$), CFF ($p = 0.12$, Partial $\eta^2 = 0.08$), and all TCSF frequences ($p=0.30$-0.70, Partial $\eta^2 = 0.01$-0.04) was not significantly different between groups.

Conclusions: This is the first study to examine this visual battery in individuals with a concussion history. The lack of group differences across all measures may indicate full visual recovery in this asymptomatic sample or that persistent perceptual visual deficits are too subtle

for detection using these methods. Future work should include acutely concussed individuals and consider additional markers of visual and neural function.

Introduction

Concussions are a growing public health concern, with millions of cases reported annually and rising rates across all age groups and levels of sport.[1–10] Concussions can result in a variety of symptoms, including headaches, dizziness, cognitive impairments, and visual dysfunction.[16–19] Among these, visual symptoms are especially common, affecting 69-90% of individuals, and can present as blurred or double vision, light sensitivity, or impaired eye tracking.[17–19] Given that more than half of the brain is involved in visual processing, these disruptions are not surprising, but they remain poorly understood.

Current post-concussion vision assessments focus largely on oculomotor control, like saccades and smooth pursuits, through tools like the Vestibular Oculor Motor Screening and King-Devick tests.[20,23,24] While useful, these assessments focus on motor output rather than sensory processing and offer limited insight into perceptual and cortical-level visual dysfunction.[21,22] This is particularly concerning given that many individuals continue to experience visual deficits, including light sensitivity, photophobia and impaired visual processing, long after concussion symptoms have resolved.[55,56] These lingering visual impairments may also contribute to increased injury risk, particularly in dynamic environments requiring rapid visual-motor integration.

To address these limitations, the field of visual psychophysics offers promising methods to quantify perceptual responses to visual stimuli and assess neural integrity. For example, visual disturbances such as glare are frequently encountered in daily life, and light sensitivity is

commonly reported well after the resolution of other concussion symptoms.[55,56] Despite its prevalence, glare sensitivity is rarely measured objectively in clinical settings. Incorporating assessments of glare sensitivity into a broader visual battery may provide valuable insight into persistent visual dysfunction and everyday challenges experienced by those following a concussion. The brain can compensate for injury allowing individuals to maintain normal function on tasks, although this compensation may come with reduced efficiency or increased cognitive or sensory load.[25,26] Therefore, assessing not only visual performance but also the magnitude of variability across tasks may provide a more detailed profile of visual system function and recovery status following concussion. This variability may reflect increased effort or instability in the system, even when overall performance appears normal.[212]

Temporal vision, an underexplored component of post-concussion visual function, refers to the ability to detect changes in visual contrast over time which is an important function for detecting motion and visual changes in a dynamic environment.[158] It is typically evaluated using the temporal contrast sensitivity function (TCSF). Additionally, critical flicker fusion threshold (CFF) does not fully characterize temporal vision, but it has been used as an index of temporal vision.[28,159] CFF is the highest flicker frequency that someone can detect at 100% modulation.[159] Both TCSF and CFF provide valuable information into the functioning of the visual system, but also cognitive processing and neural integrity.[158,159] These measures may reveal post-concussion deficits not detected by standard clinical tools. While TCSF and CFF have been minimally studied in concussion populations, evidence from aging and neurodegenerative research suggests it may be a valuable indicator of visual and cognitive performance such that lower TCSF and CFF values have been associated with worse visual and cognitive performance.[158,159,167,173,174]

Macular pigment, measured through macular pigment optical density (MPOD), has emerged as a potential noninvasive biomarker of retinal and neural health.[127,128] Composed of dietary carotenoids lutein and zeaxanthin, macular pigment plays an important protective role by filtering blue light and reducing oxidative stress.[38,115] These carotenoids are also found in the brain, and are highly correlated with the concentration found in the retina, where they offer antioxidant and anti-inflammatory benefits.[127,128] Studies in older adults and individuals with Alzheimer's disease have shown that a lower MPOD is associated with slower reaction times, poorer cognitive performance, and visual deficits.[40,125,126,129] Despite some overlap between Alzheimer's symptoms and concussion symptoms, no studies to date have investigated MPOD in individuals with a history of concussion, representing a critical gap in our understanding of post-injury neural and visual integrity.

Therefore, the purpose of this study was to assess MPOD, TCSF, CFF, glare discomfort and sensitivity, and the between trial variability on all tasks in individuals with a concussion history compared to matched healthy controls. We hypothesized that individuals with a concussion history would demonstrate lower MPOD levels, worse performance on TCSF and CFF, have higher sensitivity and discomfort to glare compared to controls and have greater variability in visual outcomes.

Methods

Participants

This cross-sectional study recruited a convenience sample of participants aged 18 years or older. Individuals were excluded if they reported any of the following within one year of testing: a significant lower extremity injury (e.g., fracture or surgery), visual impairment (worse

than 20/50 even with correction, as assessed by the test administrator), neurological disorder, or ocular disease.

Participants were assigned to either a concussion history group or a control group. To qualify for the concussion history group, individuals had to have self-reported sustaining a concussion within two years of study enrollment and be asymptomatic at the time of testing, defined as a total symptom severity score ≤ 13 on the SCAT6 symptom inventory.[202] Participants in the control group had to have no self-reported history of concussion and were matched to an individual in the concussion group based on age, sex, and height. Prior to the beginning of the study, institutional review board approval was obtained, and all participants provided signed informed consent.

Macular Pigment Optical Density

To accurately measure MPOD and account for individual differences at different loci in the fovea, customized heterochromatic flicker photometry was used and has been previously described in detail.[115,119,126,209] Previous evidence shows that this technique is highly reliable when measuring macular pigment.[119] The use of customized heterochromatic flicker photometry requires the assessment of critical flicker fusion threshold (CFF) before measuring MPOD. CFF must be obtained to reduce measurement error and facilitate good participant performance.[119,126,209] This customization helped to ensure the participant was correctly seeing and identifying what the assessment was designed for. Since the flicker stimulus presented by the macular densitometer differs slightly from that used in the full CFF assessment described below, a separate CFF value was calculated specifically to customize the MPOD measurement. This CFF value was not included in data analysis. It was used solely to tailor the flicker stimulus to each participant's threshold and ensure accurate MPOD assessment.

Participants were seated in a chair and positioned themselves in front of the macular densitometer so they could view the initial stimulus centrally in their vision using just their dominant eye.[207] Dominant eye was assessed by instructing the participant to focus with both eyes on an object smaller in size (e.g., doorknob, letter on a Snellen eye chart).[207] The participant then brought their hands together in front of them to form a triangle-shaped hole with the focal point in the center of that hole.[207] They were then instructed to close one eye at a time and verbally indicate when the target remains visible through their hands and when it does not remain in focus.[207] The eye that was open when the target was still visible through the hole is deemed the dominant eye.[207] They viewed the stimulus through a small hole while their chin rested on an adjustable chin rest. The non-dominant eye remained closed throughout the task. If the participant could not close their non-dominant eye for the duration of the task, an eye patch was provided.

The participant viewed a stimulus wavelength, centrally, at 30' (distance from the center of the fovea in arcminutes) eccentricity because that is where macular pigment is the densest.[119,126,209] The stimulus was the combination of two wavelengths, 460 and 570 nm light sources. These were presented in square-wave counter-phase orientation which gave the appearance of a uniform flicker.[119,126,209] Like with the measurement of CFF, the participant perceived this flickering stimulus while the test administrator used a rotary knob to adjust the stimulus until the flicker ceased. Once the participant said the flicker ceased, a red button linked to the densitometer was pressed to mark that moment.[119,126,209] This was done five times.

After this was completed, similar procedures were performed in the participants' peripheral vision to capture a value where there is no macular pigment present to provide a baseline comparison. The peripheral (also known as parafoveal) location was set at 7^{o} (degrees

away from the center of vision) eccentrically.[119,126,209] The participant continued looking through the same small hole, but they were presented with a small red focal light in their peripheral vision. Without moving their head, the participant adjusted their focus to this red dot for the duration of the trials, and were instructed to perform a similar task, which is to identify when the flickering of the stimulus ceased, but the stimulus was now in their peripheral vision.[119,126,209] This was also performed five times and the difference in values between locations allowed for a final MPOD calculation.[119,126,209]

Temporal Vision

The methods to measure TCSF and CFF have been previously described.[158,167,208] Although CFF is a component of the temporal contrast sensitivity function, slight modifications to the test stimulus were necessary to accurately measure CFF.[167] Participants were measured using their dominant eye.[207] Both TCSF and CFF have proven to be valid and highly reliable.[158,173,213]

Participants were then seated in front of a custom apparatus designed to accurately measure TCSF and CFF.[158,167,208] They were instructed to look through a 3 mm artificial pupil designed to control the variation in the luminance that is caused by individual differences in pupil size.[158,167,208] When measuring CFF a circular flickering stimulus, a one-degree, 660 nm target on a 5.5-degree 660 nm surround, was presented at 100% modulation, and participants were instructed to focus on the one-degree target in the center of the surround.[167] Using a method of ascending (increasing flicker frequency until the stimulus appears solid) and descending (decreasing the flicker frequency until a flicker is detected) trials, a final CFF threshold value was determined after six trials of this task were completed and averaged.

When measuring the entire TCSF the stimulus was changed to create the TCSF curve seen in previous literature.[158,208] Instead of adjusting the frequency, like CFF, the modulation was adjusted while keeping set frequencies at low (2.5 Hz), optimal (10 Hz), and high (25 Hz) frequencies. These frequencies were selected due to previous work showing the use of these being able to provide enough data in order to create the TCSF.[158] At each frequency the participant viewed a flickering stimulus through the same artificial pupil.[158,208] Just as with CFF, the test administrator used the same style of ascending and descending trials, but instead of increasing and decreasing frequency, the modulation was adjusted from 0-100%.[158,208] Six trials at each frequency were completed for a total of 24 trials total to measure the entire TCSF function and CFF.

Glare

When measuring glare discomfort participants were seated in front of a custom apparatus with a video camera directly in front of their eyes. They rested their chin on an adjustable chin rest in a manner that allowed for the video camera to view their left eye in optimal focus for data processing. Once the camera was set up to record, the participant was told to look straight ahead at the wall in front of them in the absence of the photostressor and act as they normally would.[138,210] The test administrator then introduced the photostressor (a light stimuli produced by a 1000-Watt xenon arc source) for a period of five seconds.[138,210] This was done once. After the trial, participants were given a one-item questionnaire to rate how bothersome the light was to them. The question was a Likert scale ranging from 1 to 9, with 1 indicating severe sensitivity and bother to the glare stimulus and 9 indicating little to no sensitivity and bother.[210] This value was used as the participants final glare sensitivity value.

After testing was complete, the video recording of the participants eyes was opened in QuickTime to capture the glare discomfort value..[138,210] All video analysis was completed by the same rater, who was different than the test administrator, who was blinded to group. Two frames were identified for analysis; 1) a still frame of when the participant's eye was open normally before the stimulus was triggered, and 2) a frame with the maximum level of squint in the presence of the stimulus (Figure 4.1).[138,210] The difference in distance of the palpebral fissure between the top and bottom lids was measured in millimeters for a final glare discomfort value.[138,210]

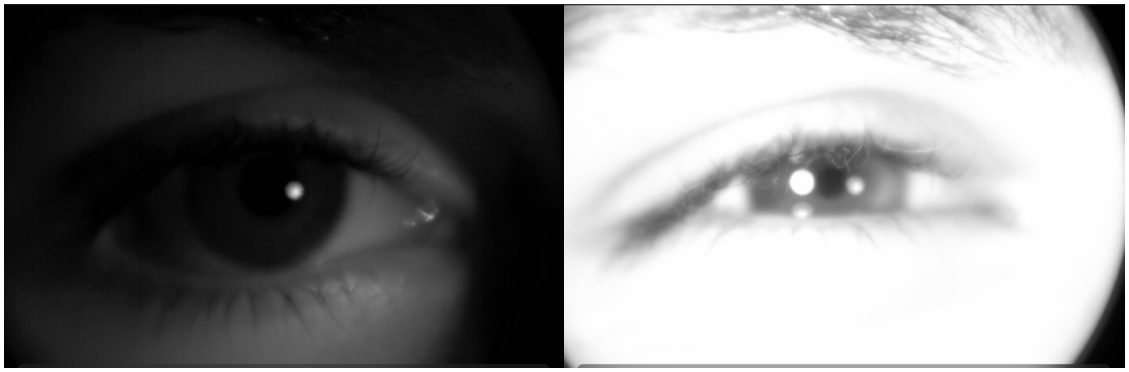

Figure 4.1: Example of squint response captured for glare discomfort analysis

Statistical Analysis

The outcomes of interest were MPOD, CFF threshold (Hz), TCSF modulation values (average modulation at 2.5, 10, and 25 Hz), glare discomfort (mm), glare sensitivity rating (Likert rating), and between trial variability (the standard deviation around the mean of each task outcome). Between trial variability was only calculated for MPOD, CFF, and TCSF outcomes since the glare outcomes only resulted from a single trial. These were compared between groups using separate ANCOVA's with effect size calculations. Days since most recent concussion was used as a covariate. The number of days post-concussion for each concussion group participant

was subtracted from the mean (252.5 days), and control participants were assigned a value of zero.[214] This created a mean centered days since concussion value which was then used for analysis.[214] For all outcomes, normality and homogeneity of variance were assessed using the Shapiro-Wilk and Levene's tests, respectively. Glare discomfort data violated the assumption of normality, so a Wilcoxon rank-sum test was used for that comparison. An alpha level of 0.05 was set a priori for statistical significance. All statistical analyses were conducted using R version 4.4.2.

Table 4.1. Group means, mean differences, and statistical findings from vision assessments.

Outcome	Control Mean (SD)	Concussion Mean (SD)	Mean Difference (95% CI)	p-value	Partial η^2
TCSF (% modulation)					
2.5 Hz	8.52 (3.79)	8.26 (3.04)	-0.26 (-2.77, 2.26)	0.84	<0.01
10 Hz	11.22 (3.86)	10.29 (3.70)	-0.93 (-3.71, 1.85)	0.50	0.02
25 Hz	79.48 (7.33)	77.31 (15.40)	-2.17 (-11.13, 6.79)	0.63	0.01
CFF	22.74 (2.67)	23.60 (2.80)	0.86 (-1.15, 2.87)	0.40	0.03
MPOD	0.61 (0.05)	0.63 (0.06)	0.02 (-0.06, 0.10)	0.62	0.01
Glare sensitivity (Likert)	4.94 (1.61)	5.38 (2.03)	0.31 (-1.03, 1.65)	0.64	0.01
Glare discomfort (mm)*	2.4* (2.71-4.38)	3.19 * (2.03-3.63)	0.77* (-1.12, 1.23)	0.83*	0.05*

Abbreviations: TCSF, temporal contrast sensitivity function; CFF, critical flicker fusion threshold; MPOD, macular pigment optical density. *denotes that the glare discomfort statistics are different. Instead of mean value (SD) it is median value (IQR). Mean difference was replaced with median difference with 95% CI. Due to the use of Wilcoxon rank-sum test r was used as effect size instead of partial eta squared.

Results

A total of 31 participants were enrolled and completed all testing procedures. The control group consisted of 15 participants (age = 20.3 ± 1.23, height = 171.92 ± 10.05 cm, sex = 67% female) and the concussion history group consisted of 16 participants (age = 21.1 ± 2.9, height = 172.83 ± 11.34 cm, sex = 67% female, average time since injury = 252.5 ± 209.12 days, median time since injury = 235 days, IQR=84.25-368 days). There were no statistically significant differences between groups for MPOD, all TCSF outcomes, CFF, and both glare outcomes (Table 4.1). The variability for MPOD ($p = 0.43$, Partial $\eta^2 = 0.02$), CFF ($p = 0.12$, Partial $\eta^2 = 0.08$), and all TCSF frequences ($p=0.30$-0.70, Partial $\eta^2 < 0.01$-0.04) were not significantly different between groups (Table 4.2).

Table 4.2. Variability group means and statistical findings from vision assessments.

