Chapter 10
Concussion in Sports: What Is It and How Is It Managed?

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Introduction: Defining Sports-Related Concussion

Sports-related concussion (SRC) is broadly defined as the immediate and transient symptoms of traumatic brain injury (TBI) [1]. The underlying issue with this definition is that it does not provide insight into the underlying processes causing brain impairment. This definition also does not distinguish between different grades of severity. The term mild traumatic brain injury (mTBI) is often used interchangeably with concussion. This term is also vague and lacks validated criteria in this context. Therefore, the term concussion with this usage is imprecise.

The most commonly recognized definition of concussion stems from the consensus statement from the 5th International Conference on Concussion in Sport, which states that sports-related concussion (SRC) is a traumatic brain injury (TBI) induced by biomechanical forces [1].

Several common features that may be utilized in clinically defining the nature of a concussive head injury include:

– SRC may be caused either by a direct blow to the head, face, neck, or elsewhere on the body with an impulsive force transmitted to the head.
– SRC typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, signs and symptoms evolve over a number of minutes to hours.

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- SRC may result in neuropathological changes, but the acute clinical signs and symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging studies.
- SRC results in a range of clinical signs and symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive features typically follows a sequential course. However, in some cases, symptoms may be prolonged.
- The clinical signs and symptoms cannot be explained by drug, alcohol, or medication use, other injuries (such as cervical injuries, peripheral vestibular dysfunction, etc.) or other comorbidities (e.g., psychological factors or coexisting medical conditions).

Several definitions of mild traumatic brain injury (mTBI) have been used over the past decades [2]. Traumatic brain injury (TBI) is typically graded from mild to severe, with use of the clinical assessment of the Glasgow Coma Scale (GCS). The GCS has a composite score ranging from 3 to 15. A numerical assignment is awarded according to the patient's eye opening, motor responses to verbal and physical stimuli, and verbal responses to questions and commands. Based on this scale, mTBI refers to patients with a GCS score of 13 to 15. The World Health Organization (WHO) Collaborating Centre for Neurotrauma Prevention, Management and Rehabilitation Task Force on Mild Traumatic Brain Injury proposed the following mTBI definition [3]:

> MTBI is an acute brain injury resulting from mechanical energy to the head from external physical forces. Operational criteria for clinical identification include: (i) 1 or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery; (ii) Glasgow Coma Scale score of 13–15 after 30 minutes post-injury or later upon presentation for health care. These manifestations of MTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries or intubation), caused by other problems (e.g. psychological trauma, language barrier or coexisting medical conditions) or caused by penetrating craniocerebral injury.

This definition set forth by the WHO was created as an attempt to standardize an operational definition of mTBI for research purposes.

MTBI may also apply to patients in whom structural lesions are found on standard neuroimaging, or those with focal neurological deficits. This is not consistent with the most commonly accepted definition of concussion. Such imaging findings may include epidural or subdural hematoma, subarachnoid hemorrhage, parenchymal contusion, skull fractures, etc. [4]. The focus of this chapter is traumatic brain injury as it relates to functional brain abnormalities, without the structural changes seen on standard neuroimaging. Please refer to Table 10.1, which provides descriptions of various levels of TBI severity according to GCS score.

Table 10.1 Classification of head injury severity

Classification	Glasgow Coma Scale Score
Mild TBI	13–15
Moderate TBI	9–12
Severe TBI	3–8

TBI traumatic brain injury

Epidemiology

TBI is a global healthcare-related problem of paramount importance [5]. An estimated 1.7 million TBIs occur annually in the United States. A majority of these TBIs are mild [6]. TBI incidence in military personnel, commonly overshadowed by the focus on sports-related injuries, has gained increased attention in recent years. Mild TBI occurred in 20% of deployed personnel, and accounted for the majority of military-associated head injuries during the conflicts in Iraq and Afghanistan [7]. Blast injury caused by improvised explosive devices (IEDs) is an especially common cause of mTBI and concussion, contributing to as much as 79% of these injuries [8–13].

The number of American patients seeking care for traumatic brain injuries, most of which are mild in nature, has been on the rise. Marin and colleagues [14] estimated that there were 2,544,087 visits to emergency departments (EDs) for TBI in 2010. This represents a 29.1% increase when compared to the number of ED visits in 2006. This rate of increase is nearly eight times greater than the rate of increase of total ED visits over the same time frame; it is most likely attributed to a combination of factors such as increases in TBI awareness, exposure, and diagnoses. Children <3 years old, and adults >60 years old had the highest rates of ED visits, with falls serving as the leading mechanisms of injury [14].

The annual incidence of sports-related concussion in the United States is estimated to be approximately 3.8 million. This number likely underestimates the true incidence given that many concussions might go unreported or affected athletes might have no gateway to formal medical follow-up [15]. Clay and associates [16] performed a systematic review of the epidemiology of concussion in sport, and reported that the highest reported incidences of concussion, in descending order, are related to football, female and male soccer, wrestling, and female basketball. Between 2% and 15% of athletes participating in organized sports will suffer a concussion during one season (Table 10.2) [17–35].

Football accounts for the highest proportion of sports-related concussion overall [16]. The incidence ranges from 0.48 to 1.03 per 1000 athletic exposures (AEs) at the high school level, 0.52 to 0.81 per 1000 AEs at the college level, and 4.56 per 1000 game AEs in the National Football League (NFL). This NFL data does not include concussions sustained during practice [16, 36–40] Linebackers are the most commonly concussed players in football in all levels of play overall. Quarterbacks are the

Table 10.2 Seasonal risk of concussion in sports

Author	Type of athletes	Years of study	Seasons (n)	Athletes (n)	Concussed	Concussed per player/season (%)
Football						
Barr and McCrea [19]	High school and college football	1997–1999	2	1313	50	1.9
McCrea [18]	High school and college football	1998–1999	2	1325	63	2.4
McCrea et al. [17]	High school and college football	1999–2001	3	2385	91	3.8
McCrea et al. [19]	College football	1999–2001	2		94	3.9
Barr et al. [16]	High school and college football	2008–2009	2	823	59	7.2
Seidman et al. [24]	High school football	2013	1	343	9	2.6
Dompier et al. [25]	Football	2012–2013	2	20,479	1178	5.8
	Youth football	2012–2013	2	4092	136	3.3
	High school football	2012–2013	2	11,957	767	6.4
	College football	2012–2013	2	4430	275	6.7
	College football	2011–2014	4	9718	518	5.3
Houck et al. [26]	College football	2006–2015	9	945[a]	118	12.5
Bretzin et al. [14]	High school football	2015–2016	1	39,520	1530	3.9
Total football				**67,133**	**3192**	**4.8**
All sports						
Galetta et al. [27]	Football, sprint football, men's and women's soccer and basketball	2010–2011	1	219	10	4.6
Marinides et al. [20]	College athletes	2011–2012	1	217	30	13.8
Galetta et al. [21]	Ice hockey/lacrosse youth and college		1	332	12	3.6
Leong et al. [28]	Football, men's and women's basketball	2012–2013	1	127	11	8.7
Putukian et al. [22]	College athletes	2011–2012	1	263	32	12.2

Table 10.2 (continued)

Author	Type of athletes	Years of study	Seasons (n)	Athletes (n)	Concussed	Concussed per player/season (%)
Chin et al. [23]	High school and college athletes	2012–2014	3	2018	166	2.7
Kerr et al. [12]	NCAA athletes	2011–2014	4	32,156	1410	4.4
	Men's baseball	2011–2014	4	1757	13	0.7
	Men's basketball	2011–2014	4	1889	74	3.9
	College football	2011–2014	4	9718	518	5.3
	Men's ice hockey	2011–2014	4	3689	253	6.9
	Men's lacrosse	2011–2014	4	1768	44	2.5
	Men's soccer	2011–2014	4	1810	29	1.6
	Men's wrestling	2011–2014	4	821	65	7.9
	Women's basketball	2011–2014	4	1690	90	5.3
	Women's ice hockey	2011–2014	4	1301	94	7.2
	Women's lacrosse	2011–2014	4	1522	49	3.2
	Women's softball	2011–2014	4	1569	38	2.4
	Women's soccer	2011–2014	4	2831	93	3.3
	Women's volleyball	2011–2014	4	1791	50	2.8
Dhawan et al. [29]	Youth hockey		1	141	20	14.2
Tsushima et al. [13]	Athletes grades 8–12	2013–2014	1	10,334	1250	12.1
Bretzin et al. [14]	High school athletes in 15 sports	2015–2016	1	193,757	3352	1.7
Total				**239,564**	**6293**	**2.6**

Table reproduced from Harmon et al. [17]

[a]Total number of athletes estimated using 105 athletes per year on football roster. NCAA, National Collegiate Athletic Association

most frequently concussed players in the NFL [16]. The majority of concussions are sustained during competition rather than practices, with high-speed player-to-player impact serving as the most common mechanism for concussive injury [41–43].

The majority of concussions are sustained during competition rather than practices, with high-speed player-to-player impact serving as the most common mechanism for concussive injury. This correlates with helmet accelerometer data collected in a study of a college football team. Data from this study demonstrated that impacts during football games are nearly three times higher than those during practice [44].

It is unclear why high school athletes report a higher incidence of concussion when compared to college and professional athletes. These findings are unexpected, given that increased high impact exposure and increased cumulative concussions are established risk factors for sustaining concussions in the future [16, 45]. Proposed explanations, which are not well established, have included the immature anatomy of younger athletes, style of play differences (more running plays vs. passing plays in high school football), injury reporting differences, and greater force per hit due to lack of experience [46]. In most sex-comparable sports (excluding lacrosse), concussion incidence is consistently higher in females than in males [17–23]. A study of the National College Athletic Association ice hockey found that in women's hockey, which prohibits body checking, player contact was the source of 41% of concussions, compared with 72% in men's hockey [47]. Additionally, women suffer fewer impacts and experience lower head acceleration than men. This suggests that these factors are not the direct cause of an overall higher incidence of concussion in female athletes [48]. Various studies have proposed that differences in incidence may relate to differences in reporting, as well as the differences in the size and strength of head and neck musculature between males and females [49].

Concussion Diagnosis

The diagnosis of concussion is based on assessment of a range of domains, which includes physical signs, cognitive impairment, clinical symptoms, neurobehavioral features, and sleep disturbance [1]. If even one of these signs is present, concussion should be suspected, and the proper management strategy should be implemented [1]. Diagnosis requires awareness of multiple symptoms that may be present with concussive injury paired with a focused clinical examination. On-site assessment tools may aid the provider in the diagnosis of concussion. Adjuncts to the clinical assessment, such as advanced neuropsychological testing, neuroimaging techniques, and blood-based biomarkers of injury, can also be used to aid in identifying injury and secure the diagnosis of concussion. Although these new technologies are not required or recommended for routine clinical care, they show promise as investigative tools to advance our understanding of the pathophysiology of concussion and recovery. Through advanced imaging techniques and analysis of biomarkers that are released following injury, research has conceded that considering concussion as only a functional entity is an inappropriate underestimate.

There is a wide range of variability in the quality of training and experience of health care professionals diagnosing and caring for concussed athletes. This

contributes to uncertainty and inconsistency in the various aspects of concussion diagnosis, prognostication, and treatment in affected athletes. In 2013, The Guideline Development Subcommittee of the American Academy of Neurology (AAN) published the "Evidence-based Guideline Update: Evaluation and Management of Concussion in Sports." [50] This comprehensive publication reviews evidence in children, adolescents, and adults from 1955 through 2012 in an attempt to bring consistency to this clinical entity.

Concussion: Signs and Symptoms

The clinical manifestations of concussion affect one or more of the domains of somatic, cognitive, behavioral, emotional, vestibular, cervical, autonomic, and sleep/wake symptoms. Common signs and symptoms of concussion are headache, fatigue, dizziness, amnesia, irritability, anxiety, poor concentration, photophobia/phonophobia, disorientation, and postural instability. See Table 10.3 for a summary of commonly reported signs and symptoms of injury.

Table 10.3 Common signs and symptoms of mild traumatic brain injury

Signs of injury
Loss of consciousness
Retrograde amnesia (forgetting events that happened before the concussion)
Anterograde or post-traumatic amnesia (forgetting events that happen after the concussion)
"Dazed" look
Confusion about injury events or details
Disorientation to person, place, or time
Emotional lability
Inappropriate emotions
Behavior or personality changes
Symptoms of injury
Headache
Dizziness
Balance problems
Fatigue
Visual changes (double or blurry vision most common)
Insomnia
Hypersomnia
Drowsiness
Attentional dysfunction
Short-term memory and learning problems
Difficulty multitasking
Phonophobia
Photophobia
Bradyphrenia
Feeling mentally "foggy"
Emotional changes

These signs and symptoms are sometimes associated with other conditions in athletes, including dehydration, exertional migraines, heat-related illness, and sleep disorders. Thus, it is important to establish a relationship between mechanism of injury and the onset or worsening of symptoms [51–55]. Physicians and others caring for athletes must remember that although symptoms typically begin immediately following the inciting trauma, some patients may not experience symptoms for several hours after the injury [56].

Headache is the most common reported symptom of concussion, present in 83–86% of patients with concussion [57, 58]. The headache characteristics experienced after concussion or mTBI may include tension-type, cluster-like, migraine-type, and mixed. The mechanism for headache following head injury is not well understood. Both migraine and mTBI have common pathophysiologic effects, including increases in intracellular sodium, calcium, chloride, and extracellular potassium. Both conditions have been linked to increased release of excitatory amino acids, such as glutamate [59].

Post-concussion headache may be associated with slowed reaction time, memory impairment, on-field anterograde amnesia, and increased overall symptoms [60]. Patients with migrainous headache after concussion scored lower on several neurocognitive measures (processing speed, verbal and visual memory, and reaction time) than those with nonmigrainous headaches or no headache after concussion [61]. Patients with migrainous headache also reported more or worse symptoms relative to the other two groups [61].

Mental status changes and subtle neurocognitive deficits are also quite common with concussion. Confusion has been a longstanding hallmark of concussion [62]. Confusion may also include a disturbance of vigilance with heightened distractibility, inability to maintain a coherent stream of thought, or the inability to carry out a sequence of goal-directed movements. Although disorientation may be present during a concussion, more subtle changes, as described previously, are more common and should be sought during examination of the patient or athlete [63]. One study highlighted that concussed athletes exhibiting "fogginess" have demonstrated poor performance on memory, reaction time, and processing speed measures, and an overall higher total symptom score [64].

Loss of consciousness (LOC) was previously viewed as a requisite for concussion, whereas the literature shows that more than 90% of concussions occur without LOC. Concussions of higher severity with corresponding signs and symptoms may occur with no LOC [65]. LOC occurs in 8–19% of sports-related concussions, with most LOC lasting less than one minute [66, 67]. The duration of LOC is not correlated to injury severity in concussion studies [68, 69]. This is contrary to what is reported in moderate and severe TBI studies [68, 69].

Posttraumatic amnesia can occur immediately, or several minutes after concussive injury in athletes [70–72]. Both retrograde and anterograde amnesia may be associated with neurocognitive deficits, and more reported symptoms in the days immediately following concussion [73]. The presence or absence of post-concussive amnesia as a predictor of long-term outcome is not currently established in the literature [1].

