



SPORTS MEDICINE AUSTRALIA

POSITION STATEMENT: CONCUSSION

Purpose of this Statement

The primary purpose of this document is to provide an evidence-based, best practice summary to assist Sports Medicine Australia members and others (coaches, parents, officials, administrators etc.) to recognise and manage sport-related concussion.

This position statement is based mainly on a review of the contents of three documents published in 2013:

- Consensus Statement on Concussion in Sport—the 4th International Conference on Concussion in Sport Held in Zurich, November 2012.¹
- American Medical Society for Sports Medicine Position Statement: Concussion in Sport.²
- 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.³

Perspective

Concussion is a relatively common injury in many sporting and recreational activities. Sports such as Australian football, rugby league and rugby union have amongst the highest rates of concussion of any team sports in the world with reported incidence ranging from about 3–10 concussive injuries per 1000 player hours⁴⁻⁷ or about five concussion injuries per team per season, regardless of the level of competition.⁸ When males and females participate in the same sports with similar rules, the reported incidence of concussion is higher among females than males.² Despite the high levels of concussion reported, there is no routine monitoring or reporting of sport-related concussion in Australia and the incidence of sport-related concussion in Australia, especially at the population level, is unknown. Nonetheless, a recent study found that Victorian hospitalisations for sports-related concussions had increased by more than 60% over 9 years.⁹ Sport-related concussion represents a significant clinical and public health issue in active communities.

Sports medicine and health care professionals are frequently involved in the care of participants with sports concussion and are often the first port of call for concussed sports participants and concerned parents. This position statement has been developed to help anyone who finds themselves in a position of making concussion-related diagnostic, management and return-to-participation (RTP) decisions and to understand the limitations of their expertise.

Definition

A concussion is a disturbance in brain function caused by a direct or indirect force to the head. Concussion is a subset of mild traumatic brain injury that is at the less severe end of the brain injury spectrum. It is characterised by a graded set of neurological symptoms and signs that typically arise rapidly and resolve spontaneously over a sequential course. The process of recovery, however, varies from person to person and injury to injury.

The pathophysiology of concussion is not well understood but the current consensus is that the injury reflects a physiological disturbance rather than a structural injury to the brain.⁴

Pathophysiology

Concussion is defined as a 'complex pathophysiological process affecting the brain, induced by biomechanical forces'.¹ The pathophysiology of concussion is not well understood although animal studies have provided some clarification.

Fundamentally, after an acute injury, the metabolic needs of the nerve cells are very high. Under normal conditions, mitochondria up-regulate production of adenosine triphosphate (ATP) to meet those metabolic demands. However, a concussion injury impairs the function of the mitochondria.

A concussion injury can be thought of as having two parts: a primary insult and a secondary inflammatory response. The primary insult triggers a pathologic release of excitatory neurotransmitters, which leads to the loss of cell wall integrity. This in turn leads to changes in the sodium–potassium balance.

During this period the mitochondria are impaired and unable to produce the large amounts of ATP needed for cellular function.¹⁰ This results in cellular damage. As severely injured cells die they increase the inflammatory process¹¹ leading to secondary injury. This cell injury may explain why concussive symptoms can worsen clinically during the first 24 to 72 hours after the initial injury.

The net result is a decrease in brain function during the first few days after the injury. Abnormalities have been demonstrated in early post-injury and in patients with persistent post-concussion symptoms.^{12,13} Additionally, there is a time window of increased susceptibility to repeat injury after the first injury. This may account for what is termed "Second Impact Syndrome" which is particularly prevalent in adolescents whose brains are still developing. Mild injury that does not cause any structural damage, when repeated again 24 hours later, results in significant nerve injury and increased memory impairment.¹⁴

Shortly after a concussion, the metabolic needs of the cell, including glucose requirements, increase dramatically.^{15,16} Glucose delivery via cerebral circulation after a brain injury is thus crucial to restoring cell membrane healing. After an injury, the brain should increase cerebral blood flow to speed the delivery of nutrients, including glucose, to the injured cells. However, the cellular response to injury restricts cerebral blood flow, and the flow metabolism coupling is disrupted.^{15,17,18} If cerebral glucose needs remain low, the cerebral perfusion and glucose delivery may be sufficient for baseline cellular needs. However, if the demand for glucose is high, the body is unable to up-regulate cerebral blood flow to meet the metabolic demand and, as a result, the glucose needs of the injured cells are not met.