Outcome	Control Mean (SD)	Concussion Mean (SD)	p-value	Partial η^2
TCSF (% modulation)				
2.5 Hz	1.47 (0.90)	1.35 (0.64)	0.67	0.01
10 Hz	1.43 (0.72)	1.21 (0.43)	0.30	0.04
25 Hz	3.93 (1.01)	3.77 (1.26)	0.70	0.01
CFF	0.76 (0.24)	0.92 (0.29)	0.12	0.08
MPOD	0.05 (0.01)	0.06 (0.03)	0.43	0.02

Abbreviations: TCSF, temporal contrast sensitivity function; CFF, critical flicker fusion threshold; MPOD, macular pigment optical density.

Discussion

This study is the first of its kind to utilize novel psychophysics measures of visual function in individuals with a concussion history compared to matched healthy controls. By examining visual performance using tools known to reflect cortical and retinal function, this study directly addresses a gap in the literature regarding how post-concussion changes in the visual system can be monitored. Despite previous literature suggesting potential long-term visual

impairments following concussion, no statistically significant differences were observed between groups across any of the measured outcomes. The assessments used were selected for their sensitivity to subtle visual and neurological changes, making them strong candidates to uncover lingering effects of concussion that might not be detected through standard clinical assessments. However, with an average time since their concussion of 252.5 days it is reasonable to suggest that participants in the concussion history group may have experienced full visual recovery by the time of testing.

No significant differences were found between groups in the variability of visual performance across tasks. The absence of group differences in variability suggests that any underlying neural adaptations or compensations had either resolved or were not severe enough to disrupt performance at the time of testing. Another potential consideration is the possibility that certain visual or neurological deficits following concussion may not emerge until long after the initial injury, potentially even beyond the average time since concussion of 252.5 days. While our findings suggest that visual function appeared normal in the concussion history group, it is possible that some visual or cognitive functions could decline gradually over many months or even years due to subtle, progressive changes in the brain or visual pathways. This reasoning aligns with research indicating that some consequences of traumatic brain injury may have delayed onset or evolve over time, especially in individuals who had experienced multiple concussions.[215–217] If this were the cause of our null findings, it may have been possible that deficits simply had not yet manifested or progressed to a degree detectable by the current methods and time frame of this study.

Although MPOD has been linked to visual performance, cognitive function, and neurodegenerative disease, our findings did not reveal differences between groups.[40,125,126,129]

This null result may indicate that concussion does not substantially affect MPOD or that any effects are too subtle to detect in asymptomatic individuals. Given that macular pigment is solely gained through dietary intake, it is possible that concussion or other forms of traumatic brain injury cannot meaningfully alter MPOD levels since they do not influence one's diet before their injury. However, without including individuals in the acute phase of concussion, it remains unclear whether any transient changes in MPOD might occur shortly after injury.

Another important consideration is that the participants in this study appeared to exhibit unusually high MPOD values. Previous studies using the same methodology as ours have reported considerably lower average MPOD values, and other regions across the United States have reported averages of 0.22 and 0.35, compared to our sample averages of 0.61 and 0.63.[218–222] These studies included a wide age range, from young adults to elderly populations (mean age = 41.5 years, range = 17-92 years), which may limit their generalizability to our sample. In younger adults similar in age to our participants (mean age = 21.3 years), average MPOD levels were reported around 0.41, which is still considerably lower than the average observed in our sample.[223,224] This suggests that our sample may have been exceptionally high performing in this regard. Since higher MPOD has been associated with enhanced visual and cognitive function it is plausible that the elevated levels in both groups could have masked any visual deficits associated with concussion history.[125,126]

Similarly, no differences were found across the three TCSF frequencies tested. TCSF is sensitive to aging and neurodegenerative conditions, but its application in post-concussion assessment had not previously been explored in the manner we tested.[167,168,171] Our results suggest that TCSF may have been fully recovered by the time of testing. It is also possible this assessment does not capture lingering visual deficits in individuals who are asymptomatic,

although acute effects and the time course of recovery remain unknown. The small effect sizes from these comparisons and modest sample size limit the strength of these conclusions and underscore the need for further research.

CFF which has shown to be a marker of cortical processing speed and neural integrity also showed no significant group differences.[167,168,171] However, the small effect size (partial η^2 = 0.03) may point to a potentially meaningful difference not detectable with the current sample size. As with TCSF, it is unclear how long any post-concussion deficits in CFF may last or whether they are only present during the acute or symptomatic phases.

Both subjective (glare sensitivity) and objective (glare discomfort) measures in response to glare revealed no differences between groups. Glare sensitivity and photophobia are commonly reported post-concussion symptoms, and this study aimed to quantify both the perception and behavioral response to glare.[158,159,173,174] The negligible group differences suggest that any glare-related impairments may resolve by the time individuals are asymptomatic. This contradicts previous research indicating lingering glare sensitivity. A prior literature review on post-concussion light sensitivity suggested that symptoms can persist for up to six months to years' post-injury.[55,56] One potential reason we did not observe group differences may be related to methodological differences between studies. For example, a previous study that identified increased light sensitivity six months post-concussion utilized five different photostressors at varying intensities to participant, with eight separate trials per intensity.[56] In contrast, our study used a single glare trial at one fixed intensity which may not have been sufficient to detect lingering differences between groups in asymptomatic individuals. Another study that reported chronic light sensitivity used a symptom questionnaire which asked participants to rate their overall sensitivity to light at multiple time points without presenting an actual photostressor.[225]

That approach focused strictly on subjective symptom tracking over time rather than assessing reactions to glare exposure. While our study also included a subjective glare sensitivity rating, our participants were asymptomatic at the time of testing, which may have limited the potential of detecting differences. Given that the average time since injury in our sample was approximately eight months, it is plausible that recovery from glare sensitivity may have occurred between the six-month mark and the time of testing. It is also worth noting that although no statistically significant differences were observed between groups, the concussion history group demonstrated an increased squint response of 0.79 mm, which is approximately a 33% increase compared to controls. This difference resulted in a small effect size (Partial η^2 = 0.05) which may indicate that a true effect exists but was not detectable with the current sample size. Prior research that reported significant differences in squint response following concussion observed a mean difference of 1.87 mm.[226] While the difference found in our study is smaller, a 33% increase in squint response may suggest a trend that could potentially reach statistical significance in a larger sample.

Several limitations must be acknowledged. The modest sample size limits statistical power and the ability to interpret our findings. Additionally, all participants were asymptomatic and tested an average of 252.5 days post-injury, which may mean that any visual dysfunctions had resolved. Without baseline or acute-phase data, the trajectory of visual recovery cannot be determined. Given the influence of diet in relation to MPOD, not tracking participants dietary habits is another limitation.

Future research should incorporate longitudinal designs beginning in the acute post-injury phase to better understand how these visual metrics may change over time. Including participants with prolonged recovery or persistent symptoms may also help identify whether these tools can

capture meaningful impairments in more affected individuals. Additionally, investigating whether dietary supplementation of macular pigment impacts recovery or visual outcomes following concussion warrants further exploration.

In conclusion, although no significant differences were found in MPOD, TCSF, CFF, or glare sensitivity between individuals with and without a history of concussion, this study contributes novel insights. As the first to apply these visual assessments to a concussed population, it lays the groundwork for future research to determine whether these measures are useful in tracking recovery, informing return-to-activity decisions, or identifying persistent visual dysfunction after concussion.

CHAPTER 5

THE IMPACT OF GLARE ON GAIT PERFORMANCE IN INDIVIDUALS WITH A HISTORY OF CONCUSSION

[1]Prato TA, Renzi-Hammond L, Hammond BR, Schmidt JD, Lynall RC. To be submitted to *Gait & Posture*

Abstract

Background: Concussions are a rising public health concern and often lead to oculomotor dysfunction and gait deficits, even after symptoms resolve. Visual disturbances, such as glare, are common in daily life but it is not clear how glare impacts movement post-concussion. Understanding how visual stimuli influence gait may help explain the increased injury risk following concussion.

Research Question: Does a history of concussion impact gait on various tasks, and does glare alter gait performance?

Methods: Thirty-one participants (16 with concussion history, 15 controls) completed three gait tasks, straight path gait, planned gait termination, and unplanned gait termination, with and without glare. Outcomes included stride velocity, length, and width, single leg support percentage, within- and between-trial variability for all gait tasks, and normalized gait termination time during planned and unplanned gait termination tasks. Separate two (glare vs. no glare) x two (concussion history vs. control) ANOVAs were conducted for each outcome.

Results: No significant interaction or main effect of concussion history was observed for any outcome. A significant main effect of glare was found for stride velocity and stride length across all gait tasks ($p<0.05$), with slower and shorter strides during glare conditions. Stride width and single leg support were unaffected. Glare also significantly increased GTT during unplanned gait termination ($p=0.044$) but not during planned termination. For the straight path task, there were no significant group or interaction effects for variability ($p = 0.295–0.684$, $d = 0.12–0.23$), but glare significantly increased variability across all spatiotemporal outcomes ($p < 0.05$, $d = 0.37–0.76$). For the planned gait termination task, there were no significant group or interaction effects for variability in stride velocity, length, single leg support, or gait termination time ($p = 0.069–$

0.841, d = 0.05–0.57). Glare significantly increased variability for stride velocity and length ($p < 0.05$, d = 0.37–0.79) but not single leg support or gait termination time ($p > 0.05$, d = 0.04-0.25). Both group ($p = 0.020$, d = 0.72) and glare effects ($p = 0.049$, d = 0.37) were observed for stride width variability. For the unplanned gait termination task, no significant group, interaction, or glare effects were observed for variability in stride velocity, stride length, single leg support, or gait termination time ($p = 0.077–0.902$, d = 0.02–0.34), but glare significantly increased stride width variability ($p < 0.001$, d = 0.71).

Significance: Glare alters gait regardless of concussion history, suggesting that visual disturbances can disrupt motor control. These findings emphasize the role of visual input in motor control and suggest that real-world visual challenges may be useful in identifying subtle deficits not captured by standard assessments.

Introduction

Concussions are a significant public health concern, with millions of cases occurring each year and rising rates across all age groups and levels of sport.[1–10] Despite advances in education and identification, many concussions go unreported suggesting that the true incidence is likely still underestimated.[1–10] Symptom presentation varies widely in severity and duration but commonly includes headaches, dizziness, cognitive impairments, functional impairments, and visual dysfunction.[7,12–15]

Visual dysfunction is highly prevalent post-concussion with up to 90% of individuals reporting oculomotor impairments.[18,19,135] Given that over half the brain is involved in visual processing, these disturbances are not surprising. However, common assessments such as the King-Devick test and Vestibular Ocular Motor Screening focus primarily on evaluating

oculomotor control and often overlook broader aspects of visual function, such as sensory integration, visual perception, and response to stimuli, all of which are critical for daily functioning and athletic performance.[17–19] Glare is a frequently encountered visual stimulus experienced in everyday life, and individuals with a concussion history often report increased sensitivity to light, or photophobia. This heightened sensitivity can persist well after individuals are deemed asymptomatic, impacting vision and potentially influencing motor control after a concussion.[20–22,24]

Also identified acutely post-concussion are altered gait characteristics, including slower walking speed, shorter stride length, and impaired postural control which can persist even after clinical recovery.[48–50] Notably, individuals with a concussion history are at an increased risk for lower extremity musculoskeletal (LEMSK) injury, with rates up to 2.5 times higher in the year following injury.[48–50] These injuries are believed to stem, at least in part, from lingering neurophysiological disruptions that affect motor coordination and balance, but specific mechanisms remain unclear. Given that specific gait patterns have been linked to injury risk in other populations, there is the potential that uncovering lingering motor control deficits during gait may shed light on the heightened LEMSK injury risk following concussion.[45,46]

Although post-concussion gait impairments may sometimes appear subtle and vary depending on the task, they often reflect meaningful disruptions in sensorimotor integration and attentional control which is essential for safe movement.[48–50] Although individuals may appear to perform normally on functional tasks after concussion, they may rely on compensatory strategies that increase cognitive or sensory demands.[48–50] As task difficulty increases, these compensations may lead to mpaired performance. Assessing not only gait performance but also variability across trials may offer a more sensitive measure of lingering deficits or increased effort, even

when outward performance appears unaffected. Traditional straight-path gait can uncover some of these deficits but more complex gait tasks, such as gait termination, can challenge higher-level motor planning and adaptability.[25,26] In particular, tasks that involve unplanned components, like unplanned gait termination, may be especially sensitive for detecting lingering functional impairments that may not otherwise be captured.[50,58,59]

Impaired visual function may disrupt gait and motor control, potentially increasing the risk of injury in unpredictable, real-world conditions. Although vision and gait are known to be linked, few studies have explored how visual disturbances, such as glare, impact gait performance.[58,59] This is a critical gap, as real-world environments are visually dynamic and often require individuals to make rapid adjustments in response to complex visual cues.

Investigating how visual challenges, such as glare, affect gait may provide a critical missing link between concussion-related visual dysfunction and the elevated risk of LEMSK injury. Therefore, the purpose of this study was to examine whether individuals with a concussion history demonstrate differences in gait performance on various gait tasks compared to controls, and to determine whether the presence of glare alters gait outcomes in both groups. A secondary aim was to explore whether individuals with a concussion history exhibited greater variability in gait performance, and if variability increased with a glare stimulus. We hypothesized that participants with a concussion history would demonstrate slower gait termination times and altered spatiotemporal gait measures relative to controls. Additionally, we hypothesized that glare conditions would result in slower and more conservative gait across all participants, with greater disruption in the concussion group. Finally, we hypothesized that individuals with a concussion history would exhibit greater variability in gait performance across all tasks, and glare would increase the variability in gait performance within trials.

Methods

Participants

This cross-sectional study recruited a convenience sample of participants aged 18 years or older. Individuals were excluded if they reported any of the following within one year of testing: a significant lower extremity injury (e.g., fracture or surgery), visual impairment (worse than 20/50 even with correction, as assessed by the test administrator), neurological disorder, or ocular disease.

Participants were assigned to either a concussion history group or a control group. To qualify for the concussion history group, individuals must have self-reported sustaining a concussion within two years of study enrollment and be asymptomatic at the time of testing, defined as a total symptom severity score ≤ 13 on the SCAT6 symptom inventory.[53,54] Participants in the control group must have no self-reported history of concussion and were matched to an individual in the concussion group based on age, sex, and height. Prior to the beginning of the study, institutional review board approval was obtained, and all participants provided signed informed consent.

Figure 5.1. Example of goggles with and without glare stimulus

Study Protocol

Participants completed three gait tasks: straight-path walking, planned gait termination, and unplanned gait termination. Each task was performed under two conditions: with glare and without glare, resulting in six total conditions. Three trials per condition were completed, totaling 18 trials per participant, with the no glare conditions always coming before the glare trials for each task. All gait tasks were conducted on a 6.1 m × 0.61 m Zeno Walkway (Protokinetics, Havertown, PA).

Prior to testing, each participant was fitted with mobile, custom made glare goggles (Figure 5.1) and a belt that held the battery pack. The goggles, designed without lenses, consisted of a circular ring of bright white LEDs housed in an adjustable goggle frame. For the no-glare conditions, the goggles still remained on the participant but were not connected to the battery pack. Participants were given practice trials for each gait task and practiced initiating and terminating gait in response to an audible buzzer, which served as the start cue for all tasks and the stop cue for the unplanned gait termination task.

Figure 5.2. Image of the mobile glare goggles during a glare trial.

For the straight path gait, the participant began at a set starting point on the walkway and was instructed to walk as they normally would throughout their everyday life while keeping their focus straight ahead of them through the end of the walkway. The first three trials were completed without glare before continuing to the glare condition (Figure 5.2). For the glare condition, participants were told to get set at the same initial starting point and close their eyes. Once their eyes were closed the glare goggles were turned on by the

test administrator. The participant was then instructed to open their eyes and begin walking once they heard the audible buzzer cue.

