Concussion Management for Primary Care

Evidence Based Answers to Cases and Questions

Deepak S. Patel
Editor

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Preface

Concussions are increasing in incidence each year. Some of this may be fueled by increased media exposure and overall public and provider awareness. Regardless of the cause, medical providers are called upon to accurately diagnose and manage these concussions. Adding to this pressure is the fact that each state in the United States has a law on management of concussions in children. These factors strengthen the need for primary care providers to be well-versed in the evaluation and management of them.

Although evidence in medicine changes and expands rapidly, this text aims to provide the latest update. Early guidelines were based on expert opinion. As new evidence has become available, guidelines have changed. This evidence has challenged several facets of concussion care and even refuted prior long-held beliefs. Despite this evidence, there are still areas of concussion care that long for better evidence and may be challenging to implement logistically.

This text provides primary care physicians and clinicians with an evidence-based yet practical approach to diagnosing and treating concussions in children and adults. Each of the authors was carefully selected based on their expertise and experience in treating concussions. In addition to being experts, they also serve as educators on concussions.

The book begins with a general overview of concussions. It then goes on to identify risks, signs, and symptoms of concussions. Next, physicians and providers learn when and how to perform appropriate physical exams for suspected concussions. The following chapters focus on finding the correct type of testing to perform in suspected concussions. The testing options addressed include diagnostic, neurocognitive, and imaging. Return-to-learn and return-to-play recommendations are then discussed to ensure that providers are able to properly educate patients about concussions. The book concludes by explaining post-concussion syndrome and identifying methods to prevent concussions and complications in the future. Each chapter presents a specific real-world patient case and addresses common questions asked by patients and family. Within each chapter are additional follow-up questions in the subsections that help the reader to answer questions. Tables and a summary of key concepts have been included for readers looking for rapid specifics.

Presented from the unique perspective of primary care physicians who also specialize in sports medicine and concussions, *Concussion Management for Primary Care* is a first-of-its-kind book that serves as a valuable resource for primary care providers, sports medicine physicians, and any other clinician treating patients suffering from a possible concussion.

Chicago, IL, USA Deepak S. Patel, MD, FAAFP, FACSM, CIC

Acknowledgments

This textbook is a culmination of effort and sacrifice from several individuals. I wanted to graciously acknowledge a few of the many involved.

The most important is the love, support, and sacrifices from my family (my wonderful wife and daughter). They are the reason I'm able to pursue an extensive project such as this. Their love and understanding helped me through this process. To my family I say, your understanding and support keep me going every day, especially on a long commitment such as this. You push me to be my best and love me just the same when I'm not. Amazingly, you both have understood the importance of concussions and how my work might contribute to it. You are able to envision the benefit to others as you sacrificed and supported me in such a challenging project. Just know that there were many times when I was working on this project that I wished I was able to spend more time with you. I love you and am eternally grateful.

I also dedicate this book to my parents. Although both have passed, they are still always in my thoughts. I'm grateful for the person and especially the physician they helped me become. They taught me the value of hard work and persistence. They instilled the value of always learning and growing. Additionally, they helped me to love being a physician by caring for the sick and injured but also to realize the importance of treating patients like family. This book is a reflection of those values.

Although I've taken on several different teaching roles, I am grateful for all of them. Whether I was teaching fellow physicians, residents, nurse practitioners, physician assistants, nurses, athletic trainers, patients, or patients' family members, I appreciated that opportunity. Each of them has taught me something and pushed me to expand my knowledge. Along with their teachings, I want to thank all of them for their trust. Because they trusted me to care for so many patients with concussions, I've been fortunate to claim a vast array of experience in concussion care.

I want to thank each of the authors who have invested numerous hours of their personal time to lend their expertise to this work and help advance the state of education on concussions. Each of them is busy in their practice and teaching roles. I know this book was an extra responsibility that reduced their personal or family time. Thank you for your dedication and effort!

Each of us has unique experiences that shaped our interests and career choices. I had several of these, including sports injuries that pushed me toward a career in sports medicine. I firmly believe these can give us empathy for our patients. There is one experience specific to concussions that I wanted to share. In this and many regards, I wish to also thank my seventh- and eighth-grade football coach and neighbor, Jerry Nichols. He probably was unaware of all the life lessons he taught his athletes and that a concussion to a future sports medicine physician would be one of them. During a practice, he chose to run a trick play, where the guard who was approximately a 6-foot, 200-pound player was handed the football. I knew as soon as I saw him with a ball in hand and a full head of steam, heading directly at me, that this tackle was going to hurt. I was significantly undersized (as I was for all my sports participations). I did my best to aim low (I was short, so that part was easy) and wrap the legs. As I attempted to do this, I felt an explosion hit my head. That explosion as his knee striking my helmet. My head hurt for several hours, but the effect thankfully was very short-lived. It was a painful lesson but a valuable one. That was my first concussion, which serves as a reminder of the cause and empathy for many of my patients' suffering.

Finally, I want to thank you, the reader. Both keeping up with advances in medicine and caring for individuals with concussions are a challenge. You've taken the initiative to seek out more education on the subject. Your patients will benefit greatly from your decision and dedication. As a fellow clinician, I especially admire that. As our knowledge of concussions evolve and change, I encourage you to continue to advance your approach and care. I hope you find information in this book that will aid you in caring for concussions.

Contents

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Chapter 1
Introduction to Concussion

Deepak S. Patel

Clinical Case

An 18-year-old female falls backward and hits her head on the basketball court. Her parents are suspicious when the athletic trainer tells them she has a concussion. They ask, "How can she have a concussion when her head looks normal, she is behaving normally, and no imaging tests were performed?"

The term *concussion* is frequently used by the media, patients, and medical personnel. Concussions are a subset of *mild traumatic brain injury* (MTBI). Although these terms are often used interchangeably, many times the term MTBI is used in the literature. The challenge for providers, patients, and those around concussed individuals is that concussions often lack outward physical findings.

What is the definition of concussion and how does it happen?

Concussion is defined by multiple international organizations as a traumatic injury to the brain that leads to a temporary impairment of brain function [1–5]. Although headache is the most common symptom, additional neurologic impairments can be demonstrated in varying severity through a number of signs and symptoms. Since some of these signs and symptoms can also occur in major traumatic brain injuries,

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it's important for providers to understand the differences. Most guidelines also differentiate major traumatic brain injuries from concussion by noting that concussions lack structural abnormalities on traditional imaging (CT or MRI). Commonly, the injury is not specific to direct head trauma but can even occur with indirect head trauma. This indirect trauma can occur with violent or rapid head movement forward, backward, or even rotationally. Such movement leads to the brain striking the inside of the cranium or being rapidly shaken.

Many of these indirect brain traumas are not specific to sports or falls. Concussions occur in all age groups in various settings. Recently, more attention has been placed on sports-related concussions. Early guidelines focused on contact sports and management of athletes with concussion. However, nonathlete concussions can result from a variety of mechanisms and often have their own associated challenges. Concussions can occur in different settings such as motor vehicle accidents, on the playground, at home, at work, and in military combat. Currently, the population most well-studied on traumatic brain injuries is the military.

Regardless of the exact injury mechanism of the concussion, this type of injury leads to microscopic axonal damage. Furthermore, the stretching of axons leads to cellular and metabolic changes, which lead to alterations in ion concentrations and neurotransmitter release. The body attempts to stabilize this damage with intracellular glucose uptake to balance sodium and potassium fluxes. This castcade likely leads to many of the signs and symptoms we observe in concussed individuals.

As our knowledge of concussions evolves, we are faced with the challenge to define how and why concussions occur. For example, why do multiple players on the team exposed to the same brain impacts vary to such a degree in the development of concussions? Various products have been proposed to measure or dissipate head injury forces, but the specific concussion threshold for each individual varies.

Which providers are best to evaluate and manage concussions?

Given the frequency with which concussions occur and the diverse populations are affected, several types of providers are expected to evaluate and manage concussions. Concussion care often falls within the expertise of neurology, neurosurgery, physical medicine and rehabilitation, and primary care sports medicine. None of these however, specialize in concussions, and they may vary in both their experience and interest. Specialties such as primary care sports medicine and neurology usually have the greatest experience in concussion care and have traditionally led in training physicians in concussion care. Since many concussions occur in a sporting environment, the sports medicine providers claimed greater experience in concussion care. Pediatrics, family medicine, and internal medicine providers care for concussions among their primary care patient population and often are the first providers to see patients after a concussion. In an acute setting, emergency and urgent care providers regularly evaluate patients for head injuries and especially determine if a concussion or a more severe, major traumatic brain injury has occurred.

Providers in other fields have risen to meet the need of concussion care by adding specialized training and certification. Examples include neuropsychologists, neuro-optometrists, and physical therapists. Physical therapists are routinely involved in the rehabilitation needs of concussed individuals and therefore are often a valuable resource to the primary care provider.

What is the role of Athletic Trainers and Nurses in evaluation and treatment of concussions?

Because they are present at athletic practices and games, athletic trainers are often the first providers to evaluate sports-related concussions. They are responsible for identifying when an injured athlete should seek immediate care or should be observed on the sideline. They also have direct knowledge of athletes' usual behaviors, emotional states, personalities, and tendencies, which can be invaluable when identifying changes after an injury. They provide an additional communication link between providers and coaching staff. Their close relationship with the players and team enhances an athlete's reincorporation into team practice and games. They are also well-versed in return-to-play protocols (see "Return to Play" Chap. 10) and can supervise the exercise progression to ensure a safe return.

Nurses are also a valuable resource in concussion treatment. In a school setting, concussed students are often sent to the nurse when new or increased symptoms arise. In many school districts, nurses are responsible for administering the return-to-learn protocol and conveying any academic and physical restrictions for students. When concussed students' symptoms increase, teachers will send students to the nurse to be evaluated. In some schools, nurses will monitor student symptoms daily and convert their office into a rest location when needed.

Are there laws related to concussions?

Every state in the United States now has a law related to concussion care directed at the school-age concussion population. These laws vary from state to state, and the authors of this text strongly encourage each reader to consult your specific state concussion law. The laws are meant to ensure appropriate evaluation and management of concussions. The priority is to ensure that anyone suspected of having suffered a concussion is not allowed to return prematurely and be thereby placed in danger of severe complications such as second-impact syndrome (see Chap. 11). Many of these laws require school districts to establish policies that address school personnel who may identify or interact with concussed students. Several states in the United States now require education for several school personnel such as administrators, coaches, and teachers.

Due to the impairments in daily functioning that occur, concussions can affect others around the concussed individual. It is important for family members and caregivers to be aware of the impairments and challenges that are involved with having a concussion. As you will read later in this book, communication with all supervisors or administrators is required. This can include parents, employers, teachers, school nurses, athletic trainers, and coaches.

The authors of this textbook believe that best concussion care involves communication and comprehensive attention from everyone surrounding the concussed individual.

Are there any good guidelines available for concussion care?

Over the years, several organizations have published and revised guidelines on concussions. At the time of the writing of this textbook, guidelines have been available from the American Academy of Neurology, American Medical Society for Sports Medicine, Centers for Disease Control and Prevention, Parachute of Canada, Ontario Neurotrauma Foundation, and International Conference on Concussion in Sport. Of note, some are more specific to certain populations, and that should be considered when reviewing those guidelines.

Where can patients find reliable information about concussions?

Although our understanding and management of concussions continue to evolve, providers and patients may need to search for additional resources. The resources may provide patients and parents further information on concussion background, warning signs, general restrictions, and recovery process. Although generic and not specific to every concussed individual, this can be a helpful addition to information and care conveyed by medical providers.

These resources may also be required by providers to consider updated information or specific resources for patients and the community. Several websites contain excellent information about concussions. Some suggestions at the time of publication of this text include:

- International Conference on Concussion in Sport: http://bjsm.bmj.com/content/bjsports/early/2017/04/26/bjsports-2017-097508CRT5.full.pdf
- American Academy of Family Physicians (AAFP): https://familydoctor.org/condition/concussion/
- Centers for Disease Control and Prevention (CDC): https://www.cdc.gov/headsup/index.html

- American Academy of Pediatrics (AAP): https://www.healthychildren.org/English/health-issues/injuries-emergencies/sports-injuries/Pages/Concussions.aspx
- Ontario Neurotrauma Foundation: https://onf.org/knowledge-mobilization/acquired-brain-injury/patient-resources/
- Parachute Canada: http://www.parachutecanada.org/home/print/2346/

Key Points

- Concussion is a type of mild traumatic brain injury (MTBI) that leads to temporary neurologic impairment.
- Several different medical specialties are involved in concussion care.
- Consult appropriate state laws related to concussions especially regarding children and students.
- Several different guidelines and websites may add additional information for both patients and providers.

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Chapter 2
Incidence and Risk Factors for Concussions

Deepak S. Patel and Natasha Ahmed

Clinical Case

A mother and her 11-year-old son are present for his well-child visit. She is concerned about her son's interest in playing football due to the risks of concussions. She wants to know what other sports are safer with a lower risk of concussions.

In general, contact sports tend to have more concussions. Concussions can still occur in lower-impact sports and non-contact sports such as swimming, cross-country, and running, but risk of sustaining concussions in these sports is much lower.

Question: Are concussions becoming more common?

Over three million sports-related concussions are estimated to occur annually within US emergency departments. In 2001–2010 adolescents made up greater than 50% of sports-related concussions (SRC) seen in the ED [1–4]. Mild TBI-related emergency department visit rates have increased from 2006 to 2012 across all age groups and gender with an increase of average annual percentage rate (AAPR) of 7.0% [3–5]. The highest rates are seen in both male and female between the ages of 0 and

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4 years and 15 and 24 years and elderly females over the age of 65 years [5]. The incidence of SRC in high school athletes has doubled over a 7-year study period, while the exposure rate has been relatively stable [4]. Concussion is a widespread form of trauma experienced among all populations regardless of age, race, gender, or mechanism of injury. Strong epidemiological framework is important for understanding the nature of mTBIs. As we will see later in this section, age, sex, and mechanism of injury-related patterns are relevant when comparing incidence rates.

At a global level, it is estimated that 100–300 per 100,000 people seek medical attention for mTBI; however this is thought to be an underestimation considering the number of unreported injuries [3, 4, 6]. For example, data collected from hospitals and emergency rooms do not account for injuries seen in outpatient settings and organized sports settings with athletic trainers. The World Health Organization (WHO) estimates a true incidence exceeding 600 per 100,000 people each year [7]. A large-scale population-based study in New Zealand attempted to correct this underreporting using a database which included all healthcare providers regardless of setting (i.e., hospitals, outpatient clinics, schools, sports clubs, etc.) and calculated a rate of 790 per 100,000, which is expected to be closer to a true incidence [7]. On the other hand, the increases in trends are thought to be multifactorial. It can be hypothesized that parents, players, coaches, and the general public are more aware of the seriousness and complications of these injuries leading to increased sensitivity in reporting these injuries [8, 9].

Children and Adolescent Age Groups

Question: Are concussions more common in older or younger children?

All age groups are at risk for experiencing mild traumatic brain injury (mTBI). Concussion experiences were found to vary by demographic as there are significant misconceptions regarding the causes, symptoms, recovery course, and risks. Children under the ages of 18 years made up an average of 283,000 ED visits from 2001 to 2016 with highest rates between 10 and 17 years old [11].

Question: Are concussions in children more common from sports or accidents?

In 2013, it was estimated that 2.3 million SRC occur annually including visits outside hospital settings [10]. According to the most recent CDC analysis, using the National Electronic Injury Surveillance System-All Injury Program (NEISS-AIP), it was found that across a 7-year study period, approximately two million children

sustained head injuries due to sports- or recreational-related activities [11]. Majority of concussions in the child and adolescents under the age of 18 are caused by sports- and recreational-related activities such as injuries occurring in football, soccer, basketball, wresting, bicycling, roller-skating, and playground activities [11, 12]. The developing brain of a child has a different physiological makeup when compared to the further developed adult and is thought to affect the threshold for head injury [2, 9, 11, 12]. Concussion rates have been found to increase with age, predominantly in older children 10–14 years old and adolescents aged 15–17 years with contact sports being twice as common when compared to non-contact sports and four times when compared to recreational activities (Fig. 2.1) [11]. High school football is the most well-studied cohort for adolescents, whereas data for the younger age group is not as well studied.

Increasing trends in SRC nearly doubled in age groups 8–13 years and in high school age groups 14–19 years from 2001 to 2012 [1, 12]. Surveillance databases have been used to conduct large-scale studies in SRC among high school and collegiate athletes. The term used to identify the rate of concussions is athlete exposure (AE) and represents one athlete participating in each practice or competition [4]. According to the National High School Sports-Related Injury Surveillance System, high school athlete rates continued to uptrend with a rate of 0.23–51/1000 AE over a 7-year academic period [13].

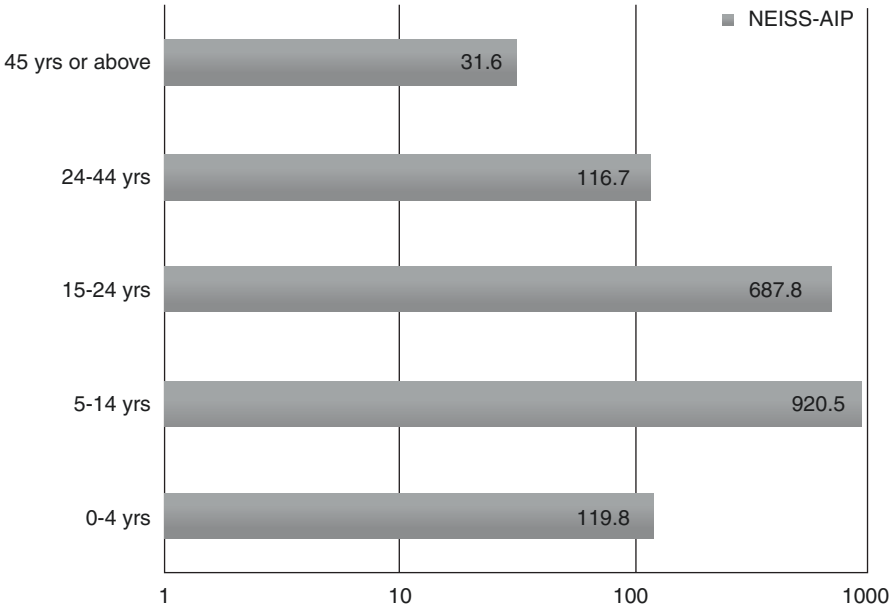

Fig. 2.1 Sports- and recreational-related mTBI rates by age in the emergency department NEISS-AIP (2012)

High School

Question: High school football is regularly identified to be associated with concussions, but are there other sports that also have a high incidence?

Nearly eight million high school students participate in organized sports, making this a large portion of the athletes at risk for SRC [9, 14]. National survey of high school students identified that over 50% of students between the ages of 15 and 19 years have played on at least one sports team with greater than 64% seen in males [12]. AE rates have almost doubled from 2.15 to 5.03 over a 10-year study period using High School Reporting Information Online (RIO). This data is consistent with the National Athletic Treatment, Injury and Outcomes (NATION) surveillance program which collects data from 147 high schools within 26 states over a 3-year academic period [9, 14]. Football has historically been shown to have the highest number of SRC (Fig. 2.2) [9, 13, 14]. It is important to also recognize the rate of head injuries sustained in less popular sports often played as club sports, most notably rugby [15]. Although rugby is a popular sport with a long history in Europe, the popularity of rugby has increased within the USA over the last 15 years.

Like football, this sport involves full contact. However, being a newer organized sport within the USA, many players often lack experience resulting in increased risk of injuries, particularly concussions [15]. The demands of daily practice, physical contact, and repetitive stress increase the risk of injury [14].

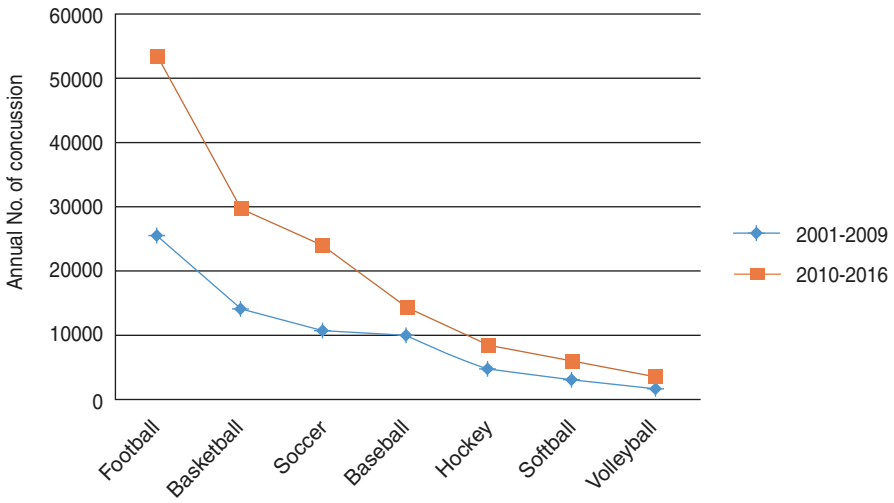

Fig. 2.2 Average annual estimates for mTBI seen in ED related to sports and recreational activities. National Electronic Injury Surveillance System, United States

Question: What are risk factors associated with higher concussion rates in athletes?

According to an evidence-based systematic review of risk factors, there is a high level of certainty extracted from level I studies that SRC during competition have a higher risk of injury as compared to practice sessions [1, 2, 7]. However, there are a few studies which contradict those findings and found a higher rate of concussions occurring during practices [16].

Additionally, there is a well-established understanding of increased risk with individuals having a history of previous concussions [1, 2]. Marshall et al. [17] found that athletes with a history of one concussion in the last 2 years had over two times the rate of concussion, and those with two or more prior concussions had up to five times higher rate when compared to individuals with no history of prior concussion. Furthermore, high school students who played on multiple sports teams were found to have positive correlation with the number of reported concussions. Athletes that played on one to three sports teams increased their concussion incidence from 16.7% to 22.9% to 30.5%, respectively [6].

Question: Although males and females may play similar sports, do their concussion rates vary?

Multiple large-scale studies conclude males are 1.4 times more likely to sustain head injuries in non-sport-related settings as compared to females. However, in gender-comparable sports such as soccer, females have the highest rate of injury, notably concussions (Fig. 2.3) [2, 7, 9, 13]. O'Connor et al. describe a 56% increase

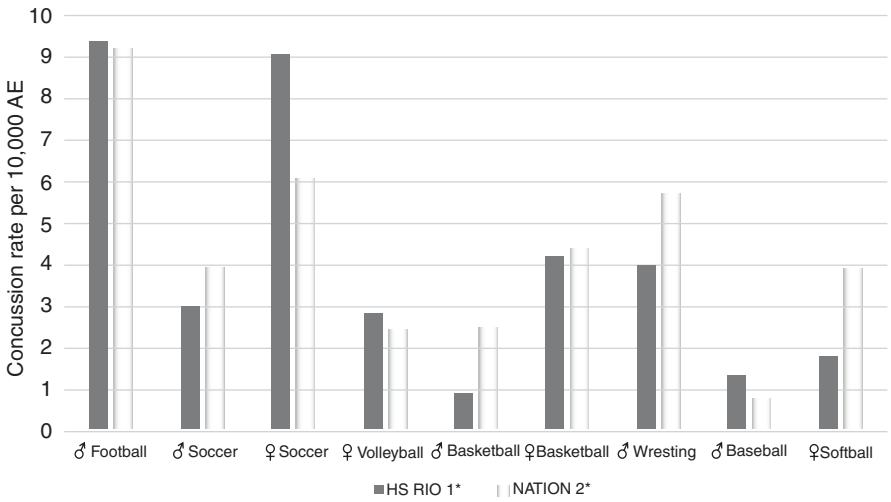

Fig. 2.3 Comparison of gender- and sport-specific differences in average concussion rates in high school from RIO vs. NATION

in injury rate in females when compared to male student athletes participating in the same sport [14]. Furthermore, Abraham et al. [1] observed that adult females were found to have a 1.5–2.5-fold greater risk in various levels of soccer and basketball. Soccer has a lack of protective gear, emphasis on in-game contact, and encouraging headers during game play compared to other contact-related sports. Females are thought to have decreased neck strength and increased head-to-ball ratio which may predispose them to have increased head acceleration during impact [14]. Similar to adolescents, adult female soccer athletes were found to have increase concussion rates per AE when compared to male counterparts: 50% vs. 27% [18].

Collegiate Sports

Question: Are rates of SRC less common in college sports?

According to the National Collegiate Athletic Association (NCAA), 460,000 athletes participate in collegiate sports annually, making this a population at high risk for developing SRC [19]. The NCAA Injury Surveillance Program (ISP) uses a convenience sample of varsity teams from 25 championship sports and found 6.2% of injuries were made up for SRC [18]. In 2010, the NCAA executive committee adopted a concussion policy that mandated strict guidelines to isolate and manage concussions. Highest rates were seen in men's wrestling, men and women's ice hockey, and men's football. Furthermore, there has been a steady increase in men's football, women's ice hockey, and men's lacrosse [18]. Men's football was found to have the highest concussion rates per team with 9.0% of injuries accounting for recurrent concussions [16–18, 21]. During the 5-year academic period from 2009 to 2014, SRC rates were found to be double during competition [21]. This study focuses on NCAA sports but does not account for popular full-contact club sports such as rugby. A prospective study comparing collegiate rugby to football found a significant rate difference in concussions with 2.5 in rugby versus 1.0 in football and a significantly higher incidence occurring during games over practice sessions [20].

Professional Sports

Question: We hear about professional players with concussions in the public media, but what is their incidence?

The National Football League (NFL) has been in the spotlight for almost two decades for its criticism regarding player safety after sustaining traumatic brain injuries, leading to changes in management policies and penalties for aggressive play [22]. 2010–2014 NFL surveillance data studies, including off-season games, concluded incidence rate of concussions to be higher than prior reports with a rate of 0.658 per NFL game as compared to 0.38 in 2002–2007 [23, 24]. Recent data

suggests no difference in injuries due to helmet-body collisions versus helmet-helmet and helmet-surface injuries [22]. A concussion is reported almost every other game in the NFL, but this number is likely to be higher due to underreporting [23, 24]. This increase in incidence set precedence to deliver safety standards; however, one can argue that the observed incidence seen may be attributed to the frequent changes in management policies [23].

Military

Question: Do military personnel have higher rates of concussions compared to civilians?

Since the conflicts in Afghanistan and Iraq, traumatic brain injuries have gained attention among military service members between 18 and 24 years old. Over 80% of traumatic brain injuries are classified as mTBI in deployed and non-deployed settings [25]. There has been an estimated 283% increase concussion rate in military service members from 2000 to 2011 [7]. The Department of Defense (DoD) has implemented its own surveillance data and policies to address these injuries [7]. Although many of the principles overlap with the demographics already discussed above, there are several unique factors that are specific to this group such as mechanism of injury, comorbidities, and military culture. The US military identifies with a culture that has its own set of values such as selfless service and mission focus over personal needs. This may affect the way these injuries are identified and managed when compared to the civilian population. These values also predispose them to recurrent injury. Non-deployed service members are also engaged in physically demanding training activities, motor vehicle crashes, sporting, and recreational events [7]. Psychiatric comorbidities, such as PTSD (post-traumatic stress disorder) commonly seen in service members, include symptoms such as sleep, mood, and cognitive disturbances can make it difficult to extrapolate true concussion symptoms [7, 24]. Data for deployed men is difficult to attain and is not included in DoD reports. According to survey data, most common cause of any traumatic brain injury is contributed to blast injuries, motor vehicle accidents, and falls. Given the context in which blast injuries occur, it is difficult to quickly identify and manage concussions from blast injuries in an acute setting, and further research is warranted.

Comorbid Neuropsychological Conditions

Question: What medical conditions predispose or complicate the management of concussions?

The prevalence of attention deficit hyperactivity disorder (ADHD) is associated with increased cognitive deficits and reporting of concussion-type symptoms [7, 26]. The American Medical Society for Sports Medicine consensus statement

classifies ADHD as a "concussion modifier," such that it is associated with increased cognitive dysfunction and prolongs recovery [2, 26]. Similarly, small-scale studies have identified challenges of diagnosis and management of concussions with comorbid conditions such as anxiety, depression, and PTSD [27, 28]. Symptoms of these conditions such as fatigue, concentration difficulty, memory impairment, and sleep disturbances often overlap with concussion symptoms. Optimizing these underlying conditions is important in the recovery of mTBI as these untreated conditions also are thought to contribute to persistence of concussion like symptoms. Alcohol is not included in any large-scale studies and has predominantly been studied in the setting of severe traumatic brain injuries rather than in mTBI. However, in the ED about one third to a quarter of patients with recreational head injuries had an elevated blood alcohol concentration [12]. One study attributed an inverse relationship with young male adults of lower socioeconomic status with alcohol use and physical assault leading to injuries including concussions [5]. The effects of alcohol including impaired motor control, lack of inhibition, and heightened risk-taking behaviors predispose predominantly younger males to violence and falls [28].

Miscellaneous or Elderly (Non-sports, Nonmilitary)

Question: What are other common causes of concussions excluding sports or military involvement?

Men older than 25 years old have a high incidence of recreational injuries. Bicycling and all-terrain vehicle (ATV) accidents contributed to most male concussion injuries in this age group [12]. Similarly, women have been affected by bicycling collisions followed by horseback riding instead of ATV [12]. As the age demographic increases greater than 45 years old, reports demonstrate decrease in sports and exercise. The third highest annual rate of mTBI is comprised of females older than 65 years old with comorbidities such as diabetes, polypharmacy, and age-related changes (i.e., hearing, vision, strength, balance, cognitive function). Increasing fall risk predisposes this age group to sustain head injuries with longer recovery times [7]. Therefore, it is reasonable to conclude elderly population to have a high incidence of mTBI. However, given the multiple comorbidities mentioned above, those conditions may take precedence during acute management.

Risk Factors (Table 2.1)

Table 2.1 Concussion risk factors

High	Moderate	Low
Prior concussion	Student male athletes (except female soccer)	Genetics *APOE gene* *Neuropsychological conditions, e.g., PTSD, anxiety, depression, ADHD*
Competition vs. practice	Body checking	Behavior *Aggressive* *Risk-taking* *Substance use*
Female soccer athletes	Participating in multiple sports	Mechanism of injury *Equipment contact* *Player to player* *Player to ball* *Ground contact*
Contact sport athletes	Older children (10–18)	Playing position *Offensive* *Defensive* *Goalie*
Military members	Males in non-sports-related setting (except for females >65 years old)	Location of head injury in conjunction with underlying neuropsychological conditions *Temporal* *Parietal*
		Match period toward end of game

Key Points

- The incidence of concussions has increased across all level of sports and recreational activities, among all genders and age groups.
- Underreporting of concussions is common.
- Adolescent age groups have higher incidence of contact SRC as compared to non-contact sports.
- Prior concussion and playing on multiple sports teams have a strong association with sustaining repeat concussions.
- Higher risk of injury during competition versus practice.
- Male football players have the highest rate of concussions.
- In gender-comparable sports, female soccer players have the highest rate of concussions.
- Military have multiple risk factors due to increased physical demands, motor vehicle crashes, and exposure to blast injuries.
- Comorbid neuropsychological conditions such as depression, anxiety, PTSD, ADHD, or being under the influence of alcohol may complicate the diagnosis and recovery of concussions.

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Chapter 3
Signs and Symptoms of Concussion

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Clinical Case

A 12-year-old female presents to the clinic with her parents for a well-child visit. She plays soccer for the local team. Her parents are concerned that she might develop a concussion through heading a soccer ball or by colliding with another girl. They also want to know what signs and symptoms they should be aware of to suspect that their child might have sustained a concussion.