Decreased postural control after concussion is well established [74]. Assessing for this may be carried out by having the patient stand upright with the eyes closed.

Eliminating visual referencing with eye closure accentuates postural control deficit that is associated with an inability to process altered sensory information [74]. Decreased postural stability symptoms typically persist up to 3 days following injury [75].

It is important to recognize that a variety of immediate post-concussive motor phenomena, such as convulsive movements and tonic posturing, may accompany a concussion [1]. These clinical features are typically benign and do not require specific management [1].

Acute Concussion: On-Field Assessment and Sideline Assessment Tools

The Consensus Statement on Concussion in Sport from the International Conference on Concussion in Sport states [1]:

When a player shows any symptoms or signs of an SRC:

(a) The player should be evaluated by a physician or other licensed healthcare provider on site using standard emergency management principles, and particular attention should be given to excluding a cervical spine injury.
(b) The appropriate disposition of the player must be determined by the treating healthcare provider in a timely manner. If no healthcare provider is available, the player should be safely removed from practice or play and urgent referral to a physician arranged.
(c) Once the first aid issues are addressed, an assessment of the concussive injury should be made using the SCAT5 or other sideline assessment tools.
(d) The player should not be left alone after the injury, and serial monitoring for deterioration is essential over the initial few hours after injury.
(e) A player with diagnosed SRC should not be allowed to return to play on the day of injury.

Signs and symptoms which are of particular concern include severe or progressively worsening headaches, positive findings on neurological examination, emesis, or rapid decline in mental status that may indicate a more life-threatening injury (e.g., epidural or subdural hematoma, intraparenchymal hemorrhage) and necessitates immediate transfer to the emergency department.

There are a variety of sideline assessment tools that gauge concussion-related symptoms, neuropsychologic function, and balance [1, 76–79]. The most commonly used sideline assessment tool is the Sports Concussion Assessment Tool, fifth edition (SCAT5). This assessment includes a standardized evaluation of signs and symptoms, neurologic function, cognition, and balance [1]. The original SCAT was developed by the second International Conference on Concussion in Sport [1]. The SCAT5 is designed for use in athletes age 13 years or older, and combines aspects of several previously published concussion tools into multiple components (Fig. 10.1) [1]. In order to eliminate the possible confounding effects of fatigue from competitive play when testing for concussive symptoms, the athlete should be

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To download a clean version of the SCAT tools please visit the journal online (http://dx.doi.org/10.1136/bjsports-2017-097506SCAT5)

SCAT5©

SPORT CONCUSSION ASSESSMENT TOOL – 5TH EDITION

DEVELOPED BY THE CONCUSSION IN SPORT GROUP
FOR USE BY MEDICAL PROFESSIONALS ONLY

supported by

FIFA

Patient details

Name: _____

DOB: _____

Address: _____

ID number: _____

Examiner: _____

Date of Injury: _____ Time: _____

WHAT IS THE SCAT5?

The SCAT5 is a standardized tool for evaluating concussions designed for use by physicians and licensed healthcare professionals[1]. The SCAT5 cannot be performed correctly in less than 10 minutes.

If you are not a physician or licensed healthcare professional, please use the Concussion Recognition Tool 5 (CRT5). The SCAT5 is to be used for evaluating athletes aged 13 years and older. For children aged 12 years or younger, please use the Child SCAT5.

Preseason SCAT5 baseline testing can be useful for interpreting post-injury test scores, but is not required for that purpose.Detailed instructions for use of the SCAT5 are provided on page 7. Please read through these instructions carefully before testing the athlete. Brief verbal instructions for each test are given in italics. The only equipment required for the tester is a watch or timer.

Recognise and Remove

A head impact by either a direct blow or indirect transmission of force can be associated with a serious and potentially fatal brain injury. If there are significant concerns, including any of the red flags listed in Box 1, then activation of emergency procedures and urgent transport to the nearest hospital should be arranged.

Key points

- Any athlete with suspected concussion should be REMOVED FROM PLAY, medically assessed and monitored for deterioration. No athlete diagnosed with concussion should be returned to play on the day of injury.
- If an athlete is suspected of having a concussion and medical personnel are not immediately available, the athlete should be referred to a medical facility for urgent assessment.
- Athletes with suspected concussion should not drink alcohol, use recreational drugs and should not drive a motor vehicle until cleared to do so by a medical professional.
- Concussion signs and symptoms evolve over time and it is important to consider repeat evaluation in the assessment of concussion.
- The diagnosis of a concussion is a clinical judgment, made by a medical professional. The SCAT5 should NOT be used by itself to make, or exclude, the diagnosis of concussion. An athlete may have a concussion even if their SCAT5 is "normal".

Remember:

- The basic principles of first aid (danger, response, airway, breathing, circulation) should be followed.
- Do not attempt to move the athlete (other than that required for airway management) unless trained to do so.
- Assessment for a spinal cord injury is a critical part of the initial on-field assessment.
- Do not remove a helmet or any other equipment unless trained to do so safely.

Davis GA, *et al. Br J Sports Med* 2017;**0**:1–8. doi:10.1136/bjsports-2017-097506SCAT5

Fig. 10.1 Developed by the concussion in sport group, SCAT5 is a standardized tool used by Medical professionals and qualified health care professionals to aid in evaluation of athletes suspected of having sustained a concussion [1]. SCAT 5 can also be used to obtain baseline data among health athletes for future reference

1

IMMEDIATE OR ON-FIELD ASSESSMENT

The following elements should be assessed for all athletes who are suspected of having a concussion prior to proceeding to the neurocognitive assessment and ideally should be done on-field after the first first aid / emergency care priorities are completed.

If any of the "Red Flags" or observable signs are noted after a direct or indirect blow to the head, the athlete should be immediately and safely removed from participation and evaluated by a physician or licensed healthcare professional.

Consideration of transportation to a medical facility should be at the discretion of the physician or licensed healthcare professional.

The GCS is important as a standard measure for all patients and can be done serially if necessary in the event of deterioration in conscious state. The Maddocks questions and cervical spine exam are critical steps of the immediate assessment; however, these do not need to be done serially.

Name: _____

DOB: _____

Address: _____

ID number: _____

Examiner: _____

Date: _____

STEP 1: RED FLAGS

RED FLAGS:

- Neck pain or tenderness
- Double vision
- Weakness or tingling/ burning in arms or legs
- Severe or increasing headache
- Seizure or convulsion
- Loss of consciousness
- Deteriorating conscious state
- Vomiting
- Increasingly restless, agitated or combative

STEP 2: OBSERVABLE SIGNS

Witnessed ☐ Observed on Video ☐

Lying motionless on the playing surface	Y	N
Balance / gait difficulties / motor incoordination: stumbling, slow / laboured movements	Y	N
Disorientation or confusion, or an inability to respond appropriately to questions	Y	N
Blank or vacant look	Y	N
Facial injury after head trauma	Y	N

STEP 3: MEMORY ASSESSMENT MADDOCKS QUESTIONS[2]

"I am going to ask you a few questions, please listen carefully and give your best effort. First, tell me what happened?"

Mark Y for correct answer / N for incorrect

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

Note: Appropriate sport-specific questions may be substituted.

STEP 4: EXAMINATION GLASGOW COMA SCALE (GCS)[3]

Time of assessment			
Date of assessment			
Best eye response (E)			
No eye opening	1	1	1
Eye opening in response to pain	2	2	2
Eye opening to speech	3	3	3
Eyes opening spontaneously	4	4	4
Best verbal response (V)			
No verbal response	1	1	1
Incomprehensible sounds	2	2	2
Inappropriate words	3	3	3
Confused	4	4	4
Oriented	5	5	5
Best motor response (M)			
No motor response	1	1	1
Extension to pain	2	2	2
Abnormal flexion to pain	3	3	3
Flexion / Withdrawal to pain	4	4	4
Localizes to pain	5	5	5
Obeys commands	6	6	6
Glasgow Coma score (E + V + M)			

CERVICAL SPINE ASSESSMENT

Does the athlete report that their neck is pain free at rest?	Y	N
If there is NO neck pain at rest, does the athlete have a full range of ACTIVE pain free movement?	Y	N
Is the limb strength and sensation normal?	Y	N

In a patient who is not lucid or fully conscious, a cervical spine injury should be assumed until proven otherwise.

Davis GA, *et al. Br J Sports Med* 2017;**0**:1–8. doi:10.1136/bjsports-2017-097506SCAT5

Fig. 10.1 (continued)

OFFICE OR OFF-FIELD ASSESSMENT

Please note that the neurocognitive assessment should be done in a distraction-free environment with the athlete in a resting state.

STEP 1: ATHLETE BACKGROUND

Sport / team / school: _____

Date / time of injury: _____

Years of education completed: _____

Age: _____

Gender: M / F / Other

Dominant hand: left / neither / right

How many diagnosed concussions has the athlete had in the past?: _____

When was the most recent concussion?: _____

How long was the recovery (time to being cleared to play) from the most recent concussion?: _____ (days)

Has the athlete ever been:

Hospitalized for a head injury?	Yes	No
Diagnosed / treated for headache disorder or migraines?	Yes	No
Diagnosed with a learning disability / dyslexia?	Yes	No
Diagnosed with ADD / ADHD?	Yes	No
Diagnosed with depression, anxiety or other psychiatric disorder?	Yes	No

Current medications? If yes, please list:

_____

_____

_____

_____

_____

_____

Name: _____

DOB: _____

Address: _____

ID number: _____

Examiner: _____

Date: _____

2

STEP 2: SYMPTOM EVALUATION

The athlete should be given the symptom form and asked to read this instruction paragraph out loud then complete the symptom scale. For the baseline assessment, the athlete should rate his/her symptoms based on how he/she typically feels and for the post injury assessment the athlete should rate their symptoms at this point in time.

Please Check: ☐ **Baseline** ☐ **Post-Injury**

Please hand the form to the athlete

	none	mild		moderate		severe	
Headache	0	1	2	3	4	5	6
"Pressure in head"	0	1	2	3	4	5	6
Neck Pain	0	1	2	3	4	5	6
Nausea or vomiting	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Blurred vision	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like "in a fog"	0	1	2	3	4	5	6
"Don't feel right"	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Fatigue or low energy	0	1	2	3	4	5	6
Confusion	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
More emotional	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervous or Anxious	0	1	2	3	4	5	6
Trouble falling asleep (if applicable)	0	1	2	3	4	5	6
Total number of symptoms:							of 22
Symptom severity score:							of 132
Do your symptoms get worse with physical activity?						Y	N
Do your symptoms get worse with mental activity?						Y	N
If 100% is feeling perfectly normal, what percent of normal do you feel?							

If not 100%, why?

_____

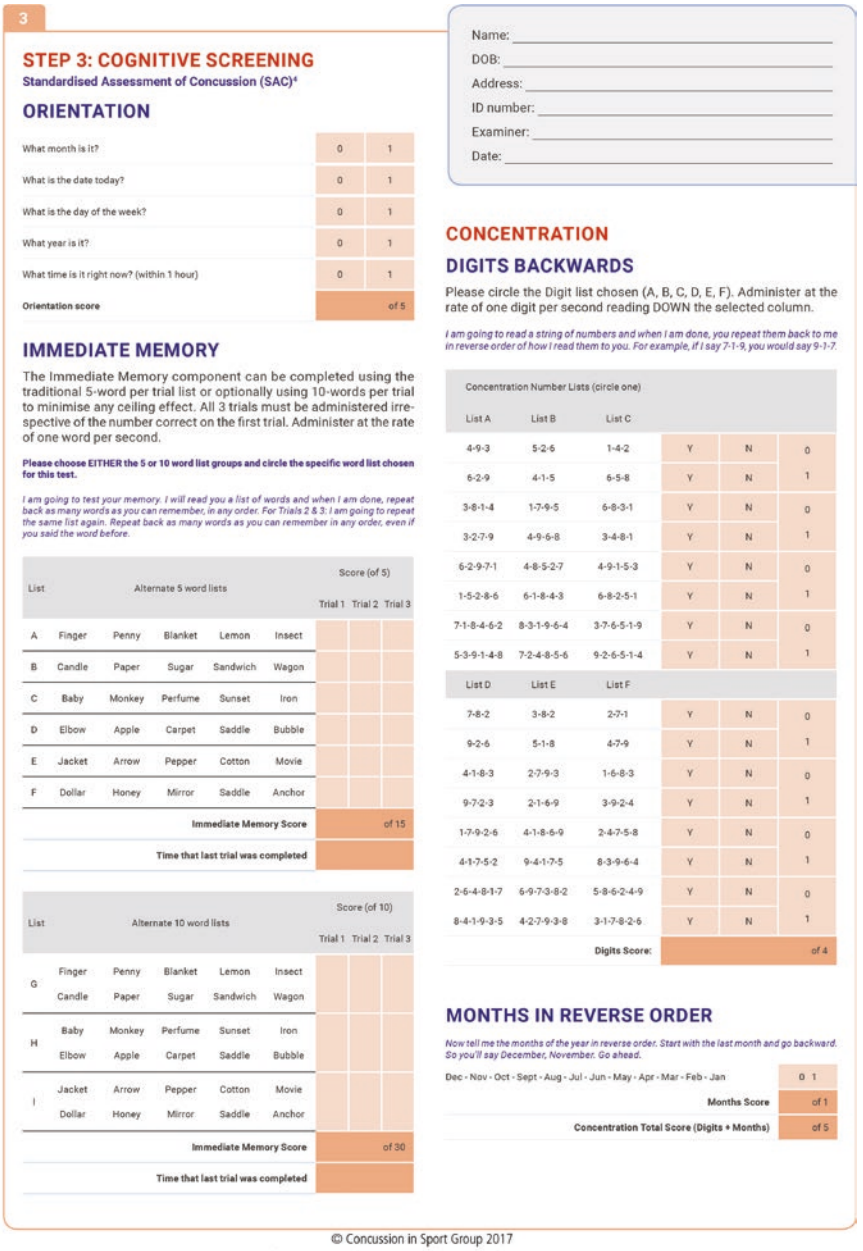
_____

_____

Please hand form back to examiner

Davis GA, *et al. Br J Sports Med* 2017;**0**:1–8. doi:10.1136/bjsports-2017-097506SCAT5

Fig. 10.1 (continued)



3

STEP 3: COGNITIVE SCREENING

Standardised Assessment of Concussion (SAC)[4]

Name: _____

DOB: _____

Address: _____

ID number: _____

Examiner: _____

Date: _____

ORIENTATION

What month is it?	0	1
What is the date today?	0	1
What is the day of the week?	0	1
What year is it?	0	1
What time is it right now? (within 1 hour)	0	1
Orientation score		of 5

IMMEDIATE MEMORY

The Immediate Memory component can be completed using the traditional 5-word per trial list or optionally using 10-words per trial to minimise any ceiling effect. All 3 trials must be administered irrespective of the number correct on the first trial. Administer at the rate of one word per second.

Please choose EITHER the 5 or 10 word list groups and circle the specific word list chosen for this test.