For the planned and unplanned gait termination tasks the participants began on the same starting point as the straight path gait, and the audible cue to begin walking remained the same. When performing planned termination trials, participants were instructed to terminate gait and come to a rest once they reach a marked location near the end of the walkway.[202] During unplanned termination trials, participants began the walk the same way as the planned trials. However, for this task they terminated their gait as soon as they heard the buzzer again. The buzzer was manually triggered upon a right heel strike, at a random point during the walk after gait initiation.[58,59] To minimize anticipation, catch trials were randomly inserted into the testing session, where no signal was used to cue gait termination and participants walked off the end of the walkway.[58,59] The glare conditions for these tasks were performed in the same manner as the straight path gait.

Data Processing

Clinical spatiotemporal gait variables such as stride velocity (cm/s), stride width (cm), stride length (cm), and percent of time in single leg stance (%) are calculated by the ProtoKinetics Movement Analysis Software (PKMAS).[58,59] The PKMAS software also calculates the within trial standard deviation for stride velocity, length, and width, which was then averaged across all trials within each task to generate a variability value for each gait variable. For the gait termination tasks, gait termination time (GTT) was calculated, in seconds, as the time from the third to last step until the center of pressure velocity matched or fell below the average center of pressure velocity prior to gait initiation.[58] GTT was then normalized to the participants' gait velocity by dividing GTT by gait velocity, resulting in a normalized GTT

outcome (GTT/velocity = normalized GTT, s^2/m).[58] Only normalized GTT was used for data analysis. Variability for GTT and single leg support percent was calculated between trials and was the standard deviation around the mean GTT.

Data Analysis

For each gait task, separate 2 (glare vs. no glare) x 2 (concussion history vs. no concussion history) mixed model analyses of variance were conducted. The gait outcomes of interest were normalized GTT, stride velocity, stride length, stride width, single leg support percent, and within trial variability for spatiotemporal variables and between trial variability for GTT. If a significant interaction was observed, post hoc testing was completed using Tukey's HSD. Comparisons of performance between each gait task were not completed because differences between tasks was not a focus of this study as they were already identified in previous literature.[58] An alpha level of 0.05 was set a priori for statistical significance. All statistical analyses were completed using R version 4.4.2.

Results

A total of 31 participants were enrolled and completed all testing procedures. The control group consisted of 15 participants (age = 20.3 ± 1.23, height = 171.92 ± 10.05 cm, sex = 67% female) and the concussion history group consisted of 16 participants (age = 21.1 ± 2.9, height = 172.83 ± 11.34 cm, sex = 67% female, time since concussion = 252.5 ± 209.12 days). There were no significant differences between groups on age, height, and sex ($p>0.05$). Output values and findings for each outcome variable across conditions are presented for straight path gait in Table 5.1, planned gait termination in Table 5.3, and unplanned gait termination in Table 5.5. Variability outcomes are presented in Table 5.2 for straight path gait, Table 5.4 for planned gait

termination, and Table 5.6 for unplanned gait termination. No significant interaction effects were observed between glare condition and concussion history group for any of the outcomes ($p>0.05$). No significant main effects of concussion history were detected for any of the outcome measures ($p>0.05$). A significant main effect of glare condition was found for stride velocity and stride length across all gait tasks, with participants exhibiting slower stride velocities and shorter stride lengths in the glare condition compared to the no-glare condition ($p<0.05$). For GTT, during the planned gait termination task there was no significant group main effect ($p=0.83$) or condition main effect ($p=0.94$). There was a significant glare condition main effect ($p=0.04$) for the unplanned GTT task such that glare increased GTT, but no group main effect ($p=0.32$).

For the straight path gait task, there were no significant interaction effects for variability ($p = 0.295–0.402$) or group main effects ($p = 0.407–0.684$, Cohen's $d = 0.12–0.23$). However, there were significant main effects of glare condition across all spatiotemporal variability measures ($p < 0.046$, Cohen's $d = 0.37–0.76$). For the planned gait termination task, there were no significant interaction effects for variability on any spatiotemporal variable or GTT ($p = 0.154–0.738$). For stride velocity and stride length variability, there were no significant group main effects ($p = 0.069–0.566$, Cohen's $d = 0.18–0.57$), but there were significant main effects of glare condition ($p < 0.012$, Cohen's $d = 0.48–0.57$). For stride width variability, there were significant group effects ($p = 0.020$, Cohen's $d = 0.72$) and glare condition effects ($p = 0.049$, Cohen's $d = 0.37$) such that the concussion history group and glare conditions resulted in significantly greater strider width variability. For single leg support and GTT variability, there were no significant group ($p = 0.147$-0.841, Cohen's $d = 0.05$-0.44) or glare condition ($p = 0.185$-0.804, Cohen's $d = 0.04$-0.25) main effects. For the unplanned gait termination task, there were no significant interaction effects for variability across all spatiotemporal variables and GTT

(p = 0.324–0.596). Additionally, there were no significant group main effects (p = 0.356–0.902, Cohen's d = 0.07–0.28) or glare condition main effects (p = 0.077–0.787, Cohen's d = 0.02–0.34) for stride velocity, stride length, single leg support, and GTT variability. However, for stride width variability, there were no significant group effects (p = 0.356, Cohen's d = 0.29), but a significant main effect of glare condition was observed such that variability was significantly increased for the glare conditions (p < 0.001, Cohen's d = 0.71).

Table 5.1. Straight path gait means and statistical findings

Outcome	Group	Condition		Group Mean Difference (95% CI)	Glare Mean Difference (95% CI)	Interaction (p)	Group Main Effect [p (Cohen's d)]	Condition Main Effect [p (Cohen's D)]
		No Glare (Mean ± SD)	Glare (Mean ± SD)					
Stride Velocity (cm/s)	Control	117.59 (15.28)	113.24 (19.03)	-6.04 (-13.59, 1.50)	-6.71 (-10.67, -2.76)	0.212	0.223 (-0.40)	0.002 (-0.61)*
	Concussion	113.77 (9.2)	104.97 (14.98)					
Stride Length (cm)	Control	134.78 (12.95)	130.97 (14.16)	-4.87 (-11.46, 1.74)	-5.71 (-8.32, -3.09)	0.125	0.252 (-0.37)	<0.001 (-0.79)*
	Concussion	131.71 (11.61)	124.32 (13.43)					
Stride Width (cm)	Control	11.37 (3.98)	11.44 (4.78)	-1.06 (-2.85, 0.73)	0.57 (-0.30, 1.43)	0.137	0.365 (-0.30)	0.099 (0.24)
	Concussion	9.84 (2.82)	10.85 (2.67)					
Single Leg Support (%)	Control	37.54 (6.13)	36.04 (1.55)	-1.03 (-2.63, 0.56)	-0.96 (-2.51, 0.56)	0.553	0.251 (-0.33)	0.203 (-0.23)
	Concussion	36.01 (1.01)	35.50 (1.36)					

*denotes statistical significance (p<0.05)

Table 5.2. Straight path gait mean variability and statistical findings

Outcome	Group	Condition		Interaction (p)	Group Main Effect [p (Cohen's d)]	Condition Main Effect [p (Cohen's D)]
		No Glare (SD)	Glare (SD)			
Stride Velocity (cm/s)	Control	3.47 (1.59)	4.45 (2.06)	0.402	0.684 (0.12)	0.002 (0.61)*
	Concussion	3.39 (1.34)	5.05 (2.90)			
Stride Length (cm)	Control	2.56 (0.90)	3.66 (1.81)	0.295	0.465 (0.20)	<0.001 (0.74)*
	Concussion	2.55 (1.02)	4.40 (2.50)			
Stride Width (cm)	Control	1.91 (0.61)	2.78 (0.98)	0.375	0.407 (0.23)	<0.001 (0.76)*
	Concussion	1.97 (0.59)	2.43 (0.97)			
Single Leg Support (%)	Control	0.43 (0.35)	0.84 (0.89)	0.334	0.634 (-0.12)	0.046 (0.37)*
	Concussion	0.50 (0.26)	0.65 (0.40)			

*denotes statistical significance ($p<0.05$). Stride velocity, length, and width were within trial variability calculated using PKMAS software. Single leg support % was between trial variability around the mean.

Table 5.3. Planned gait termination means and statistical findings

Outcome	Group	Condition		Group Mean Difference (95% CI)	Glare Mean Difference (95% CI)	Interaction (p)	Group Main Effect [p (Cohen's d)]	Condition Main Effect [p (Cohen's D)]
		No Glare (Mean ± SD)	Glare (Mean ± SD)					
Stride Velocity (cm/s)	Control	116.64 (20.09)	113.79 (20.06)	-8.37 (-17.11, 0.37)	-4.89 (-7.77, -2.01)	0.133	0.164 (-0.48)	0.001 (-0.61)*
	Concussion	110.19 (12.85)	103.50 (16.80)					
Stride Length (cm)	Control	134.13 (16.61)	131.65 (16.27)	-6.68 (-14.35, 0.98)	-4.16 (-6.37, -1.95)	0.081	0.193 (-0.44)	<0.001 (-0.68)*
	Concussion	129.03 (13.30)	123.38 (15.43)					
Stride Width (cm)	Control	11.08 (4.21)	11.99 (3.79)	0.36 (-2.15, 1.42)	0.76 (-0.01, 1.54)	0.684	0.731 (-0.10)	0.072 (0.36)
	Concussion	10.85 (3.02)	11.49 (3.41)					
Single Leg Support (%)	Control	35.94 (1.60)	35.86 (2.20)	0.86 (-1.97, 0.26)	0.17 (-0.68, 1.02)	0.588	0.241 (-0.39)	0.717 (0.07)
	Concussion	34.85 (3.17)	35.86 (2.20)					
Gait Termination Time (s)	Control	0.85 (0.38)	0.86 (0.41)	-0.01 (-0.19, 0.21)	-0.02 (-0.15, 0.12)	0.802	0.845 (0.02)	0.897 (-0.04)
	Concussion	0.88 (0.36)	0.84 (0.46)					

* denotes statistical significance ($p<0.05$)

Table 5.4. Planned gait termination task mean variability and statistical findings

Outcome	Group	Condition		Interaction (p)	Group Main Effect [p (Cohen's d)]	Condition Main Effect [p (Cohen's D)]
		No Glare (SD)	Glare (SD)			
Stride Velocity (cm/s)	Control	2.35 (1.58)	2.59 (1.53)	0.154	0.566 (0.18)	0.012 (0.48)*
	Concussion	2.29 (0.87)	3.13 (1.01)			
Stride Length (cm)	Control	1.75 (1.32)	2.67 (1.22)	0.738	0.069 (0.57)	<0.001 (0.79)*
	Concussion	2.47 (1.18)	3.55 (1.60)			
Stride Width (cm)	Control	1.41 (0.60)	1.57 (0.54)	0.357	0.020 (0.72)*	0.049 (0.37)*
	Concussion	1.82 (0.92)	2.28 (0.90)			
Single Leg Support (%)	Control	0.71 (0.35)	0.71 (0.34)	0.185	0.841 (-0.05)	0.185 (0.25)
	Concussion	0.57 (0.42)	0.82 (0.42)			
Gait Termination Time (s)	Control	0.35 (0.21)	0.30 (0.23)	0.529	0.147 (0.44)	0.804 (-0.04)
	Concussion	0.43 (0.37)	0.46 (0.20)			

*denotes statistical significance ($p<0.05$). Stride velocity, length, and width were within trial variability calculated using PKMAS software. Single leg support % was between trial variability around the mean.

Table 5.5. Unplanned gait termination means and statistical findings

Outcome	Group	Condition		Group Mean Difference (95% CI)	Glare Mean Difference (95% CI)	Interaction (p)	Group Main Effect [p (Cohen's d)]	Condition Main Effect [p (Cohen's D)]
		No Glare (Mean ± SD)	Glare (Mean ± SD)					
Stride Velocity (cm/s)	Control	113.13 (21.15)	109.77 (18.46)	-8.48 (-17.05, 0.09)	-3.81 (-6.25, -1.38)	0.717	0.183 (-0.50)	0.004 (-0.56)*
	Concussion	105.07 (14.82)	100.86 (14.53)					
Stride Length (cm)	Control	131.11 (16.72)	128.56 (16.25)	-7.28 (-15.03, 0.47)	-2.56 (-4.49, -0.64)	0.912	0.194 (-0.47)	0.011 (-0.48)*
	Concussion	123.85 (15.36)	121.27 (14.53)					
Stride Width (cm)	Control	11.03 (3.88)	11.32 (3.87)	0.19 (-1.98, 1.61)	0.29 (-0.46, 1.04)	0.873	0.869 (-0.05)	0.353 (0.14)
	Concussion	10.85 (2.84)	11.13 (3.93)					
Single Leg Support (%)	Control	34.79 (3.14)	35.35 (1.29)	0.08 (-1.01, 0.85)	0.03 (-0.70, 0.75)	0.185	0.989 (-0.04)	0.837 (0.01)
	Concussion	35.21 (1.19)	34.77 (1.30)					
Gait Termination Time (s)	Control	0.68 (0.27)	0.81 (0.32)	-0.09 (-0.06, 0.24)	0.11 (0.01, 0.22)	0.855	0.343 (0.31)	0.044 (0.39)*
	Concussion	0.78 (0.29)	0.89 (0.30)					

*denotes statistical significance ($p<0.05$)

Table 5.6. Unplanned gait termination task mean variability and statistical findings

Outcome	Group	Condition		Interaction (p)	Group Main Effect [p (Cohen's d)]	Condition Main Effect [p (Cohen's D)]
		No Glare	Glare			
Stride Velocity (cm/s)	Control	2.25 (0.64)	2.52 (1.08)	0.324	0.461 (-0.21)	0.736 (0.06)
	Concussion	2.27 (0.82)	2.14 (0.84)			
Stride Length (cm)	Control	1.70 (0.54)	1.92 (0.91)	0.596	0.359 (0.28)	0.077 (0.34)
	Concussion	1.84 (0.84)	2.25 (1.03)			
Stride Width (cm)	Control	1.55 (0.74)	1.94 (0.93)	0.387	0.356 (0.29)	<0.001 (0.71)*
	Concussion	1.69 (0.66)	2.29 (0.88)			
Single Leg Support (%)	Control	0.72 (0.53)	0.62 (0.32)	0.488	0.902 (0.07)	0.787 (-0.02)
	Concussion	0.66 (0.39)	0.73 (0.48)			
Gait Termination Time (s)	Control	0.33 (0.15)	0.44 (0.24)	0.370	0.695 (0.09)	0.366 (0.16)
	Concussion	0.40 (0.28)	0.40 (0.19)			

*denotes statistical significance ($p<0.05$). Stride velocity, length, and width were within trial variability calculated using PKMAS software. Single leg support % was between trial variability around the mean.

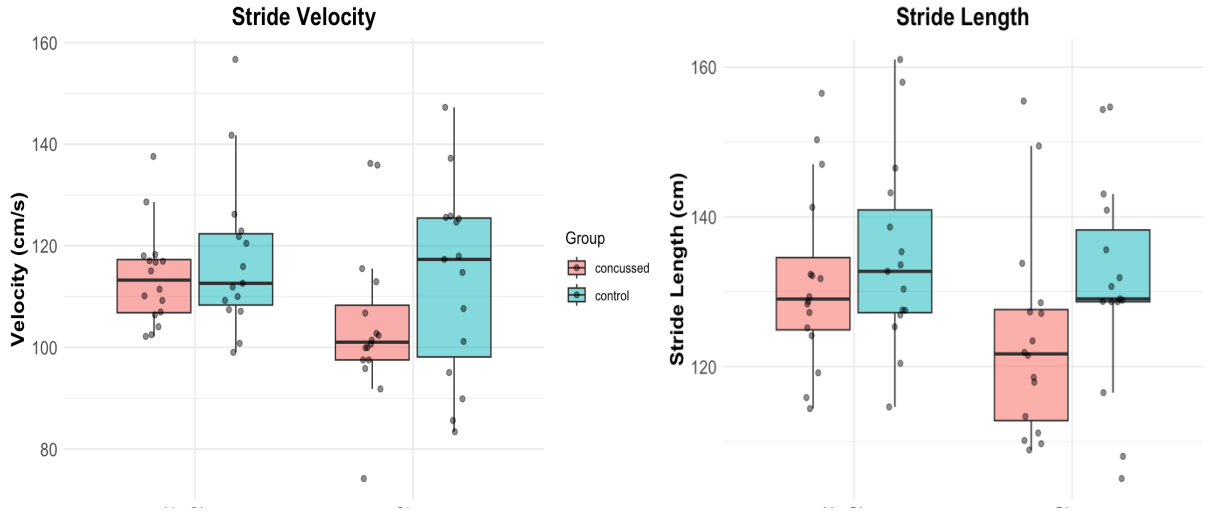

Figure 5.3. Plot of stride velocity and stride length during the straight path gait task. Glare condition significantly decreased stride velocity (p=0.002, d=0.61) and stride length (p<0.001, d=0.79), both with medium effect sizes. Group differences were not significant for stride velocity (p=0.23, d=0.40) or stride length (p=0.28, d=0.37), but small effects were observed. There were no significant interaction effects (p>0.05).