The diagnosis of concussion can be elusive and continues to be a challenge for primary care providers. Despite the public interest and plethora of research on the pathophysiology, diagnosis, and management of concussions, there remains a lot of mystery. Every year, the number of cases of concussion and the detrimental consequences of misdiagnoses continue to rise. Primary care providers are often the first to assess a concussed patient and are therefore well-positioned in the healthcare system to diagnose, treat, and prevent concussions and its sequelae. It is imperative for primary care providers to be aware of the constellation of signs and symptoms that present with a concussion.

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Question: What are the six domains of signs and symptoms of concussions?

The American Medical Society for Sports Medicine's most recent position on concussions divides the signs and symptoms of concussions into six domains [1]. Many of the symptoms of concussion are general and can overlap domains. The six domains with their symptoms are listed in Table 3.1.

Most signs and symptoms are gathered from retrospective recall studies, with a few studies using video evidence. Examples of video signs of concussion includes lying motionless for greater than 2 minutes, motor incoordination, impact seizures, tonic posturing, and blank or vacant look [2]. It is important to note that the signs and symptoms of concussions are often very general and easy to overlook. Also, many signs and symptoms can overlap into multiple categories, or a symptom in one category can compound symptoms of another category, as seen in Fig. 3.1. It is also important to clearly define symptoms that patients report. Many words to describe the symptoms are generalizable and can mean several different things, such as diz-

Table 3.1 Domains and symptoms

Domains	Examples
Headache-migraine	Headache, nausea, photosensitivity, neck pain, phonophobia, photophobia
Cognitive	Confusion, disorientation, inattention, mental fogginess, slurred speech, vacant stare
Anxiety-mood	Agitation, flat effect, depression, labile mood, anxiety
Ocular	Blurry vision, double vision, eye fatigue
Fatigue	Tiredness, decreased arousal, somnolence, difficulty sleeping
Vestibular	Imbalance, abnormal visual motion sensitivity

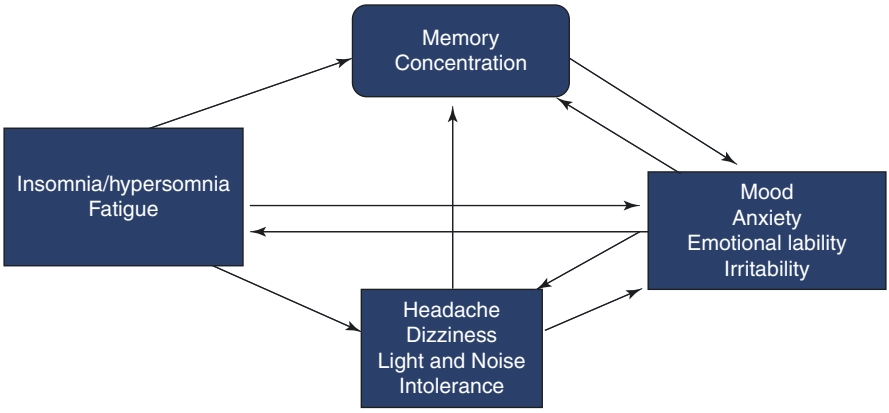

Fig. 3.1 Symptoms in one category of a concussion can interact to exacerbate symptoms in a different category. (Reprinted from Brent and Max [52] with permission from Springer Nature)

ziness or confusion. Knowing exactly what patients are experiencing is necessary as it could help guide which domain the problem fits into and the rehabilitation required.

Another important aspect when assessing concussion symptoms is to compare to a patient's baseline. A patient may have a history of headaches, attention/concentration issues, or emotional lability that could be inappropriately diagnosed as a concussive symptom. This is often an obstacle for medical providers who may be meeting the patient for the first time, so it is helpful to reach out to athletic trainers, friends, or family members to get a better understanding of the athlete's personality. For example, a survey found that almost 25% of healthy, non-concussed children reported difficulty with concentration [3]. It has also been found that athletes with a concussion history reported greater physical, emotional, and sleep-related symptoms at baseline than those with no history of concussion, with a greater endorsement in physical and sleep symptoms [4]. A wrongfully diagnosed concussion-related symptom could delay a patient's return to school and sports.

The physical symptoms associated with concussions are sometimes the most obvious to the patient and family members, especially immediately after the concussion occurs [5]. Patients and family members are more likely to recognize and report many of the physical symptoms to providers, as compared to complaints in other domains [5]. The physical signs and symptoms include headache, neck pain, drowsiness, dizziness, sensitivity to light and noise, visual and hearing changes, loss of consciousness, nausea, and vomiting [5, 6].

Signs and symptoms evolve over the subacute time period after a concussion. Furthermore, new symptoms may emerge during recovery process when the athlete starts exerting themselves. The signs and symptoms that patients experience after sustaining a concussion often start in the headache-migraine and fatigue domain and progress to the anxiety-mood domain [5, 7]. Signs and symptoms in the cognitive domain can be present both early in the clinical course and months later after the trauma [5, 8].

Question: Is loss of consciousness required for a concussion?

Loss of consciousness is another often misunderstood aspect of concussions. A common public understanding is that a concussion occurred when there was a loss of consciousness. In actuality, loss of consciousness is not a requirement for diagnosing a concussion and is a somewhat rare occurrence. Only about 10% of concussions will be accompanied by a loss of consciousness [9, 10]. When loss of consciousness occurs, even for a brief period, suspicion for a concussion is extremely high, and that player should be removed from the event for evaluation at an outside facility [11]. Loss of consciousness that is prolonged (greater than 1 minute) should raise concerns of a more serious clinical presentation that requires more immediate medical attention and deviation from standard concussion protocols [12]. However, despite the severity of its presentation, there is no evidence to suggest that loss of consciousness alone is associated with more severe brain injury or worse recovery

outcomes [11]. It is also essential to remember that the primary evaluation after a big hit or trauma is not to look for a concussion, but more serious, possibly life threatening conditions that need to be ruled out before worrying about concussions.

Question: What types of headache are common in patients that sustained a concussion?

Headaches are the most common complaint after a patient sustains a concussion [13, 14]. There is no specific type of headache classically associated with concussions. Headaches from concussion are usually classified as the common subtypes, with migraine and tension type predominating [15]. They are typically classified as secondary headaches. Headache prevalence, duration, and severity are greater in those with mild head injury compared with those with more severe trauma [16]. A significant number of patients have preexisting headaches, but studies conflict as to whether this is a risk factor for post-traumatic headaches [17]. Although new headaches can develop following a concussion, exacerbation of preexisting problems is likely more common [18]. If the patient has a history of migraine, then the current presentation should be compared closely with his or her typical migraine presentation. Post-traumatic headaches begin within 7 days after head trauma or after regaining consciousness. Acute headaches begin within 7 days of head trauma and may continue for up to 3 months after the injury [19].

For a small proportion of patients, the headache can persist beyond 2 months after the inciting trauma. These would be classified as chronic post-traumatic headaches and can often be severely debilitating [20]. Recurrent headaches can also be an important marker for physical or cognitive overexertion during recovery.

Neck pain is usually included in the headache domain as it can sometimes be the primary cause for headaches or a separate problem. It is the second most reported physical symptom of concussions [6]. Neck pain often warrants further questioning and examination to rule out other neurologic changes such as weakness, hyporeflexia, or sensory abnormalities. These findings may require a more aggressive workup with imaging and diagnostic testing [6]. It is important to keep a broad differential before concluding that a patient's neck pain is solely due to a concussion.

Question: What are cognitive symptoms of concussion?

Symptoms from the cognitive domain include decreasing mental clarity, mental fogginess, difficulty concentrating, memory issues, fatigue, and confusion. Evidence demonstrates that even a single concussion can disrupt the neurological mechanisms underlying cognition [21]. Cognitive symptoms can make it difficult to assess patients for their entire constellation of symptoms. Patients are often unable to describe how they feel – this can be problematic, particularly for children and young adolescents. Concentration difficulties and memory impairment are the most com-

mon complaints in the cognitive domain [14]. Although memory impairment may be a common complaint among patients with post-concussive symptoms, the problem may involve impaired attention and concentration, forgetfulness, distractibility, slowness of mental processing and reaction time, impaired mental flexibility, working and prospective memory, and memory retrieval [22].

In the acute setting, patients can also develop post-traumatic amnesia, which affects their ability to learn new information during this period. The post-traumatic amnesia is defined as the length of time from the injury until continuous memory resumes [23]. Retrograde and anterograde post-traumatic amnesia may diminish over time with events closest to the time of trauma being most difficult to recall. Deficits of attention, working memory and recall, and executive function will likely persist long after the post-traumatic amnesia has resolved [24].

These cognitive dysfunctions can place a heavy burden on the patient and disrupt their transition back to school or work [5]. Even after the patient becomes asymptomatic, returning to their previous activities should be handled gradually. There are more cognitive challenges during this time period, which require significantly more effort and can unmask deficits that the patient previously appeared to have overcome. Memory impairment following concussion appears to involve working memory [25]. Working memory refers to the ability to temporarily store and manipulate information that is relevant for complex cognitive processes during a task [25]. The disruption of working memory impairs executive functioning and slows processing speed [25], which may present as slow responses to question or delayed recall. Fortunately, the recovery of these deficits following concussion occurs within the first weeks to months, and recovery progresses without specific intervention [26].

Question: What are the emotional or mood symptoms of concussion?

Emotional symptoms include depression, disinhibition, irritability, mood lability, depression, frustration, and restlessness. These symptoms are not typically present immediately after the injury but arise within the first week following a concussion. Self-reported changes in mood, emotions, and behavior are typically short-lived. Symptoms peak approximately 7 days after the acquisition of a concussion and resolve over a period of weeks to a month [5, 27]. It is important to talk with family members, coaches, and teammates who know the individual well, as they may notice emotional lability that is out of character. In a retrospective review of 174 concussed athletes, 50% of the sample reported at least one new emotional symptom on the Post-Concussion Symptom Scale, with the mean sub-score of emotional symptoms being 4 [27]. Additionally, a prospective cohort study found that approximately 25% of pediatric patients that were evaluated at a children's emergency department for a concussion experienced depression, irritability, or restlessness [8]. Figure 3.2 shows the neuropsychiatric symptoms that are commonly tested for in the post-concussive clinical assessment tool.

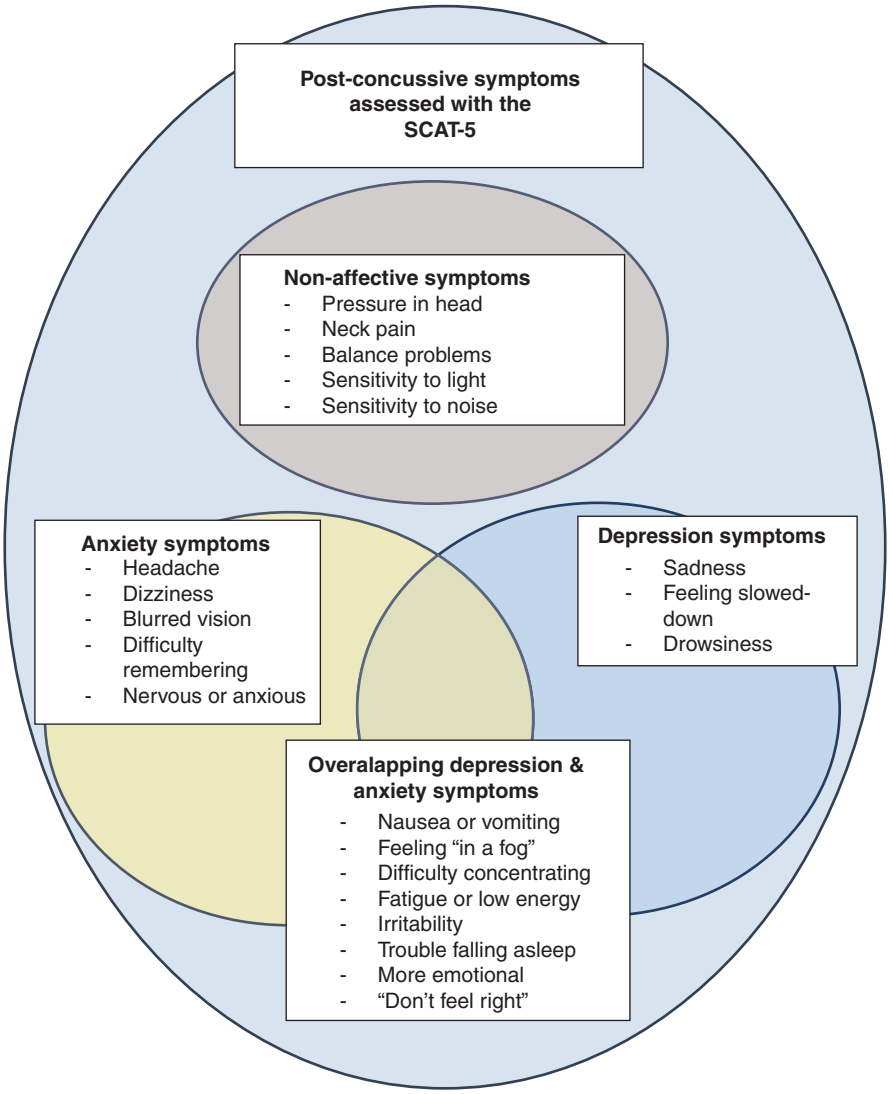

Fig. 3.2 Overlap of frequent post-concussive symptoms assessed by the Sport Concussion Assessment Tool-5. Core symptoms of mood (depression) and anxiety disorders are assessed by various psychiatric outcome measures, including the Hamilton Anxiety Scale, Beck Depression Inventory II, Generalized Anxiety Disorder-7, Brief Symptom Inventory-18, Patient Health Questionnaire-9, and the Diagnostic and Statistical Manual of Mental Disorders’ (5th revision) criteria for generalized anxiety disorder and major depression. (Reprinted from Mcallister and Wall [53] with permission from Elsevier)

It is well-established in literature that women report higher rates of concussions compared to men [12, 28, 29]. Furthermore, females report more emotional symptoms compared to their male counterparts [3]. It has been speculated that this difference in reported symptoms of concussion was secondary to hormonal differences between males and females [30]. However, it is important to understand what baseline symptoms the patient experiences as concussion-like symptoms could have been present prior to the concussion. A systematic review and meta-analysis by Brown et al. in 2015 demonstrated that females report more concussion-like symptoms and emotional symptoms prior to sustaining a concussion, but the differences of these symptoms do not persist post-concussion when compared to men [31].

Female sex, along with a prior personal or family history of a psychiatry disorder, seems to be a risk factor to develop post-concussive psychiatric disorders [24]. Some important factors that affect the risk of developing a post-concussive psychiatric disorder are the severity of concussion injury, socioeconomic status, preinjury adaptive and intellectual functioning, and psychosocial stress [27, 32]. Personality changes secondary to the concussions is the most common post-concussive psychiatric disorders, accounting for as high as 40% of pediatric patients suffering from concussions [32, 33]. Other psychiatric disorders clinically diagnosed include depressive disorders, generalized anxiety disorder, panic disorder, post-traumatic stress disorder, obsessive-compulsive disorder, somatoform disorder, attention deficit disorder, oppositional-defiant disorder and conduct disorder.

History of concussions is another possible risk factor for developing emotional symptoms secondary to a concussion. However, it is unclear if a history of concussion predisposes people to experience more emotional symptoms or if it instead results in an increased awareness of symptoms, which, in turn, leads to increase reporting. What is known is that there exists a link between sustaining multiple concussions and long-term psychiatric and neurological disorders [34, 35]. However, their association with the development of a post-concussive psychiatric disorder is undetermined [3, 27].

Concussions can cause emotional symptoms, but the diagnosis of concussion in itself can possibly affect the emotional well-being of the patient and bring out emotional symptoms. The stresses that accompany a concussion diagnosis, such as the symptoms themselves or missed school, athletics, or work, may bring out underlying mood disorders that the patient was never diagnosed with or knew they had. This may not be directly related to the concussion and therefore should not be considered a symptom that could delay rehabilitation. Nonetheless, it is imperative to understand what symptoms patients experience at baseline and be aware of the social and emotional stress that can occur with a diagnosis of a concussion.

Question: What are the ocular symptoms of concussion?

Visual deficits are more frequently being recognized and diagnosed in patients with concussions. Visual symptoms that are associated with concussions include blurry vision, double vision, and difficulty reading. Symptoms stem from a change in

coordinated eye tracking, which manifests as having trouble with reading, writing, and computer-related work in school. Master et al. (2015) found that 69% of adolescent participants had at least one visual diagnosis after a concussion and 22% had two visual diagnoses [36]. An observational study on adolescent motorcyclists found that 31% of participants reported blurry or double vision after sustaining a concussion [37].

Photophobia or light sensitivity is another common complaint in patients that sustained a concussion that can be classified in the ocular or headache-migraine domain. A third of adolescent motorcyclists previously mentioned were found to have photophobia, and one study found as high as 80% of their participants were suffering from photophobia after a concussion [14, 37]. Photophobia is usually most intense in the beginning of the clinical course and becomes less burdensome the weeks after the initial injury [5]. It is important to note that light sensitivity can be present at baseline, especially in patients that suffer from chronic migraines; therefore it is important to clarify how their current symptoms compared to their symptoms at baseline.

Identification of concussion-related symptoms is crucial as defects in vision can impair everyday tasks, such as driving a car, attending school, or performing work tasks, in both adults and children. Visual symptoms have high morbidity in the adolescent population because of the importance of vision to perform schoolwork activities and the increasing integration of technology within the education system. Increased screen time already causes significant visual symptoms such as eye strain and dry eyes [38], which can further be exacerbated by concussion-related visual symptoms. For this reason, improvements in clinical tools for the diagnosis and rehabilitation of visual deficits are needed.

Question: What other domain of concussion is closely related to fatigue?

Fatigue is often seen after a concussion and, like many other symptoms, can be multifactorial. Fatigue may be a primary entity, as opposed to being secondary to poor sleep. Patients often describe the fatigue as tiredness unrelated to physical or mental exertion, and sometimes it does not improve with rest. Concussion-related fatigue is usually less profound during the day than at morning or night [39]. Many athletes feel excessively sleepy and may report taking unusual daytime naps during the first week following the injury. However, napping can be disruptive to recovery and does not improve daytime sleepiness. It adversely affects the ability to fall asleep and causes less slow-wave sleep, which is viewed as most restorative [40]. Keeping a more regular day-to-day pattern of rest and activity relates to lower fatigue and depression scores [41].

After the acute phase of the injury, insomnia occurs in about 30% of people, and circadian rhythm shifts in approximately 36% [42]. Early morning awakenings are

also common and may, in turn, fuel daytime fatigue. Females more commonly report significant sleep disturbance after sustaining a single concussion, while males may not report sleep disturbance until cumulative concussions have been sustained [43]. Reassuringly, it has been shown that 20 years post-injury, children who sustained a TBI had similar rates of being "poor sleepers" and objective outcomes across all sleep parameters when compared to a control group [44]. Fatigue can last for months after other symptoms have improved [5].

There is also a strong correlation between sleep and symptoms in other domains, especially mood. A common neurobiological mechanism may explain why concussed individuals with abnormal sleep patterns are more susceptible to depression [45]. In addition, worsening of sleep as a result of a concussion in those with prior history of psychiatric issues could account for increased relapses of mood symptoms and treatment resistance [46]. Several hypotheses have been proposed about the etiology of post-concussive sleep regulation and psychiatric illness including neurotransmitter imbalance, dysregulation of the hypothalamic- pituitary-adrenal axis, and genetic polymorphism, but none have been verified [47].

Question: What are the vestibular symptoms of concussion?

Vestibular symptoms are usually associated with balance alterations and dizziness, which are commonly used interchangeably. The two functional aspects of the vestibular system are the vestibulo-spinal component (which helps to regulate postural stability) and the vestibulo-ocular component (which integrates vision and movement of the head) [48]. Dizziness is a blanket term used by patients, which encompasses lightheadedness, blurry vision, weakness, and vertigo or unsteadiness, so it is important to get a clear description as it could relate to different pathologies. Dizziness following concussion occurs in 50–80% of injuries [48]. Commonly tested vestibular motor symptoms include the ability to hold a tandem stance or balance on one leg. Many vestibular symptoms are evident only when provoked by stimuli or movements. Therefore, vestibular symptoms may be exacerbated by busy environments and quick head movements, such as those involved in dynamic sports or at school. Visual motion sensitivity is a phenomenon that can occur after a vestibular and/or ocular injury that refers to a heightened awareness of normal visual stimuli due to an inability to centrally integrate visual and vestibular information [48].

Balance issues can stem from the central nervous system (brain injury) or the peripheral nervous system (head injury) dysfunction [49]. Concussions can lead to balance issues from either system, but usually occur from peripheral nervous system dysfunction. Some argue that vestibular symptoms may be due to a separate injury to the neck or vestibular system that occurs along with the trauma that caused the concussion. Leddy et al. found that symptom reports from patients with delayed recovery after head injury, including cognitive symptoms, do not discriminate between those with a physiologic post-concussive disorder and those with a cervical/vestibular injury [50].

Dizziness usually occurs within 72 hours after the injury, and it usually resolves within 4 to 30 days [46]. However, dizziness can last for 6 months or longer and has been implicated as a risk factor for a prolonged recovery [49]. Reported dizziness at the time of injury can have up to a six-fold increased rate in protracted recovery (more than 21 days) and abnormalities on vestibular-ocular reflex (VOR) testing or tandem-gait performance resulted in an average recovery time of 59 days versus just 6 days for those without vestibular abnormalities [51].

Key Points

- Concussion signs and symptoms can be broken down into 6 major categories: (1) vestibular; (2) ocular; (3) cognitive; (4) headache/migraine; (5) fatigue; and (6) anxiety/mood.
- Migraine and tension-type headaches are the most common types of concussion headaches.
- Cognitive symptoms of concussion are decreasing mental clarity, mental fogginess, difficulty concentrating, memory issues, fatigue, and confusion.
- Emotional symptoms of concussion are depression, disinhibition, irritability, mood lability, depression, frustration, and restlessness.
- Ocular symptoms of concussion are blurry vision, double vision, and difficulty reading.
- Patients that suffer from fatigue after sustaining a concussion are likely to have emotional symptoms.
- Vestibular symptoms of concussion are balance alterations, lightheadedness, and dizziness.

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Chapter 4
Physical Examination

Carrie A. Jaworski and Priya Nagarajan

Clinical Case

You are about to see an adolescent patient of yours who sustained a sports-related concussion at her recent soccer game. The medical student rotating with you asks what should be included in the physical examination and if there is evidence to support this approach.

Patients with a sports-related concussion often present to the primary care office for their initial evaluation. It is imperative for the practitioner to emphasize the aspects of the physical examination that may necessitate additional evaluation and/or testing. It is also essential that a practitioner pays special attention to the areas of the physical examination that, in the concussed patient, lend themselves to an intervention that can facilitate recovery.

While much is published on the common elements utilized in the history component of concussions, little evidence exists on the validity of the approach to the physical examination of a concussed patient. In addition, many tools and applications are constantly being developed and advertised to clinicians for use in concussion evaluations that do not necessarily have strong evidence to support their use. Recent publications stress the importance of developing a more standardized

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approach to examining a concussion patient in order to allow for better research and ultimately better care of such patients.

At present, the main areas of exam that are supported in the literature include mental status testing, cervical spine evaluation, balance testing, and vestibulo-ocular examination, with orthostatic testing being appropriate in select patients (Table 4.1) [1]. The inclusion of manual muscle, cranial nerve, and reflex testing is not specific for concussion, but these tests are important to evaluate for the other etiologies in the differential of a concussion [1]. In this chapter, each of these components will be explained and evaluated in terms of the available evidence to support their use. In addition, recommendations on an approach to the overall examination will be provided.

Table 4.1 Rationale for physical exam tests in concussion

Physical exam test	Utility
Orthostatic vital signs	Recommended particularly for patients presenting with dizziness Signifies autonomic dysregulation and can help distinguish other causes from a concussion Change in heart rate is *not* consistent with concussion
Mental status	Refers to changes in cognition, memory, concentration, and mood SAC and SCAT 5 are reasonable tools for cognitive assessment Patients with a prior history of mental illness are at higher risk for post-concussive syndrome
Cranial nerve exam	Important to rule out CN injuries when evaluating for concussions as they can often be missed CN I is most commonly injured followed by CN VII, II, and IV
Vestibular-ocular evaluation	Often presents with signs of dizziness, vertigo, and balance difficulty which can persist if untreated Dynamic visual acuity should be considered for testing of vestibular-ocular dysfunction Ocular dysfunction presents with impaired near point convergence, accommodation, and oculomotor tracking
Saccades and smooth pursuits	Highly sensitive and specific to help determine prognosis with concussions Abnormal tests associated with higher risk of post-concussive syndrome Objective findings of impaired tracking useful to rule out malingering
Nystagmus	Horizontal nystagmus is a common finding in patients with head trauma Vertical nystagmus is pathologic and may indicate a brainstem lesion
Accommodation and convergence	Normal near point convergence is 6–10 cm Ocular muscle dysfunction contributes to difficulties with convergence which can affect reading speed and comprehension Helpful in determining classroom readiness
Balance assessment	Balance Error Score System (BESS) is frequently utilized despite limited reliability Romberg test is low yield Tandem gait and coordination tests may be more meaningful findings in balance assessment of concussed patient
Musculoskeletal exam	Focus on head and neck for trauma and range of motion Consider TMJ, thoracic evaluation based on situation
Neurologic assessment	MMT and DTRs do not have strong evidence for aiding diagnosis of concussion but are useful in ruling out other pathology

Question: Why are vital signs relevant in concussed patients?

Changes in vital signs may be a helpful indicator in establishing the diagnosis of a concussion as well as when evaluating persistent concussion-like symptoms. Head injuries are known to be associated with a disruption of the autonomic system which can affect one's vital signs and symptoms both with position and with activity [2]. Autonomic dysregulation following a concussion has commonly been associated with exercise intolerance that provokes an increase in symptoms [3] in addition to contributing to impaired vestibular function that leads to problems with dizziness and coordination [4]. In the case of exercise intolerance, submaximal exercise testing can be performed on a bike or treadmill to evaluate a patient for their heart rate (HR) and blood pressure (BP) response to exercise [5]. One of the most recognized, and studied, protocols is known as the Buffalo Concussion Treadmill Test [5–7]. Many providers trained in concussion care will utilize this testing in their evaluation and management of concussions, so it is worthwhile to enlist the assistance of such providers in one's community (if available). It has been demonstrated that when a patient is still suffering from autonomic dysfunction related to a concussion, they will be unable to tolerate submaximal exercise without experiencing a significant elevation in their concussion symptoms [6, 7]. Once the autonomic system has recovered from the concussion, the patient will again be able to tolerate exercise [6, 7]. At this point, if the patient still complains of symptoms outside of exercise, one must search for other contributing factors that may be causing their symptoms.

In those patients presenting with more vestibular-related symptoms, or perhaps those with less obvious reasons to have a concussion, it is essential to evaluate for other potential causes of these presenting symptoms. For instance, athletes who are dehydrated can also present with complaints of headaches and dizziness. Evaluation for orthostatic hypotension through the use of orthostatic vital signs (OVS) can help to distinguish between these entities quite easily as concussions do not result in orthostatic hypotension.

Orthostatic hypotension refers to a 20-mmHg or greater drop in systolic BP (SBP), a 10-mmHg or greater drop in diastolic BP (DBP), or a 30 beats per minute increase in pulse from supine to standing positions with associated signs and symptoms of hypoperfusion [8]. The recommended way to test for OVS is with the orthostatic stress test (OST). A patient will have their BP and HR assessed after lying supine for 2 minutes and then will have BP and HR reassessed while standing at 1 and 2 minutes after that. Symptomatic changes are not necessary to make this diagnosis [8]. Of note, it is important that the test be done without substitution of seated for supine position, as orthostatic hypotension can be missed in two-thirds of these cases [9]. In the setting of concussions, an associated change in HR is not commonly detected. An increase in the heart rate may indicate signs of hypovolemia rather than autonomic dysfunction alone, which could point to an alternate etiology rather than being neurologic in nature [10]. Postural orthostatic tachycardia syndrome (POTS) is another manifestation of autonomic dysregulation in which the HR increases without an associated drop in BP. POTS patients can often have many similar symptoms to a patient with post-concussion syndrome (PCS), so again this is where understanding these subtleties in examination findings can be helpful.

The Defense Centers of Excellence of Psychological Health and Traumatic Brain Injury recommends that patients with head injuries should be evaluated with OVS, especially when presenting with symptoms of dizziness [11]. OVS has good specificity of 75–90%, but sensitivity can be as low as 21%. Heads up tilt table testing (HUT) can be considered as an alternative test in the setting of a negative OVS where one still strongly suspects an autonomic component to the patient's symptoms [12]. HUT is not typically a first-line approach in the evaluation of concussion, but it is often used in more recalcitrant cases or when overlap between conditions makes best management approaches more difficult to determine.

Question: How does a patient's mood and cognitive function impact those with concussion?

Mental status changes in concussion can refer to cognitive abilities, memory changes, and concentration in addition to emotional affect and liability. Currently available tools, such as the Standardized Assessment of Concussion (SAC) [13] and Sport Concussion Assessment Tool (SCAT) [14] offer a fairly comprehensive assessment of the symptomology related to one's cognitive abilities. The SAC tool allows for immediate sideline mental status assessment of an athlete suspected of having a concussion. It assesses orientation, immediate memory, concentration, and delayed memory as well as includes an exertion test and a brief neurologic exam. It can be administered in approximately 5 minutes. Review of the literature suggests that, when used alone, the SAC does not reach statistical significance, but most experts still recommend its use as part of a more comprehensive evaluation of concussion [15]. According to the 5th International Conference on Concussion in Sport, the latest version of the SCAT test, the SCAT 5, is "the most well-established and rigorously developed instrument available for sideline assessment" [16]. This statement is actually in reference to the original versions of the SCAT test, the SCAT and SCAT 2, which both have strong evidence validating their use both on the sidelines and in the office. The SCAT 5 has yet to have a significant number of studies to officially validate this version of the test; however, the format is essentially the same as previous versions, so its use has been deemed appropriate [17].

Changes in emotional state, particularly depressive symptoms, can severely impact quality of life for months after the injury [18]. Those who have a history of behavioral or mental illness, including post-traumatic stress disorder, affective disorders, substance use, or attention deficit hyperactivity disorders, tend to have a higher risk of developing PCS and prolonged mood instability [19, 20]. The Patient Health Questionnaire-9 (PHQ-9) is a validated depression screening tool that can serve as a useful adjunct to concussion screening when there is a high suspicion of a depression or affective disorder that has been exacerbated by a concussion. It is self-administered and can be used in multiple settings and populations [21, 22], examples including both concussed military personnel [23] and adolescents with traumatic brain injury (TBI) [24].

Question: What cranial nerve changes can be seen in concussed patients?

It is possible for one or more cranial nerves to sustain injuries even with minor head trauma, and these may often be missed when evaluating for a concussion [25, 26]. One study showed that 12.6% of patients with head trauma, including a significant number with mild TBI, had cranial nerve (CN) injuries [26]. In this study, CNs II, CN III, and CN VII were the nerves most commonly injured [26]. In patients with mild TBI, CN I was most often found to be impaired with CNs II, IV, and VII being frequently injured as well [25]. In those with CN injuries, more than 80% showed abnormalities on head computed tomography scans [25]. This research suggests that physical examination of these nerves is particularly important to test, whereas CN V and CNs IX-XII may be less revealing [25, 26]. Interestingly, up to 30% of patients diagnosed with a concussion experience anosmia or dysosmia, and CN I has a higher likelihood of injury in mild cases [25, 27]. That being said, CN I testing is frequently excluded from cranial nerve testing on most exams based on difficulty with administration. It is unclear how injury to this nerve may affect appetite or nausea, but testing should certainly be considered in concussion patients presenting with persistent anorexia or nausea. Table 4.2 outlines the approach to cranial nerve testing.