I am going to test your memory. I will read you a list of words and when I am done, repeat back as many words as you can remember, in any order. For Trials 2 & 3: I am going to repeat the same list again. Repeat back as many words as you can remember in any order, even if you said the word before.

List	Alternate 5 word lists					Score (of 5) Trial 1	Trial 2	Trial 3
A	Finger	Penny	Blanket	Lemon	Insect			
B	Candle	Paper	Sugar	Sandwich	Wagon			
C	Baby	Monkey	Perfume	Sunset	Iron			
D	Elbow	Apple	Carpet	Saddle	Bubble			
E	Jacket	Arrow	Pepper	Cotton	Movie			
F	Dollar	Honey	Mirror	Saddle	Anchor			
					Immediate Memory Score			of 15
					Time that last trial was completed			

List	Alternate 10 word lists					Score (of 10) Trial 1	Trial 2	Trial 3
G	Finger Candle	Penny Paper	Blanket Sugar	Lemon Sandwich	Insect Wagon			
H	Baby Elbow	Monkey Apple	Perfume Carpet	Sunset Saddle	Iron Bubble			
I	Jacket Dollar	Arrow Honey	Pepper Mirror	Cotton Saddle	Movie Anchor			
					Immediate Memory Score			of 30
					Time that last trial was completed			

CONCENTRATION

DIGITS BACKWARDS

Please circle the Digit list chosen (A, B, C, D, E, F). Administer at the rate of one digit per second reading DOWN the selected column.

I am going to read a string of numbers and when I am done, you repeat them back to me in reverse order of how I read them to you. For example, if I say 7-1-9, you would say 9-1-7.

Concentration Number Lists (circle one)					
List A	List B	List C			
4-9-3	5-2-6	1-4-2	Y	N	0
6-2-9	4-1-5	6-5-8	Y	N	1
3-8-1-4	1-7-9-5	6-8-3-1	Y	N	0
3-2-7-9	4-9-6-8	3-4-8-1	Y	N	1
6-2-9-7-1	4-8-5-2-7	4-9-1-5-3	Y	N	0
1-5-2-8-6	6-1-8-4-3	6-8-2-5-1	Y	N	1
7-1-8-4-6-2	8-3-1-9-6-4	3-7-6-5-1-9	Y	N	0
5-3-9-1-4-8	7-2-4-8-5-6	9-2-6-5-1-4	Y	N	1
List D	List E	List F			
7-8-2	3-8-2	2-7-1	Y	N	0
9-2-6	5-1-8	4-7-9	Y	N	1
4-1-8-3	2-7-9-3	1-6-8-3	Y	N	0
9-7-2-3	2-1-6-9	3-9-2-4	Y	N	1
1-7-9-2-6	4-1-8-6-9	2-4-7-5-8	Y	N	0
4-1-7-5-2	9-4-1-7-5	8-3-9-6-4	Y	N	1
2-6-4-8-1-7	6-9-7-3-8-2	5-8-6-2-4-9	Y	N	0
8-4-1-9-3-5	4-2-7-9-3-8	3-1-7-8-2-6	Y	N	1
		Digits Score:			of 4

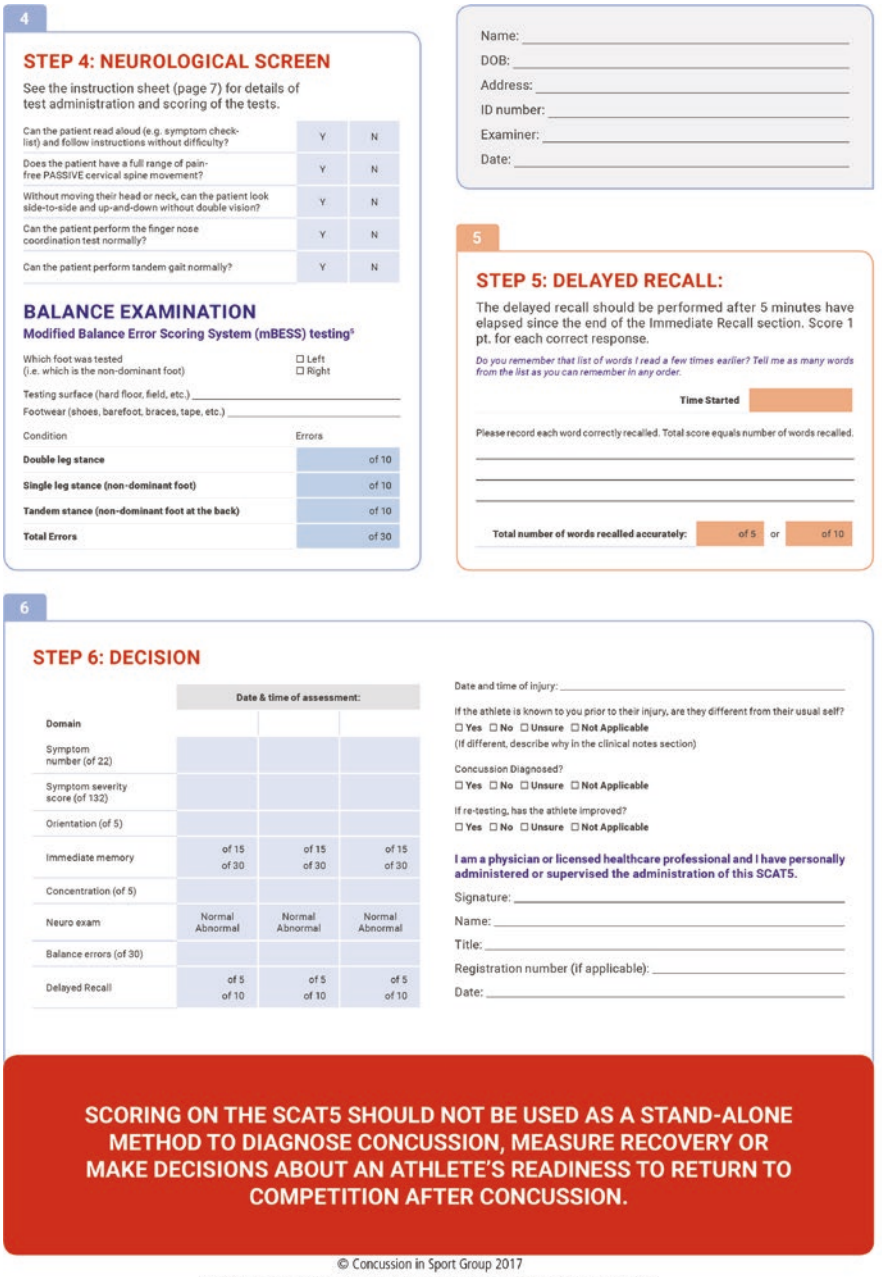
MONTHS IN REVERSE ORDER

Now tell me the months of the year in reverse order. Start with the last month and go backward. So you'll say December, November. Go ahead.

Dec - Nov - Oct - Sept - Aug - Jul - Jun - May - Apr - Mar - Feb - Jan	0 1
Months Score	of 1
Concentration Total Score (Digits + Months)	of 5

Davis GA, *et al. Br J Sports Med* 2017;**0**:1–8. doi:10.1136/bjsports-2017-097506SCAT5

Fig. 10.1 (continued)



4

STEP 4: NEUROLOGICAL SCREEN

See the instruction sheet (page 7) for details of test administration and scoring of the tests.

Can the patient read aloud (e.g. symptom check-list) and follow instructions without difficulty?	Y	N
Does the patient have a full range of pain-free PASSIVE cervical spine movement?	Y	N
Without moving their head or neck, can the patient look side-to-side and up-and-down without double vision?	Y	N
Can the patient perform the finger nose coordination test normally?	Y	N
Can the patient perform tandem gait normally?	Y	N

BALANCE EXAMINATION

Modified Balance Error Scoring System (mBESS) testing[5]

Which foot was tested (i.e. which is the non-dominant foot) ☐ Left ☐ Right

Testing surface (hard floor, field, etc.) _____

Footwear (shoes, barefoot, braces, tape, etc.) _____

Condition	Errors
Double leg stance	of 10
Single leg stance (non-dominant foot)	of 10
Tandem stance (non-dominant foot at the back)	of 10
Total Errors	of 30

Name: _____

DOB: _____

Address: _____

ID number: _____

Examiner: _____

Date: _____

5

STEP 5: DELAYED RECALL:

The delayed recall should be performed after 5 minutes have elapsed since the end of the Immediate Recall section. Score 1 pt. for each correct response.

Do you remember that list of words I read a few times earlier? Tell me as many words from the list as you can remember in any order.

Time Started

Please record each word correctly recalled. Total score equals number of words recalled.

_____

_____

_____

Total number of words recalled accurately: of 5 or of 10

6

STEP 6: DECISION

Domain	Date & time of assessment:		
Symptom number (of 22)			
Symptom severity score (of 132)			
Orientation (of 5)			
Immediate memory	of 15 of 30	of 15 of 30	of 15 of 30
Concentration (of 5)			
Neuro exam	Normal Abnormal	Normal Abnormal	Normal Abnormal
Balance errors (of 30)			
Delayed Recall	of 5 of 10	of 5 of 10	of 5 of 10

Date and time of injury: _____

If the athlete is known to you prior to their injury, are they different from their usual self?
☐ **Yes** ☐ **No** ☐ **Unsure** ☐ **Not Applicable**
(If different, describe why in the clinical notes section)

Concussion Diagnosed?
☐ **Yes** ☐ **No** ☐ **Unsure** ☐ **Not Applicable**

If re-testing, has the athlete improved?
☐ **Yes** ☐ **No** ☐ **Unsure** ☐ **Not Applicable**

I am a physician or licensed healthcare professional and I have personally administered or supervised the administration of this SCAT5.

Signature: _____

Name: _____

Title: _____

Registration number (if applicable): _____

Date: _____

SCORING ON THE SCAT5 SHOULD NOT BE USED AS A STAND-ALONE METHOD TO DIAGNOSE CONCUSSION, MEASURE RECOVERY OR MAKE DECISIONS ABOUT AN ATHLETE'S READINESS TO RETURN TO COMPETITION AFTER CONCUSSION.

Davis GA, *et al. Br J Sports Med* 2017;**0**:1–8. doi:10.1136/bjsports-2017-097506SCAT5

Fig. 10.1 (continued)

CLINICAL NOTES:

Name: _____
DOB: _____
Address: _____
ID number: _____
Examiner: _____
Date: _____

CONCUSSION INJURY ADVICE

(To be given to the person monitoring the concussed athlete)

This patient has received an injury to the head. A careful medical examination has been carried out and no sign of any serious complications has been found. Recovery time is variable across individuals and the patient will need monitoring for a further period by a responsible adult. Your treating physician will provide guidance as to this timeframe.

If you notice any change in behaviour, vomiting, worsening headache, double vision or excessive drowsiness, please telephone your doctor or the nearest hospital emergency department immediately.

Other important points:

Initial rest: Limit physical activity to routine daily activities (avoid exercise, training, sports) and limit activities such as school, work, and screen time to a level that does not worsen symptoms.

1) Avoid alcohol

2) Avoid prescription or non-prescription drugs without medical supervision. Specifically:

   a) Avoid sleeping tablets

   b) Do not use aspirin, anti-inflammatory medication or stronger pain medications such as narcotics

3) Do not drive until cleared by a healthcare professional.

4) Return to play/sport requires clearance by a healthcare professional.

Clinic phone number: _____

Patient's name: _____

Date / time of injury: _____

Date / time of medical review: _____

Healthcare Provider: _____

Contact details or stamp

Davis GA, *et al*. *Br J Sports Med* 2017;**0**:1–8. doi:10.1136/bjsports-2017-0975065CAT5

Fig. 10.1 (continued)

INSTRUCTIONS

Words in *Italics* throughout the SCAT5 are the instructions given to the athlete by the clinician

Symptom Scale

The time frame for symptoms should be based on the type of test being administered. At baseline it is advantageous to assess how an athlete "typically" feels whereas during the acute/post-acute stage it is best to ask how the athlete feels at the time of testing.

The symptom scale should be completed by the athlete, not by the examiner. In situations where the symptom scale is being completed after exercise, it should be done in a resting state, generally by approximating his/her resting heart rate.

For total number of symptoms, maximum possible is 22 except immediately post injury, if sleep item is omitted, which then creates a maximum of 21.

For Symptom severity score, add all scores in table, maximum possible is 22 x 6 = 132, except immediately post injury if sleep item is omitted, which then creates a maximum of 21x6=126.

Immediate Memory

The Immediate Memory component can be completed using the traditional 5-word per trial list or, optionally, using 10-words per trial. The literature suggests that the Immediate Memory has a notable ceiling effect when a 5-word list is used. In settings where this ceiling is prominent, the examiner may wish to make the task more difficult by incorporating two 5–word groups for a total of 10 words per trial. In this case, the maximum score per trial is 10 with a total trial maximum of 30.

Choose one of the word lists (either 5 or 10). Then perform 3 trials of immediate memory using this list.

Complete all 3 trials regardless of score on previous trials.

"I am going to test your memory. I will read you a list of words and when I am done, repeat back as many words as you can remember, in any order." The words must be read at a rate of one word per second.

Trials 2 & 3 MUST be completed regardless of score on trial 1 & 2.

Trials 2 & 3:

"I am going to repeat the same list again. Repeat back as many words as you can remember in any order, even if you said the word before."

Score 1 pt. for each correct response. Total score equals sum across all 3 trials. Do NOT inform the athlete that delayed recall will be tested.

Concentration

Digits backward

Choose one column of digits from lists A, B, C, D, E or F and administer those digits as follows:

Say: *"I am going to read a string of numbers and when I am done, you repeat them back to me in reverse order of how I read them to you. For example, if I say 7-1-9, you would say 9-1-7."*

Begin with first 3 digit string.

If correct, circle "Y" for correct and go to next string length. If incorrect, circle "N" for the first string length and read trial 2 in the same string length. One point possible for each string length. Stop after incorrect on both trials (2 N's) in a string length. The digits should be read at the rate of one per second.

Months in reverse order

"Now tell me the months of the year in reverse order. Start with the last month and go backward. So you'll say December, November ... Go ahead"

1 pt. for entire sequence correct

Delayed Recall

The delayed recall should be performed after 5 minutes have elapsed since the end of the Immediate Recall section.

"Do you remember that list of words I read a few times earlier? Tell me as many words from the list as you can remember in any order."

Score 1 pt. for each correct response

Modified Balance Error Scoring System (mBESS)[5] testing

This balance testing is based on a modified version of the Balance Error Scoring System (BESS)[3]. A timing device is required for this testing.

Each of 20-second trial/stance is scored by counting the number of errors. The examiner will begin counting errors only after the athlete has assumed the proper start position. The modified BESS is calculated by adding one error point for each error during the three 20-second tests. The maximum number of errors for any single condition is 10. If the athlete commits multiple errors simultaneously, only one error is recorded but the athlete should quickly return to the testing position, and counting should resume once the athlete is set. Athletes that are unable to maintain the testing procedure for a minimum of five seconds at the start are assigned the highest possible score, ten, for that testing condition.