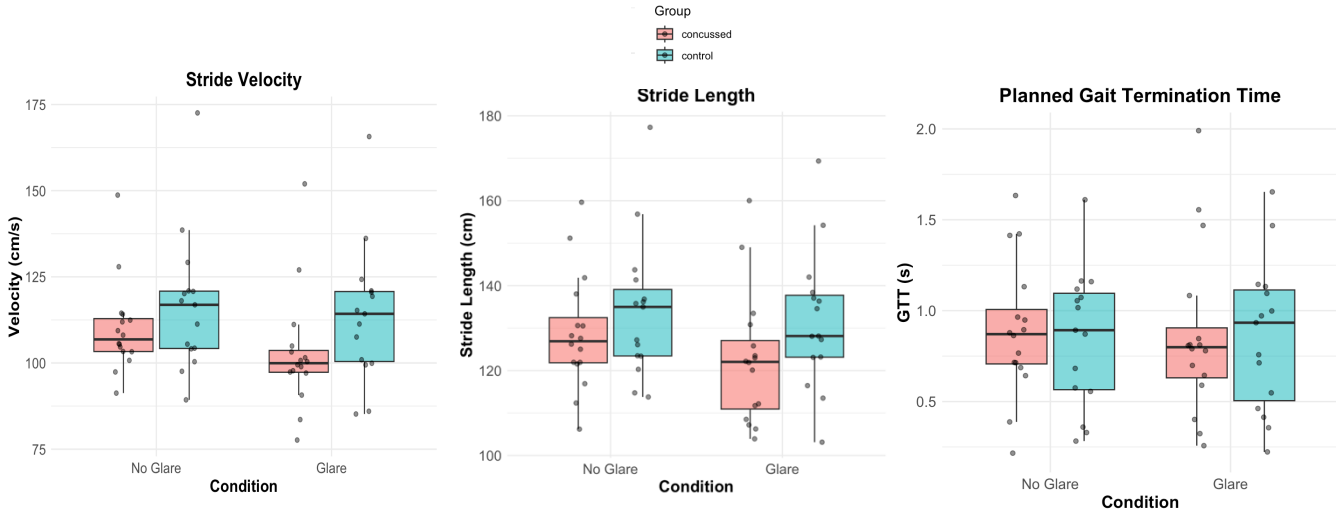

Figure 5.4. Plot of stride velocity, stride length, and gait termination time during the planned gait termination task. Glare condition significantly decreased stride velocity (p=0.002, d=0.61) and stride length (p<0.001, d=0.68), both with medium effect sizes, while no glare effect was observed for gait termination time (p=0.83, d=0.04). Group differences were not significant for any variable, though stride velocity (p=0.18, d=0.48) and stride length (p=0.22, d=0.44) showed small to medium effects and gait termination time showed a negligible group effect (p=0.94, d=0.02). There were no significant interaction effects (p>0.05).

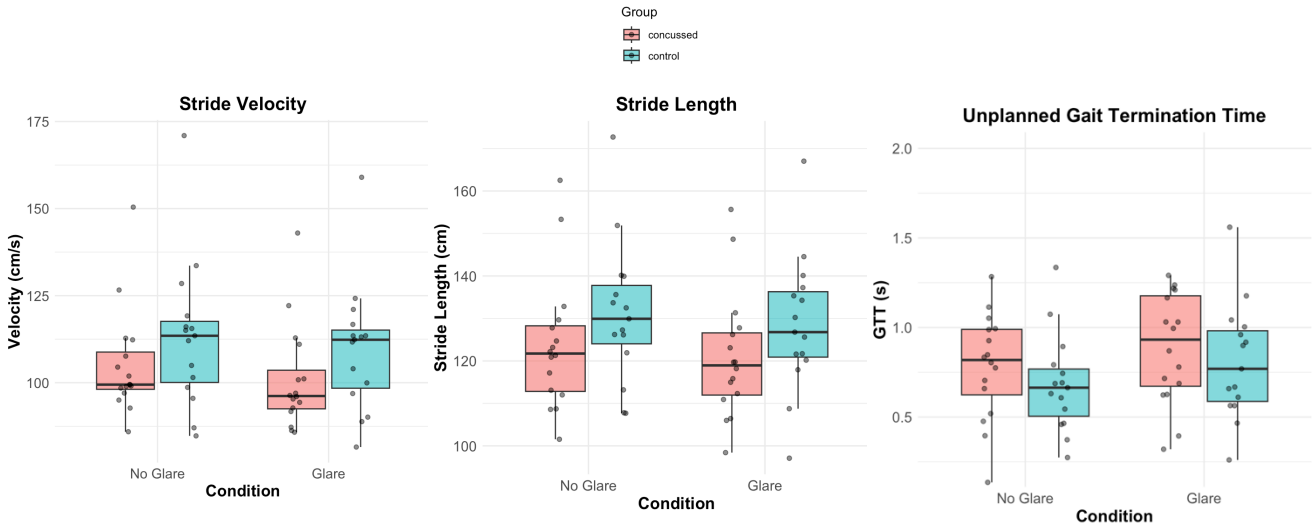

Figure 5.5. Plot of stride velocity, stride length, and gait termination time during the unplanned gait termination task. Glare condition significantly decreased stride velocity (p=0.004, d=0.56), stride length (p=0.01, d=0.48), and increased gait termination time (p=0.04, d=0.39), with small to medium effect sizes. Group differences were not significant for any variable, though stride velocity (p=0.18, d=0.50) showed a medium effect size, while stride length (p=0.19, d=0.47) and gait termination time (p=0.32, d=0.31) reflected small effects. There were no significant interaction effects (p>0.05).

Discussion

Main Findings

We aimed to examine the effects of a concussion history on various gait tasks and to identify if glare impacts gait mechanics. Our findings indicate that glare significantly impacts certain aspects of gait performance, specifically stride velocity and stride length, regardless of task, and GTT in more complex tasks like unplanned gait termination. However, there were no differences between participants with and without a concussion history, and there were no interaction effects found, suggesting that while glare negatively affects gait across tasks, a history of concussion does not appear to exacerbate this effect. This indicates that the impact of glare on gait performance may be a general sensory-motor challenge rather than one specifically heightened by prior concussion.

Concussion History Main Effect

Our group main effect findings contradict previous evidence showing that gait impairments can linger for a significant duration after symptom resolution.[59] However, they are consistent with research indicating that straight path gait impairments can resolve within a shorter timeframe after concussion, particularly in less challenging motor tasks.[187] The lack of a main effect of group may suggest that the participants in the concussion history group may be fully recovered functionally at the time of testing. All individuals in this group were asymptomatic and had sustained their concussion within the past two years, and therefore, were not still injured. With an average time since their concussion of 252.5 days it is reasonable to suggest that any potential gait differences identified on these tasks may resolve at some point between two months, when lingering gait impairments have previously been observed, and approximately eight months.

This interpretation also aligns with a previous study assessing somatosensory cortical connectivity after concussion, which may play a role in LEMSK injury risk.[228] The authors found that connectivity impairments were delayed after one month, but improvements were experienced at five months post-concussion.[228] This suggests that there is a delayed recovery, but the recovery may have occurred before the time of our testing. It is also possible that our sample size limited our ability to detect smaller but meaningful group differences which may be supported by the smaller effect sizes.

Although our findings did not reveal statistically significant group differences or interaction effects across any gait task, it is important to consider the potential trends observed in the data. While stride velocity and stride length did not differ significantly between groups, the associated effect sizes were notable. Stride velocity demonstrated small to medium effects

(Cohen's D = -0.40 to -0.50) and stride length showed small to approaching medium effects (Cohen's D = -0.37 to -0.47). These differences can be visually observed in Figures 5.3, 5.4, and 5.5. Given the magnitude of these effect sizes and the directional trends in the data, it is possible that meaningful group differences may actually exist but were not detected due to the limited sample size. This possibility suggests that individuals with a history of concussion may experience subtle lingering gait alterations that larger sample sizes could better identify.

Additionally, it is important to note that the current study used only single-task gait assessments. Prior research has demonstrated that gait differences following concussion are often most apparent under dual-task conditions, where cognitive demands may unmask motor deficits.[228] The absence of dual-task conditions in our study may have limited our ability to detect group differences.

Glare Main Effect

A significant main effect of glare was found for stride velocity and stride length, with participants demonstrating slower and shorter gait patterns in glare conditions across all tasks. This suggests that a common visual stimulus, like glare, can significantly influence motor control and efficiency, and the effect sizes for these main effects suggest that these are meaningful differences. Since visual information is fundamental to guiding safe and effective movement, visual stimuli and photostressors like glare may alter gait strategy in ways that could increase the risk of trips, falls, or other injuries in real-world environments. During the unplanned gait termination task, we observed a significant main effect of glare on GTT. The increased complexity and need for the ability to react to a random cue in this condition may increase the reliance on accurate visual input, making the impacts of glare more apparent in how quickly one comes to a stop. These results suggest that glare becomes a more meaningful factor when tasks

demand rapid adjustment and integration of sensory information, potentially mirroring the kinds of challenges individuals may encounter in their everyday life. This is particularly relevant for populations vulnerable to visual or functional disruptions, including individuals recovering from concussion.

Additionally, our analyses of spatiotemporal variability measures further supports the disruptive impact of glare on gait. We found significant main effects of glare on variability across several tasks, particularly stride velocity, stride length, and stride width. These findings suggest that glare not only alters average gait performance but also increases gait inconsistency, which may reflect heightened sensorimotor processing demands under glare conditions. Increased variability is often interpreted as a marker of reduced gait stability or increased cognitive-motor load, supporting the interpretation that glare taxes the sensory-motor system during movement.[50,57] This broad impact across tasks and gait parameters indicates that glare disrupts gait strategy and consistency, potentially elevating fall risk or injury in real-world glare settings.

We did not find significant effects of glare for stride width or single leg support percentage. This is consistent with previous research showing that, in individuals with concussions or other traumatic brain injuries, variables such as step length and velocity are more likely to change significantly, while stride width and single leg support percentage often remain unaffected.[229–231] One possible explanation is that changes in these particular gait outcomes may not be as pronounced in otherwise healthy, asymptomatic individuals with a history of concussion compared to those with more severe neurological impairments.[232,233]

Our findings add to the growing evidence highlighting the connection between vision and functional movement. While visual impairments are well-documented following concussion, few

studies have explored how everyday visual disturbances might affect motor performance. This is the first study to assess the impact of a glare on gait in individuals with a history of concussion. Our data suggests that glare is a meaningful stimulus that can alter basic gait patterns, and it may be a useful tool for uncovering subtle functional deficits. Although not a concussed population, one study focused on individuals with migraines found with increasing light intensity and light sensitivity, only step width was impacted and patients actually presented with a less conservative gait strategy.[234] This contrasts with our findings. While our study examined the impact of increased glare during walking, another study investigating gait under poor lighting conditions found that participants adopted a more cautious gait in dim settings.[235] This supports the broader link between impaired visual input and altered gait mechanics, highlighting the potential for increased fall and injury risk.

Limitations

The smaller sample size may have limited our ability to detect group differences or interactions, as reflected in the lack of statistically significant differences between groups despite small to moderate effect sizes on all gait tasks. Additionally, participants with a concussion history were asymptomatic and likely in a chronic recovery phase, which may not reflect ongoing deficits that occur closer to injury. Finally, while our chosen gait tasks captured key gait characteristics, they may not have been complex enough to fully reveal subtle impairments at the time of testing.

Future Directions

Future research should target individuals in the acute or early subacute stages of concussion, where gait and visual-motor deficits are more likely to be pronounced. Additionally, incorporating more complex tasks may increase the sensitivity of assessments and uncover

lingering impairments that are not evident during simpler gait evaluations. Other types of visual challenges, such as differing environments and other visual distractors, should also be investigated to better simulate real-world conditions and demands. Understanding how various visual stimuli affect motor control could offer insight into why individuals with concussion are at elevated risk for musculoskeletal injury upon return to activity. Assessing how visual dysfunction contributes to motor instability may help improve clinical tools and return-to-activity decision-making. Expanding the sample size may help to confirm the glare main effects and may also reveal potential interaction effects or group main effects that were not detected in this study due to limited sample size.

Conclusion

This study demonstrates that glare, a common visual stimuli, significantly alters gait by reducing stride length and stride velocity across various walking tasks, and increases GTT in an unplanned gait termination task. However, no effects were found for stride width and single leg support percentage. No significant group differences were observed between individuals with and without a history of concussion but small to moderate effect sizes for between group differences on gait outcomes may suggest there are meaningful differences that are potentially limited by the current sample size. These findings highlight that even subtle, everyday visual disturbances like glare can meaningfully alter how we move, reinforcing the critical role of vision in safe and effective motor performance.

CHAPTER 6

FINDINGS SUMMARY

Aim 1 of this study examined the visual function in individuals with and without a history of concussion using a novel battery of psychophysical tests, i.e., MPOD, TCSF, CFF, and glare responses. A total of 31 participants were enrolled (15 controls, 16 with concussion history). There were no statistically significant group differences across any visual measures. Although prior research has suggested lingering visual impairments following concussion, our findings showed no detectable deficits in MPOD, TCSF, CFF, or glare sensitivity/discomfort. These null results may reflect full recovery in our sample, as all participants were well beyond the acute phase with an average time since concussion of 252 days. The psychophysical tests used are sensitive to cortical and retinal function and have demonstrated utility in aging and neurodegenerative populations, making them strong candidates for detecting subtle changes post-concussion. However, the lack of group differences may suggest these impairments may not persist into the chronic stage, or they may only be present in individuals with ongoing symptoms.

Aim 2 looked to evaluate the effects of concussion history and visual glare on gait performance during three tasks: straight path walking, planned gait termination, and unplanned gait termination. No significant main effects of concussion history or interactions between concussion history and glare condition were found for any gait outcomes. However, a significant main effect of glare condition existed for stride velocity and stride length which were significantly reduced on all gait tasks ($p < 0.05$). Additionally, glare increased GTT during the unplanned gait termination task ($p = 0.04$). No significant effects were observed for stride width

or single leg support percentage. Furthermore, glare significantly increased gait variability for several spatiotemporal measures, including stride velocity, stride length, and stride width across tasks. Increased gait variability is often interpreted as reduced gait stability or increased cognitive-motor demand, suggesting that glare not only alters average gait performance but also taxes sensory-motor processing and consistency during movement.

The absence of group differences suggests participants with a concussion history were functionally recovered at the time of testing, consistent with previous research showing gait deficits resolve within months post-injury, especially during simple, single-task walking. Glare significantly impacted gait by reducing stride length and velocity, indicating that visual disturbances can impair motor control and efficiency even in healthy individuals. The lack of glare effects on stride width and single leg support aligns with prior findings that these parameters are less sensitive to mild neurological changes. The increased GTT during unplanned termination under glare suggests that tasks requiring rapid adjustments rely heavily on clear visual input. These findings emphasize the importance of vision in safe movement and suggest that glare could potentially elevate fall and injury risk in real-world environments.

Additionally, it is important to note the novelty in the use of glare goggles during gait tasks. To our knowledge, this is the first application of this technology in any population. These goggles provided a standardized and controlled method to bring a photostressor commonly experienced in daily life into a research setting. The successful implementation of the goggles in this sample demonstrates their feasibility for future use and their ability to produce transient visual disturbances that result in significant gait changes across all participants, regardless of concussion history.