Table 4.2 Cranial nerve exam findings

Cranial nerve	Exam testing
CN I: Olfactory nerve	Sense of smell with standardized order
CN II: Optic nerve	Visual acuity Pupillary light reflex Visual fields
CN III: Oculomotor nerve	Medial, superior, and inferior abduction movement of eye Accommodation of eyes
CN IV: Trochlear nerve	Inferior adduction movement of eye
CN V: Trigeminal nerve	Sensation of face Clamping of jaw
CN VI: Abducens nerve	Lateral movement of eyes
CN VII: Facial nerve	Facial movements (raise eyebrows, puff out cheeks, smile showing teeth) Taste of anterior 2/3 of tongue
CN VIII: Vestibulocochlear nerve	Whispered hearing Assess for nystagmus
CN IX: Glossopharyngeal nerve	Swallowing and pharyngeal gag reflex Taste of posterior 1/3 of tongue
CN X: Vagus nerve	Gag reflex Visualization of uvula and posterior pharynx with phonation
CN XI: Spinal accessory nerve	Manual muscle testing of sternocleidomastoid and trapezius
CN XII: Hypoglossal nerve	Assess for tongue deviation with protrusion

Bolded nerves: most commonly associated with concussions [25]

Question: Why is it important to assess the cervical spine in a concussion exam?

When assessing head injuries, it is essential to evaluate for any signs of neck injury as the two can closely imitate each other and coexist. In particular, cervical muscle strain caused by whiplash injuries may present with headaches, dizziness, disequilibrium, visual changes, and poor balance similar to that of concussions [28–30]. It is important to palpate the C-spine and, if negative, also apply resisted cervical isometric forces at the higher cervical levels. If pain is reproduced with this maneuver, this should prompt cervical spine x-rays with flexion and extension views as well as possibly advanced imaging [28]. The Spurling test is useful in determining signs of cervical nerve root irritation, either by a herniated disc or possible dislocation. It is performed by extending and rotating the patient's head toward the arm being tested and applying an axial force to the head. A positive test is elicited if the patient develops shooting pain down the arm with this maneuver [1].

Cervical proprioception is another important physical exam test that provides spatial orientation of how the head is positioned and moves relative to the trunk. It can be tested by having a patient close his or her eyes and try to bring his or her head and neck to a neutral position within 5° [31]. It is useful to assess as it can be associated with symptoms of disequilibrium [31]. Abnormalities with cervical proprioception can be treated with specific proprioceptive rehabilitation and, if undetected, may lead to prolonged symptoms [29]. Additionally, it is important to assess both the temporomandibular joint and thoracic abnormalities as these can also coexist with concussions and contribute to headache, tinnitus, dizziness, neck pain [32], thoracic winging [33], and thoracic trigger points [34].

Question: What is the utility of evaluating vestibulo-ocular dysfunction, and what tests are most relevant?

Vestibular dysfunction is a common presenting sign seen with concussions and includes symptoms such as dizziness, vertigo, and difficulty with balance [35]. Additionally, it can adversely affect one's quality of life with prolonged symptoms, higher risk of PCS, and increased risk of disability [36, 37]. The differential remains large for concussed individuals who have dizziness and disequilibrium. One rare manifestation of disequilibrium following concussion is known as post-traumatic benign paroxysmal vertigo (t-BPPV) which carries a much worse prognosis than idiopathic BPPV [38, 39]. Once vestibulo-ocular dysfunction is diagnosed, it can be successfully treated with a rehabilitation program. Therefore, providers need to be assessing for this in their concussion evaluations and referring to rehabilitation specialists trained in concussion and vestibular disorders [38, 40, 41].

The primary role of the vestibulo-ocular reflex is to coordinate and stabilize images on the retina during head movement. One physical exam maneuver that is useful and easy to utilize is the Halmagyi head thrust test. In this test, the patient

fixes their eyes on a specific point, while the physician holds the patient's face on either side and provides quick, unexpected jerks to the left and right. A normal test is observing whether the patient's eyes move the opposite direction of the thrust to keep the eyes centered on the retina [25].

Dynamic visual acuity should also be considered for testing of vestibulo-ocular dysfunction. This is performed with the head turning at a frequency of 2 Hz while measuring visual acuity with a Snellen chart. Abnormalities are associated with a reduction of three lines from the patient's baseline [42].

An ocular assessment with a focus on extraocular movements has become viewed as an increasingly important tool in determining the likelihood of developing post-concussive syndrome. Up to 40% of TBI patients exhibit ocular dysfunction such as impaired near point convergence, poor accommodation, and difficulties with oculomotor tracking [43, 44]. These vision problems often complicate return to school due to increased straining with computer use and reading [45]. The impairment of their function rises from shearing forces that injure the fiber tracts that connect the frontal cortex with the cerebellum. This can cause difficulty with coordination of eye traction on a fixed point, which can worsen with increased mental tasks [46].

Vestibular/Ocular Motor Screening

Vestibular/ocular motor screening (VOMS) has become a well-accepted method of evaluating a concussion patient's vestibulo-ocular system. The assessment includes five areas: (1) smooth pursuits, (2) horizontal and vertical saccades, (3) near point of convergence, (4) horizontal vestibulo-ocular reflex (VOR), and (5) visual motion sensitivity (VMS). A cross-sectional study of the VOMS assessment tool reflected good internal consistency and proved to be a reliable method of identifying patients with a concussion [47]. See Fig. 4.1 for a demonstration of VOMS testing.

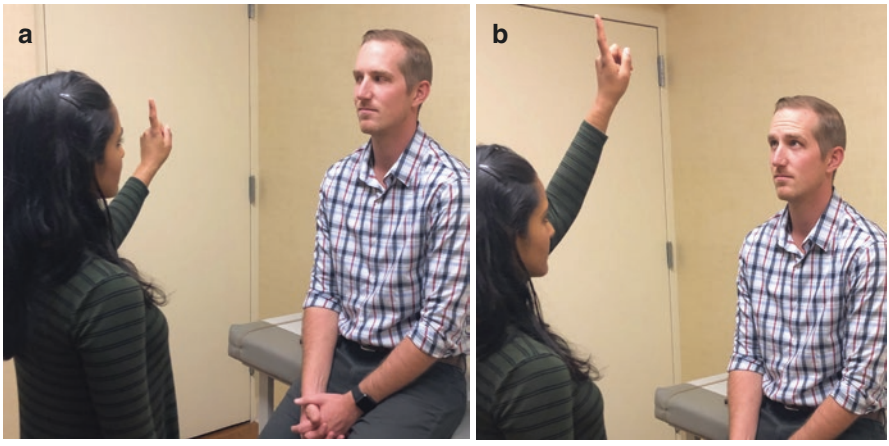

Fig. 4.1 Vestibular/ocular motor screening (VOMS) testing

Fig. 4.1 (continued)

Test	Headache	Dizziness	Nausea	Fogginess	How to perform test
Baseline symptoms (0–10)					Record baseline score for each symptom
Smooth pursuits					Tests ability to follow a slow-moving target. Examiner holds fingertip at 3 ft. from patient. Patient focuses on target as it moves slowly 1.5 ft. to the right of midline and then 1.5 ft. to the left of midline. 2 repetitions. Then same test done vertically
Saccades – horizontal					Tests ability to move quickly between targets. Examiner holds two fingers horizontally at 3 ft. from patient. One finger is 1.5 ft. to the left and the other is 1.5 ft. to the right. Patient gazes quickly left and right 10 times
Saccades – vertical					Repeated vertically
Near point convergence (NPC) in cm: Measure 1_____ Measure 2 _____ Measure 3_____					Ability to view a near target without having double vision. The patient focuses on a small object (14 pt. font) at arm's length and slowly brings it toward the tip of their nose. Patient should stop when object doubles or examiner observes outward deviation of one eye. Measure that point to tip of nose. Repeat 3 times. Abnormal is ≥6 cm from tip of nose
Vestibulo-ocular reflex (VOR) – horizontal					Ability to stabilize vision as the head moves Patient is asked to rotate their head horizontally 20° each direction while maintaining focus on an object. Speed should be ~180 beats/min. (one beat each direction); metronome can be used. 10 repetitions

(continued)

Test	Headache	Dizziness	Nausea	Fogginess	How to perform test
Visual motion sensitivity					Test ability to inhibit vestibular-induced eye movements using vision. Patient stands with arm outstretched and focuses on their thumb. Patient rotates as a unit (head, eyes, and trunk) 80° to the right and then left. Speed should be ~50 beats/min. (one beat each direction). 5 repetitions are performed

A smooth pursuits, horizontal; ***B*** smooth pursuits, vertical; ***C*** saccades, horizontal; ***D*** saccades, vertical; ***E*** near point convergence; ***F*** vestibulo-ocular reflex (VOR), horizontal; ***G*** visual motion sensitivity. Photographs by Carrie Jaworski
Provocation of symptoms is rated on a scale from 0 (no symptoms) to 10 (severe) and compared to baseline for each of the tests
Either abnormal findings/eye movements and/or increased symptoms should warrant further investigation

Saccades and Smooth Pursuits

Both saccades and smooth pursuit testing has demonstrated high sensitivity and specificity in helping to determine prognosis in recovering from concussions [48]. Abnormal extraocular tests has been associated with worse outcomes and have been more predictive for the development of PCS than neuropsychological testing, arm motor function, or patient self-report of symptoms [48]. Furthermore, patients diagnosed with PCS had worse visual tracking when compared with controls [48, 49]. The objective findings with saccades and smooth pursuit may also help to determine if there is true pathology from possible malingering [49].

Nystagmus

Nystagmus is a common finding in children who have sustained head trauma. Up to 46% of children may have spontaneous and/or positional nystagmus after head trauma, and 20% will continue to demonstrate abnormal findings for 6–12 months after injury [50]. Vertical or asymmetric nystagmus after a head injury may signify a more pathologic issue such as a brainstem lesion and should, therefore, prompt emergent imaging and/or referral [51].

Accommodation and Convergence

Normal near point of convergence ranges from 6 to 10 cm. Individuals who have sustained a concussion often develop spasm or dysfunction of the ocular muscles responsible for near point convergence [52]. This can lead a concussed patient to have double vision when focusing on an object that is greater than 6–10 cm away. Convergence insufficiency has been known to affect reading speed and comprehension [53]. This is especially problematic for adolescents and can delay their readiness for return to the classroom. Isolated convergence insufficiency has been shown to be responsible for visual symptoms in 9% of individuals who have sustained a TBI [54]. One study showed that 69% of adolescents with a concussion had one or more vision abnormalities: 51% had accommodative disorders, 49% had convergence insufficiency, and 29% had saccadic dysfunction [55].

King-Devick

The King-Devick test is a test that has been utilized more recently to track eye movements and is advertised heavily since it can be administered by a lay person such as a teacher or parent at a very low to no cost [56]. It is currently in the early stages of research regarding its effectiveness, and further research is needed before it can be considered a standard exam component [57, 58].

Fundoscopy

Fundoscopy has long been used as a tool to assess for papilledema due to increased intracranial pressure after head trauma. However, it is fairly low yield in the setting of concussions since papilledema occurs in only 3.5% of severe head trauma cases [59]. It is also very operator-dependent and relies on the experience of the physician to accurately identify signs of papilledema [60, 61]. Nonetheless, clinicians should use their best judgment in terms of utilization of this test.

Question: How useful is balance testing in a concussion evaluation?

Concussions are associated with abnormalities in balance and equilibrium both in static and dynamic testing [38, 62]. The Balance Error Scoring System (BESS) is frequently used in assessing athletes who have sustained concussions [63], but, despite this, there are several noted drawbacks including length of time of testing

which can take 5–7 minutes, low sensitivity [64], limited reliability between multiple tests [65], and high false-positive rates [66]. Additionally, sensitivity for diagnosing concussion drops days following concussions as balance is said to normalize over the first 3–5 days [67].

The Romberg test, though frequently administered in clinical office settings, also has low yield for assessing vestibular dysfunction [68]. On the other hand, tandem gait and coordination tests, such as finger to nose tests, have been found to have good reliability when used in the assessment of concussions [69]. Additional tests typically used in the assessment of balance in the elderly such as the Timed Up and Go, Gait Speed, Functional Gait Analysis, and Five Time Sit to Stand show promise in evaluating balance in the adolescent with a sports-related concussion, but are not yet considered standard of care [70].

Question: What additional neurologic evaluation should be considered in concussion testing?

There is not enough evidence to state whether manual muscle testing (MMT) or deep tendon reflexes (DTR) are affected by concussion. However, the rationale for testing muscle strength, DTRs, and the remainder of the neurologic examination is to exclude more serious pathology. Focal muscle weakness with absent or hypoactive reflexes may indicate a brainstem or cerebellar lesion [71]. On the other hand, findings such as hyperreflexia, finger rolling, pronator drift, Hoffman's sign, and a Babinski sign can signify an upper neuron injury of the cerebral cortex [71].

Key Points

- Orthostatic vital signs can be useful in evaluating patients who present with dizziness as a way to distinguish between autonomic dysregulation related to entities outside of, or in addition to, the concussion diagnosis.
- Utilizing fairly well-studied standardized tests such as the SAC or SCAT 5 can provide objective data how a concussion is impacting a patient's cognition, memory, and concentration.
- Cranial nerve injury can occur in conjunction with head injuries, and, therefore, a thorough cranial nerve examination should be included in the evaluation of a concussion.
- It is important to evaluate the cervical spine as a part of a concussion exam as these symptoms commonly mimic those seen in concussions and can lead to prolonged symptoms if not identified and treated.
- The vestibular/ocular motor screening (VOMS) evaluation utilizes tests such as smooth pursuits, saccades, and near point convergence to assess for irregular eye movements as well as complaints of headache, dizziness,

nausea, and fogginess. Abnormal exam findings can help identify those who may benefit from early vestibular-ocular rehabilitation.
- The Balance Error Score System (BESS) is most commonly used when assessing balance, but tandem gait and coordination tests may provide more insight into abnormalities with balance based on the literature.
- Neurologic assessment with manual muscle testing and deep tendon reflexes does not have sufficient evidence to help support the diagnosis of concussion but can be helpful in ruling out other neurologic injuries.

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Chapter 5
Diagnostic Tests for Concussion

Alan Shahtaji, Samuel Galloway, and Laura Murphy

Clinical Case

A 17-year-old female is brought into your outpatient primary care clinic after being elbowed in the head last night while challenging for a header in her soccer match. Her parents want to know what the best diagnostic test to confirm a concussion would be.

There is no single best diagnostic test to confirm a concussion. Rather, the diagnosis of acute concussion requires a multifaceted approach that takes into account the history surrounding the injury, signs, and symptoms at that time as well as current signs and symptoms upon evaluation in clinic. The exams described below are part of this initial evaluation and can be implemented in the clinic to help aid the primary care physician in making a diagnosis of concussion.

Table 5.1 summarizes the diagnostic utility of the tests discussed in this chapter.

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Table 5.1 Diagnostic concussion testing summary

Diagnostic tests	Sensitivity % (Sn)	Specificity % (Sp)	Pros	Cons	∗Notes
PCSS	47.4–72.2 [3]	78.6–91.7 [3]	Can track recovery; may predict prolonged recovery [14, 15]	Subjective report	Use within 3–5 days post-injury [3]
SAC	80–94 [4]	76–91 [4]	Low cost; no additional materials needed	Limited in RTP decision-making [4, 5]	Can be done on sideline
BESS/ mBESS	16–60/71 [8–10]	91–93/65 [8–10]	Sp consistent up to 7 days post-injury [8–10]	Sn decreases past 1–2 days post-injury; both lack objectivity [8–10]	
SCAT3∗	75.8–76.2 [11]	100 [11]	Good for initial diagnosis of concussion in first 3–5 days post-injury	Limited in tracking recovery and RTP	∗Data not available for SCAT5 at time of publication
MACE∗	10.7–40.4 [2]	80–96 [2]		Limited use >12–24 hours post-injury [2]	∗Scores 24 or less
VOMS∗	~60–95 [18]	–	High internal consistency; relatively low false-positive rate	Not validated for sideline use	∗Score ≥ 2 Combining multiple measures increases accuracy
K-D test	86 [22]	90 [22]	High Sn/Sp for sideline use	Requires baseline test; fee to use	Does not measure NPC, accommodation
Eye tracking	57 [18]	96 [18]	Does not require baseline test; good test-retest reliability	Expensive technology	
Force plate/ BTrackS	64 [32]	90 [32]	Objective; eliminates inter-observer variability	Expensive, requires high-tech equipment	
Orthostatic vital signs	–	–	Easy to use in clinic	Limited data re: predictive capacity	
Buffalo Concussion Treadmill Test	–	–	Helpful for diagnosis, prognosis, and developing patient-specific exercise recommendations [39]	Limited access in primary care clinic	Safety data excellent

*Please refer to Notes

Post-concussion Symptom Scale (Checklist)

Question: If someone completes a symptom checklist without any symptoms, does that rule out a concussion?

The Post-Concussion Symptoms Scale (PCSS) is comprised of a list of symptoms separated into four categories: physical, emotional, cognitive, and sleep-related. The athlete is required to grade the severity of these symptoms on a scale of 0 (absent) to 6 (most severe).

Although the PCSS has demonstrated moderate sensitivity and specificity when used within 2 days of injury, it appears to have the highest diagnostic utility when used within 3–5 days post-injury as sensitivity (47.4–72.2%) and specificity (78.6–91.7%) increase [1, 2]. This test can be particularly useful as recent research suggests that symptom burden has been shown to correlate with greater declines in clinical testing performance and may help prognosticate duration of recovery [3–5]. Nevertheless, the subjective nature of this test is dependent on the athlete's honesty and ability to correctly identify his/her own symptoms. For this reason, the PCSS alone is not useful in ruling in or out a concussion. It is important to use the symptoms scale in conjunction with physical exam findings to evaluate for impairment in order to allow for more comprehensive data when assessing for concussions.

Standardized Assessment of Concussion (SAC)

Question: Is the Standardized Assessment of Concussion (SAC) more objective than the symptom checklist?

The Standardized Assessment of Concussion (SAC) is a 5–10-minute written test with a total composite score of 30 points that includes four domains of cognitive function: orientation, immediate recall, concentration, and delayed recall [6]. This test has demonstrated its usefulness in ease of administration as well as its ability to detect subtle differences in mental status changes. It has been shown to have a high diagnostic value with a sensitivity of 80–94% and a specificity of 76–91% [7]. Unlike the PCSS, this test gathers objective data and is therefore not dependent on the subjective report from the injured athlete. This test can be used as an initial tool to help in diagnosis, as well as to track patients' recovery over time. It is limited in its ability to help guide return to play decisions, however, as research has suggested that 50% of injured athletes still had symptoms at the time that their SAC score returned to baseline with some studies reporting it to be as soon as 2 days after injury [7, 8].

BESS: Balance Error Scoring System

Question: What are the pros and cons of using the mBESS over the BESS?

The Balance Error Scoring System (BESS) is a commonly used low-cost approach in evaluating static postural stability for an athlete with a suspected concussion and can be done in any exam room. (See the Physical Examination of Concussion chapter for further details.) The exam takes up to 10 minutes to conduct and requires two different testing surfaces: a firm surface such as the ground and then again on a 2.5-inch-thick foam surface. The patient performs three different stances with hands on hips to be held 20 seconds at a time: the double-leg stance, single-leg stance, and tandem stance initially on a firm surface and then on a foam surface. Every error made (refer to Fig. 5.1) equals one point, so a higher score indicates more severe postural instability.

The BESS has demonstrated an overall low-to-moderate diagnostic accuracy for acute concussion (sensitivity 16–60% and specificity 91–93%) [9–11]. Sensitivity has been shown to decline as time progresses past 24–48 hours from onset of injury, whereas specificity remains consistent up to 7 days post-injury. This sensitivity to specificity ratio indicates high diagnostic value if positive; however, a negative test does not rule out concussion, especially as time passes between injury and evaluation.

The modified Balance Error Scoring System (mBESS) eliminates the foam pad present in the conventional BESS and is therefore more practical in the primary care or sideline/field setting. Compared with the BESS, the mBESS has a higher sensitivity (71%) within 24 hours after concussion but is less specific (65%). This is either a reflection of the test's limitations or its ability to detect lingering post-concussion stability deficits not identified on the BESS [10].

Of note, both the BESS and mBESS rely on the subjective assessment of the observer and are subject to intra-rater and inter-rater variability [10] and lack true objectivity. Some digital alternatives for balance testing are discussed later in this chapter.

SCAT5

Question: Is there a cutoff score on the SCAT5 that can be used to reliably diagnose concussion?

The Sport Concussion Assessment Tool (SCAT) is arguably the most widely used standardized method of evaluating those with a suspected concussion in patients aged 13 years and older. It has undergone numerous revisions to include more

comprehensive evaluative tools with the most current 5th edition (SCAT5) released in 2017. It is a pen and paper test that can be implemented in the clinic, requiring about 10 minutes to conduct with the patient in a resting state, and can be compared with a preseason/baseline SCAT5 assessment (although this is not required). It includes an emergency/on-field and office/off-field assessment, but for the purposes of this chapter, we will focus on the latter.

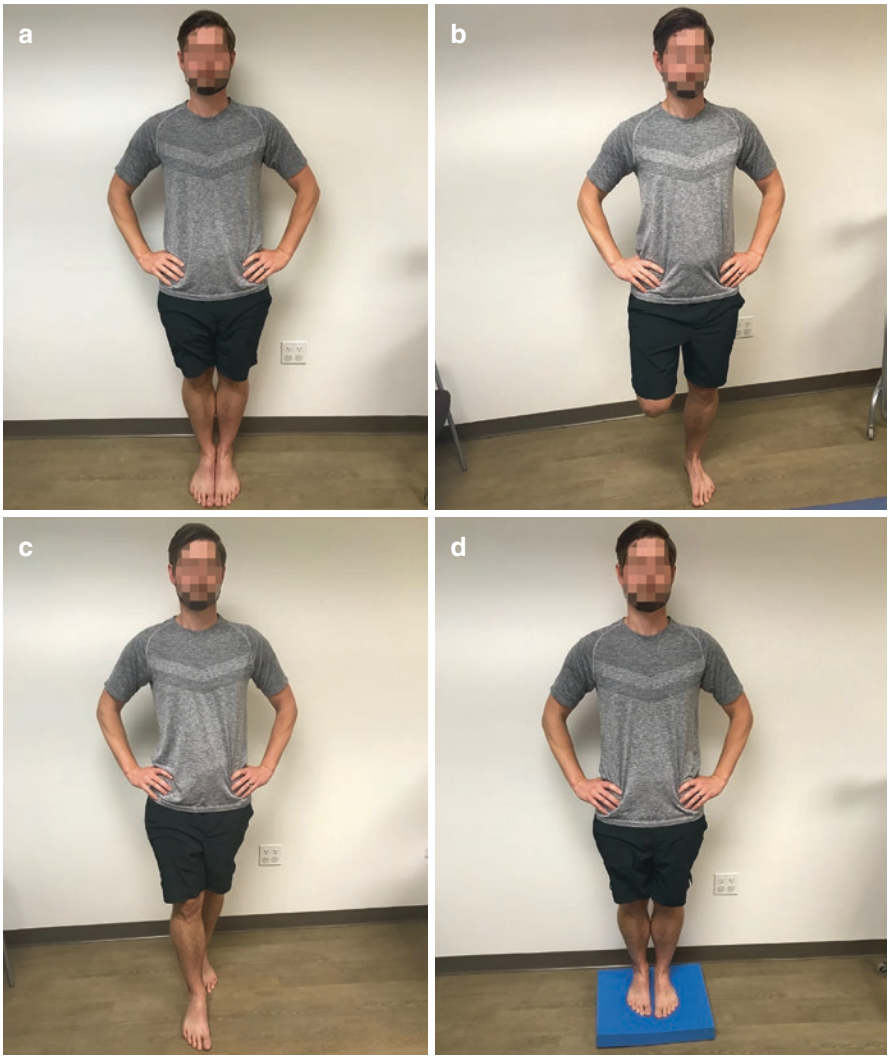

Fig. 5.1 Balance Error Scoring System (BESS). (**a**) Firm surface, double-leg stance. (**b**) Firm surface, single-leg stance. (**c**) Firm surface, tandem stance. (**d**) Foam surface, double-leg stance. (**e**) Foam surface, single-leg stance. (**f**) Foam surface, tandem stance

Fig. 5.1 (continued)

The off-field assessment implements commonly used diagnostic tools in six steps [12]:

1. Patient demographics (particularly focusing on history of head injuries and neuro/psychologically related diagnoses)
2. Symptom score
3. Cognitive screening using SAC
4. Neurologic screening (including mBESS)
5. Delayed recall
6. Decision documentation for the provider

The SCAT has high diagnostic utility that is generalizable to the larger population with only low-to-moderate levels of bias and is most reliable in the first 3–5 days post-injury [2]. As a result the SCAT is more valuable in helping to make the initial diagnosis of concussion rather than tracking recovery and assisting return to play decision-making. Additionally there are no specific cutoff scores that have been validated in diagnosing concussion, but current data does show that higher cutoff scores correlate with lower sensitivity (75.8–76.2%) [2]. Compared to prior versions, the SCAT5 adds more complexity to the cognitive component. However, more research is needed to further evaluate whether these changes help improve diagnostic utility in the acute and subacute post-injury evaluations.

While the cervical spine exam is not included in the off-field/office assessment, the chapter authors recommend that this should be included in the physical examination.

Pediatric SCAT5

Question: Is there an alternative to SCAT5 for younger children?

The pediatric SCAT5 is meant for use in children from 5 to 12 years old and is consistent in format with the traditional SCAT5. To yield the highest accuracy, the test should be conducted in no less than 10–15 minutes. Major differences compared with the traditional SCAT5 are lack of orientation questions, modification of the symptoms scale, and addition of parent observation of symptoms [13]. As with the traditional SCAT5, a noted limitation is the lack of data available in regard to its validity for patients with learning disabilities and across different cultures and languages [14].

Military Acute Concussion Evaluation (MACE)

Question: How does MACE compare to SAC when evaluating athletes more than 24–48 hours after injury?

The Military Acute Concussion Evaluation (MACE) is designed to assess the likelihood of a concussion for patients where the injury has occurred in a military operational setting. The Standardized Assessment of Concussion test, discussed above, is embedded within the MACE [15]. The test is made up of a total of 30 points with the mean non-concussed patient scoring around 27 and a score of 25 or less more indicative of an acute concussion versus other, more serious brain injury (sensitivity 11–40%, specificity 80–96%) [16]. Similar to the SAC, the MACE was designed to be administered acutely as it demonstrates declining sensitivity/specificity when administered more than 12–24 hours after the event. Furthermore, as with other diagnostic tests, there is no cutoff value that has been validated for diagnosis of acute concussion, indicating the importance that this test be used as an adjunct to help guide diagnostic reasoning.

The Vestibular/Ocular Motor Screen (VOMS)

Question: Considering that The Vestibular/Ocular Motor Screen (VOMS) partly relies on the subjective report of the patient, how useful is it in the diagnosis of concussion?

Due to the multiple areas of the brain and vast number of neural connections that are involved with the vestibular and ocular systems, these areas are commonly affected in athletes who have sustained a concussion. In one study of pediatric patients, 81%

showed an abnormality of the vestibular system on initial testing, and these athletes were shown to have longer time to return to school and play and also have lower scores on initial neurocognitive testing [17]. In another study, 69% of patients with a concussion had at least one visual abnormality, with 46% having impairment in more than one area [18].

The Vestibular/Ocular Motor Screen (VOMS) is a physical exam test to assess for impairments in the vestibular and ocular systems. It is a brief test that takes approximately 5–10 minutes to administer and generally consists of multiple components—smooth pursuits, horizontal and vertical saccades, horizontal and vertical vestibular ocular reflex (VOR), visual motion sensitivity (VMS), and near point convergence (NPC). The athlete is asked to report changes in four symptoms (headache, dizziness, nausea, and fogginess) compared to baseline using a number scale of 1–10 after each component. A visual depiction of this can be found at https://www.natafoundation.org/wp-content/uploads/VOMS-Infographic.pdf.

Impairment in any of the components of VOMS has been associated with concussion [19]. The test has high internal consistency and low false-positive rates as assessed in several recent studies [19–21]. In one study, an increase in ≥2 symptoms in any of the VOMS categories demonstrated a positive likelihood ratio of 23.9:42.8 in the diagnosis of concussion [19]. Utilizing the multiple components of VOMS increases its accuracy in detecting concussion [19].

VOMS is a reliable, validated test for the evaluation of concussion. It is easy to perform in a short amount of time and does not depend on any additional tools nor require baseline data. Recent literature has demonstrated high reliability and low false-positive rates of VOMS; thus, despite its subjective reporting of symptoms, it is a helpful test for use in the clinic to aid in the diagnosis of concussion. It has been validated for use in the clinic setting, but further research is needed to validate its use as a sideline tool. The VOMS is commonly included as part of the examination of a suspected concussion. (See the Physical Examination of Concussion chapter for further details.)

King-Devick Test

Question: Based on current literature, which diagnostic test is more appropriately applied in the clinic: King-Devick or VOMS?

Additional tools are available at certain clinics and research institutions that objectively measure eye tracking, balance, and sway. The King-Devick test (K-D) is a relatively simple rapid number naming test that is used as an objective way to assess saccadic eye movements in addition to cognitive function. It can be performed using either a computer/tablet-based application or printed flash cards. The athlete reads a series of numbers across horizontal rows that are spaced at different intervals across the page. The series of cards are meant to progressively increase in difficulty. The

total time to complete the three test cards is recorded, and the number of errors is counted. This is compared to a baseline, which should be performed prior to concussion. Slowing of the time it takes to complete, the test is a positive indicator for concussion.

The K-D test can be administered in about 2 minutes and has been studied most commonly as a sideline assessment to aid in the diagnosis of concussion, for which it has been validated in the literature [23–27]. It can be administered by healthcare professionals, athletic trainers, coaches, or other laypersons with good inter-tester and test-retest reliability [23, 27]. A recent meta-analysis found a sensitivity of 86% and a specificity of 90% in the sideline diagnosis of concussion [23]. Although this meta-analysis emphasizes the importance of comparison of data with baseline pre-injury values, there have been studies comparing data to age-matched controls without baseline data that have shown it to still be an accurate test for the diagnosis of acute concussion [28].

The K-D test is a rapid test for the diagnosis of concussion that has high sensitivity, specificity, and inter-tester and test-retest reliability. Compared to VOMS, which has been researched in the clinic setting, the K-D test is validated for use in the sideline diagnosis of concussion. It does typically require a baseline prior to concussion for comparison, and it is a licensed product that does require a fee.

Eye Tracking

Question: Are there any objective tests of ocular function that are not subject to human measurement?

It is well-known that patients with concussions have a high incidence of visual abnormalities, with one recent study reporting that 69% of adolescent athletes diagnosed with concussion had an abnormality in at least one visual test [18]. Developing reliable, objective tests for the diagnosis of concussion has been difficult. However, there has been recent evidence supporting the use of eye-tracking software to aid in the diagnosis of concussion. This can be a rapid, noninvasive way to test for concussion with the added benefit of not requiring a baseline test. A recent study reported a sensitivity of 57% and a specificity of 96% in the use of eye-tracking metrics for detection of convergence and accommodation abnormalities in a pediatric population with concussions [29]; other studies have confirmed a high sensitivity and specificity [30]. It has also been shown to have good test-retest reliability [31].