OPTION: For further assessment, the same 3 stances can be performed on a surface of medium density foam (e.g., approximately 50cm x 40cm x 6cm).

Balance testing – types of errors

1. Hands lifted off iliac crest
2. Opening eyes
3. Step, stumble, or fall
4. Moving hip into > 30 degrees abduction
5. Lifting forefoot or heel
6. Remaining out of test position > 5 sec

"I am now going to test your balance. Please take your shoes off (if applicable), roll up your pant legs above ankle (if applicable), and remove any ankle taping (if applicable). This test will consist of three twenty second tests with different stances."

(a) Double leg stance:

"The first stance is standing with your feet together with your hands on your hips and with your eyes closed. You should try to maintain stability in that position for 20 seconds. I will be counting the number of times you move out of this position. I will start timing when you are set and have closed your eyes."

(b) Single leg stance:

"If you were to kick a ball, which foot would you use? [This will be the dominant foot] *Now stand on your non-dominant foot. The dominant leg should be held in approximately 30 degrees of hip flexion and 45 degrees of knee flexion. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes and return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."*

(c) Tandem stance:

"Now stand heel-to-toe with your non-dominant foot in back. Your weight should be evenly distributed across both feet. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes and return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."

Tandem Gait

Participants are instructed to stand with their feet together behind a starting line (the test is best done with footwear removed). Then, they walk in a forward direction as quickly and as accurately as possible along a 38mm wide (sports tape), 3 metre line with an alternate foot heel-to-toe gait ensuring that they approximate their heel and toe on each step. Once they cross the end of the 3m line, they turn 180 degrees and return to the starting point using the same gait. Athletes fail the test if they step off the line, have a separation between their heel and toe, or if they touch or grab the examiner or an object.

Finger to Nose

"I am going to test your coordination now. Please sit comfortably on the chair with your eyes open and your arm (either right or left) outstretched (shoulder flexed to 90 degrees and elbow and fingers extended), pointing in front of you. When I give a start signal, I would like you to perform five successive finger to nose repetitions using your index finger to touch the tip of the nose, and then return to the starting position, as quickly and as accurately as possible."

References

1. McCrory et al. Consensus Statement On Concussion In Sport – The 5th International Conference On Concussion In Sport Held In Berlin, October 2016. British Journal of Sports Medicine 2017 (available at www.bjsm.bmj.com)
2. Maddocks, DL; Dicker, GD; Saling, MM. The assessment of orientation following concussion in athletes. Clinical Journal of Sport Medicine 1995; 5: 32-33
3. Jennett, B., Bond, M. Assessment of outcome after severe brain damage: a practical scale. Lancet 1975; i: 480-484
4. McCrea M. Standardized mental status testing of acute concussion. Clinical Journal of Sport Medicine. 2001; 11: 176-181
5. Guskiewicz KM. Assessment of postural stability following sport-related concussion. Current Sports Medicine Reports. 2003; 2: 24-30

Davis GA, *et al. Br J Sports Med* 2017;**0**:1–8. doi:10.1136/bjsports-2017-097506SCAT5

Fig. 10.1 (continued)

CONCUSSION INFORMATION

Any athlete suspected of having a concussion should be removed from play and seek medical evaluation.

Signs to watch for

Problems could arise over the first 24-48 hours. The athlete should not be left alone and must go to a hospital at once if they experience:

- Worsening headache
- Drowsiness or inability to be awakened
- Inability to recognize people or places
- Repeated vomiting
- Unusual behaviour or confusion or irritable
- Seizures (arms and legs jerk uncontrollably)
- Weakness or numbness in arms or legs
- Unsteadiness on their feet.
- Slurred speech

Consult your physician or licensed healthcare professional after a suspected concussion. Remember, it is better to be safe.

Rest & Rehabilitation

After a concussion, the athlete should have physical rest and relative cognitive rest for a few days to allow their symptoms to improve. In most cases, after no more than a few days of rest, the athlete should gradually increase their daily activity level as long as their symptoms do not worsen. Once the athlete is able to complete their usual daily activities without concussion-related symptoms, the second step of the return to play/sport progression can be started. The athlete should not return to play/sport until their concussion-related symptoms have resolved and the athlete has successfully returned to full school/learning activities.

When returning to play/sport, the athlete should follow a stepwise, **medically managed exercise progression, with increasing amounts of exercise.** For example:

Graduated Return to Sport Strategy

Exercise step	Functional exercise at each step	Goal of each step
1. Symptom-limited activity	Daily activities that do not provoke symptoms.	Gradual reintroduction of work/school activities.
2. Light aerobic exercise	Walking or stationary cycling at slow to medium pace. No resistance training.	Increase heart rate.
3. Sport-specific exercise	Running or skating drills. No head impact activities.	Add movement.
4. Non-contact training drills	Harder training drills, e.g., passing drills. May start progressive resistance training.	Exercise, coordination, and increased thinking.
5. Full contact practice	Following medical clearance, participate in normal training activities.	Restore confidence and assess functional skills by coaching staff.
6. Return to play/sport	Normal game play.	

In this example, it would be typical to have 24 hours (or longer) for each step of the progression. If any symptoms worsen while exercising, the athlete should go back to the previous step. Resistance training should be added only in the later stages (Stage 3 or 4 at the earliest).

Written clearance should be provided by a healthcare professional before return to play/sport as directed by local laws and regulations.

Graduated Return to School Strategy

Concussion may affect the ability to learn at school. The athlete may need to miss a few days of school after a concussion. When going back to school, some athletes may need to go back gradually and may need to have some changes made to their schedule so that concussion symptoms do not get worse. If a particular activity makes symptoms worse, then the athlete should stop that activity and rest until symptoms get better. To make sure that the athlete can get back to school without problems, it is important that the healthcare provider, parents, caregivers and teachers talk to each other so that everyone knows what the plan is for the athlete to go back to school.

Note: If mental activity does not cause any symptoms, the athlete may be able to skip step 2 and return to school part-time before doing school activities at home first.

Mental Activity	Activity at each step	Goal of each step
1. Daily activities that do not give the athlete symptoms	Typical activities that the athlete does during the day as long as they do not increase symptoms (e.g. reading, texting, screen time). Start with 5-15 minutes at a time and gradually build up.	Gradual return to typical activities.
2. School activities	Homework, reading or other cognitive activities outside of the classroom.	Increase tolerance to cognitive work.
3. Return to school part-time	Gradual introduction of school-work. May need to start with a partial school day or with increased breaks during the day.	Increase academic activities.
4. Return to school full-time	Gradually progress school activities until a full day can be tolerated.	Return to full academic activities and catch up on missed work.

If the athlete continues to have symptoms with mental activity, some other accomodations that can help with return to school may include:

- Starting school later, only going for half days, or going only to certain classes
- More time to finish assignments/tests
- Quiet room to finish assignments/tests
- Not going to noisy areas like the cafeteria, assembly halls, sporting events, music class, shop class, etc.
- Taking lots of breaks during class, homework, tests
- No more than one exam/day
- Shorter assignments
- Repetition/memory cues
- Use of a student helper/tutor
- Reassurance from teachers that the child will be supported while getting better

The athlete should not go back to sports until they are back to school/learning, without symptoms getting significantly worse and no longer needing any changes to their schedule.

Davis GA, *et al. Br J Sports Med* 2017;**0**:1–8. doi:10.1136/bjsports-2017-097506SCAT5

Fig. 10.1 (continued)

removed from play and be "in a resting state" before testing is performed [1]. This usually requires at least ten minutes or more, which gives time for the athlete's heart rate to return to the resting state. Therefore, it is important to recognize that the SCAT5 cannot be performed correctly if less than 10 minutes have passed since time of suspected concussion. A preseason baseline evaluation can provide tremendous benefit. Possession of a baseline "scorecard" for reference facilitates serial tracking of a concussion over time, should injury occur during competitive regular season events. This may be helpful given reports regarding the variability in baseline testing, as well as test reliability for earlier SCAT versions. One study reported baseline values for 260 collegiate athletes utilizing the original SCAT [80]. This study demonstrated gender differences in baseline values. A history of previous concussion in athletes also accounted for differences in baseline scores. Another study investigating representative baseline values on the SCAT2 reported differences in scores associated with age, gender, and self-reported concussion. This further highlights the benefit of baseline testing of athletes before initiation of competitive events during the regular season [81]. It should be noted that scores for the SCAT5 are not "pass/fail" scores, but rather, the tool is used by the healthcare professional to assist in the diagnosis of concussion, to determine overall injury severity, and management of concussion.

For children aged 5–12 years, the Child SCAT5 was developed, and contains modifications from the SCAT5 that are more appropriate to children [82]. Some of the differences between the Child SCAT5 and the SCAT5 include a child-specific symptoms list, which includes both child and parent reported symptoms, simpler concentration questions for children, removal of orientation questions for children, and simplified balance testing for younger children. A large body of evidence supports balance testing (which includes balance assessed with tandem gait.), particularly in the first few days, as a sensitive component of the diagnosis of concussion [83–85]. This influenced the inclusion of the Balance Error Scoring System (BESS) that was included in the SCAT5.

The examiner should follow this with a more detailed evaluation of cervical range of motion (ROM). Range of motion testing should assess flexion, extension, and rotation in all directions, both passively and actively [86]. This should be followed with formal assessment of motor strength testing in all muscle groups. The athlete should be withheld from participation (even in the absence of deficits on previous portions of the concussion assessment) for further evaluation, because limitations in these areas may place the athlete at risk for further injury by restricting the athlete's ability to protect the head and anticipate impacts from oncoming opponents [87]. For athletes who have performed at or above baseline for all other aspects of the assessment, functional testing marks the final step in the concussion evaluation.

The goal of functional testing is to elicit symptoms that may be exhibited with the physical and cognitive demands the athlete may experience upon return to play [87]. Simple tasks such as push-ups, and sit-ups, and Valsalva maneuver should be performed first [87].

Next, physical activity may be advanced by having the athlete jog. This may be followed by more strenuous aerobic activity such as interval sprinting exercises. Successful completion of the progression culminates with sport-specific activities (e.g., dribbling/shooting a basketball, passing drills with a soccer ball, throwing/catching a football, shooting a hockey puck, etc.) performed at a similar intensity level necessary for safe return to play [86]. With each step, the practitioner should ask the athlete whether any concussion-related symptoms have been elicited before moving on to an increase in activity level. If at any time, an athlete reports symptoms that result from the exertion, the player should not be permitted to return to play (or progressed to the next step). If no symptoms are elicited through these functional tests and all other assessments demonstrate normal findings, the athlete has not likely sustained a concussion and may be considered for return to play.

Although standardized sideline assessment tools may be useful in the evaluation of concussion, several points must be kept in mind. These tools are designed for rapid screening of concussion by the wider spectrum of practitioner types and should not be used to replace comprehensive neuropsychologic testing by a trained neuropsychologist [1]. Furthermore, no sideline assessment tool should be used for the ongoing management of sports-related concussions [1]. A standardized clinical evaluation of concussion is useful, but should not substitute for clinician's judgment [87].

Adjuncts to the Clinical Assessment for Concussion

Neuropsychologic Testing

Neuropsychologists have contributed greatly to our knowledge of the effects of concussion on cognition and emotional functioning. The application of neuropsychologic testing has been long recognized in the evaluation and treatment of individuals with concussive injuries [88] Well-known sequelae of concussion include deficits in attention, processing speed, reaction time, and learning [89–91]. If a neuropsychological assessment is deemed necessary by the treating physician, it should be performed by a trained neuropsychologist [1]. Neuropsychological assessment can be especially helpful in the cognitive assessment of athletes when distinction between true cognitive impairment secondary to a concussion versus other factors are difficult to determine.

Multiple studies have demonstrated the usefulness of neuropsychologic assessment with concussed patients and in return-to-play (RTP) protocols [1, 92–98]. Neuropsychologists employ computerized measures to examine cognitive functions such as attention, memory, language, visual abilities, and executive functioning as well as affective functioning. Computerized tests offer the advantage of decreased administration time, availability of multiple alternative forms for serial testing, and more precise measurements [99, 100]. Perhaps the most widely-used computerized

test is the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) program [101]. This is a 20-minute assessment that contains a symptom checklist, and measures visual and verbal memory, reaction time, and information processing time [102, 103]. Validity measures with computerized tests such as the ImPACT program reduce the risk of intentionally poor performance reporting on baseline testing without reaching threshold on the test validity indicators, which alert the administrator to an invalid test [104]. These validity measures may be used with or without baseline data, in which case normative data may be applied.

Various studies have demonstrated the utility of the ImPACT program in concussion [95, 103, 105–111]. Although the use of computerized instruments in concussion has become widely accepted, there are still legitimate concerns regarding this assessment method. Among some of the concerns raised are: privacy and data security, test reliability and validity, cultural, experiential, and disability effects, and checks on effort validity [112].

One study reported low reliability scores with ImPACT and other tests [113]. This study demonstrated that, even with alternative forms, practice effects can be seen on ImPACT, particularly with processing speed scores. Broglio and colleagues reported lack of test-retest reliability of three computerized tests (ImPACT, CogSport, and Headminders) in an often-cited study as argument against the validity of computerized testing [114].

Advanced Neuroimaging

Conventional neuroimaging findings seen in computed tomography (CT) and magnetic resonance imaging (MRI) are typically normal in concussion. CT and MRI of the brain are utilized when there is concern for intracerebral hemorrhage or a structural lesion is suspected in the setting of focal neurological deficit, prolonged loss of consciousness, or progressive worsening of symptoms. Several advanced neuroimaging techniques, such as diffusion tensor imaging (DTI), functional MRI (fMRI), magnetic resonance spectroscopy (MRS), and high-definition fiber tracking (HDFT), and positron emission tomography (PET), might possibly increase the sensitivity of neuroimaging to detect both structural and functional abnormalities associated with concussion—both in the acute setting and subsequently in the subacute and chronic phases of recovery.

Several studies have used advanced MRI techniques to assess for acute concussion. These studies demonstrate acute changes in brain function and structure following concussion. These acute changes were shown to gradually revert back to a normal pre-concussive state, which is comparable to non-concussed control patients. These acute MRI findings include changes in white matter integrity on diffusion imaging, changes in cerebral blood flow on arterial spin labeling (ASL), altered functional connectivity on resting state functional MRI (rs-fMRI), and appearance of microhemmorhage on perfusion imaging.

Several studies have evaluated and discussed fMRI findings in sports-related concussion [84, 115–123]. One study examined performance of memory and

sensory tasks in a group of college football players, some of whom had suffered concussion. Brain activation was found to be more widespread following concussion than before injury (baseline fMRI used for comparison). This cohort was compared to un-injured control subjects [124]. The mid-dorsolateral prefrontal cortex (DLPC) is an area associated with working memory. Less task-related activation in the DLPC was observed in subjects with persistent post-concussion symptoms 1–14 months following concussion. These findings were present in the absence of abnormalities seen on structural MRI. Of particular interest was one subject who demonstrated resolution of symptoms with associated normalization of the observed widespread activation pattern. Some studies have also demonstrated a correlation between fMRI abnormalities and cognitive test results. Athletes with post-injury fMRI findings that demonstrated hyperactivation experienced a longer clinical recovery when compared to those without hyperactivation [125].