This "metabolic mismatch" of increased cerebral metabolic needs and functionally decreased ATP production is the fundamental concept underlying acute concussion management.¹⁹ During the early phase of brain healing, a tenuous equilibrium between energy needs and energy production develops. Given sufficient time and energy to recover, the nerve cells can restore intracellular function and remain viable.

Modifying Factors in Concussion Management

A range of clinical factors that may be associated with longer duration of symptoms or increased risk of adverse outcomes are summarised in Table 1.⁴ The presence of any modifying factors after a concussive injury requires a more conservative approach, including more detailed assessment and slower return to participation. In difficult or complicated cases, a multidisciplinary team approach should be considered possibly including: referral to neuropsychologist and/or doctor with expertise in managing concussion; a physiotherapist to

coordinate a neuro-rehabilitation program; a psychologist/psychiatrist if there are prominent symptoms of depression/anxiety; and an exercise physiologist to oversee a graded sub-symptom threshold exercise program).

Table 1: Concussion modifiers⁴

Factors	Modifier
Symptoms	High number of concussions, long duration (>10 days), high severity
Signs	Prolonged loss of consciousness (>1 min), amnesia
Sequelae	Concussive convulsions
Temporal	Frequency: repeated concussions over time Timing: injuries close together in time "Recency": recent concussion or traumatic brain injury
Threshold	Repeated concussions occurring with progressively less impact force or slower recovery after each successive concussion
Age	Child and adolescent (<18 years old)
Co- and Pre-morbidities	Migraine, depression or other mental health disorders, attention deficit hyperactivity disorder, learning disabilities, sleep disorders
Medication	Psychoactive drugs, anticoagulants
Behaviour	Dangerous style of play
Sport	High-risk activity, contact and collision sport, high sporting level

Diagnosis of concussion

The diagnosis of acute concussion usually involves assessment of a range of domains including clinical symptoms, physical signs, cognitive impairment, neurobehavioral features and sleep disturbance.

The suspected diagnosis of concussion can include one more of the following features:^{1,4}

- a) Symptoms: headache, dizziness, nausea, vomiting, blurred vision, sensitivity to light and noise;
- b) Physical signs: slow to get up, holding of head, unsteadiness, dazed look, loss of consciousness (in about 10% of cases), loss of balance and poor co-ordination;
- c) Behavioural and emotional changes: irritability, sadness, anxiety;
- d) Cognitive impairment: slowed reaction times, difficulty concentrating, amnesia, feeling in a fog, confusion; and
- e) Sleep disturbances: sleeping more or less than usual, insomnia.

If any one or more of these components is present, a concussion should be suspected and the appropriate management strategy adopted.

On-field/sideline evaluation and management

If anyone sustains a knock to the head or neck while participating in sport or recreational and shows ANY of the features of a concussion listed above:



- The participant should be assessed using standard emergency first aid principles (airway, breathing and heart function) with particular attention to excluding cervical spine injury;
- Once the first aid issues have been addressed, the injured participant should be removed from the game or activity and assessed for concussion using the Concussion Recognition Tool, Sport Concussion Assessment Tool – Edition 3 (SCAT3), Standard Assessment of Concussion or other recognised sideline assessment tool;
- If concussion is suspected, the participant should be monitored closely and referred to a medical practitioner for assessment. If there is any doubt the participant should be sent by ambulance to hospital;
- The participant should not be left alone following the injury and serial monitoring is essential over the initial few hours.

Anyone evaluating concussion on the field or sideline should err on the side of safety: “when in doubt, sit them out”.

Any sports participant diagnosed with or suspected of having concussion should not be allowed to return to participation on the day of injury.

The appearance of symptoms or cognitive deficit might be delayed several hours following a concussive episode so a concussed participant should not be left alone. Appropriate arrangements should be made for the participant to be accompanied and monitored once they leave the participation area. Take home information (covering signs and symptoms that should prompt being taken to a medical facility, and the need for follow-up evaluation by a medical practitioner) should be provided in both verbal and written form to the participant and the accompanying person at the time of leaving the participation area.

Evaluation by medical personnel

A sports participant with suspected concussion should have medical follow-up. The key features of this follow-up should include:

- a) A medical assessment including a comprehensive history and detailed neurological examination.
- b) A determination of the clinical status of the athlete.
- c) Simple neurocognitive tests (where available).
- d) A determination of the need for neuroimaging in order to exclude a more severe brain injury involving a structural abnormality.

Ultimately, the diagnosis of concussion should be a clinical decision based on assessment of a range of domains including symptoms, physical signs, emotional and behavioural changes, cognitive impairment and sleep disturbances.

