

3 Head injuries in sport

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In sport medicine, doctors must be able to recognise and manage a spectrum of brain injury. Fortunately, serious brain injury is rare in sport (outside motor sports) and most head injuries seen are mild in nature.

Epidemiology

Traumatic brain injury is one of the leading causes of morbidity and mortality worldwide. The crude incidence for all traumatic brain injuries is estimated at about 300 per 100 000 per year, although this varies from country to country.

In hospital based surveys of brain trauma, sporting injuries contribute approximately 10-15% of all cases. Interestingly, the sports most commonly associated with severe brain injuries are golf, horse riding, and mountain climbing. Sporting related deaths because of brain injury are rare, although these have not been studied rigorously outside professional horse racing and American and Australian football.

Eighty per cent of all cases of sport related traumatic brain injuries are mild, so this group constitutes the greatest management problem for a team physician. Concussion, which is a subset of mild brain injury, needs accurate diagnosis and management to avoid long term problems.

Classification of head injury

The most widely accepted and validated method of classifying the spectrum of brain injury is the Glasgow coma scale. This scale uses eye opening, verbal response, and motor response to standard stimuli. These responses are measured six hours after injury after any resuscitation has been completed. The score is then used to separate the categories of injury severity. A score of 13-15 is designated as a mild injury, 8-12 as a moderate injury, and <8 as a severe injury.

The Glasgow coma scale also may be used for serial measurement of head injury status, where an immediate score is obtained during initial assessment of an injured patient and then performed serially to monitor progress.

Although the Glasgow coma scale is extremely useful for measuring moderate to severe traumatic brain injury, its usefulness is limited in the assessment of mild brain injury. In sports, most concussive injuries have recovered from their acute symptoms within six hours and as a result are unable to be classified under this scale. This group provides sports medicine practitioners with the greatest management dilemmas in terms of return to play decisions. Alternative classification systems have been proposed to assess concussive injuries; however, none of these have been validated scientifically.

Pathophysiology

Non-penetrating brain injury (or closed head injury) may be divided into primary and secondary injuries. Primary injury is the result of mechanical forces producing tissue deformation at the moment of injury. These deformations may result in either functional disturbance or structural disruption of cell membranes. The injury may also set off a complex cascade of biochemical, immunological, or coagulopathic changes that may further compromise cell integrity. Secondary damage occurs as a complication of primary injury.

All people involved in the care of athletes need to have a thorough understanding of the early management of concussed athletes and the potential sequelae of such injuries that may impact upon the athletes' ability to return to sport



Men are more than twice as likely to have a traumatic brain injury than women, with a peak incidence among those aged 15-24 years; the most common cause of these injuries is motor vehicle crashes¹

Glasgow coma scale

Category	Response	Score
Eye opening response (E)	Spontaneous	4
	To speech	3
	To pain	2
	No response	1
Verbal response (V)	Oriented	5
	Confused, disorientated	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Motor response (M)	Obeys commands	6
	Localises	5
	Withdraws (flexion)	4
	Abnormal flexion (posturing)	3
	Extension (posturing)	2
	No response	1

Score = E + M + V (maximum 15)

Secondary damage*

- Hypoxic and ischaemic damage
- Brain swelling
- Hydrocephalus
- Infection

*Hovda D et al. *J Neurotrauma* 1995;12:903-6

Concussive injury by definition has no macroscopic neuropathological damage and it is speculated that the critical physiological change occurs at the cell membrane level. Recent evidence also indicates a substantial genetic basis to the outcomes in people with head injuries²

Specific injuries

Most texts tend to focus on neurosurgical head injuries, but >95% of brain injuries seen by sports physicians and trainers are concussive injuries. In some sports, such as motor racing, more severe brain injuries occur more often, but this chapter focuses on the commonplace injuries and their management.

Concussion

More than 35 years ago, the Committee on Head Injury Nomenclature of the US Congress of Neurological Surgeons proposed a “consensus” definition of concussion. This definition was recognised as having a number of limitations, including being unable to account for the relatively minor impact injuries that result in persistent physical or cognitive symptoms, or both. Partly in response to such issues, the First World Conference on Concussion in Sport was held in Vienna in 2002. At that meeting, a consensus definition was agreed and has now become the accepted definition of this condition.³

The classification of severity of concussive injury is a contentious area. At least 35 different injury severity grading systems have been published, but none has been validated scientifically. The Vienna Expert Consensus Group recommended that no specific scale be used and that all management of concussive injuries should measure individual recovery to determine return to play rather than using anecdotal grading systems and arbitrary exclusion periods.³

The practical management of concussion can be divided into three broad areas: immediate, early and late management. In each, the issues and treatment priorities differ considerably.

Immediate management

This is where the medic is in attendance at a sporting event and is called on to manage acute brain injury. The major priorities at this early stage are the basic principles of first aid. Once the basic aspects of care have been achieved and the patient is stabilised, consideration of removal of the patient from the field to an appropriate facility is needed. At this time, careful assessment for the presence of a cervical spine injury or other injury is needed. If an alert patient complains of neck pain, has evidence of neck tenderness or deformity, or has neurological signs indicating a spinal injury, then neck bracing and transport on a suitable spinal frame is required. If the patient is unconscious, cervical spinal cord injury should be assumed until proven otherwise. Airway protection takes precedence over any potential spinal injury. In this situation, removal of helmets or other head protectors should be performed only by individuals trained in this aspect of trauma management.