PET measures certain components of cerebral metabolism, including blood flow and the cerebral metabolic oxygen consumption. Some limitations of PET include expense, exposure to radiolabeled tracers, and the time required to complete the study. Despite these limitations, studies have demonstrated interesting findings when PET is performed after mTBI. Umile et al. observed adults after mTBI who exhibited persistent post-concussion symptoms. This study found the subjects to have significantly reduced metabolism in the medial temporal lobe [126]. Chen et al. demonstrated differences in cerebral metabolism during a visual-spatial working memory task in adult subjects with persistent symptoms following mTBI [127].

MRS measures concentrations of compounds in the brain within a sampled region. It is a useful tool that is commonly used in the evaluation of brain lesions [119]. TBI is associated with specific metabolites, which may include choline, a marker of membrane damage and turnover; lactate, an indirect marker for ischemia and hypoxia; *N*-acetyl aspartate (NAA), a marker of neuronal integrity; creatine (Cr), a cellular energy marker for adenosine triphosphate (ATP) resynthesis; and myoinositol, a glial marker [117, 128].

In a study of 14 patients with sports-related concussion, reduced NAA:Cr ratios were observed 3 days after concussion, with normalization of the NAA:Cr ratio 30 days post injury [129]. This reduced ratio persisted despite resolution of symptoms which occurred, on average, after 3 days. This suggests that metabolic normalization may be different than symptom recovery. Reduction in NAA and other metabolic derangements in post-concussion patients is supported by various studies comparing athletes with and without concussion [130, 131].

HDFT and DTI, both techniques that are capable of providing information regarding fiber tract integrity and white matter microstructure, have been increasingly utilized following mTBI and concussion. Many DTI studies have shown correlation between mTBI and widespread structural changes in cortical white matter tracts [132–146].

These studies report abnormalities in various regions of the brain. These areas include the corona radiate, corpus callosum, uncinate fasciculus, inferior and superior longitudinal fasciculus, cingulum bundle, inferior fronto-occipital fasciculus, internal capsule, as well as the acoustic and posterior thalamic radiations [135]. Studies have demonstrated a correlation between DTI and injury severity with

symptoms [145] as well as functional deficits measured by neuropsychologic testing and other behavioral measures [138, 141, 147, 148].

HDFT provides high-resolution details of axonal pathways and projection fields that allow detection of the specific location and degree of damage [149, 150]. HDFT was used in one study to identify a specific lesion of the corona radiata corticospinal pathway that was associated with left-sided weakness in a patient 4 months after injury [149].

HDFT may overcome some of the shortcomings of DTI, but routine use of either modality for concussion is premature [149]. The results of initial studies using HDFT are preliminary and must be validated in larger studies, but emerging data is encouraging.

Cerebrospinal Fluid and Serum-Based Biomarkers of Injury

There has been a drastic increase in research in both cerebrospinal fluid (CSF) and serum biomarkers of neuronal, axonal, and astroglial injury with a goal of improving diagnosis and helping predict the clinical course after concussion and mTBI. Despite these efforts, there remains a lack of sufficient evidence to justify their routine clinical use [1, 151].

Recent studies have demonstrated changes in common TBI biomarkers after SRC, suggesting evidence of neuronal cell body damage, axonal injury, and neuroinflammatory response to injury. Serum may seem to be an attractive biofluid for analysis given its lower cost and lower associated risk of acquisition, but extensive studies with sampling of both biofluids suggest otherwise. Trauma to areas outside of the brain, as well as non-trauma-related athletic exertion, is associated with increased levels of certain biomarkers that may give a false representation of levels in the brain. For example, S100 calcium-binding protein (S100B) and glial fibrillary acidic protein (GFAP), both markers of astroglial injury, have been shown to be elevated in the serum of mTBI patients [152–154].

Other biomarkers, such as myelin basic protein (MBP), tau protein, and neuron-specific enolase (NSE) are being actively studied. Immunoassay technique sensitivity, validity, clinical assays for quantification, and the amount of longitudinal data in concussion need to improve before these biomarkers can be routinely used [151].

Concussion Management

The mainstays in concussion management have previously been both physical and cognitive rest. The Berlin consensus statement states [1]:

> There is currently insufficient evidence that prescribing complete rest achieves these objectives. After a brief period of rest during the acute phase (24–48 hours) after injury, patients can be encouraged to become gradually and progressively more active while staying below

their cognitive and physical symptom-exacerbation thresholds (ie, activity level should not bring on or worsen their symptoms). It is reasonable for athletes to avoid vigorous exertion while they are recovering. The exact amount and duration of rest is not yet well defined in the literature and requires further study.

Fortunately, most symptoms resolve relatively shortly over the course of several days following injury, thus allowing the patient to gradually return to social and academic activities.

A stepwise program is generally accepted with regard to return to athletic play [1]. This program begins with a period of no activity. This is followed by light aerobic exercise, followed by sport-specific exercise, noncontact training drills, full-contact practice, and finally return to play. Each of these levels is performed over a 24-hour period. The program may be initiated when the athlete is asymptomatic at rest. If the athlete exhibits any post-concussion symptoms at any of these steps, another 24-hour period of rest is completed, and the athlete reverts back to the previous step in the program. Same-day RTP should not be permitted on the day of injury. This is due to evidence that RTP on the day of injury may be associated with prolonged neuropsychological deficits with delayed onset.

It is uncommon for post-concussion symptoms to persist beyond 10 days, but it is possible in a subset of patients. This may prompt clinicians to consider pharmacologic treatments for management of symptoms. If this option is to be employed, the patient and clinician should come to a mutual agreement that that benefit of treatment outweighs any possible adverse effect of a medication under consideration [155, 156]. There are no clinically validated treatments that have demonstrated benefit with regards to expedited recovery or amelioration of deficits attributed to TBI [157]. However, athletes with post-concussion syndrome or prolonged post-concussion syndrome may experience some benefit from symptomatic medical treatment during the recovery phase [157–160].

Several factors must be considered by a physician considering use of medication for the treatment of post-concussion symptoms. Medications that lower seizure threshold, or those that contribute to fatigue, cognitive slowing, or daytime drowsiness should be avoided. Medical therapies should be initiated at the lowest effective dose, with a slow dose titration according to patient tolerability, clinical response, and side effects. In order to prevent adverse interactions, special care should be taken to review all medications and over-the counter supplements the patient is currently using [155, 156, 161].

The treatment of concussion should be specific to patient symptoms, which are grouped into the following four categories: cognitive, emotional, somatic complaints, and sleep disturbance [155, 160]. However, it should be noted that alleviating one symptom may improve upon others. A concise review of selected agents studied in mTBI or concussion for the management of various symptoms is provided below.

The most common symptom reported after concussion is post-traumatic headache, which occurs in more than 90% of patients [162–164]. Patients with persistent post-concussion headaches most commonly develop migraine-like or tension-type headaches [165–167]. Aspirin and other nonsteroidal anti-inflammatory drugs

(NSAIDs) are typically avoided in the acute period following concussion. Therefore, acetaminophen is a reasonable choice for post-concussion headache treatment in the acute period. A majority of patients experience spontaneous resolution of the headache; patients with persistent headaches may require further treatment. Amitriptyline (an antidepressant) has shown efficacy in the treatment of post-concussion headaches [161, 168, 169]. Data is currently limited regarding other treatments, such as triptans, dihydroergotamine (DHE), anticonvulsants (valproic acid, gabapentin, and topiramate), calcium channel blockers, and beta clockers [155, 166, 167, 170].

Neurostimulants are often used for moderate or severe TBI with associated fatigue, but data on their usage in mTBI is sparse. Methylphenidate (a norepinephrine–dopamine reuptake inhibitor), modafinil (an atypical, selective, and weak dopamine reuptake inhibitor), and amantadine (a weak antagonist of the NMDA-type glutamate receptor) are pharmacologic agents that have been studied in randomized trials for use in mTBI, and shown variable success [155, 171, 172].

Acutely following a concussion, difficulty falling asleep is a common source of significant morbidity. This is especially true for student athletes [155, 164]. Trazodone is an antidepressant which antagonizes serotonin 2A (5-HT_{2A}) receptors and inhibits serotonin reuptake, and is commonly used to treat sleep disturbance in post-concussion patients [155, 173]. Prazosin, zolpidem, and melatonin are other agents that have shown varied results for treatment of mTBI and concussion [173–181].

Antidepressants have been generally used for most emotional symptoms following concussion [155, 156]. Most evidence for use of selective serotonin reuptake inhibitors (SSRIs) in concussion comes from small, uncontrolled studies and case reports, thus efficacy is not well validated at this time. Despite this, SSRIs have become the primary treatment for TBI-associated depression due to their perceived clinical efficacy and relatively few side effects. A subset of patients may continue to experience cognitive deficits, as evidenced by neuropsychologic testing for a prolonged period following mTBI or concussion. Amantadine acts through several pharmacologic mechanisms and is a weak antagonist of the NMDA-type glutamate receptor, increases dopamine and norepinephrine release from free nerve endings, and blocks dopamine reuptake; it has been used to improve post-concussion neurocognitive recovery with varied success [182–185].

Recommendations for Concussion Management

The AAN makes several recommendations for health care providers regarding management of suspected concussion [50]. All athletes with suspected concussion should be removed from play immediately to avoid further injury. This should be followed by an evaluation by a provider experienced in diagnosis of concussion, and more severe TBI. Any athlete with concussion should be restricted from return to contact sports play until he or she is asymptomatic without medication use, and the concussion is resolved.

Licensed providers may use neurocognitive testing or other tools to determine concussion resolution.

The development of graded plans for return to cognitive and physical activity, in a monitored clinical setting, may help providers minimize exacerbation of early post-concussion impairments. Cognitive restructuring is a form of psychologic counseling focused on reassurance, education, and reattribution of symptoms that may help decrease the likelihood of the development of chronic post-concussion syndrome.

Post-concussion Syndrome

Symptoms associated with concussion typically resolve several days following injury. Symptoms that persist beyond 10 days occur in up to 10–15% of patients [1].

Post-concussion complications include acute symptoms, post-concussion syndrome (PCS), persistent or prolonged PCS (PPCS), mild cognitive impairment, chronic traumatic encephalopathy (CTE), and dementia pugilistica. The WHO's *International Statistical Classification of Diseases and Related Health Problems,* tenth revision (ICD-10) defines diagnosis of PCS requiring the presence of three or more of the following symptoms: headache, dizziness, fatigue, irritability, insomnia, concentration difficulty, and memory difficulty. There is no universally agreed upon time frame for symptom duration that constitutes the aforementioned syndromes; however, the persistence of symptoms for between 6 weeks and 3 months is generally consistent with PCS, and the presence of any symptom for longer than 3 months is consistent with PPCS [156–186].

There is also some controversy regarding the pathophysiology of these phenomena, with some authorities suggesting that the symptoms associated with PCS are a direct consequence of brain injury, while others propose that the symptoms are functional and represent the emotional or psychologic sequelae of the brain injury [187–189].

It is important to note that symptoms associated with concussion may also be associated with various other conditions. Differentiating among etiologies can prove to be difficult. For example, comparison of the diagnostic criteria for post-traumatic stress disorder (PTSD), major depressive disorder, acute stress disorder, anxiety disorders, and PCS shows considerable overlap [190]. The differential diagnosis also can include vestibular dysfunction, visual dysfunction, cervical injury, somatization, chronic fatigue and pain, or a combination of these in some form [191, 192]. When clinical recovery extends outside the expected window, management by a multidisciplinary team with experience in concussion management should be undertaken [1].

It is unclear which patients will continue to experience PCS spectrum symptoms far beyond their injury, but certain variables may increase the risk. These clinical variables may include prior concussions, female sex, a history of cognitive dysfunction, and the presence of affective disorders, such as depression and anxiety

[192–198]. The average time to normalization to pre-concussion baseline on neurocognitive testing has been reported as 10–14 days in high school students, 5–7 days in collegiate athletes, and 2–5 days in professional athletes [199–201].

Children may experience more severe, prolonged, and more widespread cerebral edema after brain injury [202–204]. The reasons for this are unclear, but may be due to differences in glutamate receptor expression and/or sensitivity, or weakened neck musculature that may result in greater rotational acceleration on impact when compared to that seen in adults [205–208].

There is no widely accepted explanation for the higher risk of PCS and PPCS in patients with underlying cognitive dysfunction. Patients with lower IQ scores have been found to be more likely to experience persistent post-concussion symptoms and to be diagnosed with PCS after brain injury [197, 209]. Several studies suggest that specific premorbid emotional and personality characteristics potentially put some people at increased risk for poor outcome following concussion [198, 210]. Post-concussion symptoms may be mediated by an interaction of psychologic and neurological factors after TBI. Psychiatric disease preceding or following brain injury, including PTSD, anxiety, depression, acute stress disorder, has been associated with increased risk of poor outcome following concussion [197, 209, 211].

Repetitive Mild Traumatic Brain Injury: Cumulative Effects

The short- and long-term sequela of repetitive concussion has gained increased focus in the media and medical community over the last several years. The biggest short-term fear regarding repetitive head injury is that of "second impact syndrome." This is defined as the quick deterioration and death of an athlete who experiences a second mild head injury following return to play subsequent to the first injury. Most case reports pertaining to this syndrome are not associated with space occupying lesions. Death was associated with severe and rapid cerebral edema [212]. This condition has been reported primarily in adolescent and young adult athletes. Proponents of this syndrome believe that following initial mild TBI in younger patients, there exists an increased underlying risk for development of autoregulation and catecholamine release that may result in diffuse cerebral edema and possibly death after even a mild secondary impact [213–215]. The pathophysiology leading to the severe edema and small hematomas that are often encountered is not fully understood. Furthermore, it remains unclear whether the edema or hematomas are the result of one of the individual blows to the head rather than the proposed pathophysiologic mechanisms leading to severe disability or death [215–217].

Suffering multiple concussions has been associated with increased risk for future concussion, cognitive deficit, delayed mild cognitive deficit, and sleep disruption. The association with CTE and suffering multiple concussions has gained increased attention in the media, and the mainstream public [218, 219]. First defined by Omalu et al. in 2005, CTE is defined as a progressive neurodegenerative syndrome caused

by repetitive and episodic blunt force impact to the head that cause acceleration-deceleration forces to the brain [220]. A systematic review by Manley et al. examined potential long-term sequelae of concussion by examining cognitive, mental health, neuroimaging, and neuropathological features of CTE. This review demonstrated that despite reports with a small number of retired athletes who exhibited some mental health and cognitive problems, the majority of studies demonstrated these changes to be similar to the incidence in the general population [221]. These authors concluded that while multiple concussions appear to be a risk factor for mental health problems and cognitive impairment in some individuals, more research is needed to better understand the prevalence of CTE and other neurological conditions, and the degree to which they are related to concussions and sports-related repetitive neurotrauma.