EyeBOX is a proprietary eye-tracking device that was recently FDA approved for use in the diagnosis of concussion. It is a device that utilizes a unique algorithm to track eye movements while a patient watches a 4-minute video clip. It is marketed for use in patients 5–67 years of age. Eye-Sync is another FDA approved device that uses eye-tracking software to monitor eye movements to provide an objective way of measuring smooth pursuits, saccades, and VOR.

These devices provide more objective data in the diagnosis of concussion that helps to minimize the human measurement factor. However, they require the use of proprietary software and thus can be expensive and are not readily available in the general primary care clinic.

Force Plate/BTrackS/Sway Balance

Question: Is there a more objective test of balance than the BESS test?

Balance testing is an evidence-based recommended component in the complete concussion evaluation [32]. BESS is the standard of balance testing, but due to its relatively low sensitivity and subjective nature, there is a need for a more sensitive and objective test. Standard force plate technology has been the gold standard of balance testing but can be expensive and requires high-tech equipment. BTrackS is a relatively low-cost alternative force plate that provides a higher sensitivity than BESS at 64% while still having high specificity at 90% [33]. It has also been shown to have high test-retest reliability, and there is no practice improvement with repeat testing [34]. It is a rapid (<2 minutes), portable, objective measure of balance.

Sway is an app available for Apple and Android phones that uses accelerometer data to objectively measure an athlete's balance while performing BESS testing. Studies have validated the accuracy and reliability of its data with high-tech force plates [35]; however, at the time of publication, no studies were found that independently assessed its use in the diagnosis of concussion. Sway does show promise as a low-cost objective tool for measuring balance.

There are several other force plate products each with different profiles in terms of precision, accuracy, feasibility, and cost. This is outside the scope of this chapter.

Orthostatic Vital Signs

Question: How does autonomic dysfunction manifest in orthostatic vital signs, and which measurement (BP or HR) is more important?

One of the early objective physiological signs of acute concussion is exercise intolerance, a manifestation believed to be related to impaired autonomic function. This belief is derived from studies that demonstrate a pattern of neurochemical and metabolic changes within the injured brain that result in physiologic changes largely controlled by the autonomic nervous system. The observed result, which has been confirmed across multiple studies, is an increase in sympathetic activity resulting in

reduced heart rate variability, delayed blood pressure stabilization, and changes in cerebral perfusion [36, 37]. A simple, practical screening method for these physiologic changes in the clinic setting is through orthostatic vital signs. Leddy et al. [38] describe using supine and standing (within 3 minutes) vital signs for the assessment of subjective and objective orthostatic intolerance. Subjectively, the patient may complain of dizziness, blurred vision, nausea, and/or feeling light-headed. The objective criteria is a 20 mm Hg drop of systolic blood pressure or a 10 mm Hg drop in diastolic blood pressure. In the setting of concussion evaluation, it is recommended to measure supine and standing only [39]. Heart rate is not required for the diagnosis but can be helpful in the clinical context; an increased heart rate (>20 bpm) going from supine to standing may be reflective of hypovolemia, whereas a blunted HR response or lack of elevation is associated with neurogenic etiology [37, 39].

Notably, there is limited research that directly evaluates the diagnostic utility of orthostatic vital signs as it pertains to autonomic dysfunction for the evaluation of a concussion.

Buffalo Concussion Treadmill Test

Question: What is a standardized way to test physiologic dysfunction and exercise tolerance in concussed athletes?

The Buffalo Concussion Treadmill Test (BCTT) is a standardized protocol treadmill test that can be used in the diagnosis of acute concussion and recovery prognosis. It has evidence for use in developing a heart rate-based exercise plan for concussed athletes and/or assessing physiologic recovery. Recent research has shown that rest beyond the first couple of days after concussion can be detrimental rather than beneficial for recovery [40–42]. A recent randomized controlled trial of adolescent athletes showed that those who participated in progressive sub-symptom threshold aerobic exercise had a faster recovery than those assigned to a stretching program that did not raise their heart rate [43]. (See (Chap. 9) for further details.)

The protocol (*see* Table 5.2) [40]*:*

The athlete is started at 3.6 mph at 0% incline. After the first minute, the incline is increased by 1% each minute. The athlete is assessed every minute for rating of perceived exertion (RPE, Borg scale) and for symptoms. Heart rate (and blood pressure if available) is monitored every 2 minutes. The test is stopped if the athlete has exacerbation of symptoms (≥3 points from pretest rating on 1–10 point visual analog scale) or at exhaustion (RPE of 19–20). If the maximal incline is reached without stopping criteria being met, the speed is increased by 0.4 mph every minute until stopping criteria are met. The test should not be performed if the athlete has significant baseline symptoms of ≥7 on the pretest VAS.

The BCTT has been shown to be a safe and reliable test of physiologic dysfunction in concussed athletes [40, 44]. With research continuing to support aerobic

Table 5.2 Buffalo Concussion Treadmill Test [34]

Protocol	Starting speed is 3.6 mph at 0% incline Incline is increased by 1% each minute If maximum incline is reached and patient has not reached stoppage criteria, speed is increased by 0.4 mph each minute until stoppage criteria are met
Athlete monitoring	Rating of perceived exertion (RPE, Borg scale) and symptoms assessed each minute Heart rate and blood pressure measured every 2 minutes
Stoppage criteria	Significant exacerbation of symptoms (≥3 points from baseline that day on 1–10 point visual analog scale) OR At exhaustion (RPE of 19–20)
Contraindications to testing	Absolute: Unwilling to exercise Increased cardiac risk as defined by the American College of Sports Medicine Focal neurologic deficit Significant risk of walking/running on treadmill due to orthopedic injury, balance, or visual deficit Relative: Use of beta blockers Major depression Minor risk of walking/running on treadmill due to orthopedic injury, balance or visual deficit Blood pressure > 140/90 BMI ≥ 30

exercise in the recovery of concussed athletes, the BCTT provides an objective measurement of symptom threshold and can be used for patient-specific exercise recommendations [45]. The Buffalo Concussion Bike Test is an alternative that has been validated [46] and can be very useful if vestibular dysfunction or limited mobility prohibits testing on the treadmill.

Key Points

- The Post-Concussion Symptoms Scale has the highest diagnostic utility when used within 3–5 days post-injury and may help prognosticate duration of recovery.
- The SCAT5 integrates several components (PCSS, SAC, mBESS) into one evaluation that is most reliable when used 3–5 days post-injury.
- Both the BESS and mBESS evaluate for static postural instability and rely on the subjective assessment of the observer.
- Force plate technology can provide a more objective measure of postural instability than BESS but require proprietary hardware to use.
- Orthostatic vital signs can be a useful early diagnostic test for concussion and may demonstrate autonomic dysfunction.

- VOMS is a reliable, validated test for the evaluation of concussion. No baseline is required, and there is no cost to perform the test, making this feasible and practical for most clinicians.
- The King-Devick test is a rapid (2 minutes) test for the sideline diagnosis of concussion that is a measure of saccadic eye movement as well as cognitive function.
- The Buffalo Concussion Treadmill Test is a safe and reliable way to test for physiologic dysfunction in the concussed athlete, although it is more commonly used to help determine symptom threshold and guide return to activity recommendations.

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Chapter 6
Neurocognitive Testing

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Clinical Case

An 18-year-old high school senior and his parents present in your office after being told that he needs clearance by a physician prior to returning to football practice. What single test can reliably make the diagnosis of concussion?

One of the challenges with concussion is finding objective and definitive testing to both aid the clinician in the diagnosis and help define the recovery. Multiple studies have identified blood biomarkers that rise and fall during the course of a concussion marking the metabolic changes following this injury [1–5]. However, no individual biomarker or combination of these biomarkers has yet proven to be a reliable and definitive test to rule in or rule out a concussion. Concussion research in the animal model has given details on the cellular level metabolic crisis associated with concussion and the timeline for restoration of normal metabolic activity [1, 6]. This timeline does not necessarily appear to directly correlate with the timeline of the clinical symptoms or testing abnormalities that we see in humans [7]. As noted in previous chapters, concussion can present with a wide variety of symptoms and clinical findings spread over several different physical, emotional, and cognitive domains. Therefore, a thorough evaluation for concussion should contain elements that provide information from each of these domains. Neuropsychological testing

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can provide important and useful information in the cognitive and emotional domains.

Neuropsychological testing has been used in various formats for many years to provide information for several different emotional and cognitive disease processes. These tests can assess cognition including intelligence, academic functioning, attention, working memory, processing speed, learning memory, visual spatial skills, fine and gross motor skills, and executive functioning. Current neurocognitive assessment tools utilized by providers experienced in concussion care and frontline concussion clinics are typically an abbreviated form of neuropsychological testing referred to as neurocognitive testing (NCT). These assessments are often brief enough to allow for the baseline screening of large numbers of athletes and yet still provide enough information to assist the clinician in the evaluation and management of concussions. Neurocognitive testing contains information from a limited number of the domains and *should not be used in a stand-alone manner* but rather part of a more comprehensive clinical evaluation [8].

Question: Is neuropsychological testing a new development?

Attempts at trying to localize cognitive functions in the brain started very early in the history of medicine. Herophilius is generally given credit for first attempting this evaluation process in 300 B.C. [9]. More recently, neuropsychological testing has been described as "the normatively informed application of performance-based assessments of various cognitive skills" [10]. This testing is usually accomplished through a lengthy battery of subtests covering a spectrum of cognitive ability areas. The areas evaluated may vary from test to test but typically include assessments of several cognitive domains including memory, attention, processing speed, reasoning, judgment, problem solving, spatial function, and language function (Table 6.1). This testing is currently used to assess a variety of neuropsychiatric conditions

Table 6.1 Common neuropsychological tests used in sports concussion assessments

Neuropsychological test	Cognitive domain
Controlled Oral Word Association (FAS)	Verbal fluency
Hopkins Verbal Learning Test	Verbal learning, immediate and delayed memory
Paced Auditory Serial Addition Test	Attention, concentration
Stroop Color and Word Test	Attention, information processing speed
Symbol Digit Modalities Test	Psychomotor speed, attention, concentration
Trail Making: Parts A & B	Visual scanning, attention, information processing speed, psychomotor speed
Wechsler Adult Intelligence Scale	Intelligence and cognitive ability
Wechsler Digit Span: Digits Forward and Backward	Concentration, attention
Wechsler Letter Numbering Sequencing Test	Verbal working memory

including dementia, mood disorders, stroke, and traumatic brain injury (TBI) [11]. These test batteries are most accurate when administered under controlled and standardized conditions. Current validated assessments typically provide databases with demographically matched normative data. The classic neuropsychological testing batteries are often very time-consuming and must be administered and interpreted by psychologists (usually neuropsychologists) and neuropsychiatrists with extensive training and certifications.

Question: Are computer-based neurocognitive tests a viable option in concussion evaluation?

Neurocognitive testing in the sports medicine and concussion community has been considered a cornerstone of concussion assessments [12–14]. Traditional neuropsychological testing can provide important diagnostic information for neurological, cognitive, and emotional conditions, many of which are not typically followed in a frontline concussion clinic or sports medicine clinic. The unique requirements in sports medicine have given rise to more concise and focused neurocognitive assessments that center on cognitive domains most often affected by concussion (e.g., memory, attention, processing speed, and reaction time). These newer assessments, often utilizing computer or tablet administration, can be accomplished in as little time as 5 minutes for the abbreviated field-side assessments and 10–30 minutes for more comprehensive pre- and post-injury assessment. While these sideline assessments do not yield as much information for decision-making and treatment, they provide objective information to aid clinical decision-making for a suspected concussion.

The Berlin Concussion Consensus statement and the National Athletic Trainers' Association (NATA) position statements have helped define the use of neurocognitive testing in sports medicine [13, 15]. These position statements clearly indicate that neurocognitive testing is a vital part of the overall concussion assessment but is *not intended to be used as stand-alone testing for evaluation or management*. Neurocognitive testing is best utilized when it is part of a larger multi-domain evaluation process.

Question: What is the difference between the testing administered by a neuropsychologist and computerized neurocognitive testing?

The term "neurocognitive testing" is often used interchangeably with neuropsychological testing. While both are used to evaluate the relationship between the brain and behavior, there are significant differences in the scope of use, information

derived, cost, evaluation time, and usefulness in neurorehabilitation planning. A neuropsychological examination is the gold standard for assessing all areas of brain function and includes a comprehensive evaluation of sensory/motor function, auditory and visual attention, working memory, verbal and visual memory, language, executive function, speed of processing, intellectual ability, and emotional capacity. The examination includes a combination or battery of tests that can provide an average or composite score across multiple ability areas and provide an overall index of how well a person functions cognitively at the time of testing [10]. As a result, testing is time-consuming and requires multiple hours, which may even be spread out over several days depending on the referral question and information needed. The final results are referenced to demographic groups of age, sex, race, and education levels. There are also internal checks built in to determine an examinee's effort and testing validity. The results can be interpreted by psychologists, neuropsychologists, or neuropsychiatrists and compared against known deficit profiles related to illness, disease, and injury for diagnosis. The results are detailed enough to be used for neurorehabilitation planning, special education placement, competency determination, forensic/legal purposes, drug or treatment research, and identifying functional impairments. Many of the various testing instruments were developed before the widespread use of computers and are administered using paper, pencil, and a stopwatch. In recent years there have been concerted efforts to computerize many of these tests, which require lengthy re-standardization processes. Traditional pencil-and-paper tests include those seen in Table 6.1. Most of these tests are copyright protected and require advance training and licensing to purchase, administer, and interpret. Most experts recommend a licensed psychologist, usually a board-certified neuropsychologist with clinical experience in evaluating sport-related concussion, and administer or at least supervise testing [10, 16].

Neurocognitive testing is aimed at addressing a subset of symptoms or cognitive functions related to a particular illness or injury. Most of these tests were created from the ground up for computer use to facilitate ease of administration, portability, and rapid scoring; some even include basic interpretation and provide limited age-related norms. The SCAT5, ImPACT, ANAM, Axon, C3 Logix, and other neurocognitive tests were designed and standardized to quickly assess the cognitive deficits seen with concussion, concentrating on attention, processing speed, and immediate memory. However, these devices could be inappropriate and even invalid in the evaluation of other cognitive impairments including learning disability, ADHD, brain tumor, stroke, traumatic brain injury, and other neurological conditions, due to limited domain assessment, differing comparison groups, and interpretation by those not qualified to provide a medical or psychological diagnosis. The administration of neurocognitive testing can be done by non-physician medical or athletic training staff. Some offer tablet-based administration for complete portability and provide cloud storage for universal wireless access. The health-care provider will want to become familiar with administration and interpretation documentation, especially if there are questions or concerns about effort that may impact returning to play. Lingering recovery due to comorbidities or worrisome cognitive deficits

may require a more comprehensive assessment and can always be referred for a full neuropsychological examination.

Question: What are some of the benefits of computerized neurocognitive testing?

Computerized neurocognitive assessments have found an increasingly common role in the evaluation and assessment of sport-related concussion. Different forms of these tests have increased in use for many reasons including practicality, ease of interpretation, and portability. The companies producing the CNTs suggest and provide information on how to administer and interpret the tests, but no formal training or certification is required. Programs can be downloaded to a computer or laptop or web-based programs utilized to administer the tests. Newer tablet-based tests have even improved the portability making it possible to take the test in nearly any environment. The results can then be uploaded to a central server allowing for review from any computer with web access. For example, these tests can be performed by a certified athletic trainer (ATc) at a school or sports training room environment and be remotely reviewed by a physician.

Computer-based neurocognitive assessment tools (Table 6.2) are much less time-consuming, often taking 10–30 minutes to complete, when compared to a traditional 4-hour neuropsychological test battery. These tests also differ from traditional neuropsychological testing in that they do not need to be administered or interpreted by a certified testing specialist. Scoring for these computer-based tests is automated and often produces a summary sheet for statistical analysis or automatically compared to baseline and/or normative data.

These computer-based neurocognitive tests are often useful for large group baseline testing preseason (often performed at the time of sport pre-participation examinations) and in the post-injury setting. With the relative ease of baseline testing, post-injury evaluations can readily be compared to the baseline test for an individual athlete and performed serially to assess for recovery [17]. A baseline comparison is particularly useful in those cases where learning or testing difficulties (ADHD, dyslexia, etc.) and other confounding diagnosis, such as depression, or chronic migraine, can interfere with normative testing result data. Another unique benefit to these computerized test batteries is that these tests allow for very accurate and quantified measurements of reaction time [16–19]. This is more difficult to obtain at the same level of accuracy on paper-and-pencil assessments. One of the final benefits of computer-based tests is the number of controlled test variations [17]. It may be more difficult to maintain the number of variations in traditional paper and pencil tests to accomplish this same task. Multiple retest variations are important for the athlete that is retaking tests over a relatively short time frame in order to track recovery and aid return-to-play decisions in an active management program.

Table 6.2 Computerized neurocognitive assessments

Test	Measured subtests	Summary scores
Automated Neuropsychological Assessment Metrics (ANAM)	Simple reaction time Procedural reaction time Code substitution learning Code substitution delayed Mathematical processing Matching to sample Second administration	Throughout Standardized subtest Standardized composite Composite score Classification of Impairment
AXON CogState	Processing speed Attention Learning Working memory	Subtest summary scores Composite score Classification of impairment
C^3Logix	Symbol digit coding Simple reaction time Choice reaction time Trail making A & B Verbal memory test, immediate Verbal memory test, delayed SAC concentration	Processing speed Inter-symbol response time and accuracy Simple reaction time Choice reaction time Trail A time Trail B time Trail B minus A time Immediate memory Delayed memory SAC composite score
CNS Vital Signs	Verbal memory test, immediate Visual memory test, immediate Finger tapping test Symbol digit coding Stroop test Shifting attention test Continuous performance test Verbal memory test, delayed Visual memory test, delayed	Neurocognitive index Composite memory Verbal memory Visual memory Psychomotor speed Reaction time Complex attention Cognitive flexibility Processing speed Executive function Simple attention Motor speed Composite score: IQ
HeadMinder (CRI)	Reaction time Cued reaction time Visual recognition 1 & 2 Animal decoding Symbol scanning	Psychomotor Speed index Simple reaction time
ImPACT	Word memory, immediate Design memory, immediate X's and O's Symbol match Color match Four letters Word memory, delayed Design memory, delayed	Verbal memory Visual memory Visual motor speed Reaction time Impulse control

Question: What is the role for computerized neurocognitive testing on the field side for possible concussions, and how does the field-side assessment differ from the evaluation in the clinic?

CNTs have increased in use and are part of many formal professional, collegiate, and high school concussion protocols because they can be administered relatively quickly and be performed as part of a baseline assessment and post-injury assessment. However, the requirement of a field-side test is different from a more detailed clinical assessment. Even a 10–30-minute CNT doesn't have a role in the immediate field-side evaluation. Field side, an assessment is used to evaluate an athlete and establish some validation of a concussion injury which by definition is a neurological or neurocognitive impairment from this biomechanical force applied to the head [13]. The decision to return an athlete to the field of play can be difficult and usually needs to be made rapidly. In some cases, the signs and symptoms of a concussion evolve over a number of minutes to hours [13]. Therefore, if the athlete shows enough evidence for a presumed or possible diagnosis of concussion during the assessment, then the athlete must be removed from play. A more complete neurocognitive assessment can then be performed at a later time. Neuropsychological testing and even typical CNTs do not have clinical utility for making the immediate, sideline decisions for a concussion injury. The need for immediate decision-making on the sideline has been the motivation for the development of several brief assessment tools. The functionality of these tests differs from both the traditional neuropsychological test batteries and the more recent CNTs. Brief field-side neurocognitive tests include the paper-and-pencil Standardized Assessment of Concussion (SAC) and the Sports Concussion Assessment Tool 5th Edition (SCAT5) which includes modified Maddocks questions (Table 6.3). More recently, some applications such as C3Logix and ImPACT Quick Test have incorporated similar components of these paper-and-pencil brief field-side assessments into a digital format on a computer tablet.

Table 6.3 Modified Maddocks questions from SCAT 5

What venue are we at today?
What half is it now?
Who scored last in this match?
What team did you play last week/game?
Did your team win the last game?

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Question: What are some concerns about using computerized neurocognitive testing?

With the rise in popularity and use of CNTs, some definite limitations and disadvantages have presented. The gold standard in neuropsychological testing is still considered to be the more traditional model administered and interpreted one-on-one by a trained and credentialed neuropsychologist.

There are several concerns from the neuropsychology community regarding CNTs. The first concern is that many of these tests have not yet undergone the same level of validation as the more traditional tests [16, 20]. Overall, CNTs have been found to have variable test-retest reliability and moderate sensitivity [7]. Any injury assessment tool or test is only useful if the test will reliably detect or rule out a specific injury or a finding related to a specific injury. Reliability is an extremely important concept in concussion testing due to individual serial testing strategies [21]. CNTs work most accurately when an individual has a baseline assessment and then is able to repeat a test post-injury for comparison and evaluation of the differences. The assumption is that the difference in testing scores is due to the injury, but many factors can affect testing results (Fig. 6.1). If a test has a low reliability coefficient, it may be difficult to attribute the testing result changes to the injury. When there has been no concussion, a change in scores from test to test indicates measurement error or other factors affecting the results.

A significant challenge in trying to validate CNTs has been the lack of standardization of what is being tested. Each group or company producing the CNTs uses different testing formats to test the cognitive domains deemed most important. Similar cognitive domains may be measured, but the specific subtests are different enough to make comparisons to other similar tests problematic [16]. Much of the research has been industry-sponsored, and there is little peer-reviewed work directly comparing the performance of the currently available CNTs [18]. Additionally, many of the computer-based testing systems do not even measure the same cognitive domains [7]. These differences make head-to-head comparison of results very difficult if not impossible (see Table 6.2). This can be a significant challenge if an athlete uses one CNT platform at their high school and another CNT platform for their off-campus club sport. Another example is that of an athlete who completed baseline testing on one CNT platform at school as a part of pre-participation exam, and the physician performing a post-injury examination uses a different CNT platform in their office. The tests are often not comparable.

Secondly, there is no consensus among concussion experts on the protocols for the use of the CNTs. Baseline testing and post-injury testing present some similar and some unique challenges. In post-injury evaluations, one thought is that CNTs are most accurate immediately after a concussion injury. The sensitivities for three CNTs were found to be best within 24 hours of the injury. And after 8 days, sensitivity decreased to near false-positive rate in non-injured controls [22]. Another thought is perhaps CNTs are not sensitive enough for the subtle differences seen in the later stages of concussion recovery [18]. For baseline testing, the overall

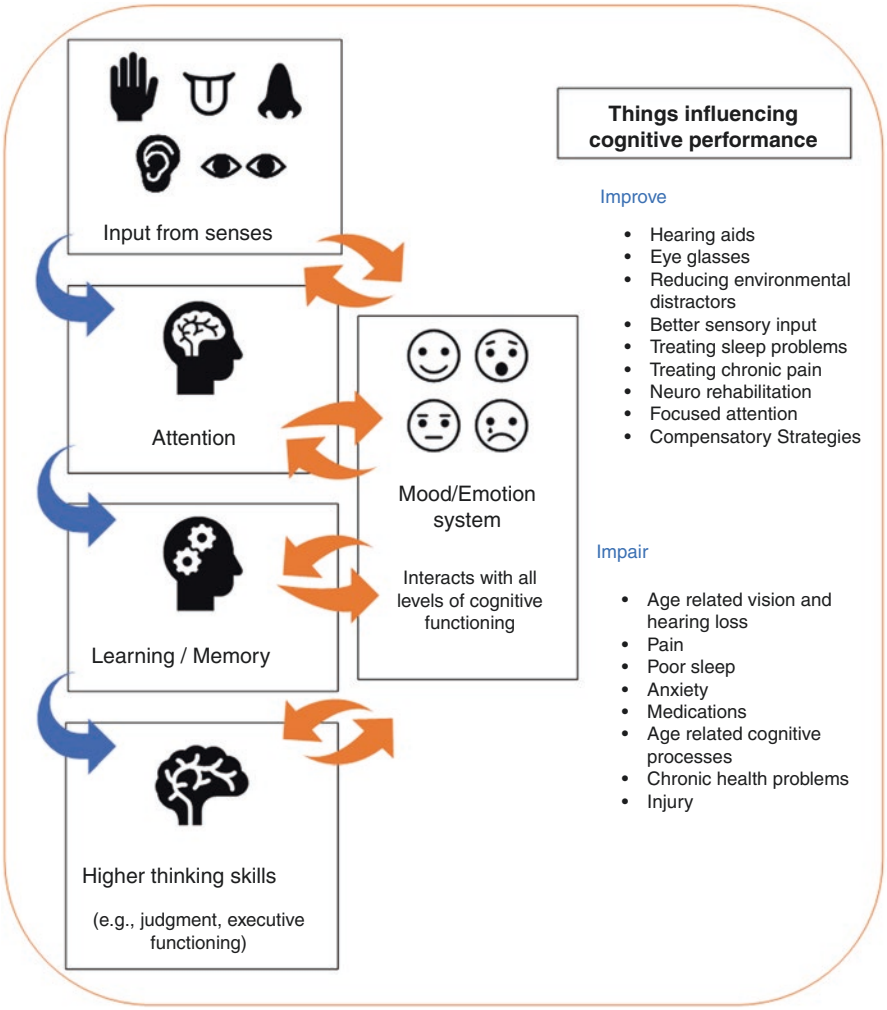

Fig. 6.1 Things that influence cognitive performance

importance and utility of baseline testing have been questioned. Baseline testing allows an individual athlete to serve as their own control rather than relying on normative data to determine when an athlete as returned to "normal" [23]. Some experts report that there is insufficient evidence to recommend the widespread routine use of baseline neuropsychological testing [20]. The concept of routine baseline testing for large populations or teams was near impossible with traditional neuropsychological testing. Newer CNTs have used the ability to perform mass baseline testing sessions as an attractive feature.

One challenge in both baseline testing and post-injury testing is the testing environment. Differences in testing environments can significantly affect the baseline

testing results and post-injury results [15]. Large group baseline evaluation can contribute to poor estimation of premorbid conditions which can have a significant effect on testing results [24]. Neuropsychological conditions are complex, and mood or motivation can affect cognitive functioning and performance on testing [10]. It is well documented that premorbid conditions, such as depression, can significantly affect performance on neurocognitive testing [25]. Other conditions (see Fig. 6.1) such as pain from injury or surgery, poor sleep, medications, fatigue, and hearing or visual deficits requiring eyeglasses or hearing aids that may have been noticed with one-on-one testing may not be recognized with large group testing. For example, sleep deprivation has been shown to have a significant effect on working memory and attention [26]. External factors include anything that can influence sensory inputs such as noise, activity, or noxious smells at the testing location. Both intrinsic and extrinsic factors can have a significant effect on attention. In a large group testing, the distractions and effort can be called into question especially for individuals with underlying attention challenges. The testing environment must allow the test taker to focus and provide the best possible effort.

Question: What do you do if you don't have a baseline test or access to baseline testing results?

In any concussion clinic environment, there will be many cases that present without baseline testing. In these cases, normative data comparison is used for scoring the testing results. Each of the commercially available CNTs have their own normative data base. However, some care should be taken using normative data comparisons. Traditional neurological tests have normative data sets typically grouped by age in 5-year blocks from age 18 to 89. Newer CNTs do not necessarily use the groupings. Some only have 3–4 groups between the ages of 10 and 24. There can be a significant difference in performance norms especially in the younger and older populations. Many factors can affect performance on baseline and post-injury testing. Despite these limitations, baseline testing is already part of many professional, collegiate, and high school concussion protocols. More rigorous scientific data is needed before we can reach a firm conclusion on its validity and use as a gold standard.

Question: Can an athlete purposefully give poor effort during baseline testing or "sandbag" in order to return to play more quickly after a concussion?

Even with an optimal pretest screening and testing environment, the test taker's effort can significantly affect testing results. It has been suggested that athletes may provide suboptimal effort—sometimes called "sandbagging"—in order to return to

their baseline cognitive scores and return to play more quickly [23]. Traditional lengthy neuropsychological test batteries contain built-in measures to assess performance validity. These measures can be useful in understanding performance inconsistencies from comorbid conditions but are also useful in picking up effort-related inconsistencies. The more commonly used CNTs do not employ all of the same measures as classical neuropsychological testing to evaluate performance, but several tests use internal validity indicators in a similar fashion. These measures are designed to identify results that may have been affected by many factors including suboptimal effort, but much of the research on these measures has been industry-sponsored. One study showed 11% of ImPACT savvy college athletes were able to successfully "sandbag" a baseline ImPACT test without activating the test internal validity indicators [27]. Another study showed 30% of ImPACT-naïve nonathlete college students were able to "sandbag" without being caught by the ImPACT validity indicators [28].

Traditional neuropsychological tests were administered and interpreted one-on-one. Even though CNTs may be administered to a large group of people at one time, it is very important that baseline testing be reviewed and examined one test at a time for valid results. In a survey of athletic trainers in 2009 reviewing the use if ImPACT testing, only 55% examined baselines for valid results [29]. If the baseline test is invalid, it cannot reliably be used for comparison as part of the return-to-play decision-making process.

Evolution of Computerized Neurocognitive Testing

Question: Why do I have to use a wired mouse with some tests when I normally use a wireless mouse with my computer?

Rapid evolution of computer systems continues to play a role in the usability and portability of neurocognitive tests. Computer-based testing has evolved as technology has advanced. Testing just a few years ago on desktop systems with wired components evolved to testing on portable laptop systems with Bluetooth or wireless components. Some neurocognitive assessments now utilize portable tablets and cell phones for testing. One of the advantages of the computer-based neurocognitive testing batteries has been the ability of a computer to assess subtests such as reaction time to a very small and sensitive level. Establishing a valid normative database for subtests such as reaction time requires very specific and standardized computer hardware configurations. The power of large normative databases comes from the number of the same tests with the same hardware configurations stored and available for reference. Changing even one component, such as a wired mouse for a Bluetooth wireless mouse, can affect the sensitive results and ultimately challenge the integrity of the normative database. It is difficult for the neurocognitive testing systems to keep up with the rapid technology changes. Newer tests are coming to market on current technology, but even these may be outdated as technology

advances. For example, the traditional ImPACT testing system requires a desktop or laptop computer with a wired mouse (not wireless or Bluetooth mouse) to assure correlation with their normative database. C3Logix and two newer ImPACT products, ImPACT quick test and ImPACT pediatric, utilize tablet-based hardware.

Key Points

- Neurocognitive testing is a well-established and important tool and can be useful to assess for dysfunction in a unique domain not evaluated with other tests.
- Computerized neurocognitive tests are evolving and have their own strengths and limitations.
- It is important to recognize the strengths and weaknesses of the testing system you are using.
- Neurocognitive testing is meant to be one part of a comprehensive concussion evaluation protocol but not as the sole determinant for diagnosis or return-to-play decisions.

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Chapter 7
Concussion Diagnostic Imaging Options

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Clinical Case

A 15-year-old football linebacker collided with another player and has some mild nausea and a headache. After appropriate sideline evaluation, diagnosis of concussion is likely. His parent asks if a diagnostic imaging test should also be performed to aid in diagnosis.

As discussed in the previous chapters, sports-related concussion is largely a clinical diagnosis. In general, the various national and international sports-related concussion guidelines do not recommend routine diagnostic imaging for concussion diagnosis [1–4]. However, as with all medicine, evaluation needs to be individualized and tailored to the specific clinical case. For the remainder of this chapter, we will review when consideration should be given to additional diagnostic imaging and which type may be appropriate in a given clinical scenario.