Concussion management

Most (80%–90%) concussions resolve in a short (7–10 day) period although the recovery time frame may be longer for children (<18 years of age).

Physical and cognitive rest until the acute symptoms resolve are the cornerstone of concussion management followed by a stepwise program of increasing exertion and medical clearance and RTP. An initial period of rest in the acute symptomatic period following injury (24–48 hours) may be of benefit. In the absence of evidence-based recommendations, a sensible approach involves the gradual return to school and social activities (prior to contact



sport) when the participant is symptom free, in a way that does not result in a significant exacerbation of symptoms.

Monitoring post-concussion symptoms and signs can be facilitated by the use of the SCAT3, a standardised method of evaluating individuals after a concussive injury. It provides a multifaceted clinical assessment, consisting of a graded symptom checklist, clinical tests of balance and a basic cognitive assessment.

Neuropsychological (NP) testing in concussion has been shown to be of clinical value and contributes significant information in concussion evaluation and in particular any RTP protocol. Formal NP testing, performed by a trained neuropsychologist, remains the clinical standard for the assessment of cognitive function and is recommended in any case where there is uncertainty about recovery or in difficult cases such as prolonged recovery.

Most concussions can be managed appropriately without the use of neuroimaging or NP testing. In general, imaging should be reserved for athletes with suspected intracranial bleeding and NP testing should be seen as an aid to clinical decision making. In some cases, properly administered and interpreted NP testing provides added value to assess cognitive function and recovery, particularly if athletes have persistent symptoms or complications.

Stepwise return-to-participation protocol

Concussion symptoms should be fully resolved before returning to any physical activity. Importantly, a concussed participant should not only be symptom free, but also should not be taking any pharmacological agents/medications that may mask or modify the symptoms of concussion.

An individualised, stepwise RTP protocol should be developed following a concussion. This should follow a process of:

1. no activity;
2. light aerobic exercise;
3. sport-specific exercise;
4. non-contact drills;
5. full contact practice; and
6. return-to-participation.

The individual should ONLY progress through the steps if asymptomatic at the current level allowing for adequate time for symptoms to develop post exercise.

Generally, each step should take 24 hours so a participant would take approximately one week to proceed through the full RTP protocol once they are asymptomatic at rest. Even where participants have expert care, these guidelines should form the basis of RTP decision-making.

If symptoms occur while in the stepwise program the progression should be halted and the participant should drop back to the previous asymptomatic level. The participant should try to progress again after a further 24 hour period of rest.

A concussed individual also requires cognitive rest and their mental workload should be reduced while they recover. For instance, students may need academic accommodation such as reduced workloads and extended rest periods while recovering from concussion.



Return-to-participation (and practice) after concussion should only occur once all symptoms are resolved and with medical clearance from a medical practitioner experienced in the management of concussion

Short term risks of premature return-to-participation

There are potential health risks associated with a sports participant returning to participation before being asymptomatic. The main concern with early RTP is the decreased reaction time leading to increased risk of repeat concussion or other injury and prolonged duration of symptoms.

There is considerable debate as to whether 'Second Impact Syndrome' (diffuse cerebral swelling) is related to a prior head injury but the association with concussion is a compelling reason why a participant should not RTP before symptoms of their concussion have completely resolved.

Prolonged concussive symptoms

Persistent symptoms (>10 days) are generally reported in 10%–15% of concussions. Post-concussion syndrome is simply defined as symptoms and signs of concussion that persist for weeks to months after the incident. Where clinical recovery takes longer than expected (i.e. approximately 10 days) participants should be managed by multidisciplinary of team health care providers with experience in sports-related concussion. The foundation of post-concussion syndrome management is time. Important components of management after the initial period of physical and cognitive rest include associated therapies such as cognitive, vestibular, physical and psychological therapy, consideration of assessment of other causes of prolonged symptoms, and consideration of commencement of a stepwise exercise program at a level that does not exacerbate symptoms.

Long-term consequences of concussion

There is increasing concern that head impact exposure and recurrent concussions contribute to long-term neurological sequelae including chronic traumatic encephalopathy (CTE) and chronic neurocognitive impairment (CNI).

While the potential for CNI and CTE is concerning, large-scale, epidemiological studies will be required to more clearly understand the causes and develop prevention strategies. At present, there are no published epidemiological, cohort, or prospective studies relating to modern CTE. Due to the nature of the case reports and pathological case series that have been published, it is not possible to determine the causality or risk factors with any certainty. It has been suggested that repeated concussion or sub-concussive impacts cause CTE and/or CNI, and further research is encouraged to explore this issue.