This study's findings are limited by a modest sample size and the inclusion of only asymptomatic individuals, which reduces the generalizability of these findings. Future research should prioritize recruiting individuals in the acute stages of concussion to better capture the recovery of visual and gait impairments. Incorporating more complex motor tasks and a variety of visual challenges, such as differing environments, glare, and other sensory distractors, may enhance sensitivity to subtle functional deficits that are not apparent during simpler assessments.

In conclusion, while this study found no lasting visual impairments or gait differences in asymptomatic individuals with a history of concussion, it highlights that everyday visual disturbances like glare can meaningfully alter gait performance in both concussed and non-concussed populations. These results highlight the critical role of vision in safe and effective motor control and suggest that visual disturbances represent a general sensory-motor challenge rather than a condition specifically exacerbated by concussion history.

REFERENCES

1. Taylor CA, Bell JM, Breiding MJ, Xu L. Traumatic Brain Injury-Related Emergency Department Visits, Hospitalizations, and Deaths - United States, 2007 and 2013. *Morb Mortal Wkly Rep Surveill Summ Wash DC 2002*. 2017;66(9):1-16. doi:10.15585/mmwr.ss6609a1

2. Baldwin GT, Breiding MJ, Dawn Comstock R. Epidemiology of sports concussion in the United States. *Handb Clin Neurol*. 2018;158:63-74. doi:10.1016/B978-0-444-63954-7.00007-0

3. Cancelliere C, Coronado VG, Taylor CA, Xu L. Epidemiology of Isolated Versus Nonisolated Mild Traumatic Brain Injury Treated in Emergency Departments in the United States, 2006-2012: Sociodemographic Characteristics. *J Head Trauma Rehabil*. 2017;32(4):E37-E46. doi:10.1097/HTR.0000000000000260

4. Daneshvar DH, Nowinski CJ, McKee AC, Cantu RC. The epidemiology of sport-related concussion. *Clin Sports Med*. 2011;30(1):1-17, vii. doi:10.1016/j.csm.2010.08.006

5. Hon KL, Leung AKC, Torres AR. Concussion: A Global Perspective. *Semin Pediatr Neurol*. 2019;30:117-127. doi:10.1016/j.spen.2019.03.017

6. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42(2):311-319.

7. Hallock H, Mantwill M, Vajkoczy P, et al. Sport-Related Concussion: A Cognitive Perspective. *Neurol Clin Pract*. 2023;13(2):e200123. doi:10.1212/CPJ.0000000000200123

8. Zuckerman SL, Kerr ZY, Yengo-Kahn A, Wasserman E, Covassin T, Solomon GS. Epidemiology of Sports-Related Concussion in NCAA Athletes From 2009-2010 to 2013-2014: Incidence, Recurrence, and Mechanisms. *Am J Sports Med*. 2015;43(11):2654-2662. doi:10.1177/0363546515599634

9. Schallmo MS, Weiner JA, Hsu WK. Sport and Sex-Specific Reporting Trends in the Epidemiology of Concussions Sustained by High School Athletes. *J Bone Joint Surg Am*. 2017;99(15):1314-1320. doi:10.2106/JBJS.16.01573

10. Pfister T, Pfister K, Hagel B, Ghali WA, Ronksley PE. The incidence of concussion in youth sports: a systematic review and meta-analysis. *Br J Sports Med*. 2016;50(5):292-297. doi:10.1136/bjsports-2015-094978

11. Nathanson JT, Connolly JG, Yuk F, et al. Concussion Incidence in Professional Football: Position-Specific Analysis With Use of a Novel Metric. *Orthop J Sports Med*. 2016;4(1):2325967115622621. doi:10.1177/2325967115622621

12. Kerr ZY, Register-Mihalik JK, Marshall SW, Evenson KR, Mihalik JP, Guskiewicz KM. Disclosure and non-disclosure of concussion and concussion symptoms in athletes: review and application of the socio-ecological framework. *Brain Inj*. 2014;28(8):1009-1021. doi:10.3109/02699052.2014.904049

13. Llewellyn T, Burdette GT, Joyner AB, Buckley TA. Concussion reporting rates at the conclusion of an intercollegiate athletic career. *Clin J Sport Med Off J Can Acad Sport Med*. 2014;24(1):76-79. doi:10.1097/01.jsm.0000432853.77520.3d

14. Register-Mihalik JK, Guskiewicz KM, McLeod TCV, Linnan LA, Mueller FO, Marshall SW. Knowledge, attitude, and concussion-reporting behaviors among high school athletes: a preliminary study. *J Athl Train*. 2013;48(5):645-653. doi:10.4085/1062-6050-48.3.20

15. Fraas MR, Coughlan GF, Hart EC, McCarthy C. Concussion history and reporting rates in elite Irish rugby union players. *Phys Ther Sport Off J Assoc Chart Physiother Sports Med*. 2014;15(3):136-142. doi:10.1016/j.ptsp.2013.08.002

16. Patricios JS, Schneider KJ, Dvorak J, et al. Consensus statement on concussion in sport: the 6th International Conference on Concussion in Sport-Amsterdam, October 2022. *Br J Sports Med*. 2023;57(11):695-711. doi:10.1136/bjsports-2023-106898

17. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S. Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. *Optom St Louis Mo*. 2007;78(4):155-161. doi:10.1016/j.optm.2006.11.011

18. Scheiman M, Grady MF, Jenewein E, et al. Frequency of oculomotor disorders in adolescents 11 to 17 years of age with concussion, 4 to 12 weeks post injury. *Vision Res*. 2021;183:73-80. doi:10.1016/j.visres.2020.09.011

19. Snegireva N, Derman W, Patricios J, Welman KE. Eye tracking technology in sports-related concussion: a systematic review and meta-analysis. *Physiol Meas*. 2018;39(12):12TR01. doi:10.1088/1361-6579/aaef44

20. Mucha A, Collins MW, Elbin RJ, et al. A Brief Vestibular/Ocular Motor Screening (VOMS) assessment to evaluate concussions: preliminary findings. *Am J Sports Med*. 2014;42(10):2479-2486. doi:10.1177/0363546514543775

21. Singman E, Quaid P. Chapter 15 - Vision Disorders in Mild Traumatic Brain Injury. In: Hoffer ME, Balaban CD, eds. *Neurosensory Disorders in Mild Traumatic Brain Injury*. Academic Press; 2019:223-244. doi:10.1016/B978-0-12-812344-7.00015-7

22. Cockerham GC, Goodrich GL, Weichel ED, et al. Eye and visual function in traumatic brain injury. *J Rehabil Res Dev*. 2009;46(6):811-818. doi:10.1682/jrrd.2008.08.0109

23. Master CL, Bacal D, Grady MF, et al. Vision and Concussion: Symptoms, Signs, Evaluation, and Treatment. *Pediatrics*. 2022;150(2):e2021056047. doi:10.1542/peds.2021-056047

24. Le RK, Ortega J, Chrisman SP, et al. King-Devick Sensitivity and Specificity to Concussion in Collegiate Athletes. *J Athl Train*. 2023;58(2):97-105. doi:10.4085/1062-6050-0063.21

25. Menorca RMG, Fussell TS, Elfar JC. Nerve physiology: mechanisms of injury and recovery. *Hand Clin*. 2013;29(3):317-330. doi:10.1016/j.hcl.2013.04.002

26. Nudo RJ. Recovery after brain injury: mechanisms and principles. *Front Hum Neurosci*. 2013;7:887. doi:10.3389/fnhum.2013.00887

27. Beydoun MA, Beydoun HA, Fanelli-Kuczmarski MT, et al. Association of Serum Antioxidant Vitamins and Carotenoids With Incident Alzheimer Disease and All-Cause Dementia Among US Adults. *Neurology*. 2022;98(21):e2150-e2162. doi:10.1212/WNL.0000000000200289

28. Hammond BR, Wooten BR. CFF thresholds: relation to macular pigment optical density. *Ophthalmic Physiol Opt J Br Coll Ophthalmic Opt Optom*. 2005;25(4):315-319. doi:10.1111/j.1475-1313.2005.00271.x

29. Meyer Zu Westrup V, Dietzel M, Zeimer M, Pauleikhoff D, Hense HW. Changes of macular pigment optical density in elderly eyes: a longitudinal analysis from the MARS study. *Int J Retina Vitr*. 2016;2:14. doi:10.1186/s40942-016-0039-6

30. Renzi-Hammond LM, Bovier ER, Fletcher LM, et al. Effects of a Lutein and Zeaxanthin Intervention on Cognitive Function: A Randomized, Double-Masked, Placebo-Controlled Trial of Younger Healthy Adults. *Nutrients*. 2017;9(11):1246. doi:10.3390/nu9111246

31. Taguchi A, Kinoshita Y, Tokumo K, et al. Usefulness of critical flicker fusion frequency measurement and its laterality for evaluating compressive optic neuropathy due to pituitary neuroendocrine tumors. *Neurosurg Rev*. 2022;46(1):4. doi:10.1007/s10143-022-01915-z

32. Algvere PV, Marshall J, Seregard S. Age-related maculopathy and the impact of blue light hazard. *Acta Ophthalmol Scand*. 2006;84(1):4-15. doi:10.1111/j.1600-0420.2005.00627.x

33. Fernandez J. What we have learnt from 30 years living with positive dysphotopsia after intraocular lens implantation?: a review: Accessed June 26, 2025. https://www.tandfonline.com/doi/full/10.1080/17469899.2021.1917997

34. Hammond BR, Gardner CR, Renzi-Hammond L. The Effects of Blue-Light Filtering Intraocular Implants on Glare Geometry. *Curr Eye Res*. 2023;48(7):639-644. doi:10.1080/02713683.2023.2192446

35. Sharpe LT, Stockman A, Knau H, Jägle H. Macular pigment densities derived from central and peripheral spectral sensitivity differences. *Vision Res*. 1998;38(21):3233-3239. doi:10.1016/s0042-6989(97)00457-4

36. Tyler CW. Two processes control variations in flicker sensitivity over the life span. *J Opt Soc Am A*. 1989;6(4):481-490. doi:10.1364/josaa.6.000481

37. Tyler CW, Likova LT. Brain trauma impacts retinal processing: photoreceptor pathway interactions in traumatic light sensitivity. *Doc Ophthalmol Adv Ophthalmol*. 2022;144(3):179-190. doi:10.1007/s10633-022-09871-1

38. Bernstein PS, Delori FC, Richer S, van Kuijk FJM, Wenzel AJ. The value of measurement of macular carotenoid pigment optical densities and distributions in age-related macular degeneration and other retinal disorders. *Vision Res*. 2010;50(7):716-728. doi:10.1016/j.visres.2009.10.014

39. Kelly D, Coen RF, Akuffo KO, et al. Cognitive Function and Its Relationship with Macular Pigment Optical Density and Serum Concentrations of its Constituent Carotenoids. *J Alzheimers Dis JAD*. 2015;48(1):261-277. doi:10.3233/JAD-150199

40. Vishwanathan R, Iannaccone A, Scott TM, et al. Macular pigment optical density is related to cognitive function in older people. *Age Ageing*. 2014;43(2):271-275. doi:10.1093/ageing/aft210

41. Giza CC, Hovda DA. The new neurometabolic cascade of concussion. *Neurosurgery*. 2014;75 Suppl 4(0 4):S24-33. doi:10.1227/NEU.0000000000000505

42. Howell DR, Lynall RC, Buckley TA, Herman DC. Neuromuscular Control Deficits and the Risk of Subsequent Injury after a Concussion: A Scoping Review. *Sports Med Auckl NZ*. 2018;48(5):1097-1115. doi:10.1007/s40279-018-0871-y

43. Powers KC, Kalmar JM, Cinelli ME. Recovery of static stability following a concussion. *Gait Posture*. 2014;39(1):611-614. doi:10.1016/j.gaitpost.2013.05.026

44. Parker TM, Osternig LR, VAN Donkelaar P, Chou LS. Gait stability following concussion. *Med Sci Sports Exerc*. 2006;38(6):1032-1040. doi:10.1249/01.mss.0000222828.56982.a4

45. Lynall RC, Mauntel TC, Pohlig RT, et al. Lower Extremity Musculoskeletal Injury Risk After Concussion Recovery in High School Athletes. *J Athl Train*. 2017;52(11):1028-1034. doi:10.4085/1062-6050-52.11.22

46. McPherson AL, Nagai T, Webster KE, Hewett TE. Musculoskeletal Injury Risk After Sport-Related Concussion: A Systematic Review and Meta-analysis. *Am J Sports Med*. 2019;47(7):1754-1762. doi:10.1177/0363546518785901

47. Pirker W, Katzenschlager R. Gait disorders in adults and the elderly : A clinical guide. *Wien Klin Wochenschr*. 2017;129(3-4):81-95. doi:10.1007/s00508-016-1096-4

48. Reidy J, Mobbs R, Kim J, Brown E, Mobbs R. Clinical gait characteristics in the early post-concussion phase: A systematic review. *J Clin Neurosci Off J Neurosurg Soc Australas*. 2023;107:184-191. doi:10.1016/j.jocn.2022.11.005

49. Oldham JR, Munkasy BA, Evans KM, Wikstrom EA, Buckley TA. Altered dynamic postural control during gait termination following concussion. *Gait Posture*. 2016;49:437-442. doi:10.1016/j.gaitpost.2016.07.327

50. Fino PC, Parrington L, Pitt W, et al. Detecting gait abnormalities after concussion or mild traumatic brain injury: A systematic review of single-task, dual-task, and complex gait. *Gait Posture*. 2018;62:157-166. doi:10.1016/j.gaitpost.2018.03.021

51. Berkner J, Meehan WP, Master CL, Howell DR. Gait and Quiet-Stance Performance Among Adolescents After Concussion-Symptom Resolution. *J Athl Train*. 2017;52(12):1089-1095. doi:10.4085/1062-6050-52.11.23

52. Kamins J, Bigler E, Covassin T, et al. What is the physiological time to recovery after concussion? A systematic review. *Br J Sports Med*. 2017;51(12):935-940. doi:10.1136/bjsports-2016-097464

53. Murray NG, Ambati VNP, Contreras MM, Salvatore AP, Reed-Jones RJ. Assessment of oculomotor control and balance post-concussion: a preliminary study for a novel approach to concussion management. *Brain Inj*. 2014;28(4):496-503. doi:10.3109/02699052.2014.887144

54. Lirani-Silva E, Stuart S, Parrington L, Campbell K, King L. Saccade and Fixation Eye Movements During Walking in People With Mild Traumatic Brain Injury. *Front Bioeng Biotechnol*. 2021;9:701712. doi:10.3389/fbioe.2021.701712

55. Digre KB, Brennan KC. Shedding light on photophobia. *J Neuro-Ophthalmol Off J North Am Neuro-Ophthalmol Soc*. 2012;32(1):68-81. doi:10.1097/WNO.0b013e3182474548

56. Abusamak M, Alrawashdeh HM. Post-concussion Syndrome Light Sensitivity: A Case Report and Review of the Literature. *Neuro-Ophthalmol Aeolus Press*. 2022;46(2):85-90. doi:10.1080/01658107.2021.1983612

57. Howell DR, Osternig LR, Chou LS. Single-task and dual-task tandem gait test performance after concussion. *J Sci Med Sport*. 2017;20(7):622-626. doi:10.1016/j.jsams.2016.11.020

58. Johnson RS, Scott KH, Lynall RC. A Proposal for Complex Gait Evaluation Using Dual-Task Gait Termination Time. *J Sport Rehabil*. 2020;30(4):525-530. doi:10.1123/jsr.2020-0080

59. Buckley TA, Munkasy BA, Tapia-Lovler TG, Wikstrom EA. Altered gait termination strategies following a concussion. *Gait Posture*. 2013;38(3):549-551. doi:10.1016/j.gaitpost.2013.02.008

60. Helmick KM, Spells CA, Malik SZ, Davies CA, Marion DW, Hinds SR. Traumatic brain injury in the US military: epidemiology and key clinical and research programs. *Brain Imaging Behav*. 2015;9(3):358-366. doi:10.1007/s11682-015-9399-z