X-Ray and Computed Tomography (CT)

Question: Should X-ray be ordered for suspected concussion?

The use of X-ray for diagnosis of concussion is not indicated. As per the guideline released by the Pediatric Mild Traumatic Brain Injury Guideline Workgroup established by the Centers for Disease Control and Prevention (CDC), the National Center for Injury Prevention and Control's Board of Scientific Counselors conducted a literature review from January 1, 1990, to July 31, 2015. The goal of this

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review was to create a guideline for recommendations on "diagnosis, prognosis, and management of pediatric mild traumatic brain injury (mTBI)" [3]. As part of this review, evidence was evaluated regarding the necessity of skull radiograph for the diagnosis of pediatric mTBI. The results of this literature review concluded that skull radiographs are not recommended to be used in the diagnosis of pediatric mTBI. Furthermore, skull radiographs are not recommended to be used as a screening tool for intracranial injury. These recommendations are based on two studies that examined the use of skull X-ray in pediatric patients after minor head injury. In one of these studies, possible skull fracture was identified in 7.1% of patients after minor head injury; however, skull radiography is not the best evaluation tool for possible skull fracture. Skull radiography successfully identifies skull fracture in children in only 63% of cases and cannot show details needed to diagnose intracranial injury, such as midline shift. Since head computed tomography (CT) will demonstrate both a skull fracture and many acute intracranial abnormalities, head CT is recommended over X-ray if skull fracture or intracranial injury is suspected [3]. Indications for head CT in mTBI will be discussed at length in the coming text. According to a consensus statement on concussion in sports released after a meeting of the Concussion in Sport Group (CISG) in 2016, there is still much investigation to be done regarding optimal imaging for concussion [4]. This sentiment is corroborated by the most recent position statement on concussion from the American Medical Society for Sports Medicine (AMSSM) [2]. Specifically, they recommend against the use of neuroimaging for suspected concussion. In this position statement, it is recommended that most athletes with suspected sports-related concussion do not need neuroimaging. However, if more serious intracranial injury is suspected, more advanced neuroimaging such as MRI or CT is recommended. Specifically, CT is recommended in suspected skull fractures or in cases of suspicion for acute brain bleed, whereas MRI is more helpful in subacute symptoms but not in the acute setting.

Question: How do I decide if a patient with suspected concussion needs a head CT?

The use of CT for diagnosis of concussion is not routinely recommended. According to guidelines released by the American Academy of Neurology (AAN) in 2013, CT imaging should not be used to diagnose sports-related concussion as currently there is no imaging modality that reliably diagnoses concussion [1]. Instead, CT imaging should be used in situations where more serious traumatic brain injury is suspected. These recommendations are echoed in the aforementioned AMSSM position statement [2]. There are multiple decision-making tools specifically aimed at helping clinicians decide if CT is needed after minor head trauma including suspected concussion. The three most common decision tools include the Canadian CT Head Rule (CCHR), the New Orleans Criteria (NOC), and the National Emergency

X-Radiography Utilization Study (NEXUS)-II. The Pediatric Emergency Care Applied Research Network (PECARN) and the Canadian Assessment of Tomography for Childhood Head Injury (CATCH) are two additional sets of decision-making rules for CT in traumatic brain injury specifically aimed at pediatric patients [5, 6].

The Canadian CT Head Rule was developed from a prospective cohort study completed in emergency departments of ten large Canadian hospitals [7]. Patients included in this study had blunt head trauma, witnessed loss of consciousness and disorientation, amnesia, an initial emergency physician determination of Glasgow Coma Scale (GCS) of 13 or more, and injury within 24 hours. The outcome measures of this study included the need for neurological intervention and clinically important brain injury on CT imaging. The need for neurological intervention was defined as death secondary to head trauma within 7 days, or need for procedures including craniotomy, elevation of skull fracture, monitoring of the intracranial pressure, or intubation secondary to head injury. Clinically important brain injuries were defined as any acute findings on CT that would typically warrant hospital admission and neurological follow-up. There were 3121 patients enrolled in this study, and there was a standardized patient assessment completed by staff physicians board certified in emergency medicine or by residents in emergency medicine residency programs that were supervised. Based on this initial assessment of patients by the physicians as above, decision was made to either obtain CT of the brain or discharge to home with a structured 14-day telephone follow-up with a registered nurse. Those patients with concerning telephone follow-up evaluation were brought back to the ER for re-evaluation and head CT. Based on the results of this study, decision rules for determining those minor head injuries that are at higher risk for clinically significant brain injury or neurosurgical intervention readily identified on CT were created. Those symptoms that stratify the patient as high risk for neurological intervention include GCS <15 2 hours after the injury, suspected open or depressed skull fracture, signs of basilar skull fracture, two or more episodes of vomiting, and age 65 or higher. Those factors leading to risk for clinically significant brain injury seen on CT include amnesia of events prior to impact of greater than 30 minutes, or a dangerous mechanism (i.e., pedestrian struck by a car, ejected car occupant, fall from height > 3 feet or five stairs). Combined, these seven factors encompass the Canadian CT Head Rule [7].

The New Orleans Criteria (NOC) were developed after completion of a two-phase study from December of 1997 to June of 1999 [8]. In the first phase of the study, patients with minor head injury (defined as loss of consciousness in combination with normal neurologic examination and GCS of 15) underwent prospective evaluation using a questionnaire to determine clinical findings that best predicted the presence of abnormalities on head CT. This questionnaire was created after review of the literature on minor head injuries and included age, presence or lack of headache, vomiting, intoxication with either drugs or alcohol, short-term memory deficits, post-traumatic seizure, history of coagulopathy, and evidence of trauma above the clavicle. Findings considered to be abnormal on CT in this study included acute traumatic intracranial lesions such as subdural, epidural, parenchymal

hematoma, subarachnoid hemorrhage, cerebral contusion, or depressed skull fracture. In the second phase of this study, a questionnaire was administered prior to CT assessing for the seven findings found to have predictive value of intracranial injury in the first phase of the study including headache, vomiting, age, drug/alcohol intoxication, deficits in the short-term memory, physical evidence of trauma about the clavicles, and seizure. In this second group of patients, they were separated into two groups that either had one or more of these findings or met none of these classifications. The rates of positive CT scans were again tallied for each group. Of the 909 patients in both phases of this study, 6.7% of those patients meeting one of more of the aforementioned criteria had abnormal findings on CT scan as compared to 0% of patients meeting none of those criteria. Derived from this study are the New Orleans Criteria, suggesting that patients who have one or more of the following should undergo CT to rule out intracranial injury: headache, vomiting, age over 60 years, drug or alcohol intoxication, deficits in short-term memory, physical evidence of trauma above the clavicles, and seizure. These criteria had a sensitivity of 100 percent for identifying patients with positive CT scans in this study. One criticism of the New Orleans Criteria is that it includes headache as a criterion for imaging, yet headache is one of the most common symptoms of concussion. Therefore, many patients with concussions will unnecessarily undergo CT based on these criteria.

The National Emergency X-Radiography Utilization Study (NEXUS)-II is another trial aimed at creating an algorithm for deciding which patients with blunt head trauma should have a head CT [9]. This was a multicenter, prospective, observational study involving 21 hospitals with a total of 13,728 patients. Clinicians decided whether or not to order CT based on clinical judgment rather than a study protocol. Any patient with acute blunt head trauma was enrolled if a CT was ordered for them, and demographic information was obtained for each of these patients in addition to a GCS score. The degree of "neurologic deficit" was recorded for each patient based on a combination of GCS score, presence or absence of motor deficit, abnormal gait, abnormal cerebellar function, and cranial nerve abnormality. If any of these issues was present, the patient was considered to have a neurologic deficit. From this study, eight criteria were identified that were highly associated with intracranial injuries including evidence of significant skull fracture, scalp hematoma, neurologic deficit, altered level of alertness, abnormal behavior, coagulopathy, persistent vomiting, and age 65 years or older. Based on these criteria, 901 of the 917 patients enrolled in this study were identified correctly.

To compare the three aforementioned CT rules for head trauma (CCHR, NOC, and NEXUS-II), a study was done by Ro et al. for the Traumatic Brain Injury Research Network of Korea from 2008 to 2009 [10]. This study was a prospective, multicenter, observational cohort study of patients with blunt head trauma. Patients were assessed using a standardized protocol in the emergency departments where this study was conducted, and then decision to image with CT was based on clinical judgment of the examining attending physician or resident rather than being dictated by the study protocol. Essentially, each of the study designs for the CCHR, NOC, and NEXUS-II was replicated with the patient population in this study.

Inclusion and exclusion criteria similar to those from each of the three original studies were used, and similar demographic and injury information were obtained as well. There were a total of 7131 patients that presented with blunt head trauma in this study, with 9.2% of patients meeting CCHR inclusion criteria, 9.8% meeting the NOC inclusion criteria, and 41.4% of patients meeting the NEXUS-II inclusion criteria. The conclusions of this comparison study were that the sensitivities of all three clinical decision-making tools are lower than reported in the original publications; however, the specificities of these three tools are similar to those figures reported in the individual studies. Notably, the CCHR and NOC did not miss any patients requiring neurosurgical intervention. Although the NEXUS-II criteria decreased the rate of head CT use, it also did miss some cases of clinically significant events requiring intervention. The CCHR criteria had a sensitivity of 100% for the five high-risk factors identified in the study with a specificity of 68.7% and a positive predictive value (PPV) of 4.37% with negative predictive value (NPV) of 100% [7]. With all seven of the CCHR criteria taken together, their sensitivity for identifying clinically important brain injury was 98.4% and specificity was 49.6%, while the PPV was 14.7% and NPV was 99.7% [7]. Regarding the NOC, the sensitivity was found to be 100% and specificity was 25%. The NPV was 100%, while the PPV was 8.2%. The NEXUS-II criteria had a sensitivity of 98.3%, a specificity of 13.7%, and PPV of 50% with NPV of 99.1% [8]. Table 7.1 compares the components of each of these decision-making tools, while Table 7.2 offers a comparison of the sensitivities, specificities, and positive predictive values and negative predictive values of each of these three decision-making tools.

A more recent systematic review done in 2015 by Easter et al. compared the three aforementioned clinical decision-making tools as part of its analysis. The authors of this study performed a search of MEDLINE and the Cochrane Library from 1966 to 2015 to find studies dealing with diagnosis of intracranial injuries.

Table 7.1 Comparison of the CCHR, NOC, and NEXUS-II criteria [7–9]

	Canadian Head CT Rule (CCHR)	New Orleans Criteria (NOC)	National Emergency X-Radiography Utilization Study II (NEXUS-II)
Age	≥65 years	≥60 years	≥65 years
Evidence of trauma	Suspected open or depressed skull fracture Signs of basilar skull fracture	Physical evidence of trauma above the clavicles	Evidence of significant skull fracture Scalp hematoma
Neurologic assessment	GCS <15 at 2 hours after the injury Amnesia of events prior to impact of greater than 30 minutes	Deficits in short-term memory Drug or alcohol intoxication Seizure	Altered level of alertness Abnormal behavior Neurologic deficit
Emesis	Two or more episodes of vomiting	Vomiting	Persistent vomiting
Other	Dangerous mechanism of injury	Headache	Coagulopathy

Table 7.2 Comparing the sensitivities, specificities, positive predictive values, and negative predictive values of the Canadian Head CT Rule (CCHR), New Orleans Criteria (NOC), and the National Emergency X-Radiography Utilization Study II criteria [7–9]. Note that the reported values for the CCHR are for all of the seven previously discussed risk factors taken together for prediction of clinically important brain injury [7]

	Sensitivity (%)	Specificity (%)	Positive predictive value (PPV) (%)	Negative predictive value (NPV)(%)
CCHR	98.4	49.6	14.7	99.7
NOC	100	25	8.2	100
NEXUS-II	98.3	13.7	50	99.1

There were 14 studies that met inclusion criteria with a total of 23,079 patients. In assessing the accuracy of CCHR, NOC, and NEXUS-II in predicting those patients who are at low risk for significant intracranial injury, it was found that in the absence of meeting any criteria of the CCHR, the probability of severe intracranial injury was 0.31%. In the absence of meeting any criteria of the NOC, the probability of severe intracranial injury was 0.61%, and without any positive criteria from the NEXUS-II criteria, this probability was 3.5% [11].

Question: Are there also CT decision-making rules for children with a head injury?

The Pediatric Emergency Care Applied Research Network (PECARN) group conducted a prospective, cohort study of pediatric patients from 2004 to 2006 to develop guidelines for CT imaging in acute, suspected TBI specifically in children [5]. This study enrolled patients under 18 years of age with head trauma in 25 emergency departments that were part of the PECARN group across the USA. The patient had to be within 24 hours of traumatic event, and the outcomes assessed in this study were death from TBI, need for neurosurgery, intubation greater than 24 hours, and hospital admission for greater than or equal to two nights. There were a total of 43,904 patients ultimately enrolled in this study. Emerging from this study were seven predictors of more serious injury in pediatric patients including age younger than 2 years, vomiting, loss of consciousness, severe mechanism of injury, severe or worsening headache, amnesia, non-frontal scalp hematoma GCS score less than 15, and clinical suspicion for skull fracture.

The Canadian Assessment of Tomography for Childhood Head Injury (CATCH) rules were presented after a study was done by Osmond et al. published in 2010. This was a prospective, cohort study carried out in ten Canadian pediatric emergency departments, with patients 0–16 years of age enrolled in this study presenting with minor head injuries. There was a total of 3866 patients participating in this study. There were four high-risk factors and three medium-risk factors that were

identified for intracranial injury. The high-risk factors were those that put patient at increased risk for need for neurologic intervention, and the medium-risk factors placed the patient at higher risk for brain injury on CT. The four high-risk factors include "failure to reach GCS of 15 within 2 hours of injury, suspicion of open skull fracture, worsening headache, and irritability." The three medium-risk factors identified were "large scalp hematoma, signs of basilar skull fracture, or a dangerous mechanism of injury." Those mechanisms that were deemed dangerous included injuries sustained in a motor vehicle crash, a fall from greater than or equal to 3 feet or five stairs, and a fall from a bike without a helmet. It was found that those high-risk factors identified above had a 100% sensitivity for predicting the need for neurologic intervention with a specificity of 70.2%. A patient having any of the four high-risk factors above, or any of the three medium-risk factors, had a sensitivity of 98.1% for identifying CT evidence of brain injury with a specificity of 50.1% [6]. Table 7.3 provides a helpful overview of the PECARN and CATCH criteria, while Table 7.4 compares the sensitivities and specificities of the two decision rules.

A prospective, cohort study done by Easter et al. in 2014 also compared the diagnostic accuracy of PECARN and CATCH in diagnosing traumatic brain injury in children. This study was carried out at a Denver hospital and enrolled children younger than 18 years. There were 1009 patients ultimately included in this study. When PECARN and CATCH were compared regarding the ability to identify clinically important TBI, PECARN had a sensitivity of 100% with a specificity of 62% and a positive likelihood ratio of 2.7. The CATCH decision rules had a sensitivity of

Table 7.3 The PECARN and CATCH clinical decision-making rules [5, 6]

PECARN	CATCH
<2 years of age	Failure to reach GCS of 15 within 2 hours of injury (H)
Vomiting	Suspicion of open skull fracture (H)
Loss of consciousness	Worsening headache (H)
Severe mechanism of injury	Irritability (H)
Severe/worsening headache	Large scalp hematoma (M)
Amnesia	Signs of basilar skull fracture (M)
Non-frontal scalp hematoma	Dangerous mechanism of injury (M)
GCS < 15	
Clinical suspicion for skull fracture	

H indicates those factors that place a patient at higher risk for need for neurologic intervention, while M denotes those at "medium risk" who will likely have a traumatic finding on brain CT

Table 7.4 Comparing the CATCH and PECARN clinical decision-making tools regarding their sensitivity and specificity. CATCH sensitivities and specificities based on primary outcome of need for neurologic intervention [6]. PECARN sensitivities and specificities reported from validation group in the study [5]

	Sensitivity (%)	Specificity (%)
CATCH	100	70.2
PECARN	96.8[1]–100[2]	53.7[2]–59.8[1]

[1]Those older than 2 years old in validation groups
[2]Those less than 2 years old

91%, with a specificity of 44% and a positive likelihood ratio of 1.6. For those injuries requiring neurosurgical intervention, PECARN had a sensitivity of 100%, with a specificity of 61% and a likelihood ratio of 2.6. CATCH, however, had a sensitivity of 75% with a specificity of 43% and a likelihood ratio of 1.3 [12].

In summary, and in accordance with the National Institute for Health and Care Excellence (NICE) recommendations for investigation of clinically important brain injuries, non-contrast CT is the test of choice for detecting clinically important intracranial injuries given its shorter examination time requirements and ability to reveal neurosurgical emergencies [13]. The decision on whether or not to obtain CT in those patients with head trauma can be guided by the use of one or more of the aforementioned clinical decision-making tools in addition to general clinical judgment.

Magnetic Resonance Imaging (MRI) and Magnetic Resonance Angiography (MRA)

Question: Is an MRI or MRA helpful for diagnosis of concussion?

In acute evaluation of suspected concussion or mTBI, MRI is not recommended based on multiple guidelines [1–4]. According to previously mentioned CDC guidelines on diagnosis and management of mild traumatic brain injury among children, MRI should not be routinely obtained in patients with acute symptoms of mTBI [3]. These sentiments are echoed in recommendations from the AMSSM position statement on concussion in sports [2]. MRI should be reserved for patients with recalcitrant symptoms or for those with suspected underlying chronic brain pathology. Most MRIs done for mTBI fail to reveal any pathology or demonstrate findings that may not be clinically significant such as petechiae or white matter shearing injury [14]. Additionally, a publication from the American Society of Radiologic Technologists (ASRT) on neuroimaging of sports concussions points out that while MRI is better at detecting small lesions in TBI, the amount of time needed in the acute setting to complete the test especially in an emergent setting is unacceptable [15].

There is little evidence to suggest the use of magnetic resonance angiography (MRA) in acute concussion. If there are findings concerning for vascular injury on non-contrast CT, either contrast computed tomographic angiography (CTA) or MRA (depending on acuity) should be obtained. MRA is helpful in further elucidating the definition of the vascular walls, whereas CTA is subject to fewer flow artifacts than MRA and also offers better resolution [14].

Other Imaging Modalities

Question: Are there other imaging modalities I should consider obtaining to help diagnose concussion?

Diffusion Tensor Imaging (DTI)

Diffusion tensor imaging (DTI) is a MR method of imaging measuring the diffusion of water, often measured as fractional anisotropy (FA) [16]. This can allow for detection of specific structural brain abnormalities, such as diffuse axonal injury and white matter changes [16, 17]. Changes in FA have been reported after mTBI in both the acute and chronic settings, but the values are variable. Both elevated and reduced values of FA are seen during the acute injury phase [17–19]. DTI is typically used to compare one group to another and not an individual to a group [17, 20]. Standards for the use of DTI are still being developed, but many feel this is a very promising imaging modality. Some studies have shown this modality to be superior to other more common imaging modalities, such as CT or MRI, for detecting subtle white matter injuries [20]. Additional, longitudinal studies are needed to determine whether DTI should be clinically utilized in the future.

Functional MRI (fMRI)

Functional MRI works by assessing regional blood flow variation in the brain with more active brain areas having more blood flow. It can be used to monitor areas of brain activation during specific tasks. Although there is evidence for some changes in the prefrontal cortex of athletes with post-concussive syndrome and it may be able to predict recovery of concussion with changes seen in some athletes with chronic concussive symptoms [20], additional studies are needed to further assess its use as a clinical diagnostic tool.

Single-Photon Emission Tomography (SPECT) and Positron Emission Tomography (PET)

Both single-photon emission tomography (SPECT) and positron emission tomography (PET) are functional imaging modalities. SPECT uses radiotracers to indicate where blood flow is in the brain. Brain injury is inferred by decreased blood flow. Its use seems most helpful in cases of moderate or severe injury.

Positron emission tomography (PET) also uses radiotracers, but these radiotracers indicate areas of binding or substance uptake in the brain. The information it provides is linked to levels of a specific chemical or substance in the brain.

Both SPECT and PET produce images that are of poorer resolution compared to images generated by CT or MRI [16, 21]. These tests are quite expensive, involve radiation, and are not widely available. For these reasons and others, SPECT and PET are not recommended for use in sports mTBI.

Magnetic Resonance Spectroscopy (MRS)

Magnetic resonance spectroscopy (MRS) is a metabolic imaging technique that uses chemical shift information to combine neurochemical information with neuroanatomy. Using the standard clinical strength of MRI, this method can typically detect the following substances in neural tissue: N-acetyl-aspartate (NAA), creatine (Cr), choline (Cho), myoinositol (mI), and glutamate (Glu) plus glutamine (Gln), known as Glx. Some studies have shown lower NAA levels and higher Cho levels after sports mTBI in both gray and white matter; however these levels seem to be influenced by history of mTBI with the biggest changes typically seen after the first, clinically diagnosed mTBI. It also seems that these levels also change over time, regardless of mTBI history. Head impacts that do not lead to clinically diagnosed mTBI have also shown some changes in NAA and Cho levels [19]. In a review in the pediatric population, no significant differences were found when comparing those with and without mTBI [18].

For the above reasons, single-use MRS does not currently appear to be helpful for sports mTBI diagnosis in the clinical setting. Additional studies investigating longitudinal changes and standardization would be helpful to further clarify the role of MRS in the clinical assessment of sports mTBI.

Key Points

- Diagnostic imaging is not routinely recommended for use in concussion.
- If there is concern for another diagnosis, such as skull fracture or hemorrhage, a CT scan may be indicated.
- There are multiple decision rules available to determine the need for a head CT in adults (CCHR, NOC, and NEXUS-II) and children (PECARN and CATCH).
- Other diagnostic imaging modalities such as DTI, fMRI, PET, SPECT, MRS, and others are being investigated to determine what, if any, clinical role they may play in the diagnosis of concussion.

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Chapter 8
Concussion Grading and Prognostic Factors

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Clinical Case

A 15-year-old female with a past medical history of untreated anxiety and a single concussion with protracted recovery earlier this season is brought to your office after getting hit on the back of the head by a volleyball during a serve on practice 2 days ago. The athletic trainer suspected a concussion without loss of consciousness (LOC) and took her off the court for sideline evaluation. Her immediate post-concussion symptom score (PCSS) was elevated, mostly complaining of significant headache, dizziness, and nausea.

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What defines prolonged recovery after a concussion?

The most recent International Consensus Statement on Concussion in Sport reports full recovery of symptoms following sport-related concussions (SRC) typically occurs approximately 10–14 days after injury [1] and within 1 month in children aged 0–18 years [2]. Delayed recovery occurs when symptoms persist beyond these expected time frames. Current literature trends suggest using the term persistent post-concussive symptoms (PPCS) (i.e., in the physical, cognitive, sleep, and mood domains) following SRC to reflect the failure of normal clinical recovery [1]. Research has increasingly focused on determining which risk factors increase the likelihood of PPCS. Nonetheless, concussion literature regarding recovery time is complex, mixed, and difficult to interpret definitively, whereas each recovery is unique and will follow its own trajectory [3].

Do medical providers still use a concussion grading system to identify severity and prognosis?

In the late 1990s, multiple concussion grading systems were created to classify concussion severity in the attempt to predict outcomes and to help guide return-to-play protocols. These grading systems included the Colorado Medical Society Guidelines, American Academy of Neurology (AAN) Guidelines, and the Cantu Guidelines [4, 5]. They incorporated a time frame and symptomatological variation of LOC, post-traumatic amnesia (PTA), and post-concussion signs/symptoms. The importance of LOC and PTA as reliable markers of severity has been questioned, and recent evidence has failed to correlate the grading systems with injury severity and has not been found to be a reliable predictor of recovery time [6, 7]. These grading systems are no longer used among concussion providers nor recommended by position statements. Current guidelines recommend using different prognostic factors such as age, sex, and past history, among others, to help predict clinical recovery by use of symptom scales and cognitive testing [3, 8].

Should clinicians screen for prognostic factors?

Screening for the presence of prognostic risk factors (Table 8.1) is recommended to initiate earlier intervention and/or appropriate referrals. Both the AMSSM consensus guideline for SRC and the most recent CDC guidelines for pediatric concussion recommend screening for potential risk factors that may contribute to prolonged recovery [3, 8]. These will be discussed in further detail later in this chapter.

Table 8.1 Risk factors that may predispose to a prolonged recovery

Age < 18
Female gender
Symptom burden (multiple symptoms)
History of mental health disorders
Past history of concussion
Severe on-field signs and symptoms
Past history of migraines
Post-traumatic headaches (especially delated onset or persistent)
Prolonged rest >5 days
History of learning disability and attention deficit disorder
Cervical spine dysfunction
Vestibular and oculomotor dysfunction
Delayed reporting or "playing through it"
History of substance abuse
Suboptimal heart rate threshold on BCTT

In the case above, which are the patient's risk factors that may predispose her to a prolonged recovery?

Age < 18 Age is one of the most studied factors related to outcomes after SRC. The few available studies directly examining age-related differences in SRC risk and recovery have produced inconsistent results [9]. Yet, there is some evidence for an age-gradient and level-of-play association with clinical recovery. Overall, trends show faster recovery in professional athletes, followed by college athletes and then HS athletes [6, 10]. Moreover, a large prospective multicenter study suggested that the teenage and high school years might represent a more vulnerable stage for slow recovery, than those of primary school age [11]. However, literature documenting differences in SRC recovery outcomes among adolescents and adults is limited. More research in this area is needed to establish a stronger correlation.

Female Sex The literature on sex differences regarding recovery time and persistent symptoms is mixed. Overall evidence supports that females take longer to recover, are more likely to have prolonged symptoms, and have greater neurocognitive impairment than their male counterparts [11–14]. Multiple sex-determined differences have been compared and may account for this overall trend. These include differences in neck strength, injury biomechanics, and injury rates in females, as well as higher pre- and post-injury symptom reporting [15]. Ongoing basic science and clinical research may help solidify these underlying differences [16].

Symptom Burden In an adult cohort with concussion, initial symptom burden (i.e., higher PCCS at first clinical visit within 2 weeks of injury) was more predictive of

a SRC recovery >28 days than age, sex, concussion history, and migraine/headache history [17]. This agrees with most studies to date, which consistently show that the strongest predictor of slower recovery from concussion is greater symptoms severity following injury both immediately and in the first few days following the injury [1, 6].

History of Mental Health Disorders Behavioral problems after a concussion are very common and may include depression and anxiety. There is some evidence that pre-injury mental health problems [18], particularly depression and anxiety, have been associated with persistent symptoms after a concussion [11, 19]. Short-term elevated anxiety levels and new-onset anxiety disorders are four times more likely after a mild TBI than after an orthopedic injury [20]. A threefold risk of depression in an adolescent cohort after concussion has also been reported [21]. Prolonged rest or activity restriction has been well described to contribute to anxiety and depressive symptoms in the general population after injury/illness [22]. This is similar after concussions, highlighting the importance of returning patients to their normal environment and activity as soon as medically possible. It is not yet clear, however, whether these are new diagnoses after a concussion or an exacerbation of an underlying predisposition or a subclinical premorbid condition. Referral to a mental health provider for cognitive behavioral therapy should, therefore, be considered when appropriate.

Past History of Concussion Literature is mixed with regard to associating prior concussion history with clinical outcomes. Whereas some studies have not shown correlation with prolonged recovery [23–25], large-scale retrospective analyses show a link between concussion history and increased risk for symptoms lasting more than 4 weeks [11, 14, 26, 27]. In addition, having a previous concussion with symptoms for ≥1 week also correlated with longer recovery in children [11]. Prospective research is needed to strengthen these associations; however, we must certainly consider past concussion history in the clinical setting.

On-Field Signs and Symptoms

Acute signs and symptoms, including presumed injury severity factors, have been greatly scrutinized in literature. While there are mixed findings, most studies have not associated LOC or PTA with recovery [6]. On the other hand, there is some evidence linking presence of retrograde amnesia and slower recovery time, yet a strong relationship has not been demonstrated [6]. A recent systematic review supports that on-field dizziness in children was a consistent predictor for prolonged recovery [28]. Other physical symptoms including nausea, fogginess, and balance have also been associated with prolonged recovery in very few studies [9]. Overall, literature studying individual symptom association with outcome is limited.

What other prognostic factors are associated with prolonged recovery after concussions?

Past History of Migraines Pre-injury history of migraine has not been related to outcome in most studies [6]. However, a large, multisite, prospective, and well-powered study reported that a pre-injury history of migraine was associated with risk for symptoms lasting more than 4 weeks [11]. More research is needed to definitively establish this association.

Post-traumatic Headaches Post-traumatic headaches (PTHs) are a common clinical finding in patients who suffered sport-related concussions [9] with approximately 90% of patients experiencing them. Most studies show that acute and subacute PTHs are associated with persistent symptoms [6]. Persistent headaches (>7 days) have been linked to increased recovery time, delayed return to sport, poor cognition, and more symptomatology [9]. It has been reported that PTH with migraine features makes an athlete 7 times more prone to recover in >21 days compared to those without these types of headaches [29]. Moreover, the emergence of PTH after 7 days of injury was linked to longer recovery, slow reaction time, reduced memory performance, anterograde amnesia, and an increase in the number of reported symptoms such as nausea, dizziness, and visual changes, among others [9, 30]. More evidence is needed, but all of these factors related to PTH seem to contribute to prolonged recovery and return-to-play.

Prolonged Rest Basic science models hypothesize that neurophysiologic disturbances may return to baseline with restriction of neurometabolic demand, i.e., rest. A brief period of 24–48 hours of physical and cognitive rest remains the initial step in concussion management [1, 8, 22]. Although immediate removal from the sport and relative rest have shown to be necessary to prevent prolonged recovery [6, 31], a randomized controlled trial of patients aged 11–22 with acute concussion showed that those who underwent strict rest for 5 days vs. usual care (24–48 hours of rest followed by stepwise return-to-activity) had more daily persistent symptom score and slower recovery [32]. For this reason, strict rest protocols are currently being revised [22]. Many studies have reported that extreme withdrawal or "cocooning" patients from daily activities such as school, sports, technology, and the usual pattern of socializing can be linked to psychological issues [8, 33]. Moreover, these issues may translate into poor coping mechanisms which can further lead to increased anxiety and depression—both associated with prolonged recovery time [22]. These maladaptive neuropsychiatric disorders combined with physical deconditioning may exacerbate post-concussive symptoms and delay recovery.

History of Learning Disability and Attention Deficit Disorder Pediatric patients with a history of learning disabilities have an overall greater lifetime concussion incidence [34]. Interestingly, this population has a high prevalence of concussion-like symptoms even in the absence of a concussive injury [35]. Yet, studies have not

clearly linked learning disabilities with having more post-concussive symptoms [36]. Nonetheless, in young patients, a history of attention deficit hyperactivity disorder (ADHD) has been associated with prolonged recovery from concussion [36]. A single-center case-control study involving pediatric patients found that a prior history of ADHD predicted a prolonged recovery (>28 days) [14]. Another study reported that HS students who sustained a concussion and had pre-injury history of ADHD exhibited a longer, though not statistically significant, time of recovery. In this study, the athletes with a pre-injury history of ADHD recovered in 16.5 days compared with the control group who took an average of 13.5 days [37]. In addition, athletes with ADHD can have worsening of the ADHD symptoms after a concussion [38]. Special consideration is required in order to analyze neurocognitive testing in this population. If the patient is taking a stimulant medication, both baseline and post-concussion testing should be performed after the patient has taken their medication to permit consistent interpretation [38]. More information is needed to strengthen the correlation between ADHD (among other learning disabilities) and prolonged recovery.