Injury prevention

There is no good clinical evidence that currently available protective equipment will prevent concussion. Strict enforcement of participation rules by coaches and officials, and strict adherence to the rules and the principles of fair play by participants will reduce the incidence of concussion.

This document was prepared by a working group at the request of Sports Medicine Australia. Members of the working group were: Gaery Barber, Deb Bow, Fiona Boys, Adrian Cohen, Alasdair Dempsey, Alex Donaldson, Mark Hecimovich and Eric Rosario.

References

1. McCrory P, Meeuwisse WH, Aubry M, et al. Consensus statement on concussion in sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012. *British Journal of Sports Medicine*. 2013;47(5):250-258.
2. Harmon KG, Drezner J, Gammons M, et al. American Medical Society for Sports Medicine position statement: Concussion in sport. *Clinical Journal of Sport Medicine*. 2013;23(1):1-18.
3. Giza CC, Kutcher JS, Ashwal S, et al. Summary of evidence-based guideline update: Evaluation and management of concussion in sports. *Neurology*. 2013;80(24):2250-2257.
4. Makdissi M, Davis G, McCrory P. Updated guidelines for the management of sports-related concussion in general practice. *Australian Family Physician*. 2014;43:94-99.
5. Makdissi M, McCrory P, Ugoni A, Darby D, Brukner P. A prospective study of postconcussive outcomes after return to play in Australian football. *Am J Sports Med*. 2009;37(5):877-883.
6. Kemp SPT, Hudson Z, Brooks JHM, Fuller CW. The epidemiology of head injuries in English professional rugby union. *Clinical Journal of Sport Medicine*. 2008;18(3):227-234.
7. Hinton-Bayre AD, Geffen G, Friis P. Presentation and mechanisms of concussion in professional Rugby League Football. *J Sci Med Sport*. 2004;7(3):400-404.
8. Makdissi M, Davis G, McCrory P. Updated guidelines for the management of sports-related concussion in general practice. *Australian Family Physician*. 2014;43(3):94-99.
9. Caroline F Finch, Clapperton AJ, McCrory P. Increasing incidence of hospitalisation for sport-related concussion in Victoria, Australia. *Med J Aust* 2013;198(8):427-430.
10. Thompson HJ, Lifshitz J, Marklund N, et al. Lateral fluid percussion brain injury: a 15-year review and evaluation. *Journal of neurotrauma*. 2005;22(1):42-75.
11. Schmidt OI, Heyde CE, Ertel W, Stahel PF. Closed head injury—an inflammatory disease? *Brain Research Reviews*. 2005;48(2):388-399.
12. Niogi SN, Mukherjee P. Diffusion tensor imaging of mild traumatic brain injury. *The Journal of head trauma rehabilitation*. 2010;25(4):241-255.
13. Wilde EA, McCauley SR, Barnes A, et al. Serial measurement of memory and diffusion tensor imaging changes within the first week following uncomplicated mild traumatic brain injury. *Brain imaging and behavior*. 2012;6(2):319-328.
14. Smith DH, Meaney DF, Shull WH. Diffuse axonal injury in head trauma. *The Journal of head trauma rehabilitation*. 2003;18(4):307-316.
15. Gurkoff GG, Giza CC, Hovda DA. Lateral fluid percussion injury in the developing rat causes an acute, mild behavioral dysfunction in the absence of significant cell death. *Brain research*. 2006;1077(1):24-36.
16. Yuen TJ, Browne KD, Iwata A, Smith DH. Sodium channelopathy induced by mild axonal trauma worsens outcome after a repeat injury. *Journal of neuroscience research*. 2009;87(16):3620-3625.
17. Bergsneider M, Hovda DA, Shalmon E, et al. Cerebral hyperglycolysis following severe traumatic brain injury in humans: a positron emission tomography study. *Journal of neurosurgery*. 1997;86(2):241-251.
18. Mintun MA, Lundstrom BN, Snyder AZ, Vlassenko AG, Shulman GL, Raichle ME. Blood flow and oxygen delivery to human brain during functional activity: theoretical modeling and experimental data. *Proceedings of the National Academy of Sciences*. 2001;98(12):6859-6864.
19. Donat CK, Walter B, Kayser T, et al. Effects of lateral fluid percussion injury on cholinergic markers in the newborn piglet brain. *International Journal of Developmental Neuroscience*. 2010;28(1):31-38.