The clinical management may involve the treatment of disorientated, confused, unconscious, uncooperative, or convulsing patients. The immediate treatment priorities remain the basic first aid principles of “ABC—airway, breathing, and circulation.” Once this has been established and the patient is stabilised, a full medical and neurological assessment examination should follow. On site doctors are in an ideal position to initiate the critical early steps of medical care to ensure optimal recovery from a head injury.

Early management

This refers to the situation when an athlete is brought to the medical room for assessment or to an emergency department or medical facility after the injury. Assessment of injury severity is best performed in a quiet medical room rather than the field of play. This assessment should be performed by a medical practitioner. If no doctor is available for this assessment, the athlete should be referred to a suitable facility (for example, a hospital emergency department).

Vienna expert consensus definition of concussion

Concussion is defined as “a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces.” Several common features that incorporate clinical, pathological, and biomechanical injury constructs and may be used to define the nature of a concussive head injury including:

- Concussion may be caused by a direct blow to the head, face, neck, or elsewhere on the body with an “impulsive” force transmitted to the head.
- Concussion typically results in rapid onset of short lived impairment of neurological function that resolves spontaneously.
- Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.
- Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
- Concussion typically is associated with grossly normal structural neuroimaging studies

Initial on field assessment of concussion

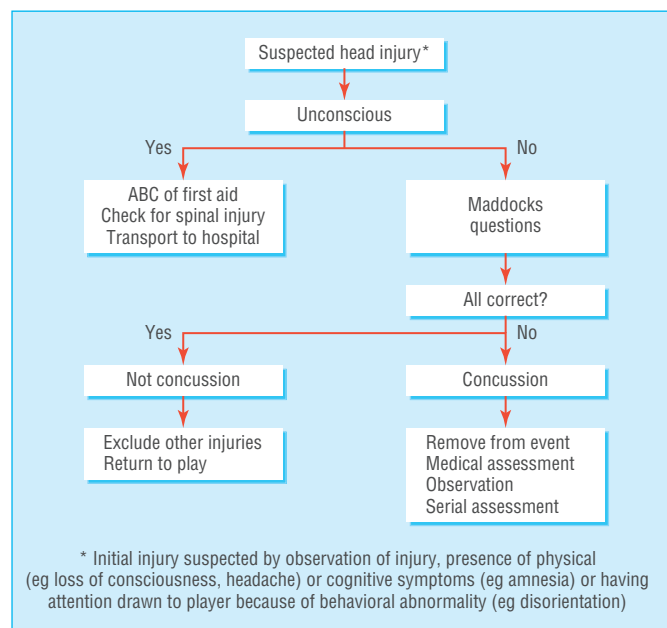
D—Danger: ensure no immediate environmental dangers that may potentially injure the patient or treatment team. This may involve stopping play in a football match or marshalling cars on a motor racetrack.

R—Response: is the patient conscious? Can they talk?

A—Airway: ensure a clear and unobstructed airway. Remove any mouthguard or dental device that may be present.

B—Breathing: ensure patient is breathing adequately.

C—Circulation: ensure adequate circulation.



Acute concussion management

When an acutely concussed player is assessed, various aspects of the history and examination are important. The common symptoms of concussion have been examined in prospective studies and include headache, dizziness, blurred vision, and nausea. The presence of headache is not confined to concussion, however, with up to 50% of sporting athletes reporting exercise related headache. As much emphasis is placed on headache as an important symptom of concussion, medical assessment needs to be accurate in ascertaining the nature and cause of the player's symptoms.

Also important to note is that neither the presence nor duration of loss of consciousness is a critical aspect of concussion assessment. Numerous studies have shown that loss of consciousness is not prognostic in this setting, and, accordingly, injury classification and management algorithms should not be based on this symptom.

A full neurological examination is important when a concussed athlete is examined. The major management priorities at this stage are to establish an accurate diagnosis and exclude a catastrophic intracranial injury, so this part of the examination should be particularly thorough.

Recently, simple neuropsychological tests have created considerable interest as a means of objectively assessing concussed athletes. The standard approach of asking the orientation items (for example, day, date, year, time, date of birth, and so on) has been shown to be unreliable after concussive injury. This aspect of memory remains relatively intact in the face of concussive injury and should not be used. More useful, as demonstrated in prospective studies, are questions of recent memory.⁴ A typical question battery is rapid to administer and validated scientifically in making a diagnosis of concussion. Alternative systems have been proposed, but these are lengthier to administer and not suited to most sports.⁵

Although a trainer or non-medical person may use the Maddocks questions, or other similar tools, to diagnose concussion (or suspect the diagnosis), all concussed athletes should be referred for urgent medical assessment. Most high level amateur and professional teams in fact will have their own medical staff to make the diagnosis; however, where teams lack this facility, concussed athletes need to be referred to a local medical provider or hospital emergency department to undergo a full assessment.