Cervical Spine Dysfunction Due to the close proximity of the skull to the cervical spine, the medical provider must consider cervicogenic dysfunction as a possible contributing factor for persistent post-concussive symptoms. At the time this chapter was written, there is no standard definition for cervical spine dysfunction (CSD) [39]. One study defines CSD as at least one subjective symptom of cervical spine injury that may include neck pain, headache, or dizziness. In addition, the patient must have some evidence of cervical spine injury during physical examination [39]. Both conditions can have similar mechanism of action and present with nearly similar symptoms [40]. Early findings suggest that CSD may contribute to the development of PPCS, for which more research is needed to establish a stronger correlation [39, 40]. Early intervention with physical rehabilitation including cervical manipulation and muscle strengthening is an important aspect of treatment in this population [40]. Identifying the etiology of certain concussion symptoms from cervicogenic dysfunction source is vital on the evaluation of concussion patients to ensure proper diagnosis and early management.

Vestibular and Oculomotor Dysfunction Vestibular and oculomotor dysfunction are common complaints in the concussed patient. Studies have identified that 81% of concussion patients may develop some form of vestibular dysfunction [41]. Moreover, patients found with vestibular dysfunction or dizziness on initial evaluation tend to take more time to return to school/sports than those without [41–43]. In regard to oculomotor complaints, convergence insufficiency is one of the main causes for eye strain, headaches, and blurred vision [44]. Oculomotor disturbances related to concussion may affect reading, note-taking, and technology use among other daily life activities [41]. More specifically, as seen in a retrospective study, vestibulo-ocular deficits are one of the many risk factors that may extend recovery [45]. Literature has well-documented deficient performances on neurocognitive

tests among patients with receded near point of convergence (NPC) [46]. Additionally, a recent study reported that patients with convergence insufficiency revealed gait deficits not present in either healthy controls or those without NPC insufficiency [47]. For this reason, early symptom identification with subsequent vestibulo-ocular rehabilitation is important to improve the patient's vestibulo-ocular symptoms and prognosis [48].

Delayed Reporting or "Playing Through It" Underreporting of concussion symptoms is an issue that can curtail the removal of an athlete from physical activity following concussion [49]. Studies have revealed that between 30% and 50% of concussions go unreported by athletes, displaying a dependence on self-reported symptoms and the need for objective diagnostic measures [49]. A recent study indicated that athletes who were not immediately removed from activity following concussion had a longer recovery and missed more days of activity versus those who were quickly removed from play [31]. Delayed reporting of symptoms was linked to a recovery that lasted 5 days longer than those who immediately reported symptoms [31]. Other related studies have also found that delayed removal from play has been associated with a longer recovery time [6].

Substance Abuse In many cases, substance abuse may mask the symptoms of mild TBI thus challenging the assessment of risk factors that may alter prognosis [50]. Literature has reported deficits in executive functioning and memory among abusers, as well as poor post-concussive mood stability among patients with a history of abuse. All of these deficits exacerbate post-concussive symptomatology and have been shown to prolong recovery [51, 52]. Moreover, patients who have a history of mood disorders under treatment may exhibit chronic alterations of brain structures that regulate emotions caused by long-term substance (i.e., medication) use. These alterations along with post-injury substance abuse also curtail concussion rehabilitation [52, 53].

Heart Rate Threshold The Buffalo Concussion Treadmill Test (BCTT) is a validated analysis that allows measuring the amount of aerobic exercise that is safe to perform following concussion. It is based on the maximum heart rate (termed heart rate threshold [HRt]) achieved when exacerbation of symptoms occurs [54]. In a randomized controlled trial in acutely concussed adolescents, a HRt of < 135 bpm during BCTT was associated with a recovery of >21 days [55, 56]. This cutoff value was only studied in adolescents and may not be useful in other patient populations due to differences in cardiovascular fitness and those with baseline heart rate variability [57]. These variables must be taken into account in order to reach a more standardized cutoff. Moreover, a recent study on concussions, patients discussed that a heart rate change (HRt—baseline HR) of less than 50 bpm was 73% sensitive and 78% specific for predicting prolonged recovery (>30 days) [58]. More prospective studies are needed in this regard to further elucidate these associations.

Is there any evidence for risk factor modification to improve outcomes after concussion?

Although age and gender are not modifiable, early risk factor modification is key in the management of concussion. The correct identification of these prognostic factors can help guide rehabilitation in concussed athletes. *Return-to-activity/play programs need to be individualized for each patient in order to achieve better outcomes.* As described above, there is strong evidence for immediate removal of a person from high-risk contact activity, short term (48–72 hours) of rest, followed by symptom limited activity with the goal of avoiding protracted recovery. Reinforcing these recommendations, a prospective multicenter cohort study by Grool and colleagues showed that children and adolescents who participated in at least light aerobic exercise within 7 days following an acute concussion had a lower risk of PPCS at 28 days vs. controls who didn't engage in physical activity [59]. In addition, there is increasing support for use of therapeutic submaximal HRt activity in patients with chronic symptoms or PPCS to facilitate recovery [55, 56, 60]. Overall, growing evidence, current consensus, and clinical experience suggest that gradual return of physical activity should begin as soon as tolerated following concussion while avoiding high-risk activities that could result in another TBI [1, 22].

Each concussive injury is unique in its mechanism, symptomatology, and formulation of modifiers that may impact prognosis. Management should be individualized according to the athlete's presentation (Table 8.2). Clinicians should be aware of their State law and league guidelines for safe return-to-play.

Table 8.2 Treatment options for persistent post-concussive symptoms (PPCS)

Early identification of concussion symptoms and screening of risk factors for prolong recovery
Adequate referral and management of previously diagnosed mental health disorders
Appropriate management of past history of migraines or headaches
Avoidance of prolonged strict rest
Referral if needed for evaluation of learning disability and treatment of attention deficit disorder
Physical therapy to address cervical spine, vestibular, and/or oculomotor dysfunction
Identification or management of substance abuse
Buffalo Concussion Treadmill Test (BCTT) to develop an early return-to-physical activity program

Key Points

- Concussion grading systems are no longer used in the medical community.
- Screening for the presence of prognostic risk factors is recommended to initiate earlier intervention and/or appropriate referrals.
- Demographic characteristics such as younger age (<18 years old) and female sex may be associated with longer recoveries.

- Past history of concussion, migraines, psychiatric, and learning disabilities (e.g., anxiety, depression, ADHD) may be linked with persistent post-concussive symptoms.
- Individual symptoms have variable association with longer recovery (e.g., post-traumatic headache, dizziness, fogginess, etc.) as well as cervicogenic and vestibular/oculomotor dysfunction after a concussion.
- Higher initial concussion symptom scores (i.e., symptom burden) have the strongest association with prolonged recovery.
- While prolonged rest for >5 days has negative correlation with recovery, early progression of physical activity within the first week (guided by the symptom exacerbation threshold) shows an inverse relationship.
- Return-to-activity/play programs need to be individualized for each patient in order to achieve better outcomes.

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Chapter 9
Concussion Treatment

Matt Roth and Drew Nowakowski

Clinical Case

A mother and her 11-year-old son present for an acute visit and are concerned that he may have sustained a concussion the day prior after colliding with another player at soccer practice. Currently he states that he has a headache, has sensitivity to light, and has been feeling fatigued. He is currently attending school, and the mother wants to know when he can return to normal activity.

The current management of concussions is based upon consensus guidelines, clinical experience, and observational studies. Currently there is not a standardized way of managing concussions, as they are all different in severity, degree of symptoms, and progression/recovery. Initial management though focuses around protecting the patient from sustaining additional injury by removing them from competition or restricting them from physical activity. Additionally, cognitive rest is advised early following the injury. With this comes a great deal of education about what levels of activity are acceptable in the period of time while the brain is still recovering. General brain health measures such as adequate hydration, healthy dietary intake, and adequate sleep (see more below) are encouraged as these can be overlooked as a patient is dealing with multiple other symptoms.

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Question: If rest is good, should patients be advised to sleep or rest all day in a dark and quiet room?

Regarding rest, it is generally recommended that the individual completely rest for a 24–48-hour period and gradually ease back into physical activity that involves noncontact activities that do not exacerbate their symptoms. A rapid return to activity is more likely to exacerbate symptoms than a gradual return [1]. Patients and their loved ones may have concerns about the potential for permanent symptoms after a brain injury. It is important to reassure the patient and their parents that their symptoms will almost certainly resolve with appropriate conservative treatment. Often most symptoms resolve within the first 10–14 days. However, 15–20% of individuals may require more than 30 days before a safe return to play is achieved, with a higher percentage of these more prolonged recovery patients being in the youth and high school population [2–4]. Patients often feel pressured in doing more activity beyond their symptom threshold because of the outside demand from school and by parents, their team, work, or family. Previous recommendations centered around complete physical rest. However new research has highlighted that light physical activity such as biking, jogging, and household chores that don't aggravate symptoms sustained after a concussion, improved symptoms at a faster rate, and decreased the risk for post-concussion symptoms [5].

Symptoms of a concussion can also be exacerbated with cognitive stimulation. Depending on the severity of symptoms, the physician may need to prescribe varying amounts of brain rest. Much attention has been given to completely refraining from phones, video games, and other forms of screen time. Current literature shows that patients who engage in high cognitive levels have longer recovery times compared to patients who only engaged in low levels of function [6]. However, avoiding all cognitive activity may be detrimental as well [1]. A more accurate recommendation may be to limit activities that aggravate the patient's particular symptoms, but not put patients at risk for complete social and cognitive isolation. For example, if phonophobia is present, reducing volume on devices and using noise cancelling headphones or ear plugs may be of benefit. If photophobia or bright screens are aggravating, screens can be dimmed, and duration of exposure can be limited. If there are eye tracking or balance issues, certain content can be limited such as avoiding watching fast action sports and video games while the brain recovers. School or work may need to be restricted if the patient is having difficulty concentrating or is having worsening symptoms due to increased screen time or reading (for further details, see return to learn Chap. 10). These are important patient education areas as many students are tempted to substitute their reduced school time with recreational screen time. Results from computerized neurocognitive testing may help direct what specific accommodations may be required or helpful. During the period of rest, light mental activities that do not aggravate symptoms are allowed. Close follow-up is also required in order to assure the patient's symptoms are improving and not worsening. Light mental activities may include board games, puzzles, or light reading (even on paper if better tolerated).

Timing of follow-up is determined by severity of initial symptoms, within a week in most cases but sometimes as soon as 2–3 days later if symptoms are severe. During the recovery phase, if the patient's symptoms begin to recur or worsen, then they need to decrease their physical or mental exertional level to a more manageable level that is not exacerbating their symptoms. The Buffalo Concussion Treadmill Test (BCTT) is a formal test to measure the amount of aerobic exercise that is safe to perform so as not to exacerbate symptoms of concussion [7]. The BCTT is not widely utilized at this time, although as the test becomes more validated, its simple equipment requirement of heart rate monitoring and a treadmill make it a potentially promising protocol to individualize recommendations to determine safe amounts of subthreshold exercise parameters for patients. Also, recent studies have shown that people who practice complete physical and mental brain rest for greater than 5 days have shown longer recovery times [6]. For patients that want to return to sport, they need to follow a return to play protocol that is specifically tailored to the activity that they are in. For example, someone who is a swimmer will have an entirely different return to play protocol than someone who plays contact sports such as football (see return to play Chap. 10).

Medications

Question: The mother proceeds to ask what medications would be safe for him to take for his headache?

Concussions can cause a wide variety of symptoms, but the most common symptom that concerns patients is a headache. By practicing relative mental and physical rest from activities, the majority of symptoms should resolve on their own with time. The current literature on medical management of concussions is lacking, but some medications are used in moderation for patient symptoms. It is important to differentiate whether the headache is more migraine or tension related. Migrainous headaches typically have more of a throbbing sensation often associated with nausea and can be aggravated with light, sound, or certain smells. Current treatment of headaches is largely empiric, but more specific therapy can be guided depending on what is actually causing the headache. In the acute setting, acetaminophen and NSAIDs can be used. Typically, the NSAID medications are withheld for the first 24 hours after injury to limit the possibility of bleeding. Chronic use of NSAIDs and acetaminophen should be avoided and limited to shortest duration possible. They can cause rebound headaches after stopping them if they have been used for a sustained period of time [8]. For headaches that are not alleviated by simple analgesics and have migraine features, triptans may be the best treatment option. For headaches that are occurring more frequently and persistent (for several weeks), preventive medications such as tricyclic antidepressants may be considered. These medications usually are not initiated during the initial acute treatment phase for the first few

weeks as most symptoms generally resolve within a few weeks. These medications also typically require several weeks duration to demonstrate some benefit. There are retrospective studies published in regard to amitriptyline having a positive and beneficial effect on concussion recovery. However, there is contrary literature showing minimal benefit. Currently, there have not been any controlled trials published in regard to the use of amitriptyline in the pediatric population [9–11]. Further treatment of persistent concussion symptoms and post-concussion syndrome can be found in Chap. 11.

Tension-related headaches typically are described as a tight- or pressure-like sensation and may involve cervical pain with exacerbation with neck movement. If neck symptoms and tension-related headaches are predominant symptoms, education about posture, early neck stretches, and range of motion exercises are important. Also, use of physical therapy and Osteopathic Manipulation Treatment (OMT) in combination with analgesics for tension-related headaches may also be helpful. Physical therapy and OMT may be initiated early in the treatment process and may be particularly beneficial if there is a cervical component to their headaches that can frequently occur when there is a whiplash-type mechanism at the time of their concussion injury. Muscle relaxers and antispasmodic medications are rarely used because their side effects often compound other concussive symptoms.

Question: What treatments are available for dizziness from a concussion?

For prolonged symptoms of a concussion, a multidisciplinary team is often required. Patients who have an initial complaint of dizziness typically have a prolonged recovery time, and this may influence the decision to be more aggressive with treatment at the follow-up visit if symptoms persist. In the acute setting, dizziness should be managed conservatively. Simple measures recommended for other patients with dizziness not due to concussion may be considered, like reminding patients to be cautious with rapid position changes and rapid head movements.

Although meclizine is not FDA approved for concussion-related dizziness, it may be considered if symptoms are severe. Patients should be cautioned about possible sedation side effects. This may further exacerbate the pre-existing fatigue. For chronic dizziness and visual disturbance, a referral to vestibular and/or oculomotor rehabilitation may need to be made. There are many different causes of dizziness in concussions including benign paroxysmal positional vertigo, balance dysfunction, cervicogenic dizziness, exercise-induced dizziness, visual motion sensitivity, and vestibulo-ocular reflex impairment. Depending on the cause, there are a variety of different physical therapy treatments that can be applied including balance training, gaze stability training, manual therapy, progressive dynamic exercise, and gradual exposure to visually stimulating environments. Recent literature has shown that early incorporation of vestibular rehabilitation can improve

recovery times. A recent review article analyzing four different trials was published showing that there is moderate evidence for the incorporation of vestibular rehabilitation in the recovery of concussions for patients experiencing recurrent dizziness [12].

Question: Is there any medication that can improve the cognitive symptoms of a concussion?

Currently, there are no medications that are approved for the treatment of the cognitive symptoms associated with concussions. Most of the speculation regarding medication usefulness in concussion management is derived from evidence with animal studies or from human studies in patients with more severe traumatic brain injury. Extrapolating this data for use with concussion is difficult due to the variety of symptom presentation and relatively short duration in most mild brain injuries, making demonstration of significant symptom improvement difficult. In general, consideration of daily medications to manage cognitive symptoms should only be initiated if symptoms are prolonged. The authors suggest medications may be considered when symptoms have been present for at least a month after concussion. In addition, symptoms should be severe enough that it impacts ability to function with school or work. Prescribing providers should be familiar with both the medication and concussion management. Of note, medication initiation may also complicate return to play decisions. Determination of whether symptoms are controlled only because medications are masking symptoms or their brain has truly recovered is often difficult when taking additional medications. In such cases, patients will often need to be given time to reduce or wean off medications to verify they have recovered.

Some medications have been evaluated for the management of cognitive deficits with concussion although their use is controversial and routine use is not advised. Amantadine is used as a medication by some practitioners to help speed cognitive efficiency and decrease overall symptom scores. Some early literature had shown that amantadine may have an effect on overall functional improvement [13]. It is traditionally used as an antiparkinsonian agent as well as an antiviral agent. Amantadine causes the release of dopamine and is thought to have NMDA receptor antagonist effects. This may help contribute to its proposed neuroprotective effects after sustaining an injury. A randomized control trial designed by Meythaler et al. showed that when compared to a placebo group, there was a consistent trend toward more rapid functional improvements [14]. In this study regardless of the timing of medication initiation (days or even several weeks after the initial injury), there was still a statistically significant benefit. Another more recent study showed that amantadine compared to a control group had greater improvements in reported symptoms, verbal memory, and reaction time performance. Alternatively, a recent publication by Hammond et al. shows no benefit versus

placebo in regard to improved cognitive function. Their study measured 119 participants over the course of a 60-day period. The treatment group was given amantadine 100 mg twice daily. Their general cognitive index, learning memory index, and attention/processing speed index were measured at day 28 and day 60 and showed no statistical benefit [15]. Amantadine currently is not on patent and is relatively affordable for the patient. Along with this, it has only a mild side effect profile. Treatment for concussion with amantadine is still considered off-label use, and there is not a specific time frame for which the patient should be on the medication, but duration of use is often tailored around school or work requirements. Given amantadine's conflicting data, it deserves further study and consideration for use in concussion.

Question: Do Attention Deficit Disorder medications have a role in treating attention symptoms of a concussion?

Patients often complain of difficulty with processing as well as inattentiveness after sustaining a concussion, and this often brings up attention deficit medications as a consideration in the treatment of concussions as well. Methylphenidate is a frequent consideration in this situation because it is well tolerated and has been shown to be effective with severe brain injury. Some studies have been published which show the added benefit of using this medication to help with concentration and processing speed during recovery [16]. There is literature which has shown that the use of methylphenidate is best used in the subacute treatment phase of a concussion. Long-term use of stimulants has not shown any added benefit compared to patients not taking this medication [17–20]. Providers should keep in mind that methylphenidate is not FDA approved specifically for inattentiveness due to concussion. Atomoxetine is another medication that may be attractive especially in the pediatric population because of its effectiveness for managing inattentiveness in this age group and the fact that it is not a controlled medication. However, data regarding its use in concussion symptom management is lacking [16]. Use of medications affecting cognition during concussion recovery is controversial and should be used with caution, weighing risks, and benefits with individual patients.

Question: Should nausea medications be given during a concussion? (Table 9.1)

Nausea is another common symptom seen in concussions. Ondansetron is frequently used in the acute setting but should be limited due to potential side effects such as drowsiness, dizziness, and low blood pressure. Other strategies such as smaller more frequent meals may also be helpful. Focus on hydration and healthy fluid consumption as a more practical approach.

Table 9.1 Concussion medications

Medication[a]	Mechanism of action/ typical indication	Targeted concussion symptoms	Typical dosing	Potential side effects
Amantadine	Potentiates CNS dopaminergic response, used in Parkinson's disease	Improved cognitive efficiency	100 mg twice daily	Dizziness, dry mouth, nausea, constipation, visual disturbance
Methylphenidate	CNS sympathomimetic stimulant, used in ADHD	Improved processing speed and inattentiveness	5–15 mg twice to three times daily (immediate release) 10–20 mg once or twice daily (extended release)	Abdominal pain, nausea, decreased appetite, tachycardia, headache exacerbation, nervousness
Atomoxetine	Inhibits norepinephrine reuptake, used in ADHD	Improved inattentiveness	40 mg once or twice daily	Dry mouth, headache, abdominal pain, decreased appetite, nausea, insomnia, fatigue
Zolpidem	Interacts with GABA-benzodiazepine receptors, used for insomnia	Difficulty with sleep	5–10 mg before bedtime	Headache, drowsiness, dizziness, lethargy, lightheadedness, decreased mood
Trazodone	Antagonizes serotonin and alpha adrenergic receptor, inhibiting serotonin reuptake, used for insomnia and major depressive disorder	Difficulty with sleep	25–50 mg before bedtime	Drowsiness, dry mouth headache, nausea, blurred vision. Black box warning for increased suicide risk in children, adolescents, and young adults
Amitriptyline	Tricyclic antidepressant, inhibits norepinephrine and serotonin reuptake, used in migraine headache prophylaxis among other indications	Headache management and/or difficulty with sleep	10–25 mg nightly	Drowsiness, dry mouth, dizziness, constipation, blurred vision
Ondansetron	Selectively antagonizes serotonin 5-HT3 receptors, used as antiemetic	Nausea	4–8 mg every 8 hours as needed for nausea	Headache, constipation, fatigue, dizziness

[a] No medications are FDA approved to treat concussion-specific symptoms

Question: How does sleep play a role in recovery?

Sleep disturbance is also common in acute brain injuries. It is important to educate the patient on proper sleep hygiene and the importance of rest and good sleep in the recovery of concussions. Short naps, limited to around 20 minutes, may be appropriate in the acute recovery phase. It should be clarified that daytime sleep or naps should not be so prolonged or frequent that it interferes with a sustained sleep overnight. In addition to this, educating the patient in regard to caffeine consumption and alcohol use can have a profound effect on adjusting sleeping patterns. Therefore, both caffeine and alcohol should be avoided. Melatonin may be considered when symptoms are not improving and sleep disturbance persists despite proper education and good sleep hygiene. Typical dosages for melatonin are 3 mg for children and 5 mg for adolescents. Currently though, there is minimal evidence to suggest its benefit in recovery long term. The majority of studies that have been published mainly involve the use of melatonin in the acute phase and have not been studied long term [21]. Studies have also shown that trazodone may be of benefit. However, some concerns with the use of trazodone are the wider side effect profile, and it can cause the patient to have less REM sleep. Zolpidem is a medication that is sometimes considered in the adult population. It also should be used with caution given its unfavorable side effect profile including next-day drowsiness and mental fogginess that can be confused for worsening concussive symptoms. Zolpidem is a controlled substance that requires close monitoring and has concerns for possible dependence.

Supplements

Question: Are any supplements (fish oil, turmeric, resveratrol) recommended to help with recovery?

There is limited research on supplements and vitamins that can be used in concussion treatment. There is considerable evidence that omega-3 fatty acids may be beneficial in the recovery of more significant brain injuries but lacks evidence with less severe injuries such as concussion. Many practitioners often recommend omega-3 fatty acids because of its potential to assist with brain recovery, but there are limited human studies suggesting the added benefit of this supplement [22]. Turmeric is used for its anti-inflammatory effects and has been shown in animal studies to reduce neural inflammation; however, there is no human evidence of its effectiveness with concussion [23]. Resveratrol is an antioxidant found in red wine and certain plants and nuts with potential protective effects in chronic neurologic disease and in animal studies following brain injury although evidence in acute head injury in humans is lacking [23]. Along with supplementation, there has been question as to whether hyperbaric oxygen (HBO) may be beneficial in the recovery of

concussion. HBO seems very attractive but can be an expensive treatment option. A recent meta-analysis published showed that there is no added benefit in the use of HBO in the treatment of concussion [24].

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Chapter 10
Concussion Return to Learn or Work and Return to Play

Suraj Achar, William Timothy Ward, and Rachel Buehler Van Hollebeke

Clinical Case

You are seeing a 16-year-old track-and-field athlete for concussion evaluation after a fall 2 weeks ago at practice. She denies any loss of consciousness or post-traumatic amnesia. She has been staying home from school after being prescribed strict brain rest by her Primary Physician, but symptoms have yet to fully resolve. The patient and her mother are asking when it will be safe to leave the house.

Consensus guidelines endorse 24–48 hours of symptom-limited cognitive and physical rest followed by a gradual increase in activity staying below symptom exacerbation thresholds.

The rationale for post-injury rest related to both symptoms and recovery. Rest was believed to lessen post-concussion symptoms, thus easing discomfort during the acute recovery phase. Secondly, rest was thought to promote recovery by minimizing brain energy demands following concussion. Unfortunately, evidence is insufficient that prescribed rest achieved either one of these objectives. To the contrary, in 2015 a prospective randomized control trial showed that post SRC, recommending strict rest for 5 days, offered no added benefit over 1–2 days rest followed by gradual return to activity [1]. The strict rest group actually reported more daily post-concussive symptoms and had slower symptom resolution [1]. A follow-up multicenter prospective cohort study in 2016 reconfirmed that prolonged rest

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(avoidance of physical activity for 7 days post-concussion) was associated with increased incidence of persistent post-concussive symptoms [2]. As such, the most recent fifth consensus statement on concussion in sport in 2016 recommended that further studies are needed to determine the exact amount and duration of rest. Current recommendations include a brief period of rest (24–48 hours) during the acute phase after injury, followed by gradual increase in activity while staying below symptom exacerbation thresholds [3].

Question: Are there risks associated with prolonged rest?

Since 2001, when the Concussion in Sport Group (CISG) released its consensus statement encouraging active rest until asymptomatic, much has changed regarding our approach to managing return to learn and return to play in athletes who are recovering from sport-related concussion (SRC).

There are multiple factors that may be contributing to the negative outcomes associated with prolonged rest after concussion. The inactivity contributes to not only exercise intolerance and physical deconditioning [4], but there are many other psychosocial stressors to consider. Missed school and social isolation, leading to increased anxiety, depression, discouragement about recovery, and catastrophizing, may contribute to perpetual symptoms [5–7].

With more understanding of the adverse effects of prolonged rest, researchers set out to determine if there were any potential adverse effects of early return to activities in symptomatic patients. Brooks et al. examined whether cognitive exertion by an acutely or subacutely injured brain may negatively alter outcomes. They used computerized cognitive testing on adolescents in the emergency department and showed that the increased cognitive stress (by way of computerized neurocognitive testing immediately post diagnosis of concussion) did not result in worsening of symptoms at follow-up (7–10 days, 1 month, 2 months, and 3 months) or prolong symptom recovery compared to the control group [8]. In 2017, Leddy et al. set out to determine if there was any relationship between early physical activity and concussion symptom recovery/duration. Using a prospective randomized control trial, they determined that exercise using the Buffalo Concussion Treadmill Test within 1 week of diagnosis of SRC did not affect recovery. Additionally, they found that the degree of early exercise intolerance after sport-related concussion was an important prognostic factor with implications for academic and team preparation [9].

Question: Are there any advantages of early light aerobic activity?

As research continued to emerge showing that sport-related concussion patients did not seem to have adverse effects with early subthreshold physical activity, Leddy et al. continued to assess the potential positive aspects of early aerobic exercise. In

2019, they published a randomized control trial of concussed adolescents showing that individualized sub-symptom threshold aerobic exercise treatment during the first week after SRC sped up recovery and may reduce the incidence of delayed recovery [10].

Question: When is an athlete fully recovered and how long does it typically take?

The latest CISG guidelines state that athletes should return to a baseline level of symptoms, but they do not provide objective definitions to establish when an athlete is fully recovered. Resolution of symptoms is a critical part of recovery, but symptom reporting alone may be problematic if athletes are underreporting symptoms. Additionally, the term "asymptomatic" itself is problematic as it has not been formally defined and agreed upon by experts. What makes the term potentially confusing and misleading is the fact that post-concussion-like symptoms are present in the non-concussed population at rest and with exercise [11]. Healthy adolescents, for example, have been shown to have symptom severity scores up to 6 (out of a maximal 132) when given a concussion checklist to complete [12]. As such, some studies use a symptom cutoff score of less than 7 to define symptom recovery consistent with "baseline level of symptoms" in the latest CISG guidelines. Clinical recovery is defined functionally as a return to normal activities, including school, work, and sport after injury. It includes a resolution of post-concussion-related symptoms and return to clinically normal balance and cognitive functioning [3].

Many adolescents with SRC recover within 7–10 days; however, studies have shown that complete resolution of physiological/visual and balance function may take up to 3–4 weeks [13]. The latest concussion in sport guidelines have defined persistent post-concussive symptoms, which are a failure of "normal" clinical recovery, as symptoms lasting greater than 10–14 days in adults and 4 weeks in children [3].

Question: Are there additional metrics useful for informing return to learn/play decisions?

Neurocognitive testing has been widely used to assess concussion and track cognitive recovery. It is discussed in depth in a separate chapter; however, we address it briefly here as it is commonly used in return to play decisions (see "Concussion Neurocognitive Testing" Chap. 6). In fact, a 2010 national survey of concussed high school athletes found that approximately 40% of US high schools that employ an athletic trainer use computerized neurocognitive tests when managing sport-related concussions [14].

Neurocognitive tests can provide a more objective measure of brain-behavior relationships and are believed to be important given the potential unreliability of

self-reporting symptoms after injury. In a case-control study comparing concussed and non-concussed athletes, at 2 days post injury, the addition of neurocognitive testing resulted in an increase in sensitivity of 19%, leading authors to conclude that neurocognitive testing can increase diagnostic accuracy when used in conjunction with self-reported symptoms [15].

In the 2018 American Medical Society for Sports Medicine (AMSSM) position statement on concussion in sport, it was asserted that most concussions can be managed appropriately without the use of neurocognitive testing. According to the AMSSM, when used, neurocognitive testing should be part of a comprehensive concussion management strategy. The statement reiterates that testing should be interpreted by healthcare professionals trained and familiar with the type of test and individual test limitations. It also highlights that comprehensive neurocognitive evaluation can be helpful in management of athletes with persistent symptoms or complicated courses [16]. In our clinic for patients with prolonged concussion, we take a multidisciplinary approach with neurocognitive testing administered and evaluated by a neuropsychologist. We track changes in performance over time with testing providing another data point to help guide our management.

Question: How do you implement return to learn?

To date, evidence shows that SRC can have short-term effects on learning with possible academic dysfunction for up to 1 month [17] but overall minimal impact on long-term academic performance [18]. One large-scale study of 5- to 18-year-olds followed after an emergency visit for concussion showed that 70% had symptom resolution after 28 days. For students transitioning back to the classroom, return to learn should be performed in an individualized but yet stepwise process. In our practice we follow the protocol outlined in Table 10.1. Immediately post injury, an initial stage of brain rest for 24–48 hours is recommended. Students then advance to partial school participation where they attend a few hours of school and only complete necessary assignments, avoid testing and loud areas, and have breaks available as needed. If symptoms are significant and exacerbated at school, students may need to rest in a quiet area versus the nurse's office or leave school for the day, and ideally return the next day. As patients tolerate increasing demands without symptom exacerbation, they gradually return to a normal course and activity load.

During transition back to the classroom following a concussion, a clear set of academic supports should be provided to allow for early screening, quick intervention, and progress monitoring (Table 10.2). In order for the return to learn to be immediate and effective, general education teachers must be trained and empowered to front-load academic supports within the first 4 weeks and should fade academic supports as the concussion symptoms resolve [19]. If these supports are not adequate, then increasing to a more formal intervention in the way of an individualized education plan (IEP) or a 504 plan is warranted. Section 504 of the Rehabilitation Act is a federal civil rights law that provides protection if a person has a physical or

mental impairment. A 504 plan may be considered if a medical condition substantially limits at least one of the major life activities (i.e., thinking, concentrating, reading, sleeping, or learning). 504 plans are an ideal mechanism for use in the return to learn process for the remaining 30% of patients with symptoms that are severe and or lasting longer than the expected recovery of 4 weeks. However, requesting a 504 plan or IEP too soon following a concussion can delay rapidly needed academic supports for students by diverting time and energy into legal or policy-based processes [19].