A systematic review examining potential long term sequelae of concussion studied the neuroimaging, cognitive, mental health, and neuropathological features of CTE, and identified that whilst a very small number of retired athletes have some mental health and cognitive problems, the majority of studies demonstrate these changes to be similar to the incidence in the general population and the Berlin consensus paper stated that: [1, 221]

> However, there is much more to learn about the potential cause-and-effect relationships of repetitive head-impact exposure and concussions. The potential for developing chronic traumatic encephalopathy (CTE) must be a consideration, as this condition appears to represent a distinct tauopathy with an unknown incidence in athletic populations.
>
> A cause-and-effect relationship has not yet been demonstrated between CTE and SRCs or exposure to contact sports. As such, the notion that repeated concussion or sub-concussive impacts cause CTE remains unknown.

There is significant debate regarding both the clinical and pathological diagnostic criteria for CTE. There is also debate over the theory around a causal and exclusive link between exposure to repetitive head impact and risk for CTE. While case studies have received a great deal of attention, several studies have failed to find a link [222]. Tauopathy changes in the brain that meet the pathological criteria of CTE have been identified in multiple cases with a notable absence of history of head trauma [223, 224]. Therefore, the causal association between the pathological findings of CTE and the clinical condition requires continuing study.

The clinical features of CTE involve a prolonged latency period prior to a composite syndrome of neuropsychiatric disturbance, mood disorder, and cognitive impairment [225, 226]. Insidious onset of poor attention, loss of recent memory, frequent headaches, and bouts of confusion and disorientation are common early in the syndrome [226]. There is often a progression of effects referable to poor prefrontal cortex executive function and limbic system dysfunction, including worsening disorientation and confusion, poor insight and judgment, and the onset of antisocial behaviors [226]. The pathophysiologic mechanisms responsible for the clinical sequelae associated with repetitive brain injury are not entirely understood, but microvascular changes, blood-brain barrier (BBB) breakdown, diffuse axonal injury (DAI), and immunoexcitotoxicity have been implicated [227]. Currently, the

diagnosis of CTE relies on autopsy and subsequent neuropathologic examination, with the defining feature of CTE being that of tauopathy, which may be seen in the form of neuropil threads (NT), glial tangles (GTs), or neurofibrillary tangles (NFTs), in an irregular, superficial cortical distribution [226, 228]. The development of tau-binding radiolabeled ligands will likely enhance the ability to diagnose CTE in living patients [218, 229]. Limited cases have demonstrated the ability to identify an increased tau burden within the brains of retired professional football players subjected to repetitive neurotrauma [229]. This work represents the first effort to clearly show the potential of premortem CTE diagnosis [219, 229].

Conclusion

The definition of concussion continues to evolve. Concussion epidemiology, signs and symptoms, and on-field assessment are important for the sports neurosurgeon. Concussion clinical assessment can be supported by neuropsychological testing, advanced neuroimaging, and cerebrospinal fluid and serum-based biomarkers. The American Academy of Neurology Guideline Recommendations for Concussion Management should be heeded by the sports neurosurgeon.

References

1. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016. Br J Sports Med. 2017;51:838–47.
2. Kristman VL, Borg J, Godbolt AK, et al. Methodological issues and research recommendations for prognosis after mild traumatic brain injury: results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. Arch Phys Med Rehabil. 2014;95(suppl):S265–77.
3. Carroll LJ, Cassidy JD, Holm L, et al. Injury WHOCCTFoMTB. Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. J Rehabil Med. 2004;36(suppl 43):113–25.
4. Tator CH. Let's standardize the definition of concussion and get reliable incidence data. Can J Neurol Sci. 2009;36(4):405–6.
5. Lopez AD, Murray CC. The global burden of disease, 1990–2020. Nat Med. 1998;4(11):1241–3.
6. Thurman DJ, Alverson C, Dunn KA, et al. Traumatic brain injury in the United States: a public health perspective. J Head Trauma Rehabil. 1999;14(6):602–15.
7. Bryan CJ, Clemans TA. Repetitive traumatic brain injury, psychological symptoms, and suicide risk in a clinical sample of deployed military personnel. JAMA Psychiat. 2013;70(7):686–91.
8. Erickson JC. Treatment outcomes of chronic post-traumatic headaches after mild head trauma in US soldiers: an observational study. Headache. 2011;51(6):932–44.
9. Hoge CW, McGurk D, Thomas JL, et al. Mild traumatic brain injury in U.S. Soldiers returning from Iraq. N Engl J Med. 2008;358(5):453–63.

10. Okie S. Traumatic brain injury in the war zone. N Engl J Med. 2005;352(20):2043–7.
11. Schwab KA, Ivins B, Cramer G, et al. Screening for traumatic brain injury in troops returning from deployment in Afghanistan and Iraq: initial investigation of the usefulness of a short screening tool for traumatic brain injury. J Head Trauma Rehabil. 2007;22(6):377–89.
12. Terrio H, Brenner LA, Ivins BJ, et al. Traumatic brain injury screening: preliminary findings in a US Army Brigade Combat Team. J Head Trauma Rehabil. 2009;24(1):14–23.
13. Felber ES. Combat-related posttraumatic headache: diagnosis, mechanisms of injury, and challenges to treatment. J Am Osteopath Assoc. 2010;110(12):737–8.
14. Marin JR, Weaver MD, Yealy DM, et al. Trends in visits for traumatic brain injury to emergency departments in the United States. JAMA. 2014;311(18):1917–9.
15. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. J Head Trauma Rehabil. 2006;21(5):375–8.
16. Clay MB, Glover KL, Lowe DT. Epidemiology of concussion in sport: a literature review. J Chiropr Med. 2013;12(4):230–51.
17. Harmon KG, Drezner JA, Gammons M Endorsed by the National Trainers' Athletic Association and the American College of Sports Medicine, et al. American Medical Society for Sports Medicine position statement: concussion in sport. Br J Sports Med. 2013;47:15–26.
18. Kerr ZY, Roos KG, Djoko A, et al. Epidemiologic measures for quantifying the incidence of concussion in national collegiate athletic association sports. J Athl Train. 2017;52:167–74. https://doi.org/10.4085/1062-6050-51.6.05.
19. Tsushima WT, Siu AM, Ahn HJ, et al. Incidence and risk of concussions in youth athletes: comparisons of age, sex, concussion history, sport, and football position. Arch Clin Neuropsychol. 2018;126 https://doi.org/10.1093/arclin/acy019.
20. Bretzin AC, Covassin T, Fox ME, et al. Sex differences in the clinical incidence of concussions, missed school days, and time loss in high school student-athletes: part 1. Am J Sports Med. 2018;46:2263–9. https://doi.org/10.1177/0363546518778251.
21. Barr WB, McCrea M. Sensitivity and specificity of standardized neurocognitive testing immediately following sports concussion. J Int Neuropsychol Soc. 2001;7:693–702. https://doi.org/10.1017/S1355617701766052.
22. Barr WB, Prichep LS, Chabot R, et al. Measuring brain electrical activity to track recovery from sport-related concussion. Brain Inj. 2012;26:58–66. https://doi.org/10.3109/02699052.2011.608216.
23. McCrea M, Kelly JP, Randolph C, et al. Immediate neurocognitive effects of concussion. Neurosurgery. 2002;50:1032–40.
24. McCrea M. Standardized mental status assessment of sports concussion. Clin J Sport Med. 2001;11:176–81. https://doi.org/10.1097/00042752-200107000-00008.
25. McCrea M, Barr WB, Guskiewicz K, et al. Standard regression-based methods for measuring recovery after sport-related concussion. J Int Neuropsychol Soc. 2005;11:58–69. https://doi.org/10.1017/S1355617705050083.
26. Marinides Z, Galetta KM, Andrews CN, et al. Vision testing is additive to the sideline assessment of sports-related concussion. Neurol Clin Pract. 2015;5:25–34. https://doi.org/10.1212/CPJ.0000000000000060.
27. Galetta KM, Morganroth J, Moehringer N, et al. Adding vision to concussion testing: a prospective study of sideline testing in youth and collegiate athletes. J Neuroophthalmol. 2015;35:235–41. https://doi.org/10.1097/WNO.0000000000000226.
28. Putukian M, Echemendia R, Dettwiler-Danspeckgruber A, et al. Prospective clinical assessment using Sideline Concussion Assessment Tool-2 testing in the evaluation of sport-related concussion in college athletes. Clin J Sport Med. 2015;25:36–42. https://doi.org/10.1097/JSM.0000000000000102.
29. Chin EY, Nelson LD, Barr WB, et al. Reliability and validity of the sport concussion assessment tool-3 (SCAT3) in high school and collegiate athletes. Am J Sports Med. 2016;44:2276–85. https://doi.org/10.1177/0363546516648141.

30. Seidman DH, Burlingame J, Yousif LR, et al. Evaluation of the King-devick test as a concussion screening tool in high school football players. J Neurol Sci. 2015;356:97–101. https://doi.org/10.1016/j.jns.2015.06.021.
31. Dompier TP, Kerr ZY, Marshall SW, et al. Incidence of concussion during practice and games in youth, high school, and collegiate american football players. JAMA Pediatr. 2015;169:659–65. https://doi.org/10.1001/jamapediatrics.2015.0210.
32. Houck Z, Asken B, Bauer R, et al. Epidemiology of sport-related concussion in an ncaa division i football bowl subdivision sample. Am J Sports Med. 2016;44:2269–75. https://doi.org/10.1177/0363546516645070.
33. Galetta KM, Brandes LE, Maki K, et al. The King-Devick test and sports-related concussion: study of a rapid visual screening tool in a collegiate cohort. J Neurol Sci. 2011;309(1–2):34–9. https://doi.org/10.1016/j.jns.2011.07.039.
34. Leong DF, Balcer LJ, Galetta SL, et al. The King-Devick test for sideline concussion screening in collegiate football. J Optom. 2015;8:131–9. https://doi.org/10.1016/j.optom.2014.12.005.
35. Dhawan PS, Leong D, Tapsell L, et al. King-devick test identifies real-time concussion and asymptomatic concussion in youth athletes. Neurol Clin Pract. 2017;7:1–73. https://doi.org/10.1212/CPJ.0000000000000381.
36. Shankar PR, Fields SK, Collins CL, et al. Epidemiology of high school and collegiate football injuries in the United States, 2005–2006. Am J Sports Med. 2007;35(8):1295–303.
37. Guskiewicz KM, Weaver NL, Padua DA, et al. Epidemiology of concussion in collegiate and high school football players. Am J Sports Med. 2000;28(5):643–50.
38. Yard EE, Comstock RD. Effects of field location, time in competition, and phase of play on injury severity in high school football. Res Sports Med. 2009;17(1):35–49.
39. Guskiewicz KM, McCrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study. JAMA. 2003;290(19):2549–55.
40. Pellman EJ, Powell JW, Viano DC, et al. Concussion in professional football: epidemiological features of game injuries and review of the literature—part 3. Neurosurgery. 2004;54(1):81–94; discussion 94–86.
41. Rechel JA, Yard EE, Comstock RD. An epidemiologic comparison of high school sports injuries sustained in practice and competition. J Athl Train. 2008;43(2):197–204.
42. Wisniewski JF, Guskiewicz K, Trope M, et al. Incidence of cerebral concussions associated with type of mouthguard used in college football. Dent Traumatol. 2004;20(3):143–9.
43. Meehan WP 3rd, d'Hemecourt P, Comstock RD. High school concussions in the 2008–2009 academic year: mechanism, symptoms, and management. Am J Sports Med. 2010;38(12):2405–9.
44. Crisco JJ, Fiore R, Beckwith JG, et al. Frequency and location of head impact exposures in individual collegiate football players. J Athl Train. 2010;45(6):549–59.
45. Gessel LM, Fields SK, Collins CL, et al. Concussions among United States high school and collegiate athletes. J Athl Train. 2007;42(4):495–503.
46. Kimbler DE, Murphy M, Dhandapani KM. Concussion and the adolescent athlete. J Neurosci Nurs. 2011;43(6):286–90.
47. Agel J, Harvey EJ. A 7-year review of men's and women's ice hockey injuries in the NCAA. Can J Surg. 2010;53(5):319–23.
48. Brainard LL, Beckwith JG, Chu JJ, et al. Gender differences in head impacts sustained by collegiate ice hockey players. Med Sci Sports Exerc. 2012;44(2):297–304.
49. Niedfeldt MW. Head injuries, heading, and the use of headgear in soccer. Curr Sports Med Rep. 2011;10(6):324–9.
50. Giza CC, Kutcher JS, Ashwal S, et al. Summary of evidence-based guideline update: evaluation and management of concussion in sports: report of the Guideline Development Subcommittee of the American Academy of Neurology. Neurology. 2013;80(24):2250–7.
51. Ellemberg D, Henry LC, Macciocchi SN, et al. Advances in sport concussion assessment: from behavioral to brain imaging measures. J Neurotrauma. 2009;26(12):2365–82.
52. Meehan WP 3rd, Bachur RG. Sport-related concussion. Pediatrics. 2009;123(1):114–23.

53. Patel AV, Mihalik JP, Notebaert AJ, et al. Neuropsychological performance, postural stability, and symptoms after dehydration. J Athl Train. 2007;42(1):66–75.
54. Patel DR, Reddy V. Sport-related concussion in adolescents. Pediatr Clin N Am. 2010;57(3):649–70.
55. Scorza KA, Raleigh MF, O'Connor FG. Current concepts in concussion: evaluation and management. Am Fam Physician. 2012;85(2):123–32.
56. McCrea M, Guskiewicz KM, Marshall SW, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. JAMA. 2003;290(19):2556–63.
57. McCrory PR, Ariens T, Berkovic SF. The nature and duration of acute concussive symptoms in Australian football. Clin J Sport Med. 2000;10(4):235–8.
58. Pardini J, Bailes JE, Maroon JC. Mild traumatic brain injury in adults and concussion in sports. In: Winn HR, editor. Youmans neurological surgery, vol. 4. 6th ed. Philadelphia, PA: Saunders; 2011. p. 3380–9.
59. Packard RC. Epidemiology and pathogenesis of posttraumatic headache. J Head Trauma Rehabil. 1999;14(1):9–21.
60. Collins MW, Field M, Lovell MR, et al. Relationship between postconcussion headache and neuropsychological test performance in high school athletes. Am J Sports Med. 2003;31(2):168–73.
61. Mihalik JP, Stump JE, Collins MW, et al. Posttraumatic migraine characteristics in athletes following sports-related concussion. J Neurosurg. 2005;102(5):850–5.
62. McCrea M. Sideline assessment of concussion. In: Bailes JE, Day AL, editors. Neurological sports medicine: a guide for physicians and trainers. Rolling Meadows, IL: American Association of Neurological Surgeons; 2001. p. 109–22.
63. McCrea M, Kelly JP, Randolph C, et al. Standardized assessment of concussion (SAC): on-site mental status evaluation of the athlete. J Head Trauma Rehabil. 1998;13(2):27–35.
64. Iverson GL, Gaetz M, Lovell MR, et al. Relation between subjective fogginess and neuropsychological testing following concussion. J Int Neuropsychol Soc. 2004;10(6):904–6.
65. Cantu RC. Return to play guidelines after a head injury. Clin Sports Med. 1998;17:46–60.
66. Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. Clin J Sport Med. 1995;5:32–5.
67. Halstead ME, Walter KD, Council on Sports Medicine and Fitness. American Academy of Pediatrics. Clinical report—sport-related concussion in children and adolescents. Pediatrics. 2010;126(3):597–615.
68. Leininger BE, Gramling SE, Farrell AD, et al. Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. J Neurol Neurosurg Psychiatry. 1990;53(4):293–6.
69. Lovell MR, Iverson GL, Collins MW, et al. Does loss of consciousness predict neuropsychological decrements after concussion? Clin J Sport Med. 1999;9(4):193–8.
70. Fisher CM. Concussion amnesia. Neurology. 1966;16:826–30.
71. Yarnell PR, Lynch S. Retrograde memory immediately after concussion. Lancet. 1970;1(7652):863–4.
72. Yarnell PR, Lynch S. Progressive retrograde amnesia in concussed football players: observation shortly postimpact. Neurology. 1970;20(4):416–7.
73. McCrea M, Kelly JP, Kluge J, et al. Standardized assessment of concussion in football players. Neurology. 1997;48(3):586–8.
74. Peterson CL, Ferrara MS, Mrazik M, et al. Evaluation of neuropsychological domain scores and postural stability following cerebral concussion in sports. Clin J Sport Med. 2003;13(4):230–7.
75. Riemann BL, Guskiewicz KM. Effects of mild head injury on postural stability as measured through clinical balance testing. J Athl Train. 2000;35(1):19–25.
76. Guskiewicz KM, Bruce SL, Cantu RC, et al. National Athletic Trainers' Association position statement: management of sport-related concussion. J Athl Train. 2004;39(3):280–97.

77. Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. Clin J Sport Med. 1995;5(1):32–5.
78. McCrea M. Standardized mental status assessment of sports concussion. Clin J Sport Med. 2001;11(3):176–81.
79. Guskiewicz KM. Assessment of postural stability following sport-related concussion. Curr Sports Med Rep. 2003;2(1):24–30.
80. Shehata N, Wiley JP, Richea S, et al. Sport concussion assessment tool: baseline values for varsity collision sport athletes. Br J Sports Med. 2009;43(10):730–4.
81. Valovich McLeod TC, Bay RC, Lam KC, et al. Representative baseline values on the Sport Concussion Assessment Tool 2 (SCAT2) in adolescent athletes vary by gender, grade, and concussion history. Am J Sports Med. 2012;40(4):927–33.
82. Davis GA, Purcell L, Schneider KJ, et al. The child sport concussion assessment tool 5th edition (child SCAT5): background and rationale. Br J Sports Med. 2017;51:859–61.
83. Cavanaugh JT, Guskiewicz KM, Giuliani C, et al. Detecting altered postural control after cerebral concussion in athletes with normal postural stability. Br J Sports Med. 2005;39(11):805–11.
84. Guskiewicz KM, Ross SE, Marshall SW. Postural stability and neuropsychological deficits after concussion in collegiate athletes. J Athl Train. 2001;36(3):263–73.
85. Davis GA, Iverson GL, Guskiewicz KM, et al. Contributions of neuroimaging, balance testing, electrophysiology and blood markers to the assessment of sport-related concussion. Br J Sports Med. 2009;43(suppl 1):i36–45.
86. Arfanakis K, Haughton VM, Carew JD, et al. Diffusion tensor MR imaging in diffuse axonal injury. AJNR Am J Neuroradiol. 2002;23(5):794–802.
87. Putukian M, Raftery M, Guskiewicz K, et al. Onfield assessment of concussion in the adult athlete. Br J Sports Med. 2013;47(5):285–8.
88. Echemendia RJ, Herring S, Bailes J. Who should conduct and interpret the neuropsychological assessment in sports-related concussion? Br J Sports Med. 2009;43:i32–5.
89. Belanger HG, Curtis G, Demery JA, et al. Factors moderating neuropsychological outcomes following mild traumatic brain injury: a meta-analysis. J Int Neuropsychol Soc. 2005;11(3):215–7.
90. Iverson GL, Brooks BL, Collins MW, et al. Tracking neuropsychological recovery following concussion in sport. Brain Inj. 2006;20(3):245–52.
91. Iverson GL, Zasler NF, Lange RT. Post-concussion disorder. New York: Demos Medical Publishing; 2006.
92. Belanger HG, Vanderploeg RD. The neuropsychological impact of sports-related concussion: a meta-analysis. J Int Neuropsychol Soc. 2005;11(4):345–7.
93. Broglio SP, Sosnoff JJ, Ferrara MS. The relationship of athlete-reported concussion symptoms and objective measures of neurocognitive function and postural control. Clin J Sport Med. 2009;19(5):377–82.
94. Collie A, Makdissi M, Maruff P, et al. Cognition in the days following concussion: comparison of symptomatic versus asymptomatic athletes. J Neurol Neurosurg Psychiatry. 2006;77(2):241–5.
95. Fazio VC, Lovell MR, Pardini JE, et al. The relation between post concussion symptoms and neurocognitive performance in concussed athletes. NeuroRehabilitation. 2007;22(3):207–16.
96. Iverson GL, Lovell MR, Collins MW. Validity of ImPACT for measuring processing speed following sports-related concussion. J Clin Exp Neuropsychol. 2005;27(6):683–9.
97. Van Kampen DA, Lovell MR, Pardini JE, et al. The "value added" of neurocognitive testing after sports-related concussion. Am J Sports Med. 2006;34(10):1630–5.
98. Echemendia RJ, Iverson GL, McCrea M, et al. Role of neuropsychologists in the evaluation and management of sport-related concussion: an inter-organization position statement. Arch Clin Neuropsychol. 2012;27:119–22.
99. McCrory P, Johnston P, Meeuwisse W, et al. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. Br J Sports Med. 2005;43(suppl 1):i76–84.

100. McCrory P, Makdissi M, Davis G, et al. Value of neuropsychological testing after head injuries in football. Br J Sports Med. 2005;39:i58–63.
101. Meehan WP, d'Hemecourt P, Collins CL, et al. Computerized neurocognitive testing for the management of sport-related concussions. Pediatrics. 2012;129(1):38–44.
102. ImPACT 6.0 [computer program]. Pittsburgh, PA; NeuroHealth Systems; 2007.
103. ImPACT: Immediate post-concussion assessment and cognitive testing. [computer program]. Pittsburgh, PA: NeuroHealth Systems; 2000.
104. Erdal K. Neuropsychological testing for sports-related concussion: how athletes can sandbag their baseline testing without detection. Arch Clin Neuropsychol. 2012;27:473–9.
105. Elbin RJ, Schatz P, Covassin T. One-year test-retest reliability of the online version of ImPACT in high school athletes. Am J Sports Med. 2011;39(11):2319–24.
106. Gl I, Lange RT. Examination of "post-concussion-like" symptoms in a healthy sample. Appl Neuropsychol. 2003;21:303–10.
107. Lau BC, Collins MW, Lovell MR. Sensitivity and specificity of subacute computerized neurocognitive testing and symptom evaluation in predicting outcomes after sport-related concussion. Am J Sports Med. 2011;39(6):1209–16.
108. Schatz P. Long-term test-retest reliability of baseline cognitive assessments using ImPACT. Am J Sports Med. 2010;38(1):47–53.
109. Schatz P, Pardini JE, Lovell MR, et al. Sensitivity and specificity of the ImPACT test battery for concussion in athletes. Arch Clin Neuropsychol. 2006;21:91–9.
110. Schatz P, Putz BO. Cross-validation of measures used for computer-based assessment of concussion. Appl Neuropsychol. 2006;13(3):151–9.
111. Schatz P, Sandel N. Sensitivity and specificity of the online version of ImPACT in high school and collegiate athletes. Am J Sports Med. 2013;41:321–6.
112. Bauer RM, Iverson GL, Cernich AN, et al. Computerized neuropsychological assessment devices: joint position paper of the American Academy of Clinical Neuropsychology and the National Academy of Neuropsychology. Clin Neuropsychol. 2012;26(2):177–96.
113. Register-Mihalik JK, Kontos DL, Guskiewicz KM, et al. Age-related differences and reliability on computerized and paper-pencil neurocognitive assessment batteries. J Athl Train. 2012;47(3):297–305.
114. Broglio SP, Ferrara MS, Macciocchi SN, et al. Test-retest reliability of computerized concussion assessment programs. J Athl Train. 2007;42(4):509–14.
115. Gosselin N, Saluja RS, Chen JK, et al. Brain functions after sports-related concussion: insights from event-related potentials and functional MRI. Phys Sportsmed. 2010;38(3):27–37.
116. Johnston KM, Ptito A, Chankowsky J, et al. New frontiers in diagnostic imaging in concussive head injury. Clin J Sport Med. 2001;11(3):166–75.
117. Khurana VG, Kaye AH. An overview of concussion in sport. J Clin Neurosci. 2012;19(1):1–11.
118. Mendez CV, Hurley RA, Lassonde M, et al. Mild traumatic brain injury: neuroimaging of sports-related concussion. J Neuropsychiatry Clin Neurosci. 2005;17(3):297–303.
119. Prabhu SP. The role of neuroimaging in sport-related concussion. Clin Sports Med. 2011;30(1):103–14, ix.
120. Pulsipher DT, Campbell RA, Thoma R, et al. A critical review of neuroimaging applications in sports concussion. Curr Sports Med Rep. 2011;10(1):14–20.
121. Chen JK, Johnston KM, Collie A, et al. A validation of the post concussion symptom scale in the assessment of complex concussion using cognitive testing and functional MRI. J Neurol Neurosurg Psychiatry. 2007;78(11):1231–8.
122. Chen JK, Johnston KM, Frey S, et al. Functional abnormalities in symptomatic concussed athletes: an fMRI study. NeuroImage. 2004;22(1):68–82.
123. Ptito A, Chen JK, Johnston KM. Contributions of functional magnetic resonance imaging (fMRI) to sport concussion evaluation. NeuroRehabilitation. 2007;22(3):217–27.
124. Jantzen KJ, Anderson B, Steinberg FL, et al. A prospective functional MR imaging study of mild traumatic brain injury in college football players. AJNR Am J Neuroradiol. 2004;25(5):738–45.

125. Lovell MR, Pardini JE, Welling J, et al. Functional brain abnormalities are related to clinical recovery and time to return-to-play in athletes. Neurosurgery. 2007;61(2):352–9; discussion 359–360.
126. Umile EM, Sandel ME, Alavi A, et al. Dynamic imaging in mild traumatic brain injury: support for the theory of medial temporal vulnerability. Arch Phys Med Rehabil. 2002;83(11):1506–13.
127. Chen SH, Kareken DA, Fastenau PS, et al. A study of persistent post-concussion symptoms in mild head trauma using positron emission tomography. J Neurol Neurosurg Psychiatry. 2003;74(3):326–32.
128. DiFiori JP, Giza CC. New techniques in concussion imaging. Curr Sports Med Rep. 2010;9(1):35–9.
129. Vagnozzi R, Signoretti S, Tavazzi B, et al. Temporal window of metabolic brain vulnerability to concussion: a pilot 1H-magnetic resonance spectroscopic study in concussed athletes—part III. Neurosurgery. 2008;62(6):1286–95; discussion 1295–1286.
130. Cimatti M. Assessment of metabolic cerebral damage using proton magnetic resonance spectroscopy in mild traumatic brain injury. J Neurosurg Sci. 2006;50(4):83–8.
131. Henry LC, Tremblay S, Boulanger Y, et al. Neurometabolic changes in the acute phase after sports concussions correlate with symptom severity. J Neurotrauma. 2010;27(1):65–76.
132. Krishna R, Grinn M, Giordano N, et al. Diagnostic confirmation of mild traumatic brain injury by diffusion tensor imaging: a case report. J Med Case Rep. 2012;6(1):66.
133. Bazarian JJ, Zhong J, Blyth B, et al. Diffusion tensor imaging detects clinically important axonal damage after mild traumatic brain injury: a pilot study. J Neurotrauma. 2007;24(9):1447–59.
134. Bazarian JJ, Zhu T, Blyth B, et al. Subject-specific changes in brain white matter on diffusion tensor imaging after sports-related concussion. Magn Reson Imaging. 2012;30(2):171–80.
135. Cubon VA, Putukian M, Boyer C, et al. A diffusion tensor imaging study on the white matter skeleton in individuals with sports-related concussion. J Neurotrauma. 2011;28(2):189–201.
136. Henry LC, Tremblay J, Tremblay S, et al. Acute and chronic changes in diffusivity measures after sports concussion. J Neurotrauma. 2011;28(10):2049–59.
137. Huisman TA, Schwamm LH, Schaefer PW, et al. Diffusion tensor imaging as potential biomarker of white matter injury in diffuse axonal injury. AJNR Am J Neuroradiol. 2004;25(3):370–6.
138. Lipton ML, Gellella E, Lo C, et al. Multifocal white matter ultrastructural abnormalities in mild traumatic brain injury with cognitive disability: a voxel-wise analysis of diffusion tensor imaging. J Neurotrauma. 2008;25(11):1335–42.
139. Mayer AR, Ling J, Mannell MV, et al. A prospective diffusion tensor imaging study in mild traumatic brain injury. Neurology. 2010;74(8):643–50.
140. Messe A, Caplain S, Paradot G, et al. Diffusion tensor imaging and white matter lesions at the subacute stage in mild traumatic brain injury with persistent neurobehavioral impairment. Hum Brain Mapp. 2011;32(6):999–1011.
141. Niogi SN, Mukherjee P, Ghajar J, et al. Extent of microstructural white matter injury in post-concussive syndrome correlates with impaired cognitive reaction time: a 3T diffusion tensor imaging study of mild traumatic brain injury. AJNR Am J Neuroradiol. 2008;29(5):967–73.
142. Niogi SN, Mukherjee P, McCandliss BD. Diffusion tensor imaging segmentation of white matter structures using a Reproducible Objective Quantification Scheme (ROQS). NeuroImage. 2007;35(1):166–74.
143. Rutgers DR, Fillard P, Paradot G, et al. Diffusion tensor imaging characteristics of the corpus callosum in mild, moderate, and severe traumatic brain injury. AJNR Am J Neuroradiol. 2008;29(9):1730–5.
144. Rutgers DR, Toulgoat F, Cazejust J, et al. White matter abnormalities in mild traumatic brain injury: a diffusion tensor imaging study. AJNR Am J Neuroradiol. 2008;29(3):514–9.
145. Smits M, Houston GC, Dippel DW, et al. Microstructural brain injury in post-concussion syndrome after minor head injury. Neuroradiology. 2011;53(8):553–63.