61. Mack CD, Solomon G, Covassin T, Theodore N, Cárdenas J, Sills A. Epidemiology of Concussion in the National Football League, 2015-2019. *Sports Health*. 2021;13(5):423-430. doi:10.1177/19417381211011446

62. Benson BW, Meeuwisse WH, Rizos J, Kang J, Burke CJ. A prospective study of concussions among National Hockey League players during regular season games: the NHL-NHLPA Concussion Program. *CMAJ Can Med Assoc J J Assoc Medicale Can*. 2011;183(8):905-911. doi:10.1503/cmaj.092190

63. Viano DC, Parenteau CS. Concussion, Diffuse Axonal Injury, and AIS4+ Head Injury in Motor Vehicle Crashes. *Traffic Inj Prev*. 2015;16(8):747-753. doi:10.1080/15389588.2015.1013188

64. DOD TBI Worldwide Numbers. Military Health System. Accessed June 26, 2025. https://www.health.mil/Military-Health-Topics/Centers-of-Excellence/Traumatic-Brain-Injury-Center-of-Excellence/DOD-TBI-Worldwide-Numbers

65. Breck J, Bohr A, Poddar S, McQueen MB, Casault T. Characteristics and Incidence of Concussion Among a US Collegiate Undergraduate Population. *JAMA Netw Open*. 2019;2(12):e1917626. doi:10.1001/jamanetworkopen.2019.17626

66. Katayama Y, Becker DP, Tamura T, Hovda DA. Massive increases in extracellular potassium and the indiscriminate release of glutamate following concussive brain injury. *J Neurosurg*. 1990;73(6):889-900. doi:10.3171/jns.1990.73.6.0889

67. Takahashi H, Manaka S, Sano K. Changes in extracellular potassium concentration in cortex and brain stem during the acute phase of experimental closed head injury. *J Neurosurg*. 1981;55(5):708-717. doi:10.3171/jns.1981.55.5.0708

68. Yoshino A, Hovda DA, Kawamata T, Katayama Y, Becker DP. Dynamic changes in local cerebral glucose utilization following cerebral conclusion in rats: evidence of a hyper- and subsequent hypometabolic state. *Brain Res*. 1991;561(1):106-119. doi:10.1016/0006-8993(91)90755-k

69. Hovda DA. Metabolic Dysfunction. In: *Neurotrauma*. McGraw-Hill; 1996:1459-1478.

70. Pettus EH, Christman CW, Giebel ML, Povlishock JT. Traumatically induced altered membrane permeability: its relationship to traumatically induced reactive axonal change. *J Neurotrauma*. 1994;11(5):507-522. doi:10.1089/neu.1994.11.507

71. Pettus EH, Povlishock JT. Characterization of a distinct set of intra-axonal ultrastructural changes associated with traumatically induced alteration in axolemmal permeability. *Brain Res*. 1996;722(1-2):1-11. doi:10.1016/0006-8993(96)00113-8

72. Giza CC, Maria NSS, Hovda DA. N-methyl-D-aspartate receptor subunit changes after traumatic injury to the developing brain. *J Neurotrauma*. 2006;23(6):950-961. doi:10.1089/neu.2006.23.950

73. Atkins CM, Falo MC, Alonso OF, Bramlett HM, Dietrich WD. Deficits in ERK and CREB activation in the hippocampus after traumatic brain injury. *Neurosci Lett*. 2009;459(2):52-56. doi:10.1016/j.neulet.2009.04.064

74. Osteen CL, Giza CC, Hovda DA. Injury-induced alterations in N-methyl-D-aspartate receptor subunit composition contribute to prolonged 45calcium accumulation following lateral fluid percussion. *Neuroscience*. 2004;128(2):305-322. doi:10.1016/j.neuroscience.2004.06.034

75. Kumar A, Zou L, Yuan X, Long Y, Yang K. N-methyl-D-aspartate receptors: transient loss of NR1/NR2A/NR2B subunits after traumatic brain injury in a rodent model. *J Neurosci Res*. 2002;67(6):781-786. doi:10.1002/jnr.10181

76. Lowenstein DH, Thomas MJ, Smith DH, McIntosh TK. Selective vulnerability of dentate hilar neurons following traumatic brain injury: a potential mechanistic link between head trauma and disorders of the hippocampus. *J Neurosci Off J Soc Neurosci*. 1992;12(12):4846-4853. doi:10.1523/JNEUROSCI.12-12-04846.1992

77. Zanier ER, Lee SM, Vespa PM, Giza CC, Hovda DA. Increased hippocampal CA3 vulnerability to low-level kainic acid following lateral fluid percussion injury. *J Neurotrauma*. 2003;20(5):409-420. doi:10.1089/089771503765355496

78. Reger ML, Poulos AM, Buen F, Giza CC, Hovda DA, Fanselow MS. Concussive brain injury enhances fear learning and excitatory processes in the amygdala. *Biol Psychiatry*. 2012;71(4):335-343. doi:10.1016/j.biopsych.2011.11.007

79. Fendt M, Fanselow MS. The neuroanatomical and neurochemical basis of conditioned fear. *Neurosci Biobehav Rev*. 1999;23(5):743-760. doi:10.1016/s0149-7634(99)00016-0

80. Koenigs M, Huey ED, Raymont V, et al. Focal brain damage protects against post-traumatic stress disorder in combat veterans. *Nat Neurosci*. 2008;11(2):232-237. doi:10.1038/nn2032

81. Li HH, Lee SM, Cai Y, Sutton RL, Hovda DA. Differential gene expression in hippocampus following experimental brain trauma reveals distinct features of moderate and severe injuries. *J Neurotrauma*. 2004;21(9):1141-1153. doi:10.1089/neu.2004.21.1141

82. Giza CC, Prins ML. Is being plastic fantastic? Mechanisms of altered plasticity after developmental traumatic brain injury. *Dev Neurosci*. 2006;28(4-5):364-379. doi:10.1159/000094163

83. Hutson CB, Lazo CR, Mortazavi F, Giza CC, Hovda D, Chesselet MF. Traumatic brain injury in adult rats causes progressive nigrostriatal dopaminergic cell loss and enhanced vulnerability to the pesticide paraquat. *J Neurotrauma*. 2011;28(9):1783-1801. doi:10.1089/neu.2010.1723

84. Israelsson C, Bengtsson H, Kylberg A, et al. Distinct cellular patterns of upregulated chemokine expression supporting a prominent inflammatory role in traumatic brain injury. *J Neurotrauma*. 2008;25(8):959-974. doi:10.1089/neu.2008.0562

85. Leao A a. P. Further observations on the spreading depression of activity in the cerebral cortex. *J Neurophysiol*. 1947;10(6):409-414. doi:10.1152/jn.1947.10.6.409

86. Mihalik JP, Stump JE, Collins MW, Lovell MR, Field M, Maroon JC. Posttraumatic migraine characteristics in athletes following sports-related concussion. *J Neurosurg*. 2005;102(5):850-855. doi:10.3171/jns.2005.102.5.0850

87. Oni MB, Wilde EA, Bigler ED, et al. Diffusion tensor imaging analysis of frontal lobes in pediatric traumatic brain injury. *J Child Neurol*. 2010;25(8):976-984. doi:10.1177/0883073809356034

88. Wood J, Chaparro A, Anstey K, et al. Simulated visual impairment leads to cognitive slowing in older adults. *Optom Vis Sci Off Publ Am Acad Optom*. 2010;87(12):1037-1043. doi:10.1097/OPX.0b013e3181fe64d7

89. Ruiz-Rizzo AL, Bublak P, Redel P, et al. Simultaneous object perception deficits are related to reduced visual processing speed in amnestic mild cognitive impairment. *Neurobiol Aging*. 2017;55:132-142. doi:10.1016/j.neurobiolaging.2017.03.029

90. Akhand O, Balcer LJ, Galetta SL. Assessment of vision in concussion. *Curr Opin Neurol*. 2019;32(1):68-74. doi:10.1097/WCO.0000000000000654

91. Daiber HF, Gnugnoli DM. Visual Acuity. In: *StatPearls*. StatPearls Publishing; 2025. Accessed June 26, 2025. http://www.ncbi.nlm.nih.gov/books/NBK563298/

92. Cronin TW, Porter ML. Visual System: Invertebrates. In: Squire LR, ed. *Encyclopedia of Neuroscience*. Academic Press; 2009:351-358. doi:10.1016/B978-008045046-9.00905-0

93. Kaur K, Gurnani B. Contrast Sensitivity. In: *StatPearls*. StatPearls Publishing; 2025. Accessed June 26, 2025. http://www.ncbi.nlm.nih.gov/books/NBK580542/

94. Spector RH. Visual Fields. In: Walker HK, Hall WD, Hurst JW, eds. *Clinical Methods: The History, Physical, and Laboratory Examinations*. 3rd ed. Butterworths; 1990. Accessed June 26, 2025. http://www.ncbi.nlm.nih.gov/books/NBK220/

95. Zebhauser PT, Vernet M, Unterburger E, Brem AK. Visuospatial Neglect - a Theory-Informed Overview of Current and Emerging Strategies and a Systematic Review on the Therapeutic Use of Non-invasive Brain Stimulation. *Neuropsychol Rev*. 2019;29(4):397-420. doi:10.1007/s11065-019-09417-4

96. Price TD, Khan R. Evolution of Visual Processing in the Human Retina. *Trends Ecol Evol*. 2017;32(11):810-813. doi:10.1016/j.tree.2017.09.001

97. Wilcox LM, Harris JM. Fundamentals of Stereopsis. In: Dartt DA, ed. *Encyclopedia of the Eye*. Academic Press; 2010:164-171. doi:10.1016/B978-0-12-374203-2.00237-2

98. Belliveau AP, Somani AN, Dossani RH. Pupillary Light Reflex. In: *StatPearls*. StatPearls Publishing; 2025. Accessed June 26, 2025. http://www.ncbi.nlm.nih.gov/books/NBK537180/

99. Pillai C, Gittinger JW. Vision Testing in the Evaluation of Concussion. *Semin Ophthalmol*. 2017;32(1):144-152. doi:10.1080/08820538.2016.1228412

100. Master CL, Scheiman M, Gallaway M, et al. Vision Diagnoses Are Common After Concussion in Adolescents. *Clin Pediatr (Phila)*. 2016;55(3):260-267. doi:10.1177/0009922815594367

101. Hunt AW, Mah K, Reed N, Engel L, Keightley M. Oculomotor-Based Vision Assessment in Mild Traumatic Brain Injury: A Systematic Review. *J Head Trauma Rehabil*. 2016;31(4):252-261. doi:10.1097/HTR.0000000000000174

102. Ventura RE, Balcer LJ, Galetta SL, Rucker JC. Ocular motor assessment in concussion: Current status and future directions. *J Neurol Sci*. 2016;361:79-86. doi:10.1016/j.jns.2015.12.010

103. Galetta KM, Brandes LE, Maki K, et al. The King-Devick test and sports-related concussion: study of a rapid visual screening tool in a collegiate cohort. *J Neurol Sci*. 2011;309(1-2):34-39. doi:10.1016/j.jns.2011.07.039

104. Fetter M. Vestibulo-ocular reflex. *Dev Ophthalmol*. 2007;40:35-51. doi:10.1159/000100348

105. Elbin RJ, Schatz P, Mohler S, Covassin T, Herrington J, Kontos AP. Establishing Test-Retest Reliability and Reliable Change for the King-Devick Test in High School Athletes. *Clin J Sport Med Off J Can Acad Sport Med*. 2021;31(5):e235-e239. doi:10.1097/JSM.0000000000000772

106. Thomas CE, Thomas SH, Bloom B. Vestibular/ocular motor screening (VOMS) score for identification of concussion in cases of non-severe head injury: A systematic review. *J Concussion*. 2023;7:20597002231160941. doi:10.1177/20597002231160941

107. Whelan BM, Gause EL, Ortega JD, et al. King-Devick testing and concussion recovery time in collegiate athletes. *J Sci Med Sport*. 2022;25(11):930-934. doi:10.1016/j.jsams.2022.08.012

108. Knell G, Caze T, Burkhart SO. Evaluation of the vestibular and ocular motor screening (VOMS) as a prognostic tool for protracted recovery following paediatric sports-related concussion. *BMJ Open Sport Exerc Med*. 2021;7(1):e000970. doi:10.1136/bmjsem-2020-000970

109. Liversedge SP, Gilchrist I, Everling S, eds. *The Oxford Handbook of Eye Movements*. Oxford University Press; 2011. doi:10.1093/oxfordhb/9780199539789.001.0001

110. Mele ML, Federici S. Gaze and eye-tracking solutions for psychological research. *Cogn Process*. 2012;13 Suppl 1:S261-265. doi:10.1007/s10339-012-0499-z

111. Ting WKC, Perez Velazquez JL, Cusimano MD. Eye movement measurement in diagnostic assessment of disorders of consciousness. *Front Neurol*. 2014;5:137. doi:10.3389/fneur.2014.00137

112. Albrecht TJ, Makwana Mehmel B, Rossi EA, Trbovich AM, Eagle SR, Kontos AP. Temporal Changes in Fixational Eye Movements After Concussion in Adolescents and Adults: Preliminary Findings. *J Neurotrauma*. 2024;41(1-2):199-208. doi:10.1089/neu.2023.0080

113. Urosevich TG, Boscarino JJ, Hoffman SN, et al. Visual Dysfunction and Associated Co-morbidities as Predictors of Mild Traumatic Brain Injury Seen Among Veterans in Non-VA Facilities: Implications for Clinical Practice. *Mil Med*. 2018;183(11-12):e564-e570. doi:10.1093/milmed/usy102

114. D'Silva LJ, Siengsukon CF, Devos H. Gaze stability in young adults with previous concussion history. *J Vestib Res Equilib Orientat*. 2020;30(4):259-266. doi:10.3233/VES-200706

115. Wooten BR, Hammond BR, Land RI, Snodderly DM. A practical method for measuring macular pigment optical density. *Invest Ophthalmol Vis Sci*. 1999;40(11):2481-2489.

116. Snodderly DM, Auran JD, Delori FC. The macular pigment. II. Spatial distribution in primate retinas. *Invest Ophthalmol Vis Sci*. 1984;25(6):674-685.

117. Hammond BR, Wooten BR, Snodderly DM. Individual variations in the spatial profile of human macular pigment. *J Opt Soc Am A Opt Image Sci Vis*. 1997;14(6):1187-1196. doi:10.1364/josaa.14.001187

118. Purves D, Augustine GJ, Fitzpatrick D, et al. Cones and Color Vision. In: *Neuroscience. 2nd Edition*. Sinauer Associates; 2001. Accessed June 26, 2025. https://www.ncbi.nlm.nih.gov/books/NBK11059/

119. Stringham JM, Hammond BR, Nolan JM, et al. The utility of using customized heterochromatic flicker photometry (cHFP) to measure macular pigment in patients with age-related macular degeneration. *Exp Eye Res*. 2008;87(5):445-453. doi:10.1016/j.exer.2008.08.005

120. Congdon N, O'Colmain B, Klaver CCW, et al. Causes and prevalence of visual impairment among adults in the United States. *Arch Ophthalmol Chic Ill 1960*. 2004;122(4):477-485. doi:10.1001/archopht.122.4.477

121. Rein DB, Wittenborn JS, Zhang X, et al. Forecasting age-related macular degeneration through the year 2050: the potential impact of new treatments. *Arch Ophthalmol Chic Ill 1960*. 2009;127(4):533-540. doi:10.1001/archophthalmol.2009.58

122. Seddon JM, Ajani UA, Sperduto RD, et al. Dietary carotenoids, vitamins A, C, and E, and advanced age-related macular degeneration. Eye Disease Case-Control Study Group. *JAMA*. 1994;272(18):1413-1420.