Table 10.1 Return to learn table

Stage	Home activity	School activity	Physical activity	Goal of step
Brain rest	Sleep as much as needed (at least 8 hours) Allow short naps (<1 hour) during day Start transition toward regular sleep/wake cycle Avoid bright light if bothersome Stay well hydrated and eat healthy snacks q3–4 hours Limit screen time; use large font	No school No homework May begin easy tasks at home (drawing, baking, cooking) Soft music/books on tape ok Limit reading of text as symptoms tolerate (i.e., 10–15 minutes intervals)	Walking short distances to get around (RTP stage 1) No strenuous exercise or contact sports No driving	Gradual return to typical activities
This stage should be limited to 24–48 hours post injury				
Return to school (partial day)	Strict sleep wake cycle with goal 8 hours of sleep at night Avoid napping during day Stay well hydrated and eat healthy snacks q3–4 hours Limit screen time and social activities outside of school as symptoms tolerate	Start with a few hours/ half days Sit in front of class Take a break in nurse's office or quiet room q2 hours PRN Avoid loud areas (music, band, choir, shop class, locker room, cafeteria loud hallway, and gym) Sunglasses/brimmed hat/ ear plugs PRN Preprinted (large font – 18) class notes Complete necessary assignments only Limit homework No tests or quizzes Tutoring or note taker as needed Stop work if symptoms increase	No strenuous physical activity or contact sports (RTP stage 2) No driving	Increase tolerance to cognitive work

(continued)

Table 10.1 (continued)

Stage	Home activity	School activity	Physical activity	Goal of step
Progress to next stage as symptoms improve and can tolerate activities without increasing symptoms				
Return to school Full day	Strict sleep wake cycle with 8 hours of sleep at night Avoid napping during day Stay well hydrated and eat healthy snacks q3–4 hours Screen time and social activities outside of school as symptoms tolerate	Progress to attending core classes for full days Add in electives when tolerated No more than 1 test or quiz per day Give extra time or untimed homework/tests Tutoring or help as needed Stop work if symptoms increase	No strenuous physical activity or contact sports (Ok for RTP stage 2) OK to drive	Increase academic activities
Progress to next stage when in school full time and completing all assignments without symptoms				
Full recovery	Return to normal home and social activities	Return to normal school schedule and course load	Ok to progress to return to play stage 3	Return to full academic activities and catch up on missed work

Table 10.2 Example of physician recommended school accommodations following concussion

Area	Physician-requested modifications
Breaks	If symptoms worsen during class, allow student to go to quiet area or nurse's office; if no improvement after 30 minutes, dismissal to home Allow breaks during day as deemed necessary by student or teachers/school personnel
Visual stimulus	Enlarged print (18 font) copies of textbook material/assignments Preprinted notes (18 font) or notetaker for class material Limited computer, TV screen, bright screen use Allow handwritten assignments (as opposed to typed on a computer) Allow student to wear sunglasses/hat in school; seat student away from bright windows and bright lights Reduce brightness on monitors/screens Change classroom seating to front of room as necessary
Auditory stimulus	Avoid loud classroom activities Lunch in a quiet place with a friend Avoid loud classes/places (music, band, choir, shop class, gym, cafeteria) Allow student to wear earplugs as needed Allow class transitions before the bell
School work	Simplify tasks (i.e., three-step instructions) Short breaks (5 minutes) between tasks Reduce overall amount of in class work Prorate workload (only core or important tasks) Reduce overall amount of in class work No homework Reduce amount of nightly homework Will attempt homework but will stop if symptoms occur Extra tutoring/assistance requested May begin make up of essential work

Table 10.2 (continued)

Area	Physician-requested modifications
Testing	No testing Additional time for testing/untimed testing Alternative testing methods: oral delivery of questions, oral response, or scribe No more than one test per day No standardized testing
Education plan	Student is in need of a 504 plan and/or IEP (if prolonged symptoms are interfering with academic performance)

Table 10.3 Return to work table

Phase	Activity	When to progress
1	Total rest. No mental exertion, computer, texting, video games, or work projects	24–48 hours
2	Light mental activity. Up to 30 minutes of mental activity that does not worsen symptoms	Progress if 30 minutes of mental activity does not worsen symptoms
3	Part-time work with adjustments; breaks as needed, no formal presentations, modify/simplify tasks as needed, provide extra time for projects, and modify deadlines if needed	Progress if 45 minutes of sustained mental activity does not worsen symptoms
4	Continue part-time work with moderate adjustments and begin to reduce extra time for projects as needed	Progress if 60 minutes of mental activity does not worsen symptoms
5	Attempt full-time work and continue to scale back occupation-specific accommodations	Progress if 60 minutes of mental activity does not cause symptoms and when accommodations are no longer needed
6	Full-time work with normal workload and no modifications	n/a

Question: How do you implement return to work?

The return to work process, outlined in Table 10.3, is very similar to the return to learn protocol just discussed. Since work demands will vary significantly, and individualized approach is even more important in the work environment. Again, after 24–48 hours of brain rest, a patient is encouraged to progress to light mental activity and then part-time work, full-time work, and eventually full-time work with a normal workload. At each step the patient should progress if sustained mental activity does not worsen symptoms. Although the formal school supports seen in the return to learn protocol generally do not exist in the work environment, patients should have extra breaks, modified/simplified tasks, extra time, and extended deadlines as they progress through the return to work process. These recommendations can be further challenged as some employers may not be able to accommodate that.

Question: When do you start progressing through the return to play and why is it necessary?

As supported by the CISG, a successful return to learn is necessary prior to progressing through the return to sport program. Unfortunately, there is no physiological time window for SRC recovery, and research indicates that physiological dysfunction may outlast current clinical measures of recovery [20, 21]. The consequences of an athlete returning to sport with continued underlying physiologic dysfunction have yet to be understood fully, but possible outcomes of athletes returning to play while there is continued brain dysfunction include repeat injury, prolonged symptoms, increased risk of musculoskeletal injury, more severe physiologic dysfunction, or increased risk of neurodegenerative disease [22]. As such, a properly implemented return to play program serves as a buffer zone of gradually increasing activity before full-contact exposure risk.

The standard road to recovery after sport-related concussion follows a graduated stepwise rehabilitation strategy (an example is outlined in Table 10.4). After a 24–48-hour period of initial rest, stage 1 can be started with light activities of daily living and the goal of staying below the cognitive and physical threshold for

Table 10.4 Example of return to play table

Stage	Aim	Activity	Goal of step
N/A	24–48 hours rest	None	Relative physical/cognitive rest
1	Symptom-limited activity	Daily activities that do not worsen symptoms	Gradual reintroduction of work and or school activities
2	Light aerobic exercise (2A)	Walking or stationary cycling at slow-to-medium pace; no resistance training	Keep HR below 70% of max predicted HR
Return to learn protocol must be completed prior to advancing to stage 2b			
	Moderate aerobic exercise (2B)	Walking, swimming, or stationary cycling at increased pace; light resistance training with bodyweight squats and pushups (1 set of 10 reps each)	Increase heart rate
3	Sport-specific exercise	Running or sport-specific drills; no activities with risk of head impact	Add movement
4	Noncontact training drills	Harder drills (e.g., passing drills and team drills) may begin progressive resistance training	Exercise, coordination, and increased thinking during sport
5	Full-contact practice	After medical clearance, participate in full/normal training activities	Restore confidence and allow coaching staff to assess functional skills
6	Return to sport	Normal game play	Full clearance/ participation

exacerbating symptoms. We explain to our patients that symptoms are going to be present during the recovery period. It is okay to participate in light activity while experiencing baseline symptoms. Rather, the goal is to avoid any activities that acutely worsen symptoms.

Once the patient is tolerating regular daily activities at school and home, we recommend starting supervised light aerobic activity (stage 2A) with the goal of increasing heart rate via light walking or stationary cycling at a low to medium pace. This step is best coordinated with the athlete's certified athletic trainer. Activities should be kept subthreshold, and any activity exacerbating symptoms should be discontinued. This step in particular is the most in contrast with previous recommendations in which light aerobic activity was not initiated until the athlete was completely asymptomatic. In our institution, athletes are held in the supervised stage 2A until they have successfully completed the return to learn and are generally asymptomatic. Once this is achieved, they are then progressed to stage 3. Each step of the return to play should take at least 24 hours, so at least 1 week is required to progress through all stages of the protocol. We do not allow Thursday night clearance, so high school football players who have a concussion on Friday night are not eligible to return to play the following Friday. The best athletes are at the highest risk of being returned too soon because coaches and parents often want them back out on the field early. Troy Aikman from the Dallas Cowboys specifically mentioned if he was the backup quarterback, he would have had more time to rest from his frequent SRC. Because he was the starter, he felt he was encouraged and supported to return too quickly.

When implementing a return to play protocol, each patient's recovery is individual. We try to not place high expectations on a specific return to sport date, as this may cause increased stress and anxiety if actual recovery is not as fast as desired. It must be reiterated that the timeframe for the return to play protocol will vary based on an athlete's age, history, and level of sport. If the patient experiences any concussion-related symptoms during the stepwise return to play, then the athlete drops back to the previous asymptomatic level and attempts to progress again after being free of symptoms for another 24-hour period at the lower level. If patients experience prolonged symptoms and resultant inactivity, each step may take longer than 24 hours simply because of physical deconditioning that occurred during recovery. A great way to avoid this particular scenario is through a customized prescription for a symptom-limited exercise program.

In 2014, May et al. also proposed sport-specific return to play protocols for sports with higher risk of head injury including football, gymnastics, cheerleading, wrestling, soccer, basketball, lacrosse, baseball, softball, and ice hockey. These guidelines seek to maintain the integrity of the current six-step model (described in Table 10.4); however they add a moderate activity phase highlighted by resistance training and provide contact drills specific to the athlete's sport [23].

Question: How do you generate an exercise prescription? What is a Buffalo Concussion Treadmill Test and how is it useful?

In order to create an individualized exercise prescription, clinicians must be familiar with the Buffalo Concussion Treadmill Test (BCTT). The BCTT (Table 10.5) is a validated treadmill test that may be utilized [9, 27]. First the BCTT can be used to diagnose physiologic dysfunction in concussion patients. As previously mentioned, it may be used to generate an individualized exercise prescription for those same patients suffering from prolonged concussion symptoms.

The BCTT may help differentiate concussion from other diagnoses such as cervical injury, vestibular/ocular dysfunction, depression, or post-traumatic headache syndrome such as migraines. If patients can exercise to exhaustion without reproduction or exacerbation of headache or other symptoms, and they demonstrate a normal physiologic response to exercise, then symptoms are not likely due to sport-related concussion. It is also useful in quantifying the clinical severity and exercise capacity of concussed patients.

The test itself was developed by John J. Leddy at the University of Buffalo. It is based on the standard Balke Cardiac Treadmill Test, which has been shown to be safe in patients with cardiac and orthopedic issues. Absolute and relative contraindications are in Table 10.6. The starting speed is 3.6 mph at 0% incline, but the starting speed can be slightly increased for taller or athletic persons and reduced for shorter or sedentary persons. During the first minute, the patient walks at 0% incline. The incline is increased to 1% at minute 2 and subsequently increased by 1% each minute thereafter. The speed remains constant until the maximum incline is reached or the patient cannot continue. Ratings of perceived exertion (using a Borg Scale) and symptom score are assessed every minute (Table 10.5). Heart rate (by monitor)

Table 10.5 Summary of Buffalo Treadmill Test

Test duration (minutes)	Speed (mph)	Incline (%)
1st minute	3.6	0%
2nd minute	3.6	1%
Subsequent minutes ➔ variation in incline		Incline is increased by 1% each minute until maximum incline is reached or stopping criteria fulfilled
Subsequent minutes ➔ variation in speed	If maximum incline is reached, then speed is increased by 0.4 mph every minute until stopping criteria fulfilled	

Stopping criteria
1. Significant exacerbation of symptoms
   3-point increase from that day's pre-treadmill resting symptom sore on visual analog scale
2. Exhaustion
   RPE of 19–20 on Borg Scale

Table 10.6 Absolute and relative contraindications to performing the BCTT

Absolute contraindications to performing the BCTT	
History	Unwilling to exercise Increased risk for cardiopulmonary disease as defined by the ACSM[a]
Physical exam	Focal neurological deficit Significant balance deficit, visual deficit, or orthopedic injury that would represent a significant risk for walking/running on a treadmill
Relative contraindications to performing the BCTT	
History	Beta-blocker use Active major depression (may not comply with directions or prescription)
Physical exam	Minor balance deficit, visual deficit, or orthopedic injury that increases risk for walking/running on a treadmill (potential use of cycle ergometry) SBP >140 mm Hg or DBP >90 mm Hg Severe obesity

[a]Patients with known cardiovascular, pulmonary, or metabolic disease, signs, and symptoms suggestive of cardiovascular or pulmonary disease, or individual aged >/= 45 years who have more than one risk factor to include (1) family history of myocardial infarction, coronary revascularization, or sudden death before 55 years of age; (2) cigarette smoking; (3) hypertension; (4) hypercholesterolemia; (5) impaired fasting glucose; or (6) obesity

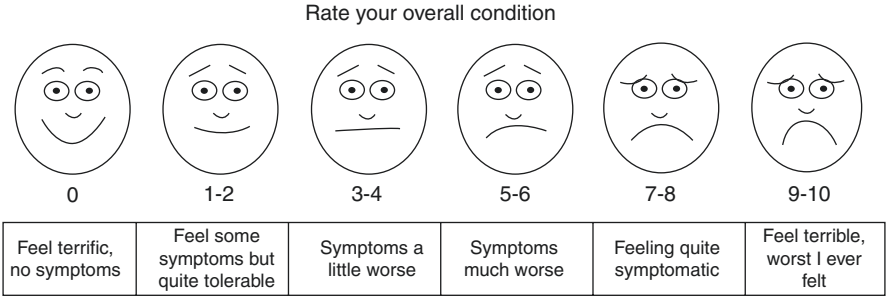

Fig. 10.1 Visual analog symptom score scale

and blood pressure (by automated cuff) are measured every 2 minutes. The test is stopped with significant exacerbation of symptoms (defined as a 3-point increase from that day's pre-treadmill test resting overall symptom score on a 1–10 point visual analog scale) or at exhaustion (RPE of 19–20 on the Borg Scale). If the patient achieves the maximum incline without reaching either stopping criteria, then the treadmill speed is increased by 0.4 mph every minute until stopping criteria are fulfilled. The test is deferred with any patient who has significant pretest resting symptoms defined as greater than or equal to 7 on the pretest visual analog scale. A sample visual analog scale can be seen in Fig. 10.1.

The BCTT may be used clinically to gauge recovery. If the patient is able to exercise to exhaustion (measured as reaching 85–90% of theoretic maximum HR) without symptom recurrence for 20 minutes, then it is safe to declare them physiologically recovered, and they can begin the graduated return to play protocol. Alternatively, if the patient develops symptoms prior to peak exertion, then you

have objective evidence that the athlete is not physiologically ready and will need more recovery time. Per Leddy and Willer 2013, the most commonly reported symptoms indicating that concussion has not resolved are worsening headache, dizziness/lightheadedness, and/or a sensation that the head feels full [24].

Additionally, the BCTT may be used to generate an exercise prescription while recovering. If a submaximal symptom exacerbation threshold is identified, then the patient is given a prescription to perform aerobic exercise on a stationary bicycle, treadmill, or elliptical for 20 minutes a day at subthreshold intensity (i.e., 80% of the threshold HR achieved on the BCTT) for 5–6 days per a week. The BCTT may be repeated every 2–3 weeks to establish a new symptom-limited threshold HR. Alternatively, in order to avoid repeating the BCTT, once the practitioner has a baseline threshold HR and the patient is responding favorably, it is reasonable to increase the exercise HR target by 5–10 bpm every 2 weeks via phone or email.

Question: Why is return to play important to primary care providers and their patients?

Many concussions will only present to primary care providers and not providers well versed in concussion care. Regrettably, concussion management and recommendations continue to elude translation into practice as many recent surveys of both US and Canadian physicians continue to indicate that not only is physical rest the most commonly prescribed treatment for concussion but also cognitive rest and return to learn/return to play guidelines are overlooked all together [25, 26].

Key Points (Answers to Questions)

- Complete rest should be no longer than 24–48 hours.
- Risks associated with prolonged rest include worsening exercise intolerance, physical deconditioning, and worsening of psychosocial stressors.
- Individualized sub-symptom threshold aerobic exercise treatment may speed up recovery and reduce delayed recovery.
- Implementing a successful return to learn strategy includes starting with a general set of academic supports for transitioning back into the classroom. If symptoms are lasting longer than expected, then increasing to more formal interventions in the way of an individualized education or a 504 plan is warranted.
- Progression through the return to play is attempted when the patient has returned to his or her baseline and is no longer experiencing post-concussion-related symptoms. The stepwise return to play is necessary because physiological dysfunction may outlast current clinical measures of recovery and returning to sport too early with continued physiologic dysfunction may lead to negative outcomes.

- The Buffalo Concussion Treadmill Test is a useful tool for generated individualized exercise prescriptions, assessing physiologic recovery post-concussion, and may also help differentiate concussion from other potential diagnoses.
- Return to work follows the same principals of return to learn with gradual return, allowing for breaks and extra time to complete assignments until symptoms resolve.

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Chapter 11
Post-concussion Syndrome

Jack Spittler and Lindsey Kolar

Clinical Case

A 17-year-old high school athlete missed the majority of the fall soccer season due to a concussion she sustained in the second game. She is considering trying out for track and field but still suffers from headaches 4 months later and simply does not feel as competitive as she used to. She wants to know if these symptoms are due to her concussion and what she can do to feel better before the spring tryouts.

When does a concussion evolve into post-concussion syndrome?

There is no consensus definition for post-concussion syndrome—making diagnosis, treatment, and broader scientific study a challenge. Historically, the most commonly used diagnostic criteria come from the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-4) and the *International Statistical Classification of Diseases and Related Health Problems* (ICD-10). The updated DSM-5 has abandoned its former classification system, and now the greater scientific community has adopted the more clinically relevant term "persistent post-concussive symptoms" (PPCS) [1, 2].

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DSM Criteria

Previously, the DSM-4 utilized the term "postconcussional disorder." In short, this diagnosis required that cognitive impairment plus a certain amount of new or worsening specific symptoms occur after a concussion, causing some degree of disability for at least 3 months [1]. The DSM-5 discards this term in favor of "major or mild neurocognitive disorder due to traumatic brain injury." Under these criteria, there must be evidence of a traumatic brain injury with at least one of the following: loss of consciousness, post-traumatic amnesia, disorientation and confusion, or other neurologic signs. In the immediate period following the injury, the patient must also demonstrate a decline in at least one of six cognitive domains: complex attention, executive function, learning and memory, language, perceptual motor, or social cognition. This disorder is designated as either mild or major based on the severity of symptoms and degree of functional deficit. Although specific symptom durations are not required for diagnosis, the DSM-5 suggests that symptoms lasting beyond 3 months exceed the expected recovery time of a concussion [3].

ICD-10 Criteria

The ICD-10 uses the terminology "postconcussional syndrome." Postconcussional syndrome is defined as a history of TBI that is usually severe enough to cause loss of consciousness and is followed by three or more of the following eight features: headache, dizziness, fatigue, irritability, insomnia, difficulty concentrating, memory deficits, and decreased tolerance to stress, emotion, or alcohol [2].

Persistent Post-Concussive Symptoms (PPCS)

The prior criteria have varying requirements for symptom duration, initial injury, symptomatology, and objective findings. These discrepancies make it difficult to determine which patients warrant further evaluation and when this evaluation should occur. As a result, the 5th International Conference on Concussion in Sport and the American Medical Society for Sports Medicine favor the term persistent post-concussive symptoms (PPCS), which is the term that will be utilized for the remainder of this chapter [4, 5]. Patients with PPCS experience symptoms that extend beyond the expected recovery time of 2 weeks for adults and 4 weeks for children. This patient-centric diagnosis captures all patients who do not recover as anticipated following a concussion—targeting this population for further evaluation and treatment.

Who is at risk for developing post-concussion symptoms?

The majority of adults and older adolescents (~80–90%) will achieve clinical recovery from a concussion within 2 weeks, while the majority of children and younger adolescents will require 4 weeks to return to baseline [6, 7]. The remaining ~10–15% of patients will experience PPCS ranging from weeks to years following their initial injury [8]. Identifying patients at risk for developing PPCS is important so that aggressive and individualized treatment strategies can be employed to reduce the duration of symptoms and burden of disability. A great deal of effort has been dedicated to identifying such risk factors; however, many results have been inconsistent. Heterogeneous diagnostic criteria (as described above) make it difficult to compare and pool research. Overall, however, it appears that increased initial symptom burden and severity, female sex, adolescent age, preinjury mental health diagnoses, and specific post-injury symptoms (such as headache, migraine, dizziness, oculomotor dysfunction, and depression) may all increase the risk of prolonged recovery [7, 9]. It is of note that patients with learning disabilities or attention-deficit/hyperactivity disorder are not known to be at greater risk for developing PPCS [9].

Tables 11.1 and 11.2 summarize possible PPCS symptoms and potential referrals.

Table 11.1 Possible symptoms in PPCS

Musculoskeletal	Whiplash/cervical dysfunction
Psychologic	Anxiety Depression Concentration difficulties
Neurologic	Vestibulo-ocular dysfunction Dizziness Photophobia Phonophobia Headaches Memory problems
Others	Sleep disturbance Fatigue/exercise intolerance

Table 11.2 Potential referrals for PPCS

Referral	Reasons for referral
Neuropsychology or psychology	Neuropsychological evaluation Depression, anxiety, or other psych symptoms
Psychiatry	Depression, anxiety, or other psych symptoms
Neurology	Headaches Dizziness/vestibular dysfunction Memory issues
Ophthalmology	Oculomotor dysfunction
Optometry	Light/vision dysfunction
Sleep medicine	Sleep disturbance
Physical or occupational therapy	Cervical dysfunction Balance issues Impaired activities of daily living (ADLs)

What causes persistent symptoms following a concussion?

Knowledge of acute concussion pathophysiology continues to grow. It is thought that temporary mechanical forces on neurons incite a cascade of events that include inappropriate ion shifts, diffuse neuronal depolarization, abnormal neurotransmitter release, autoregulatory disturbances and altered cerebral blood flow (CBF), neuro-inflammation, and metabolic mismatch and depression. It is postulated that axonal injury, impaired neuroplasticity, blood-brain barrier dysfunction, and even some degree of cell death may also play a role in concussion [10]. Alternatively, the exact pathophysiology of PPCS remains unknown. It has been hypothesized that continued physiologic derangements, neuro-inflammation, axonal injury, whiplash injury, vestibulo-ocular dysfunction, and psychological factors may all be contributors.

Physiologic Derangements

Persistent physiologic derangements such as autonomic, CBF, and metabolic dysfunction have all been postulated as causes of PPCS. This idea is extrapolated from the better-known pathophysiology of acute concussion and based on the observation that some patients with PPCS experience symptoms largely with physical or cognitive exertion rather than at rest [11]. Indeed, higher resting heart rates following TBI and increased heart rates during cognitive exertion in PPCS suggest continued autonomic dysfunction in this patient population [12, 13]. The role of CBF dysfunction is less certain. Reduced CBF is found in 40–60% of patients with PPCS using single-positron emission computed tomography (SPECT) [8]. Interestingly, similar findings have been noted in disease states such as neck and back pain, whiplash injuries, obsessive-compulsive disorder, depression, chronic fatigue, AIDS dementia complex, and more [14–20]. In light of its seeming ubiquity in other disorders, it is uncertain whether CBF dysfunction plays a causative role in PPCS. Similarly, available evidence does not clearly support metabolic dysfunction as a cause of PPCS. Research utilizing magnetic resonance spectroscopy demonstrates reduced N-acetyl aspartate (NAA) (a correlate of ATP) in patients with PPCS; however, these metabolic changes are also seen in asymptomatic patients who have recovered appropriately from a concussion—calling to question the strength of the relationship between symptomology and metabolic dysfunction in PPCS as measured by NAA [8, 21, 22]. This does not disprove that continued metabolic depression has a role in PPCS but may rather demonstrate that a more indicative biomarker has not been found.

Neuro-inflammation

Neuro-inflammation may also contribute to PPCS. Increased levels of the serum inflammatory marker, C-reactive protein (CRP), are associated with PPCS as well as persistent cognitive dysfunction and psychological issues following TBI [23]. It

has also been considered that systemic inflammation—even in the absence of TBI—may be the true underlying source of PPCS-like symptoms. In this model, concussion is merely one of many possible inciting etiologies [24]. This concept is in part based upon research showing that post-concussive symptoms occur after both concussion and polytrauma without brain injury [25]. It must be noted, however, that this study evaluated symptoms within 2 weeks of initial injuries. Therefore, results may not be generalizable to time frames typically encompassed by PPCS (>2–4 weeks). Overall, more research is needed to elucidate the relationship between inflammation and PPCS. It is possible that better understanding this potential pathophysiology may lead to novel anti-inflammatory treatments for PPCS in the future.

Axonal Injury

It has also been theorized that axonal dysfunction contributes to PPCS. In acute concussion, rapid head deceleration produces shearing forces that can result in structural axonal damage, subsequently increased calcium, and resultant axonal dysfunction [8]. Diffusion tensor imaging (DTI) shows that patients who suffer a concussion demonstrate white matter changes beyond expected recovery times compared to patients with non-brain injuries (cervical injuries excluded). Interestingly, these concussion-related white matter changes are present in patients who develop PPCS as well as those who do not [26]. These results suggest that continued axonal dysfunction does not appear to play a role in PPCS.

Whiplash Injury

It is likely that whiplash injuries often occur concurrently with concussions and therefore may play a role in PPCS. Isolated whiplash injuries and concussions share many associated symptoms such as headache, neck pain, nausea/vomiting, dizziness, instability, vision changes, memory deficits, decreased concentration, and more [8]. Limited research shows that, when compared to controls, patients with PPCS suffer higher levels of painful upper cervical joint dysfunction, reduced neck flexor endurance, and increased neck spasm [27]. Furthermore, patients with PPCS experience significant pain relief from therapies directed at the cervical spine such as cervical mobilization and assisted muscle-stretching therapy [28]. Cervico-vestibular rehabilitation therapy for persistent post-concussive symptoms such as neck pain, headache, and/or dizziness results in a dramatic increase in patients who are able to return to sport [29]. Whiplash may further contribute to persistent post-concussive symptoms by altering cervical proprioception. Cervical muscle and joint mechanoreceptors feed proprioceptive information to multiple levels of the central nervous system (CNS) and are responsible for informing the cervicocollic, vestibulocollic, and cervico-ocular reflexes. These reflexes work to

stabilize the head during complex movements [11]. Although the mechanisms behind cervical proprioceptive dysfunction remain unknown, it is postulated that alterations therein could lead to common concussion symptoms such as dizziness and imbalance. Taken together, these results expose the possibility that some persistent post-concussive symptoms may have a cervicogenic etiology.

Vestibulo-Ocular Dysfunction

Dysfunction of the vestibulo-ocular system (VOS) may also contribute to PPCS [11]. The VOS consists of peripheral inputs from the inner ears, eyes, and musculoskeletal mechanoreceptors that feed processing centers in the CNS [30]. Efferent signals from the CNS then complete pathways for important reflexes such as the vestibulo-ocular reflex (VOR) and the vestibulo-spinal reflex (VSR). The VOR coordinates fixed gaze during head acceleration. The VSR consists of compensatory body movements induced by changes in head position to maintain balance and posture. Like other potential causes of PPCS, the pathophysiology of persistent VOS dysfunction after concussion is not fully understood; however, complications such as labyrinthine concussion, perilymphatic fistula, endolymphatic hydrops, or central vestibular lesions may be contributors [31]. It seems logical that alterations of this complex system could account for persistent post-concussive symptoms such as dizziness, imbalance, fogginess, nausea/vomiting, blurred vision, headache, and "not feeling right" [5].

Psychological Dysfunction

Finally, it is likely that psychological factors have an etiologic role in persistent post-concussive symptoms, as suggested by the increased risk of developing PPCS in those with underlying mental health diagnoses [32]. In some patients, persistent post-concussion symptoms may include depressed mood, anxiety, irritability, decreased concentration, fatigue, and sleep disturbances. In these situations, it may be difficult to determine whether symptoms are due to a concussion or preexisting psychological disorder. This highlights the importance of knowing a patient's baseline mental health status and diagnoses.

How can a clinician evaluate persistent post-concussive symptoms?

It is important to understand that persistent post-concussion symptoms usually have several contributing etiologies, as described above, resulting in heterogeneous clinical presentations. "Clinical profiles" are a developing concept in concussion care

that reflects this. These profiles categorize symptoms as cognitive, ocular, vestibular, affective (anxiety/mood), somatic (headache), and fatigue-related [11, 33, 34]. Symptoms may be unique to one profile or shared. These profiles provide a conceptual framework for evaluation and treatment of PPCS.

As with acute concussion, evaluation starts with a thorough history of the initial injury and following events. Clinical inventories such as the symptom checklist on the Sports Concussion Assessment Tool Fifth Edition (SCAT5) can help identify and track the progression of collective post-concussive symptoms [35] (see Chaps. 5 and 6 for further details). A complete physical exam should be done with special attention to the neurologic and vestibulo-ocular systems, the cervical spine, and cognition. (See Chap. 4 on Concussion Physical Exam for further details.) Focal findings warrant advanced imaging; however, the neurologic exam will be unremarkable in the vast majority of patients with PPCS (See Chap. 7) [36].

Clinical profiles, as mentioned above, may help guide further testing. Formal and computerized neuropsychological testing measures cognitive deficits [37]. The Beck Depression Inventory-II (BDI-II), Patient Health Questionnaire (PHQ-9), and Brief Symptom Inventory-18 (BSI-18) evaluate affective symptoms [37]. Exercise testing with the Buffalo Concussion Treadmill Test and autonomic evaluation with tilt table testing can help detect physiologic dysfunction [37].

Standard computed tomography (CT) and magnetic resonance imaging (MRI) are generally uninformative in PPCS evaluation; whereas, functional MRI, diffusion tensor imaging, magnetic resonance spectroscopy, and quantitative EEG have all demonstrated measurable differences in patients with PPCS versus controls. These findings are important in the research setting, however, often do not change clinical management. Similarly, genetic testing and biomarkers continue to be subjects of study but do not yet have a role in clinical medicine [37].

Finally, it is important to remember that if a patient is not recovering as expected following a concussion, preexisting and alternative diagnoses, apart from PPCS, should also be considered.

What is the best approach to treatment of prolonged concussion symptoms?

Treatment for prolonged concussion symptoms has traditionally been based on an extension of the guidelines for treating acute concussion or those used in treating mild traumatic brain injury (TBI) [38]. This is usually not effective for prolonged symptoms, however, since treatment for acute concussion usually consists of more passive treatment with a short period of rest, followed by gradual return to activity as symptoms improve. Current evidence suggests that management of prolonged symptoms should focus on active treatment of specific primary and secondary diagnoses identified on assessment. Recent systematic reviews have advocated use of somatic, cognitive, mental health, physiological (exercise), cervical spine, vestibular, oculomotor, autonomic, sleep, hormonal, and loss of cognitive and/or physical stamina in the assessment in order to facilitate individualized and targeted management of

post-concussion syndrome [37]. It is ideal for those with PPCS to be evaluated by a provider or multidisciplinary team with expertise in complicated concussion management [5]. A collaborative treatment approach that includes exercise, therapy, mental health, and possibly pharmacotherapy can be then utilized. Further high-quality studies are still needed, but there is some good data to help guide treatment for prolonged concussion symptoms.