146. Wilde EA, McCauley SR, Hunter JV, et al. Diffusion tensor imaging of acute mild traumatic brain injury in adolescents. Neurology. 2008;70(12):948–55.
147. Kraus MF, Susmaras T, Caughlin BP, et al. White matter integrity and cognition in chronic traumatic brain injury: a diffusion tensor imaging study. Brain. 2007;130(Pt 10):2508–19.
148. Niogi SN, Mukherjee P, Ghajar J, et al. Structural dissociation of attentional control and memory in adults with and without mild traumatic brain injury. Brain. 2008;131(Pt 12):3209–21.
149. Shin SS, Verstynen T, Pathak S, et al. High-definition fiber tracking for assessment of neurological deficit in a case of traumatic brain injury: finding, visualizing, and interpreting small sites of damage. J Neurosurg. 2012;116(5):1062–9.
150. Fernandez-Miranda JC, Pathak S, Engh J, et al. High-definition fiber tractography of the human brain: neuroanatomical validation and neurosurgical applications. Neurosurgery. 2012;71(2):430–53.
151. Zetterberg H, Smith DH, Blennow K. Biomarkers of mild traumatic brain injury in cerebrospinal fluid and blood. Nat Rev Neurol. 2013;9(4):201–10.
152. Mussack T, Kirchhoff C, Buhmann S, et al. Significance of Elecsys S100 immunoassay for real-time assessment of traumatic brain damage in multiple trauma patients. Clin Chem Lab Med. 2006;44(9):1140–5.
153. Stalnacke BM, Ohlsson A, Tegner Y, et al. Serum concentrations of two biochemical markers of brain tissue damage S-100B and neurone specific enolase are increased in elite female soccer players after a competitive game. Br J Sports Med. 2006;40(4):313–6.
154. Metting Z, Wilczak N, Rodiger LA, et al. GFAP and S100B in the acute phase of mild traumatic brain injury. Neurology. 2012;78(18):1428–33.
155. Meehan WP 3rd. Medical therapies for concussion. Clin Sports Med. 2011;30(1):115–24, ix.
156. Petraglia AL, Maroon JC, Bailes JE. From the field of play to the field of combat: a review of the pharmacological management of concussion. Neurosurgery. 2012;70(6):1520–33; discussion 1533.
157. Beauchamp K, Mutlak H, Smith WR, et al. Pharmacology of traumatic brain injury: where is the "golden bullet"? Mol Med. 2008;14(11–12):731–40.
158. Mittenberg W, Burton DB. A survey of treatments for post-concussion syndrome. Brain Inj. 1994;8(5):429–37.
159. Mittenberg W, Canyock EM, Condit D, et al. Treatment of post-concussion syndrome following mild head injury. J Clin Exp Neuropsychol. 2001;23(6):829–36.
160. Reddy C. A treatment paradigm for sports concussion. Brain Inj Professional. 2004;4:24–5.
161. Comper P, Bisschop SM, Carnide N, et al. A systematic review of treatments for mild traumatic brain injury. Brain Inj. 2005;19(11):863–80.
162. Arciniegas DB, Anderson CA, Topkoff J, et al. Mild traumatic brain injury: a neuropsychiatric approach to diagnosis, evaluation, and treatment. Neuropsychiatr Dis Treat. 2005;1(4):311–27.
163. Faux S, Sheedy J. A prospective controlled study in the prevalence of posttraumatic headache following mild traumatic brain injury. Pain Med. 2008;9(8):1001–11.
164. Paniak C, Reynolds S, Phillips K, et al. Patient complaints within 1 month of mild traumatic brain injury: a controlled study. Arch Clin Neuropsychol. 2002;17(4):319–34.
165. Haas DC. Chronic post-traumatic headaches classified and compared with natural headaches. Cephalalgia. 1996;16(7):486–93.
166. Lenaerts ME, Couch JR. Posttraumatic headache. Curr Treat Options Neurol. 2004;6(6):507–17.
167. Lew HL, Lin PH, Fuh JL, et al. Characteristics and treatment of headache after traumatic brain injury: a focused review. Am J Phys Med Rehabil. 2006;85(7):619–27.
168. Tyler GS, McNeely HE, Dick ML. Treatment of post-traumatic headache with amitriptyline. Headache. 1980;20(4):213–6.
169. Weiss HD, Stern BJ, Goldberg J. Post-traumatic migraine: chronic migraine precipitated by minor head or neck trauma. Headache. 1991;31(7):451–6.

170. Lane JC, Arciniegas DB. Post-traumatic headache. Curr Treat Options Neurol. 2002;4(1):89–104.
171. Jha A, Weintraub A, Allshouse A, et al. A randomized trial of modafinil for the treatment of fatigue and excessive daytime sleepiness in individuals with chronic traumatic brain injury. J Head Trauma Rehabil. 2008;23(1):52–63.
172. Kaiser PR, Valko PO, Werth E, et al. Modafinil ameliorates excessive daytime sleepiness after traumatic brain injury. Neurology. 2010;75(20):1780–5.
173. Flanagan SR, Greenwald B, Wieber S. Pharmacological treatment of insomnia for individuals with brain injury. J Head Trauma Rehabil. 2007;22(1):67–70.
174. Rao V, Rollings P. Sleep disturbances following traumatic brain injury. Curr Treat Options Neurol. 2002;4(1):77–87.
175. Nutritional support after spinal cord injury. Neurosurgery. 2002;50(suppl 3):S81–S84.
176. Ruff RL, Ruff SS, Wang XF. Improving sleep: initial headache treatment in OIF/OEF veterans with blast-induced mild traumatic brain injury. J Rehabil Res Dev. 2009;46(9):1071–84.
177. Samantaray S, Das A, Thakore NP, et al. Therapeutic potential of melatonin in traumatic central nervous system injury. J Pineal Res. 2009;47(2):134–42.
178. Orff HJ, Ayalon L, Drummond SP. Traumatic brain injury and sleep disturbance: a review of current research. J Head Trauma Rehabil. 2009;24(3):155–65.
179. Maldonado MD, Murillo-Cabezas F, Terron MP, et al. The potential of melatonin in reducing morbidity-mortality after craniocerebral trauma. J Pineal Res. 2007;42(1):1–11.
180. Kemp S, Biswas R, Neumann V, et al. The value of melatonin for sleep disorders occurring post-head injury: a pilot RCT. Brain Inj. 2004;18(9):911–9.
181. Buscemi N, Vandermeer B, Pandya R, et al. Melatonin for treatment of sleep disorders. Evid Rep Technol Assess (Summ). 2004;108:1–7.
182. Leone H, Polsonetti BW. Amantadine for traumatic brain injury: does it improve cognition and reduce agitation? J Clin Pharm Ther. 2005;30(2):101–4.
183. Sawyer E, Mauro LS, Ohlinger MJ. Amantadine enhancement of arousal and cognition after traumatic brain injury. Ann Pharmacother. 2008;42(2):247–52.
184. Kraus MF, Smith GS, Butters M, et al. Effects of the dopaminergic agent and NMDA receptor antagonist amantadine on cognitive function, cerebral glucose metabolism and D2 receptor availability in chronic traumatic brain injury: a study using positron emission tomography (PET). Brain Inj. 2005;19(7):471–9.
185. Meythaler JM, Brunner RC, Johnson A, et al. Amantadine to improve neurorecovery in traumatic brain injury-associated diffuse axonal injury: a pilot double-blind randomized trial. J Head Trauma Rehabil. 2002;17(4):300–13.
186. Sedney CL, Orphanos J, Bailes JE. When to consider retiring an athlete after sports-related concussion. Clin Sports Med. 2011;30(1):189–200, xi.
187. Binder LM. Persisting symptoms after mild head injury: a review of the postconcussive syndrome. J Clin Exp Neuropsychol. 1986;8(4):323–46.
188. Binder LM, Rohling ML. Money matters: a meta-analytic review of the effects of financial incentives on recovery after closed-head injury. Am J Psychiatry. 1996;153(1):7–10.
189. Johnston KM, McCrory P, Mohtadi NG, et al. Evidence-based review of sport-related concussion: clinical science. Clin J Sport Med. 2001;11(3):150–9.
190. Silverberg ND, Iverson GL. Etiology of the post-concussion syndrome: physiogenesis and psychogenesis revisited. NeuroRehabilitation. 2011;29(4):317–29.
191. Al Sayegh A, Sandford D, Carson AJ. Psychological approaches to treatment of postconcussion syndrome: a systematic review. J Neurol Neurosurg Psychiatry. 2010;81(10):1128–34.
192. Leddy JJ, Sandhu H, Sodhi V, et al. Rehabilitation of concussion and post-concussion syndrome. Sports Health. 2012;4(2):147–54.
193. Guskiewicz KM, Marshall SW, Bailes J, et al. Recurrent concussion and risk of depression in retired professional football players. Med Sci Sports Exerc. 2007;39(6):903–9.
194. Ponsford J, Willmott C, Rothwell A, et al. Factors influencing outcome following mild traumatic brain injury in adults. J Int Neuropsychol Soc. 2000;6(5):568–79.

195. Preiss-Farzanegan SJ, Chapman B, Wong TM, et al. The relationship between gender and postconcussion symptoms after sport-related mild traumatic brain injury. PM R. 2009;1(3):245–53.
196. Chamelian L, Feinstein A. The effect of major depression on subjective and objective cognitive deficits in mild to moderate traumatic brain injury. J Neuropsychiatry Clin Neurosci. 2006;18(1):33–8.
197. Luis CA, Vanderploeg RD, Curtiss G. Predictors of postconcussion symptom complex in community dwelling male veterans. J Int Neuropsychol Soc. 2003;9(7):1001–15.
198. McCauley SR, Boake C, Levin HS, et al. Postconcussional disorder following mild to moderate traumatic brain injury: anxiety, depression, and social support as risk factors and comorbidities. J Clin Exp Neuropsychol. 2001;23(6):792–808.
199. Field M, Collins MW, Lovell MR, et al. Does age play a role in recovery from sports-related concussion? A comparison of high school and collegiate athletes. J Pediatr. 2003;142(5):546–53.
200. Grady MF. Concussion in the adolescent athlete. Curr Probl Pediatr Adolesc Health Care. 2010;40(7):154–69.
201. Pellman EJ, Lovell MR, Viano DC, et al. Concussion in professional football: recovery of NFL and high school athletes assessed by computerized neuropsychological testing—Part 12. Neurosurgery. 2006;58(2):263–74; discussion 263–274.
202. Pickles W. Acute general edema of the brain in children with head injuries. N Engl J Med. 1950;242(16):607–11.
203. Aldrich EF, Eisenberg HM, Saydjari C, et al. Diffuse brain swelling in severely head-injured children. A report from the NIH Traumatic Coma Data Bank. J Neurosurg. 1992;76(3):450–4.
204. Bruce DA, Alavi A, Bilaniuk L, et al. Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema". J Neurosurg. 1981;54(2):170–8.
205. Bauer R, Fritz H. Pathophysiology of traumatic injury in the developing brain: an introduction and short update. Exp Toxicol Pathol. 2004;56(1–2):65–73.
206. McDonald JW, Johnston MV. Physiological and pathophysiological roles of excitatory amino acids during central nervous system development. Brain Res Brain Res Rev. 1990;15(1):41–70.
207. Mihalik JP, Blackburn JT, Greenwald RM, et al. Collision type and player anticipation affect head impact severity among youth ice hockey players. Pediatrics. 2010;125(6):e1394–401.
208. Ommaya AK, Goldsmith W, Thibault L. Biomechanics and neuropathology of adult and paediatric head injury. Br J Neurosurg. 2002;16(3):220–42.
209. Meares S, Shores EA, Taylor AJ, et al. Mild traumatic brain injury does not predict acute postconcussion syndrome. J Neurol Neurosurg Psychiatry. 2008;79(3):300–6.
210. Ruff RM, Camenzuli L, Mueller J. Miserable minority: emotional risk factors that influence the outcome of a mild traumatic brain injury. Brain Inj. 1996;10(8):551–65.
211. Bryant RA, Harvey AG. Postconcussive symptoms and posttraumatic stress disorder after mild traumatic brain injury. J Nerv Ment Dis. 1999;187(5):302–5.
212. Wetjen NM, Pichelmann MA, Atkinson JL. Second impact syndrome: concussion and second injury brain complications. J Am Coll Surg. 2010;211(4):553–7.
213. Giza CC, Hovda DA. The neurometabolic cascade of concussion. J Athl Train. 2001;36(3):228–35.
214. Cantu RC. Second-impact syndrome. Clin Sports Med. 1998;17(1):37–44.
215. McCrea HJ, Perrine K, Niogi S, et al. Concussion in sports. Sports Health. 2013;5(2):160–4.
216. Thomas M, Haas TS, Doerer JJ, et al. Epidemiology of sudden death in young, competitive athletes due to blunt trauma. Pediatrics. 2011;128(1):e1–8.
217. McCrory P, Davis G, Makdissi M. Second impact syndrome or cerebral swelling after sporting head injury. Curr Sports Med Rep. 2012;11(1):21–3.
218. Bailes JE, Dashnaw ML, Petraglia AL, et al. Cumulative effects of repetitive mild traumatic brain injury. Prog Neurol Surg. 2014;28:50–62.

219. Bailes JE, Petraglia AL, Omalu BI, et al. Talavage T. Role of subconcussion in repetitive mild traumatic brain injury. J Neurosurg. 2013;119(5):1235–45.
220. Omalu BI, DeKosky ST, Minster RL, et al. Chronic traumatic encephalopathy in a National Football League player. Neurosurgery. 2005;57(1):128–34; discussion 128–134.
221. Manley G, Gardner AJ, Schneider KJ, et al. A systematic review of potential long-term effects of sport-related concussion. Br J Sports Med. 2017;51:969–77.
222. McAllister T, McCrea M. Long-term cognitive and neuropsychiatric consequences of repetitive concussion and head-impact exposure. J Athl Train. 2017;52(3):309–17. https://doi.org/10.4085/1062-6050-52.1.14.
223. Noy S, Krawitz S, Del Bigio MR. Chronic traumatic encephalopathy-like abnormalities in a routine neuropathology service. J Neuropathol Exp Neurol. 2016;75(12):1145–54. https://doi.org/10.1093/jnen/nlw092.
224. Iverson GL, Luoto TM, Karhunen PJ, Castellani RJ. Mild chronic traumatic encephalopathy neuropathology in people with no known participation in contact sports or history of repetitive neurotrauma. J Neuropathol Exp Neurol. 2019;78(7):615–25. https://doi.org/10.1093/jnen/nlz045.
225. Dashnaw ML, Petraglia AL, Bailes JE. An overview of the basic science of concussion and subconcussion: where we are and where we are going. Neurosurg Focus. 2012;33(6):E5: 1–9.
226. Saulle M, Greenwald BD. Chronic traumatic encephalopathy: a review. Rehabil Res Pract. 2012;2012:816069.
227. Blaylock RL, Maroon J. Immunoexcitotoxicity as a central mechanism in chronic traumatic encephalopathy—a unifying hypothesis. Surg Neurol Int. 2011;2:107.
228. Turner RC, Lucke-Wold BP, Robson MJ, et al. Repetitive traumatic brain injury and development of chronic traumatic encephalopathy: a potential role for biomarkers in diagnosis, prognosis, and treatment? Front Neurol. 2012;3:186.
229. Small GW, Kepe V, Siddarth P, et al. PET scanning of brain tau in retired national football league players: preliminary findings. Am J Geriatr Psychiatry. 2013;21(2):138–44.