123. Antioxidant Status and Neovascular Age-Related Macular Degeneration. *Arch Ophthalmol*. 1993;111(1):104-109. doi:10.1001/archopht.1993.01090010108035

124. Snellen ELM, Verbeek ALM, Van Den Hoogen GWP, Cruysberg JRM, Hoyng CB. Neovascular age-related macular degeneration and its relationship to antioxidant intake. *Acta Ophthalmol Scand*. 2002;80(4):368-371. doi:10.1034/j.1600-0420.2002.800404.x

125. Feeney J, Finucane C, Savva GM, et al. Low macular pigment optical density is associated with lower cognitive performance in a large, population-based sample of older adults. *Neurobiol Aging*. 2013;34(11):2449-2456. doi:10.1016/j.neurobiolaging.2013.05.007

126. Renzi LM, Dengler MJ, Puente A, Miller LS, Hammond BR. Relationships between macular pigment optical density and cognitive function in unimpaired and mildly cognitively impaired older adults. *Neurobiol Aging*. 2014;35(7):1695-1699. doi:10.1016/j.neurobiolaging.2013.12.024

127. Craft NE, Haitema TB, Garnett KM, Fitch KA, Dorey CK. Carotenoid, tocopherol, and retinol concentrations in elderly human brain. *J Nutr Health Aging*. 2004;8(3):156-162.

128. Vishwanathan R, Neuringer M, Snodderly DM, Schalch W, Johnson EJ. Macular lutein and zeaxanthin are related to brain lutein and zeaxanthin in primates. *Nutr Neurosci*. 2013;16(1):21-29. doi:10.1179/1476830512Y.0000000024

129. Nolan JM, Loskutova E, Howard AN, et al. Macular pigment, visual function, and macular disease among subjects with Alzheimer's disease: an exploratory study. *J Alzheimers Dis JAD*. 2014;42(4):1191-1202. doi:10.3233/JAD-140507

130. Nolan JM, Loskutova E, Howard A, et al. The impact of supplemental macular carotenoids in Alzheimer's disease: a randomized clinical trial. *J Alzheimers Dis JAD*. 2015;44(4):1157-1169. doi:10.3233/JAD-142265

131. Hume C, Mitra B, Wright B, Kinsella GJ. Cognitive performance in older people after mild traumatic brain injury: Trauma effects and other risk factors. *J Int Neuropsychol Soc JINS*. 2023;29(7):651-661. doi:10.1017/S1355617722000674

132. Martini DN, Broglio SP. Long-term effects of sport concussion on cognitive and motor performance: A review. *Int J Psychophysiol Off J Int Organ Psychophysiol*. 2018;132(Pt A):25-30. doi:10.1016/j.ijpsycho.2017.09.019

133. Guskiewicz KM, Marshall SW, Bailes J, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. *Neurosurgery*. 2005;57(4):719-726; discussion 719-726. doi:10.1093/neurosurgery/57.4.719

134. Willer BS, Zivadinov R, Haider MN, Miecznikowski JC, Leddy JJ. A Preliminary Study of Early-Onset Dementia of Former Professional Football and Hockey Players. *J Head Trauma Rehabil*. 2018;33(5):E1-E8. doi:10.1097/HTR.0000000000000421

135. Gallo V, Motley K, Kemp SPT, et al. Concussion and long-term cognitive impairment among professional or elite sport-persons: a systematic review. *J Neurol Neurosurg Psychiatry*. 2020;91(5):455-468. doi:10.1136/jnnp-2019-321170

136. Hammond BR, Fletcher LM, Roos F, Wittwer J, Schalch W. A double-blind, placebo-controlled study on the effects of lutein and zeaxanthin on photostress recovery, glare disability, and chromatic contrast. *Invest Ophthalmol Vis Sci*. 2014;55(12):8583-8589. doi:10.1167/iovs.14-15573

137. Mainster MA, Turner PL. Glare's causes, consequences, and clinical challenges after a century of ophthalmic study. *Am J Ophthalmol*. 2012;153(4):587-593. doi:10.1016/j.ajo.2012.01.008

138. Renzi-Hammond LM, Buch J, Xu J, Hammond BR. Reduction of Glare Discomfort and Photostress Recovery Time Through the Use of a High-Energy Visible-Filtering Contact Lens. *Eye Contact Lens*. 2022;48(12):516-520. doi:10.1097/ICL.0000000000000935

139. Pierson C, Wienold ,Jan, and Bodart M. Review of Factors Influencing Discomfort Glare Perception from Daylight. *LEUKOS*. 2018;14(3):111-148. doi:10.1080/15502724.2018.1428617

140. Sutter P. Impact of Lutein-Based Food Supplement on Macular Pigment, Glare and Contrast Vision. *Open J Ophthalmol*. 2023;13(3):316-326. doi:10.4236/ojoph.2023.133030

141. Hammond BR, Fletcher LM, Elliott JG. Glare disability, photostress recovery, and chromatic contrast: relation to macular pigment and serum lutein and zeaxanthin. *Invest Ophthalmol Vis Sci*. 2013;54(1):476-481. doi:10.1167/iovs.12-10411

142. Stringham JM, Garcia PV, Smith PA, McLin LN, Foutch BK. Macular pigment and visual performance in glare: benefits for photostress recovery, disability glare, and visual discomfort. *Invest Ophthalmol Vis Sci*. 2011;52(10):7406-7415. doi:10.1167/iovs.10-6699

143. Hashemi H, Pakzad R, Yekta A, et al. Global and regional prevalence of age-related cataract: a comprehensive systematic review and meta-analysis. *Eye Lond Engl*. 2020;34(8):1357-1370. doi:10.1038/s41433-020-0806-3

144. Babizhayev MA. Glare disability and driving safety. *Ophthalmic Res*. 2003;35(1):19-25. doi:10.1159/000068199

145. Brandl C, Zimmermann ME, Herold JM, Helbig H, Stark KJ, Heid IM. Photostress Recovery Time as a Potential Predictive Biomarker for Age-Related Macular Degeneration. *Transl Vis Sci Technol*. 2023;12(2):15. doi:10.1167/tvst.12.2.15

146. Sandberg MA, Gaudio AR. Slow photostress recovery and disease severity in age-related macular degeneration. *Retina Phila Pa*. 1995;15(5):407-412. doi:10.1097/00006982-199515050-00006

147. Huang W, Yang Y, Luo MR. Discomfort glare caused by white LEDs having different spectral power distributions. *Light Res Technol*. 2018;50(6):921-936. doi:10.1177/1477153517704996

148. Shang YM, Wang GS, Sliney D, Yang CH, Lee LL. White Light–Emitting Diodes (LEDs) at Domestic Lighting Levels and Retinal Injury in a Rat Model. *Environ Health Perspect*. 2014;122(3):269-276. doi:10.1289/ehp.1307294

149. (PDF) Basics of Light Emitting diodes, Characterizations and Applications. ResearchGate. Accessed June 28, 2025. https://www.researchgate.net/publication/200071029_Basics_of_Light_Emitting_diodes_Characterizations_and_Applications

150. Burstein R, Noseda R, Fulton AB. Neurobiology of Photophobia. *J Neuro-Ophthalmol Off J North Am Neuro-Ophthalmol Soc*. 2019;39(1):94-102. doi:10.1097/WNO.0000000000000766

151. Bohnen N, Twijnstra A, Wijnen G, Jolles J. Tolerance for light and sound of patients with persistent post-concussional symptoms 6 months after mild head injury. *J Neurol*. 1991;238(8):443-446. doi:10.1007/BF00314651

152. Suter PS, Harvey LH, eds. Chapter Photophobia, Light, and Color in Acquired Brain Injury. In: *Vision Rehabilitation*. Routledge; 2011.

153. Theis J. Differential diagnosis and theories of pathophysiology of post-traumatic photophobia: A review. *NeuroRehabilitation*. 2022;50(3):309-319. doi:10.3233/NRE-228014

154. Echemendia RJ, Brett BL, Broglio S, et al. Introducing the Sport Concussion Assessment Tool 6 (SCAT6). *Br J Sports Med*. 2023;57(11):619-621. doi:10.1136/bjsports-2023-106849

155. de Boer JB, Schreuder DA. Glare as a Criterion for Quality in Street Lighting. *Trans Illum Eng Soc*. 1967;32(2_IEStrans):117-135. doi:10.1177/147715356703200205

156. Harriss AB, Abbott KC, Humphreys D, et al. Concussion Symptoms Predictive of Adolescent Sport-Related Concussion Injury. *Clin J Sport Med Off J Can Acad Sport Med*. 2020;30(5):e147-e149. doi:10.1097/JSM.0000000000000714

157. Pappas TN, Safranek RJ, Chen J. Perceptual Criteria for Image Quality Evaluation. In: *Handbook of Image and Video Processing*. Elsevier Inc.; 2005:939-959. doi:10.1016/B978-012119792-6/50118-2

158. Wooten BR, Renzi LM, Moore R, Hammond BR. A practical method of measuring the human temporal contrast sensitivity function. *Biomed Opt Express*. 2010;1(1):47-58. doi:10.1364/BOE.1.000047

159. Renzi LM, Hammond BR. The relation between the macular carotenoids, lutein and zeaxanthin, and temporal vision. *Ophthalmic Physiol Opt J Br Coll Ophthalmic Opt Optom*. 2010;30(4):351-357. doi:10.1111/j.1475-1313.2010.00720.x

160. Powell RR. Flicker fusion as a typological index of nervous system “reactivity.” *Percept Mot Skills*. 1983;57(3 Pt 1):701-702. doi:10.2466/pms.1983.57.3.701

161. Görtelmeyer R, Wiemann H. Retest Reliability and Construct Validity of Critical Flicker Fusion Frequency. *Pharmacopsychiatry*. 2008;15:24-28. doi:10.1055/s-2007-1019545

162. Grünberger J, Saletu B, Berner P, Stöhr H. CFF and Assessment of Pharmacodynamics: Role and Relationship to Psychometric, EEG and Pharmacokinetic Variables. *Pharmacopsychiatry*. 2008;15:29-35. doi:10.1055/s-2007-1019546

163. Granit R, Harper P. Comparative studies on the peripheral and central retina. *Am J Physiol-Leg Content*. 1930;95(1):211-228. doi:10.1152/ajplegacy.1930.95.1.211

164. Tyler CW, Hamer RD. Eccentricity and the Ferry-Porter law. *J Opt Soc Am A Opt Image Sci Vis*. 1993;10(9):2084-2087. doi:10.1364/josaa.10.002084

165. Fernandez-Alonso M, Innes W, Read JCA. Peripheral Flicker Fusion at High Luminance: Beyond the Ferry-Porter Law. *Vis Basel Switz*. 2023;7(1):26. doi:10.3390/vision7010026

166. Venkataraman AP, Lewis P, Unsbo P, Lundström L. Peripheral resolution and contrast sensitivity: Effects of stimulus drift. *Vision Res*. 2017;133:145-149. doi:10.1016/j.visres.2017.02.002

167. Mewborn C, Renzi LM, Hammond BR, Miller LS. Critical Flicker Fusion Predicts Executive Function in Younger and Older Adults. *Arch Clin Neuropsychol*. 2015;30(7):605-610. doi:10.1093/arclin/acv054

168. Neelam K, Nolan J, Chakravarthy U, Beatty S. Psychophysical function in age-related maculopathy. *Surv Ophthalmol*. 2009;54(2):167-210. doi:10.1016/j.survophthal.2008.12.003

169. Peters A. Structural changes that occur during normal aging of primate cerebral hemispheres. *Neurosci Biobehav Rev*. 2002;26(7):733-741. doi:10.1016/s0149-7634(02)00060-x

170. Tyler CW. Specific deficits of flicker sensitivity in glaucoma and ocular hypertension. *Invest Ophthalmol Vis Sci*. 1981;20(2):204-212.

171. Mayer MJ, Ward B, Klein R, Talcott JB, Dougherty RF, Glucs A. Flicker sensitivity and fundus appearance in pre-exudative age-related maculopathy. *Invest Ophthalmol Vis Sci*. 1994;35(3):1138-1149.

172. Phipps JA, Dang TM, Vingrys AJ, Guymer RH. Flicker perimetry losses in age-related macular degeneration. *Invest Ophthalmol Vis Sci*. 2004;45(9):3355-3360. doi:10.1167/iovs.04-0253

173. Curran S, Wattis JP. Critical flicker fusion threshold: a useful research tool in patients with Alzheimer's disease. *Hum Psychopharmacol Clin Exp*. 1998;13(5):337-355. doi:10.1002/(SICI)1099-1077(199807)13:5<337::AID-HUP7>3.0.CO;2-P

174. Curran S, Hindmarch I, Wattis JP, Shillingford C. Critical flicker fusion in normal elderly subjects; a cross-sectional community study. *Curr Psychol*. 1990;9(1):25-34. doi:10.1007/BF02686765

175. Chang TTL, Ciuffreda KJ, Kapoor N. Critical flicker frequency and related symptoms in mild traumatic brain injury. *Brain Inj*. 2007;21(10):1055-1062. doi:10.1080/02699050701591437

176. Schrupp LE, Ciuffreda KJ, Kapoor N. Foveal versus eccentric retinal critical flicker frequency in mild traumatic brain injury. *Optom St Louis Mo*. 2009;80(11):642-650. doi:10.1016/j.optm.2009.04.097

177. Benassi M, Frattini D, Garofalo S, Bolzani R, Pansell T. Visuo-motor integration, vision perception and attention in mTBI patients. Preliminary findings. *PloS One*. 2021;16(4):e0250598. doi:10.1371/journal.pone.0250598

178. Littleton AC, Register-Mihalik JK, Guskiewicz KM. Test-Retest Reliability of a Computerized Concussion Test: CNS Vital Signs. *Sports Health*. 2015;7(5):443-447. doi:10.1177/1941738115586997

179. Ataullah AHM, De Jesus O. Gait Disturbances. In: *StatPearls*. StatPearls Publishing; 2025. Accessed June 26, 2025. http://www.ncbi.nlm.nih.gov/books/NBK560610/

180. Manickam A, Gardiner MD. Gait assessment in general practice. *Aust J Gen Pract*. 2021;50(11):801-806. doi:10.31128/AJGP-12-20-5777

181. Scott D, McLaughlin P, Nicholson GC, et al. Changes in gait performance over several years are associated with recurrent falls status in community-dwelling older women at high risk of fracture. *Age Ageing*. 2015;44(2):287-293. doi:10.1093/ageing/afu169

182. Fernandez NB, Hars M, Trombetti A, Vuilleumier P. Age-related changes in attention control and their relationship with gait performance in older adults with high risk of falls. *NeuroImage*. 2019;189:551-559. doi:10.1016/j.neuroimage.2019.01.030

183. Ali A, Sundaraj K, Ahmad B, Ahamed N, Islam A. Gait disorder rehabilitation using vision and non-vision based sensors: a systematic review. *Bosn J Basic Med Sci*. 2012;12(3):193-202. doi:10.17305/bjbms.2012.2484

184. Fukuchi CA, Fukuchi RK, Duarte M. Effects of walking speed on gait biomechanics in healthy participants: a systematic review and meta-analysis. *Syst Rev*. 2019;8(1):153. doi:10.1186/s13643-019-1063-z

185. von der Recke F, Warmerdam E, Hansen C, Romijnders R, Maetzler W. Reduced Range of Gait Speed: A Parkinson's Disease-Specific Symptom? *J Park Dis*. 2023;13(2):197-202. doi:10.3233/JPD-223535

186. Peterson DS, Mancini M, Fino PC, Horak F, Smulders K. Speeding Up Gait in Parkinson's Disease. *J Park Dis*. 2020;10(1):245-253. doi:10.3233/JPD-191682

187. Büttner F, Howell DR, Ardern CL, et al. Concussed athletes walk slower than non-concussed athletes during cognitive-motor dual-task assessments but not during single-task assessments 2 months after sports concussion: a systematic review and meta-analysis using individual participant data. *Br J Sports Med*. 2020;54(2):94-101. doi:10.1136/bjsports-2018-100164

188. Oldham JR, Howell DR, Knight CA, Crenshaw JR, Buckley TA. Single-Task and Dual-Task Tandem Gait Performance Across Clinical Concussion Milestones in Collegiate Student-Athletes. *Clin J Sport Med Off J Can Acad Sport Med*. 2021;31(6):e392-e397. doi:10.1097/JSM.0000000000000836