Exercise

Initially the mainstay of treatment for concussion was "cocoon therapy" which involved complete rest with avoidance of activity and stimulation [39, 40]. Especially for prolonged concussion symptoms, there is emerging evidence that voluntary and controlled exercise, not rest, is a much better treatment option. Exercise intolerance may actually be a physiologic biomarker of an ongoing concussion, and return of normal exercise tolerance may help to establish recovery from a concussion [41]. An individualized, symptom-limited aerobic exercise program has been shown to be safe and effective in improving persistent symptoms compared to controls [42–44]. One such program is the Buffalo Concussion Exercise Treatment Protocol. This is a progressive sub-symptom threshold aerobic exercise program based upon systematically establishing the level of exercise tolerance on the Buffalo Concussion Treadmill Test (BCCT), which is the most studied, controlled exercise program [45]. Along these lines, there is also evidence that inactivity may prolong concussion symptoms, such as vestibular dysfunction and depression/anxiety [46].

Vestibular and Oculomotor/Vision Therapy

Vestibular and oculomotor symptoms occur in about 60% of athletes with sports-related post-concussive symptoms [47, 48]. This may include dizziness, vertigo, disequilibrium, impaired balance, blurry/unstable vision, and nausea. Presence of these symptoms at time of injury, especially dizziness, is predictive of a prolonged concussion recovery [49]. Therapy should focus on specific deficits identified and utilize an "expose-recover" model performed by clinicians with expertise in vestibular rehabilitation [50, 51]. There is some evidence that addressing vestibular dysfunction with a targeted physical therapy program improves outcomes in those with post-concussive symptoms [29, 52].

The control of eye movements is guided by several parts of the brain that are particularly vulnerable to concussion [53]. Physical examination may reveal deficits in saccades, anti-saccades, smooth visual pursuits, vergence, accommodation, vestibular-ocular reflex, visual fields, and photosensitivity. The most commonly diagnosed vision problems in athletes with PPCS are convergence insufficiency (CI) and accommodative insufficiency (AI) [54]. If there is a deficit, referral to an

eye care specialist (optometrist or ophthalmologist) should be considered. Therapy directed at certain ocular deficits or more general oculomotor training (OMT) is shown to be beneficial for individuals with oculomotor issues with PPCS [55, 56].

Light Therapy

Light affects many aspects of human physiology including circadian rhythm, sleep-wake cycles, alertness, cognition, and mood. There is some evidence that light therapy may be helpful in treating PPCS. Athletes with concussion who received daily blue wavelength light therapy have shown decreased daytime fatigue, improved sleep, greater alertness, and reduced anxiety compared to controls [57–59]. In addition, emerging evidence suggests that green light may be an effective sleep promoter and reduce headache symptoms, while red light may aid in alertness [57, 60]. Mitigation of certain light wavelengths may also be helpful. In one small study of athletes with photophobia following concussion, 85% experienced symptom relief when using glasses of one or more colors. The colors that provided the most relief were blue, green, red, and purple, and no adverse events were reported [61]. More investigation is needed in regard to certain light wavelengths for treatment of PPCS, as it may be an inexpensive therapy with minimal side effects.

Physical/Occupational Therapy

As mentioned earlier, concomitant injury to the cervical spine (resembling whiplash) may occur with concussion [62]. The neck and upper cervical spine are particularly vulnerable to injury as they are the most mobile part of the vertebral column. Cervical spine injuries have been linked with headache, blurry vision, dizziness, and vertigo, and therefore these symptoms of a concussion may be derived from injury to the brain, neck, or both [63]. If neck pathology is felt to be contributing to PPCS, the athlete should be referred to physical therapy with focus on neck position, manual therapy, neuromotor/sensorimotor retraining, and postural stability [64].

Headaches

In many cases, it can be difficult to distinguish PPCS from a migraine headache. Athletes with prolonged headache following concussion should be evaluated for underlying headache disorders, cervical dysfunction causing headache, and other possible contributors [37]. Obtaining a thorough history of previous headache disorders and treatment is essential with special attention paid to any differences in

symptomatology. Pharmacologic treatment of prolonged concussion-related headaches often mimics that of chronic migraines and may include prophylactic treatment with tricyclic antidepressants (TCAs, i.e., nortriptyline), beta blockers (i.e., propranolol), and anticonvulsants (i.e., gabapentin) and/or abortive treatment with NSAIDs, acetaminophen, and triptans [65]. Both gabapentin and TCAs have immediate impact on symptom burden following concussion; however, they are not proven to reduce symptoms in PPCS [66]. Medications such as these may have side effects including dizziness, drowsiness, and blurry vision, which could potentially exacerbate other PPCS symptoms. Therefore, they should be used very judiciously and with close monitoring of symptoms. Athletes with PPCS should be asked about current medication use (including over-the-counter medications)—medication, dose, and frequency. The athlete may be overusing medications, which could exacerbate symptoms or cause rebound headaches. It has been shown that greater initial PPCS scores result in higher likelihood of receiving medications for treatment, which may not be helpful or even detrimental in PPCS treatment [67].

Sleep Disturbance

High-quality sleep has emerged as an incredibly important factor in athletics and many other aspects of human performance. Sleep disruption is a common symptom in athletes with PPCS. Prevalence and severity are not well studied, but some newer data show that sleep disturbance may be present in over half of athletes with slow recovery from concussion [68]. Problems with sleep may be associated with higher overall symptom severity burden and worsened reaction time during recovery [69, 70]. In addition, history of repeated concussion is associated with longer concussion duration and higher reported sleep disturbance. Those athletes with sleep disturbance also exhibit more severe headaches, mood disturbance, and cognitive dysfunction [71]. In athletes with sleep disturbance, sleep hygiene should first be addressed [72]. This may include enacting a regular daily bedtime, avoiding stimulating activities immediately before bed, not consuming caffeine for 4–6 hours prior to bedtime, and avoiding daytime naps, among other strategies. Pharmacotherapy should be used judiciously as most medication use is off-label to treat sleep following concussion. Medications may include analgesics, muscle relaxants, melatonin, and hypnotics. While not all of the reasons are clear, reduced evening melatonin production may be present in the body following concussion, which could consequently alter circadian rhythm [73]. A recent systematic review confirmed, however, that there is no current evidence that melatonin is beneficial in sleep disturbance in PPCS [74]. There is an ongoing randomized, double-blinded, placebo-controlled trial regarding melatonin use in PPCS that is slated to finish soon and may shed some light on this treatment option [75]. Sedatives should be avoided, but

if used, only for a short (2–3 weeks) period. Antidepressants may be considered to treat sleep, especially if concurrent mood alterations are present [76].

Psychological Symptoms

Individuals experiencing prolonged psychological symptoms such as irritability, sadness, and anxiety after a concussion should be evaluated further and offered appropriate treatment. These symptoms (especially if untreated) may lead to depression, post-traumatic stress disorder, and behavioral disorders, which can be short- and longer-term sequelae of concussion [72]. It has been shown that a collaborative care model can improve outcomes in those with prolonged psychological symptoms [77]. An important initial intervention can simply be discussing the concussion diagnosis and prognosis more thoroughly with the individual and their family or providing them with educational materials. Reassurance, discussing expected recovery time, and education about coping strategies have been shown to improve PPCS [78]. Non-pharmacologic treatment options also include cognitive-behavioral therapy (CBT). There is some limited evidence that CBT may help treat post-concussive symptoms and reduce or prevent PPCS in individuals who are "at-risk" (initial high symptom severity and belief that symptoms will persist) for developing PPCS [79, 80]. Neuropsychological evaluation should be considered early on in PPCS if psychological symptoms are present. Neuropsychologists can provide bio-psychosocial evaluation and treatment services to non-athletes and athletes. In athletes, they are able to provide post-injury education and emotional reassurance, guidance on symptom management and emotional support, and assistance with return-to-play and return-to-school processes [81]. Pharmacologic therapy can be directed at the type of psychological disturbance. Generally, selective serotonin reuptake inhibitors (SSRIs) are recommended as first-line treatment, if depression and anxiety symptoms are present post-concussion. Efficacy and tolerability of serotonin and norepinephrine reuptake inhibitors (SNRIs) are less established, but expert consensus guidelines endorse their use for these symptoms. Tricyclic antidepressants (i.e., amitriptyline) may be considered, especially if there are concomitant headache issues, but should be used cautiously due to a less favorable risk-benefit profile [82]. Different pharmacologic therapies can be considered if the main psychological symptoms are related to attention difficulties. In a meta-analysis of randomized controlled trials, methylphenidate (stimulant used for ADHD/ADD treatment) was found to be effective for attention deficits after mTBI [83]. Comorbid fatigue, daytime sleepiness, and apathy may also respond favorably to stimulant treatment [84, 85]. As stimulants may have significant side effects (poor appetite, insomnia, weight loss, etc.) and could result in dependence, they should be used judiciously with careful monitoring of symptoms.

What are potential complications associated with prolonged post-concussion symptoms?

Second Impact Syndrome

Second impact syndrome is a condition in which an individual experiences a second head injury prior to full recovery from the initial head injury. This is a rare, but catastrophic, complication that could result from returning an individual too quickly to athletics. If after a concussion the athlete is still symptomatic, this indicates that the brain is still in a fragile state due to altered cerebral metabolism. A second impact during this period could potentially cause complete dysregulation of cerebral perfusion and pressure control. This leads to rapid swelling in the brain and increased intracranial pressure, consequently leading to brain herniation and death. Because it is so rare, there is little epidemiologic data about second impact syndrome and its existence somewhat controversial [86]. A recent systematic review found 36 cases of second impact syndrome in the medical literature. It demonstrated that risk factors may include age 16–19 years old, participation in American football, and male gender [87]. There is currently no consensus on the length of time in between hits that an individual can suffer second impact syndrome; but if an athlete is still symptomatic, they may be at risk [88]. Cases of second impact syndrome that resulted in death have unsurprisingly garnered significant media attention and concern in many communities. As such, every state in the United States has adopted specific legislation regarding diagnosis and return-to-play following concussion to help protect athletes from returning to sport too quickly. Providers should become familiar with their respective state law in regard to concussions.

Chronic Traumatic Encephalopathy

Chronic traumatic encephalopathy (CTE) is a neurodegenerative condition that demonstrates deposition of tau proteins on autopsy. Therefore it can only be diagnosed postmortem. It is hypothesized to be caused by repetitive mild traumatic brain injuries or concussions. CTE has been described in former athletes with a history of concussion or repetitive head impact exposure, often accompanied by behavioral change [89]. Direct cause and effect has not been confirmed, though, making CTE a diagnosis that is currently widely debated. Advocates of CTE as a disease describe it as athletes presenting with behavioral disturbance, increased suicidality, and neurodegenerative disease leading to dementia [90]. A cause-and-effect relationship between postmortem CTE changes and antemortem behavioral and cognitive manifestations has not been demonstrated yet. Many asymptomatic players have had confirmed CTE pathology at autopsy [91, 92]. It is also unknown if CTE is a progressive disease and whether tau deposition is the cause of CTE or a by-product or marker of a disease [93]. The incidence and prevalence of CTE in athletes and the general population also remain unknown.

CTE-associated symptoms may be related to numerous different factors. These include impact load and type, career length, genetic predisposition, or other lifestyle behaviors including alcohol, drug and anabolic steroid use, general health, and psychiatric disease [5]. Some retrospective studies have reported increased risk of neurodegenerative disease in former professional football players; however, former high school football players do not show a higher prevalence of neurodegenerative disease when compared to non-football peers [94, 95]. Concurrent extensive exposure to repetitive head impacts and number of diagnosed concussions is the most cited risk factor CTE, but the degree of exposure is likely specific to each individual, with multiple modifying risk factors [96]. A contact sport athlete or former athlete that presents with prolonged neuropsychiatric symptoms should be evaluated for potentially treatable comorbid conditions and not be assumed to have CTE [97].

Suicidality

In recent years, there have been several prominent professional athletes that have committed suicide. Many of these cases have been hypothesized to be related to repetitive head injuries or CTE; however, no clear link has yet been established [98]. Depression is a clear risk factor for suicide, but underlying causes of depression in athletes and former athletes are likely multifactorial. A large number of retired National Football League (NFL) players have experienced difficulty adjusting to life after sports, trouble finding employment, financial struggles, chronic pain, and use of prescription painkillers [99, 100]. All of these factors, in addition to recurrent head injury, could contribute to depression symptoms. A recent systematic review found that there is evidence of some former athletes in contact, collision, and combat sports who suffer from depression later in life, and there may be an association between this and a history of multiple concussions [101, 102]. However, former athletes are not at an increased risk of death by suicide [103]. More prospective research is needed regarding the relationship between recurrent head injury and suicide in athletes, but what is clear is that any individual who suffers from depression should be treated appropriately and monitored for warning signs of suicide.

Key Points

- The term persistent post-concussive symptoms (PPCS) is currently favored in the sports medicine community as it captures all patients who do not recover as anticipated following a concussion.
- The exact pathophysiology behind PPCS is unknown, but is likely multifactorial.
- Current evidence suggests that management of prolonged symptoms should focus on active treatment of specific primary and secondary diagnoses identified on assessment.

- Emerging evidence suggests that light exercise, rather than prolonged rest, is helpful in recovery from PPCS.
- Referrals should be considered for PPCS and guided toward specific symptoms, which may include neuropsychology/psychology, psychiatry, neurology, ophthalmology, optometry (vision/light therapy) sleep medicine, physical therapy, and occupational therapy.
- Second impact syndrome is a rare but catastrophic condition that could occur if an athlete is returned to activity prior to full recovery from a concussion.
- Long-term sequelae of repetitive concussions and PPCS are not clear, but potentially include depression, anxiety, suicidality, and chronic traumatic encephalopathy (CTE).

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Chapter 12
Concussion Prevention

Kathleen M. Weber and Elizabeth B. Portin

Clinical Case

A mother and father accompany their 14-year-old son to his preseason pre-participation physical for high school sports. They inquire if there are any strategies or interventions that the high school is implementing to prevent concussions.

The following discussion will provide an overview of prevention strategies developed to reduce SRCs among athletes.

Protective Gear

Question: Are helmets effective at preventing concussions?

Helmets and Headgear

There is consensus among researchers, clinicians, and industry professionals that proper equipment, particularly helmets and headgear, protect against head injuries. The American Medical Society for Sports Medicine, for example, endorses helmet use to reduce scalp lacerations, skull fractures, and intracranial bleeds [1]. For a helmet or headgear to prevent an SRC, it must attenuate linear and rotational acceleration, which are the primary underlying mechanisms of SRCs [2].

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Researchers have not been able to prove that helmets reduce SRCs, notwithstanding anecdotal evidence to the contrary. Investigating the effect of helmets to reduce SRC has proven to be challenging, especially in football, hockey, and other team collision sports, due to co-variability and ethical issues around having cohorts of athletes sustain from wearing helmets and comparing the rate of concussions in this group with athletes that are wearing helmets [3]. Players wear a variety of helmet brands and models, and different helmets may perform better under certain circumstances or when used by athletes in specific positions.

Changes to helmet design often involve tradeoffs. For example, heavier helmets typically attenuate linear acceleration but are less comfortable and accelerate rotational momentum and the duration of such rotation [2]. Many new helmet designs significantly reduce direct focal external force transfers, but these designs have not been proven to prevent linear and rotational acceleration. Interestingly, some argue that wearing seemingly more protective equipment, such as full-face masks in hockey and thicker padding in football helmets may, in fact, increase risky behaviors in wearers stemming from a false sense of security thereby exposing them to a greater risk for SRCs and other injuries [1, 4].

Football

Question: Are there specific football helmets that prevent concussions?

Football helmet manufacturers have developed and continue to develop helmets incorporating new designs and materials intended to reduce SRCs. The National Football League ("NFL"), in conjunction with the National Football League Players Association, the NFL players' union, conducts an annual laboratory test, the NFL/NFLPA Helmet Performance Test, which stimulates certain concussion-causing impacts to assess the performance of helmets worn by NFL players [5]. According to the 2019 NFL/NFLPA Helmet Performance Test, approximately 50% of NFL players upgraded to a better performing helmet after reviewing the study's results, and those wearing top performing helmets reported fewer concussions [5]. Nevertheless, research unaffiliated with the NFL has failed to show a meaningful difference in the incidence of SRCs with newer helmet models [6].

Other Sports

Question: Should athletes in soccer and other sports wear headgear to prevent concussions?

Studies have found that ski and snowboard helmets provide protection against head injuries, but, as another study pointed out, the studies did not separate concussions from other head injuries when looking at their efficacy towards concussion prevention

[3, 7, 8]. Several bicycle helmets, especially newer designs, have built-in mechanisms to reduce rotational head acceleration caused by an oblique impact [9–11]. Wearing headgear in soccer has been explored with conflicting evidence for SRC reduction benefit [12]. In the past, rugby headgear has not provided any protection against concussions [13]. However, newer headgear using a viscoelastic material has shown the ability to reduce linear and rotational impact energy in the test setting, which could potentially help reduce the rate of developing and the severity of concussions in rugby [14]. As stated above, the added protection may also inadvertently promote more aggressive and risky behavior.

Mouth Guards

Question: Do mouth guards have any affect in preventing or reducing the severity of concussions?

It is widely accepted that mouth guards protect against overall head injury, especially dental injuries, while playing contact sports. However, the effectiveness of mouth guards in reducing concussions specifically is less clear [15, 16]. A meta-analysis did suggest a nonsignificant trend towards a protective effect of mouth guards against concussions in contact sports, specifically when looking at basketball, ice hockey, and rugby [3].

Fit and Maintenance

Question: Is there any reputable sources to assist in properly fitting headgear?

Ensuring the proper fit of equipment is paramount. The use of an ill-fitting helmet, for example, is a risk factor for concussions with more symptoms and of longer duration [17]. The Centers for Disease Control and Prevention launched a mobile web-based application as part of their larger Heads Up: Concussion program to help find a properly fitting helmet for various sports [18]. It is also important to periodically inspect helmets, mouth guards, and other equipment throughout sports seasons in addition to at the beginning of each season and repair or replace whatever has become deformed, worn down, or no longer fits [17]. Moreover, designating a specific person to monitor for proper fit is essential, especially in youth and high school athletes where head size and hairstyle may change throughout the season.

Cost

Unfortunately, protective equipment in contact sports can be expensive. In 2016, *The Gadsden Times*, an Alabama newspaper, estimated that the cost of outfitting a high school football player for a practice and a game might be between $800 and

$1000 [19]. The list price of the top-ranked helmet in the 2019 NFL/NFLPA Helmet Performance Test is $950 [20].

Such expenses typically present, at minimum, financial hardship to parents and/or school districts and may even bar youth participation in the sport altogether. Programs that help provide youth athletes or youth sports teams with new or gently used equipment, such as Good Sports [21], Sports Matter [22], and The Sports Shed [23], may help reduce these costs. Additional care is required to ensure that such equipment is appropriate and fits correctly [24].

Athlete Bias

Question: Do athletes use the safety-related equipment that is designed to reduce the risk of concussion?

Countless examples exist of professional athletes resisting safety-related equipment upgrades for behavioral and even cosmetic reasons [25, 26]. For example, the overwhelming majority of Major League Baseball pitchers do not wear protective liners and caps designed to protect them from dangerous line drives hit back at them. Such intransigence has been addressed through a combination of rule changes at professional and youth levels designed to socialize these players from a young age to the more protective equipment [27].

Technique

Question: Are organized sports organization incorporating skill instruction in an attempt to reduce the risk of sport-related concussions?

More than ever, organized sports have largely replaced neighborhood pickup games beloved by previous generations of Americans. While the proliferation of organized sport has its detractors and disadvantages, one potential benefit is an increase in opportunities for youth participants to learn proper techniques, especially in high-risk maneuvers associated with SRCs, such as football tackling, hockey body checking, and soccer heading [1, 28, 29].

Using this time for skill development and instruction instead of games may reduce SRCs. Athletes learn better body control to prepare them for the inevitable collisions that will occur when they grow older and join leagues that permit these maneuvers. Additionally, this training now incorporates sport-specific techniques to reduce head acceleration.

Football

USA Football, a youth football governing body, heavily promotes Heads Up Football ("HUF"), a series of online and in-person courses for coaches to learn about proper equipment fitting, tackling technique, and instruction in drills designed to reduce head contact [30]. HUF's tackling module includes fundamentals of and systems for teaching shoulder tackling and draws on principles of rugby tackling, which does not use the head [31]. A 2015 study found that HUF reduced injury rates but *The New York Times* raised issues with how USA Football and the study's authors presented certain data from that study [28, 32, 33]. Further, that study evaluated the entire HUF program so the efficacy, if any, of the HUF tackling model on reducing SRCs was debated. A more recent prospective study demonstrated a 33% reduction in SRC with HUF, providing some potential for benefit [34].

Soccer

Strategies to reduce head acceleration when heading the ball in soccer include achieving head-neck-torso alignment and neck strengthening (discussed below) [35, 36]. Other experts recommend using lightweight soccer balls to teach and perfect heading techniques [36]. In theory these recommendations make sense but lack the support of strong evidence-based clinical research.

Hockey

Coaching and education programs in the United States and Canada emphasize teaching youth to keep their heads up, especially when about to receive a check. Since the implementation of programs emphasizing these skills there has been a decrease in cervical spine injuries, but the effectiveness of these techniques have not been formally assessed [37]. USA Hockey's American Development model emphasizes skill and skating development including proper body control, angling, and body contact but does not teach body checking skills until 11–12-year-old age group [37].

Neck Strengthening

Question: Do neck strengthening exercise prevent concussions?

Small and/or weak neck musculature is an SRCs risk factor [38]. Neck strengthening may help prevent SRCs by reducing head acceleration [39, 40]. Support for neck strengthening exists primarily based on anecdotal evidence- and lab-based testing,

but not clinical evidence. One such lab study found that every 1-pound increase in neck strength contributes to a 5% decrease in odds for a concussive event to occur [38]. This protective benefit is likely due to the decreased kinematic response of the head to controlled impulsive loading with greater neck strength [39]. It is also believed that neck-strengthening exercises are more likely to reduce SRCs in females than males due to females' weaker necks [1, 38, 41–44]. Multiple neck strengthening programs have been proposed that show promise [39, 45]. However, further research is required on the benefit of neck strengthening on concussion prevention during actual play.

Rule Changes

Question: How effective have rule changes been in reducing the number of concussions? Is there a difference in the effectiveness from sport to sport?

Rule changes intended to reduce collisions that often result in concussion have been proposed and implemented in several sports. The difficulty in coming up with new rules is finding a balance between limiting the number of head collisions while maintaining the game's integrity. Additionally, rule changes in specific sports cannot be generally applied across all levels of play from youth to the elite level.

Football

Football has implemented several safety-related rule changes in an effort to reduce SRCs and other injuries. In 2011, the NFL moved the kickoff line forward by 5 yards in an attempt to prevent concussions. That rule change was intended to increase rates of touchbacks on kickoffs, a play involving high-speed collisions in which a disproportionate number of SRCs have been found to occur. In 2016, the Ivy League, a Division I National Collegiate Athletic Association conference, moved the kickoff and touchback lines up to the 40-yard and 25-yard lines, respectively, for the same reasons. A 2018 study found these rule changes reduce the average annual concussion rate in Ivy League football by more than 68% [46].

Certain youth football leagues have postponed tackling until a certain age and/or reduced the number of contact practices [47].The NFL also adopted a rule beginning in the 2018–2019 season making it a foul for a player to lower his head to initiate and make contact with his helmet against an opponent [48].

Hockey

Body checking and fighting, which are associated with higher risk of SRCs, are hot button, safety-related issues debated in hockey. Until the 2010–2011 season, a body check to an opponent's head as the primary point of contact was legal in the National Hockey League ("NHL"). Beginning that season, in an effort to prevent SRCs and other head injuries, the NHL adopted Rule 48.1, which made *targeting* an opponent's head from the blind side illegal. An independent study released in July 2013 found no decrease in concussion incidence among NHL players following implementation of Rule 48 [49].[1]

Removing body checking at the youth level has also gained popularity [37]. Since 2011, USA Hockey, the American youth hockey governing board, has prohibited all body checking in players 12 and younger [37]. In Canada, the age at which body checking is allowed has also increased to 13 years old [37]. A 2011 Canadian study found that eliminating body checking under age 13 lead to significant reduction in SRCs among youth 13 and under compared to SRCs among similarly aged youth playing in leagues that allowed checking [50]. Opponents of the youth body checking ban were concerned that such a ban would increase the risk of injury, including SRCs, to young players by depriving them of the chance to learn proper body checking technique once they grew and joined leagues in which body checking is permissible. The authors of this chapter are not aware of any research that substantiates this concern and the same 2011 Canadian study specifically refuted it [37, 50]. Disallowing body checking has also been incorporated into older age groups in non-elite levels, but the impact on concussions in these groups requires further investigation [51].

In an effort to reduce fighting, beginning the 2016–17 season, the American Hockey League adopted Rule 23.7, which provides an automatic one-game suspension after a player incurs ten fighting major penalties during the regular season [52]. The Ontario Hockey League adopted a similar rule in 2011 [53].

Other Sports

Other sports have also instituted rule changes in an effort to reduce SRCs. For example, since 2014, Major League Baseball has banned avoidable collisions between catchers and base runners at home plate by initiating a rule that disallows runners attempting to score from deviating from his direct pathway to the plate [54].

[1] Rule 48 has been revised several times since the 2010–2011 and season and currently to define an illegal check to the head as "a hit resulting in contact with an opponent's head where the head was the main point of contact and such contact to the head was *avoidable*." http://www.nhl.com/nhl/en/v3/ext/rules/2018-2019-NHL-rulebook.pdf; https://www.cbc.ca/sports-content/hockey/opinion/2013/09/30-thoughts-nhl-clarifies-illegal-check-to-head-rule.htm

In 2015, US Soccer, the soccer governing body in the United States, banned heading among players younger than age 10 and limited the amount of heading in practice among players ages 11–13 [55, 56].

Rule Enforcement

Question: How effective have rule changes been in reducing illegal play?

While the rules of play form the basis of safer play, athletes, coaches, and officials need to adhere to the rules in order for them to make a difference [37]. In high school athletes, illegal activity contributed to over 10% of injuries in boys and girls soccer and basketball, and concussions made up the greatest percentage of those injuries [57]. The injuries related to illegal play are even greater in other leagues and have been reported to be as high as 50% [57, 58]. Promoting fair and safer play requires an attitude shift and modeling by coaches, parents, officials, and managers in addition to the athletes. In ice hockey, fair play rules, a program developed to reward teams with good sportsmanship, has contributed to a significant reduction in injuries including concussions [37]. Additionally, the zero tolerance to head contact rule change in the NHL has led to a 36% reduction in concussion risk [49]. In soccer, stricter enforcement of red cards for high elbows during heading duals has led to a slightly reduced risk of concussion [3, 59].

Legislation

Question: Have states implemented legislation to protect children from SRCs?

Is medical clearance needed prior to returning to sports following a concussion?

Concussion management in youth sports is subject to a state-by-state patchwork of laws and regulations [60, 61]. The most well-known of these laws is Washington State's Lystedt Law [62]. The Lystedt Law was enacted in 2009 in the aftermath of the tragic death of Zackery Lystedt who suffered multiple concussions in a single game resulting in intracranial hemorrhage and severe traumatic brain injury. The Lystedt Law has three main components: (1) removal; (2) medical clearance for return-to-play; and (3) education [60, 62]. Since the Lystedt Law's enactment, all 50 states have enacted some form of legislation to protect children from SRCs. Many of these are patterned on the Lystedt Law but significant differences exist.

Several studies have looked at the impact of the Lystedt Law in Washington State and similar laws in other states on concussions in high school athletes [61, 63–65]. Studies have found an increase both in the frequency of concussions and the mean number of days during which concussed youths are held out of play. This is likely due to the increased awareness of concussions in addition to the need for medical clearance prior to returning to play. In regards to implementation, high school football and soccer coaches in Washington State endorsed receiving appropriate concussion education 3-years after the Lystedt Law was passed [66]. However, there is still a lot that must be done to in regards to implementation of the laws [61, 63]. One study found that after enacting a concussion law in Ohio the rate of follow-up after an initial ED visit for concussion increased from 44% pre-law to 58% post-law, which means that nearly 40% of concussed players did not receive appropriate follow-up and therefore clearance [61, 64].

Education

Question: Do formal concussion education programs improve concussion knowledge? Does education decrease the rate of concussions?

Several formal education programs have been established that focus on both primary and secondary prevention strategies with specific information geared towards the athletes, coaches/staff, parents, healthcare providers, and/or the public. There is consensus among policies, guidelines, and consensus statements on school sport injury prevention that education is the mainstay of concussion prevention [24]. A large component of programs is teaching improved identification and reporting of concussions to protect against athletes the potential consequences of playing with concussions. Most also emphasize strict adherence to return to play guidelines (refer to chapter on return to play). Programs also aim to educate on the short and long-term consequences of suffering a concussion with the hope of changing attitudes on the playing field to prevent risky behaviors that may lead to concussions. While several educational programs have been developed and most show increased rates of concussion knowledge after the programs, further research into whether they actually decrease the rates of concussions is required. The specific education method or program should be tailored to each individual group to optimize learning. Table 12.1 lists several education programs and resources that currently exist.

The annual pre-participation physical exam (PPE) can be very useful in identifying athletes who have a history of concussion and who may be at increased risk for concussion due to involvement in contact sports. The PPE provides an excellent educative opportunity to inform athletes and their parents of the significance of concussions [51]. The medical professional performing the exam should ask concussion-related questions including past history of concussion, duration of

Table 12.1 Education programs and resources

Program	Target audience	Website
Barrow Brainbook	High school athletes	https://concussion.barrowneuro.org
Brain 101: The Concussion Playbook	Coaches Educators Parents Teen athletes	http://brain101.orcasinc.com/1000/
Concussion Legacy Foundation Team Up against Concussions Advanced Concussion Training	Schools, community centers, and athletic programs with youth in grades 4–12 Families, coaches, teachers, medical professionals, and athletes beyond high school	https://concussionfoundation.org/programs/education
The Center for Disease Control and Prevention's Heads Up	Coaches Parents Athletes School professionals Healthcare provider	www.cdc.gov/headsup/index.html
Heads Up Football	Coaches	usafootball.com/programs/heads-up-football/
National Federation of State High School Associations' Concussion for Students	High school athletes	https://nfhslearn.com/courses?searchText=Concussion
NCAA Concussion Educational Resources	College athletes Coaches Athletic trainers Team physicians Athletic directors	http://www.ncaa.org/sport-science-institute/concussion-educational-resources
Rugbysmart	Coaches Referees	www.rugbysmart.co.nz
ThinkFirst About Concussion	Youth and teens	http://thinkfirst.org/concussion

Note: This table is not exhaustive

symptoms, and the presence of mood, learning attention, or migraine disorders, which have been shown to complicate the diagnosis and management of concussed athletes [1]. However, there is no evidence that pre-existing mood or learning disorders predisposes athletes to concussions.

Conclusion

Researchers have made considerable progress over the past 20 or so years in determining the causes of SRCs but have made fewer inroads in determining how to prevent them. There is no cure-all to prevent SRCs. This much is known.

Nevertheless, much of the existing research is imprecise and/or unsatisfying and questions abound. More research is required, and until such research becomes available, PCPs are left to endorse SRC prevention strategies based largely on intuition and anecdotal evidence.

Key Points

- Investigating the effect of helmets to reduce SRC has proven to be challenging and requires on-going research.
- Teaching proper technique in sports activities such as sport-specific techniques to reduce head acceleration may help to reduce concussions.
- Rule changes intended to reduce collisions that often result in concussion have been proposed and implemented in several sports.
- More research is required and until such research becomes available, PCPs are left to endorse SRC prevention strategies based largely on intuition and anecdotal evidence.

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