189. Howell DR, Osternig LR, Chou LS. Return to activity after concussion affects dual-task gait balance control recovery. *Med Sci Sports Exerc*. 2015;47(4):673-680. doi:10.1249/MSS.0000000000000462

190. Roeing KL, Moon Y, Sosnoff JJ. Unplanned gait termination in individuals with multiple sclerosis. *Gait Posture*. 2017;53:168-172. doi:10.1016/j.gaitpost.2017.01.016

191. Sparrow WA, Tirosh O. Gait termination: a review of experimental methods and the effects of ageing and gait pathologies. *Gait Posture*. 2005;22(4):362-371. doi:10.1016/j.gaitpost.2004.11.005

192. Srivastava A, Ahmad OF, Pacia CP, Hallett M, Lungu C. The Relationship between Saccades and Locomotion. *J Mov Disord*. 2018;11(3):93-106. doi:10.14802/jmd.18018

193. Matthis JS, Barton SL, Fajen BR. The biomechanics of walking shape the use of visual information during locomotion over complex terrain. *J Vis*. 2015;15(3):10. doi:10.1167/15.3.10

194. Marigold DS, Patla AE. Gaze fixation patterns for negotiating complex ground terrain. *Neuroscience*. 2007;144(1):302-313. doi:10.1016/j.neuroscience.2006.09.006

195. Hollands MA, Marple-Horvat DE. Coordination of eye and leg movements during visually guided stepping. *J Mot Behav*. 2001;33(2):205-216. doi:10.1080/00222890109603151

196. Grasso R, Prévost P, Ivanenko YP, Berthoz A. Eye-head coordination for the steering of locomotion in humans: an anticipatory synergy. *Neurosci Lett*. 1998;253(2):115-118. doi:10.1016/s0304-3940(98)00625-9

197. Imai T, Moore ST, Raphan T, Cohen B. Interaction of the body, head, and eyes during walking and turning. *Exp Brain Res*. 2001;136(1):1-18. doi:10.1007/s002210000533

198. Yamada M, Higuchi T, Mori S, et al. Maladaptive turning and gaze behavior induces impaired stepping on multiple footfall targets during gait in older individuals who are at high risk of falling. *Arch Gerontol Geriatr*. 2012;54(2):e102-108. doi:10.1016/j.archger.2011.08.012

199. Patla AE, Vickers JN. How far ahead do we look when required to step on specific locations in the travel path during locomotion? *Exp Brain Res*. 2003;148(1):133-138. doi:10.1007/s00221-002-1246-y

200. Nemanich ST, Earhart GM. Freezing of gait is associated with increased saccade latency and variability in Parkinson's disease. *Clin Neurophysiol Off J Int Fed Clin Neurophysiol*. 2016;127(6):2394-2401. doi:10.1016/j.clinph.2016.03.017

201. Broglio SP, Kontos AP, Levin H, et al. National Institute of Neurological Disorders and Stroke and Department of Defense Sport-Related Concussion Common Data Elements Version 1.0 Recommendations. *J Neurotrauma*. 2018;35(23):2776-2783. doi:10.1089/neu.2018.5643

202. Schmidt JD, Register-Mihalik JK, Mihalik JP, Kerr ZY, Guskiewicz KM. Identifying Impairments after concussion: normative data versus individualized baselines. *Med Sci Sports Exerc*. 2012;44(9):1621-1628. doi:10.1249/MSS.0b013e318258a9fb

203. Lempke LB, Johnson RS, Schmidt JD, Lynall RC. Clinical versus Functional Reaction Time: Implications for Postconcussion Management. *Med Sci Sports Exerc*. 2020;52(8):1650-1657. doi:10.1249/MSS.0000000000002300

204. Lempke LB, Oh J, Johnson RS, Schmidt JD, Lynall RC. Single- Versus Dual-Task Functional Movement Paradigms: A Biomechanical Analysis. *J Sport Rehabil*. 2021;30(5):774-785. doi:10.1123/jsr.2020-0310

205. Chin EY, Nelson LD, Barr WB, McCrory P, McCrea MA. Reliability and Validity of the Sport Concussion Assessment Tool-3 (SCAT3) in High School and Collegiate Athletes. *Am J Sports Med*. 2016;44(9):2276-2285. doi:10.1177/0363546516648141

206. Hänninen T, Parkkari J, Howell DR, et al. Reliability of the Sport Concussion Assessment Tool 5 baseline testing: A 2-week test-retest study. *J Sci Med Sport*. 2021;24(2):129-134. doi:10.1016/j.jsams.2020.07.014

207. Lopes-Ferreira D, Neves H, Queiros A, Faria-Ribeiro M, Peixoto-de-Matos SC, González-Méijome JM. Ocular dominance and visual function testing. *BioMed Res Int*. 2013;2013:238943. doi:10.1155/2013/238943

208. Dengler M, Thorne A, Puente A, et al. Measuring the Temporal Contrast Sensitivity Function and Macular Pigment Optical Density in Older Adults with and Without Cognitive Impairment. *J Vis*. 2011;11(15):35. doi:10.1167/11.15.35

209. Bovier ER, Renzi LM, Hammond BR. A double-blind, placebo-controlled study on the effects of lutein and zeaxanthin on neural processing speed and efficiency. *PloS One*. 2014;9(9):e108178. doi:10.1371/journal.pone.0108178

210. Renzi-Hammond LM, Hammond BR. The effects of photochromic lenses on visual performance. *Clin Exp Optom*. 2016;99(6):568-574. doi:10.1111/cxo.12394

211. Measuring Spatial Parameters, Speed and Walk Ratio » ProtoKinetics. ProtoKinetics. December 21, 2018. Accessed July 6, 2023. https://www.protokinetics.com/measuring-spatiotemporal-parameters-spatial-parameters-speed-and-walk-ratio/

212. Gamaldo AA, An Y, Allaire JC, Kitner-Triolo MH, Zonderman AB. Variability in performance: Identifying early signs of future cognitive impairment. *Neuropsychology*. 2012;26(4):534-540. doi:10.1037/a0028686

213. Curran S, Wilson S, Musa S, Wattis J. Critical Flicker Fusion Threshold in patients with Alzheimer's disease and vascular dementia. *Int J Geriatr Psychiatry*. 2004;19(6):575-581. doi:10.1002/gps.1134

214. Functional Movement Deficits In Relation to Sport-Related Concussion.

215. Lennon MJ, Brooker H, Creese B, et al. Lifetime Traumatic Brain Injury and Cognitive Domain Deficits in Late Life: The PROTECT-TBI Cohort Study. *J Neurotrauma*. 2023;40(13-14):1423-1435. doi:10.1089/neu.2022.0360

216. Cole JH, Leech R, Sharp DJ. Prediction of brain age suggests accelerated atrophy after traumatic brain injury. *Ann Neurol*. 2015;77(4):571-581. doi:10.1002/ana.24367

217. Vincent AS, Roebuck-Spencer TM, Cernich A. Cognitive changes and dementia risk after traumatic brain injury: Implications for aging military personnel. *Alzheimers Dement*. 2014;10(3, Supplement):S174-S187. doi:10.1016/j.jalz.2014.04.006

218. Hammond BR, Fuld K, Snodderly DM. Iris color and macular pigment optical density. *Exp Eye Res*. 1996;62(3):293-297. doi:10.1006/exer.1996.0035

219. Hammond BR, Johnson EJ, Russell RM, et al. Dietary modification of human macular pigment density. *Invest Ophthalmol Vis Sci*. 1997;38(9):1795-1801.

220. Hammond BR, Wooten BR, Snodderly DM. Cigarette smoking and retinal carotenoids: implications for age-related macular degeneration. *Vision Res*. 1996;36(18):3003-3009. doi:10.1016/0042-6989(96)00008-9

221. Hammond BR, Curran-Celentano J, Judd S, et al. Sex differences in macular pigment optical density: relation to plasma carotenoid concentrations and dietary patterns. *Vision Res*. 1996;36(13):2001-2012. doi:10.1016/0042-6989(95)00290-1

222. Hammond BR Jr, Caruso–Avery M. Macular Pigment Optical Density in a Southwestern Sample. *Invest Ophthalmol Vis Sci*. 2000;41(6):1492-1497.

223. Howells O, Eperjesi F, Bartlett H. Macular Pigment Optical Density in Young Adults of South Asian Origin. *Invest Ophthalmol Vis Sci*. 2013;54(4):2711-2719. doi:10.1167/iovs.12-10957

224. Ramirez C, Lightfield K, Zuniga K. Macular Carotenoids and Cognitive Function in a Young Adult Population (FS05-07-19). *Curr Dev Nutr*. 2019;3:nzz052.FS05-07-19. doi:10.1093/cdn/nzz052.FS05-07-19

225. Bohnen N, Twijnstra A, Wijnen G, Jolles J. Tolerance for light and sound of patients with persistent post-concussional symptoms 6 months after mild head injury. *J Neurol*. 1991;238(8):443-446. doi:10.1007/BF00314651

226. Wade DT, King NS, Wenden FJ, Crawford S, Caldwell FE. Routine follow up after head injury: a second randomised controlled trial. *J Neurol Neurosurg Psychiatry*. 1998;65(2):177-183. doi:10.1136/jnnp.65.2.177

227. Shumski EJ, Barany DA, Schmidt JD, Lynall RC. The influence of concussion history on turning gait performance. *Gait Posture*. 2025;121:93-100. doi:10.1016/j.gaitpost.2025.04.026

228. Chong CD, Wang L, Wang K, Traub S, Li J. Homotopic region connectivity during concussion recovery: A longitudinal fMRI study. *PloS One*. 2019;14(10):e0221892. doi:10.1371/journal.pone.0221892

229. Moon Y, Sung J, An R, Hernandez ME, Sosnoff JJ. Gait variability in people with neurological disorders: A systematic review and meta-analysis. *Hum Mov Sci*. 2016;47:197-208. doi:10.1016/j.humov.2016.03.010

230. Lamoth CJ, van Deudekom FJ, van Campen JP, Appels BA, de Vries OJ, Pijnappels M. Gait stability and variability measures show effects of impaired cognition and dual tasking in frail people. *J Neuroengineering Rehabil*. 2011;8:2. doi:10.1186/1743-0003-8-2

231. Rajachandrakumar R, Mann J, Schinkel-Ivy A, Mansfield A. Exploring the relationship between stability and variability of the centre of mass and centre of pressure. *Gait Posture*. 2018;63:254-259. doi:10.1016/j.gaitpost.2018.05.008

232. Niechwiej-Szwedo E, Inness EL, Howe JA, Jaglal S, McIlroy WE, Verrier MC. Changes in gait variability during different challenges to mobility in patients with traumatic brain injury. *Gait Posture*. 2007;25(1):70-77. doi:10.1016/j.gaitpost.2006.01.002

233. Martini DN, Sabin MJ, DePesa SA, et al. The chronic effects of concussion on gait. *Arch Phys Med Rehabil*. 2011;92(4):585-589. doi:10.1016/j.apmr.2010.11.029

234. Nadkarni NK, Mawji E, McIlroy WE, Black SE. Spatial and temporal gait parameters in Alzheimer's disease and aging. *Gait Posture*. 2009;30(4):452-454. doi:10.1016/j.gaitpost.2009.07.003

235. Machado Maciel N, Ferreira Carvalho G, Ferreira Pinheiro C, van Emmerik R, Moraes R, Bevilaqua Grossi D. Gait control of migraine patients with increasing light and sound levels. *Gait Posture*. 2022;92:480-486. doi:10.1016/j.gaitpost.2021.04.003

236. Alexander MS, Lajoie K, Neima DR, Strath RA, Robinovitch SN, Marigold DS. Effects of age-related macular degeneration and ambient light on curb negotiation. *Optom Vis Sci Off Publ Am Acad Optom*. 2014;91(8):975-989. doi:10.1097/OPX.0000000000000286

APPENDIX A

MICHIGAN TBI IDENTIFICATION METHOD

Michigan TBI Identification Method

CONCUSSION HISTORY									
	Mechanism	The concussion was diagnosed or undiagnosed	Approximate date of injury (mm/yyyy)	Age at time of injury	Did you lose consciousness (i.e. knocked out/blacked out)?	How long were you unconsciousness (seconds)?	Did/do you have difficulty remembering things before or after the injury?	How many minutes do you not remember (min)	How many days did you experience symptoms related to the injury?
Injury #1	☐ Blow to head or neck ☐ Motor vehicle crash - pedestrian/ bicyclist ☐ Motor vehicle crash occupant ☐ Sport / recreation ☐ Fall ☐ Fight or being hit ☐ Explosion / Blast ☐ Other	☐ Diagnosed ☐ Undiagnosed	_____/_____		☐Yes ☐No	_____(sec) ☐ Unknown	☐Yes ☐No	_____(min) ☐ Unknown	_____(days) ☐ Unknown
Injury #2	☐ Blow to head or neck ☐ Motor vehicle crash - pedestrian/ bicyclist ☐ Motor vehicle crash occupant ☐ Sport / recreation ☐ Fall ☐ Fight or being hit ☐ Explosion / Blast ☐ Other	☐ Diagnosed ☐ Undiagnosed	_____/_____		☐Yes ☐No	_____(sec) ☐ Unknown	☐Yes ☐No	_____(min) ☐ Unknown	_____(days) ☐ Unknown

** Additional Injury lines can be added as needed

Mechanism Prompts:

Blow to the head or neck: Have you ever been hospitalized or treated in an emergency room following an injury to your head or neck? Think about any childhood injuries you remember or were told about.

Car / vehicle accident: Have you ever injured your head or neck in a car accident or from some other moving vehicle accident (e.g. motorcycle, ATV)?

Sport / recreation: Have you ever been hit in the head or fallen on your head while participating in an organized sport or recreational activity, including on the playground?

Fall: Have you ever injured from falling?

Fight or being hit: Have you ever injured your head or neck in a fight, from being hit by someone/something, or from being shaken violently?

Explosion / Blast: Have you ever been nearby when an explosion or a blast occurred? If you served in the military, think about any combat- or training-related incidents.

* adapted from the Ohio State University TBI Identification Method (Corrigan, J.D., Bogner, J.A. (2007). Initial reliability and validity of the OSU TBI Identification Method. *J Head Trauma Rehabil, 22*(6):318-329,

SRC Version 1.0

APPENDIX B

SCAT6 SYMPTOM INVENTORY

Symptom	Rating
Headaches	0 1 2 3 4 5 6
Pressure in head	0 1 2 3 4 5 6
Neck pain	0 1 2 3 4 5 6
Nausea or vomiting	0 1 2 3 4 5 6
Dizziness	0 1 2 3 4 5 6
Blurred vision	0 1 2 3 4 5 6
Balance problems	0 1 2 3 4 5 6
Sensitivity to light	0 1 2 3 4 5 6
Sensitivity to noise	0 1 2 3 4 5 6
Feeling slowed down	0 1 2 3 4 5 6
Feeling like "in a fog"	0 1 2 3 4 5 6
"Don't feel right"	0 1 2 3 4 5 6
Difficulty concentrating	0 1 2 3 4 5 6
Difficulty remembering	0 1 2 3 4 5 6
Fatigue or low energy	0 1 2 3 4 5 6
Confusion	0 1 2 3 4 5 6
Drowsiness	0 1 2 3 4 5 6
More emotional	0 1 2 3 4 5 6
Irritability	0 1 2 3 4 5 6
Sadness	0 1 2 3 4 5 6
Nervous or anxious	0 1 2 3 4 5 6
Trouble falling asleep (if applicable)	0 1 2 3 4 5 6

Do your symptoms get worse with physical activity?	Y	N
Do your symptoms get worse with mental activity?	Y	N

If 100% is feeling perfectly normal, what percent of normal do you feel?

If not 100%, why?

PLEASE HAND THE FORM BACK TO THE EXAMINER

Once the athlete has completed answering all symptom items, it may be useful for the clinician to revisit items that were endorsed positively to gather more detail about each symptom.

Total number of symptoms: _____ **of 22**

Symptom severity score: _____ **of 132**