When the presence of a concussive injury is determined, the patient needs to be monitored serially until full recovery ensues. If the concussed player is discharged home after recovery, they should be in the care of a responsible adult. The author's policy is to give the patient and their attendant a head injury advice card upon discharge.

The treating doctor also must decide who should be referred to a hospital emergency facility or neurosurgical centre. A number of urgent indications exist. Apart from these "cookbook" type approaches, referral to such a centre depends on the experience, ability, and competency of the doctor at hand. The overall approach should be "when in doubt, refer."

Late management and return to play

This refers to when a player has sustained a concussive injury previously and now is presenting for advice or clearance before resuming sport. The main management priorities at this stage are assessment of recovery and application of the appropriate return to sport guidelines. Any clearance to return to sport is the province of a medical practitioner, ideally with experience of these sporting injuries, and it should not be undertaken by non-medical personnel.

Criteria for return to sport after a concussion remain the most contentious area of debate. Although the traditional

Early assessment of concussion—history

- Time and place of injury
- Mechanism of injury (eyewitness or video)
- Presence or duration of loss of consciousness
- Behaviour after injury
- Presence of convulsions post-injury
- Past medical history
- Drug use

Post concussion memory assessment (Maddocks questions)

- Which ground are we at?
- Which team are we playing today?
- Who is your opponent at present?
- Which quarter is it?
- How far into the quarter is it?
- Which side scored the last goal?
- Which team did we play last week?
- Did we win last week?

Head injury advice

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 is expected to be rapid, but in such cases to be quite certain is not possible.

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

Other important points:

- No alcohol
- No analgesics or pain killers
- No driving

Patient's name: _____

Date and time of injury: _____

Date of medical review: _____

Treating doctor: _____

Clinic telephone number _____

Indications for urgent referral

- Fractured skull
- Penetrating skull trauma
- Deterioration in conscious state after injury
- Focal neurological signs
- Confusion or impairment of consciousness >30 minutes
- Loss of consciousness >5 minutes
- Persistent vomiting or increasing headache post injury
- Any convulsive movements
- More than one episode of concussive injury in a session
- Where assessment is difficult (for example, in an intoxicated patient)
- Children with head injuries
- High risk patients (for example, haemophilia or anticoagulant use)
- Inadequate post injury supervision
- High risk injury mechanism (for example, high velocity impact)

approach is to advocate a mandatory arbitrary exclusion period from sport, this strategy has been rejected by the Vienna Expert Consensus Group.³ Use of individualised neuropsychological testing in conjunction with clinical assessment currently is the recommended basis for return to play. Where any doubt exists, clinical judgment should prevail.

Neuropsychological testing to determine recovery and guide return to play is increasingly accepted worldwide. In Australian and American football, such strategies have been used since 1985. More recently, professional horse racing, ice hockey, and a number of other sports have followed similar strategies. Post-injury tests usually are compared with a player's baseline or preseason performance. The most important conceptual point is the understanding that these tests are not designed to be used as a diagnostic test for concussion in acute situations. In practice, the test battery is performed once all post-concussive symptoms have resolved, as a means of objectively measuring return to baseline level of function. Although these are not yet in widespread use, they may provide a simple aid for medical practitioners to objectively measure recovery from concussion.

Specific post-concussion risks

Second impact syndrome—Diffuse cerebral oedema is a rare but well recognised complication of mild traumatic brain injury in sport that occurs predominantly in children and teenagers. The impact, however trivial, sets in train the rapid development of cerebral swelling that usually results in brainstem herniation and death. Its cause is unknown, but it is thought to involve disordered cerebral vascular autoregulation.

Concussive or impact convulsions—Concussive convulsions in collision sport are an uncommon but dramatic association with minor head injury. They are thought to represent a reflex phenomenon and are not associated with structural brain injury. From a clinical standpoint, late seizures do not occur, antiepileptic therapy is not indicated, and prohibition from collision sport is unwarranted. The treating doctor can reassure the patient that concussive convulsions are benign, and the overall management should centre on appropriate treatment of the concussive injury itself.

Prevention of concussion

Concussive brain injury may be minimised in sport by relatively few methods. The brain is not an organ that can be conditioned to withstand injury. Thus, extrinsic mechanisms of injury prevention must be sought.

Helmets have been proposed as a means of protecting the head and theoretically reducing the risk of brain injury. In sports in which there are high speed collisions or that have the potential for missile injuries (for example, in baseball) or falls onto hard surfaces, published evidence shows the effectiveness of sport specific helmets in reducing head injuries, particularly skull fractures.² For other sports, such as football and rugby, no sport specific helmet has been shown to be of proven benefit in reducing rates of head injury. Some believe that the use of protective equipment may alter playing behaviour deleteriously, so that the athlete actually has an increased risk of brain injury. This is particularly an issue with children and adolescents who may adopt risk taking behaviour when wearing protective equipment.

Although the use of correctly fitting mouth guards can reduce the rate of dental, orofacial and mandibular injuries, evidence that they reduce cerebral injuries largely is theoretical. Clinical evidence for a beneficial effect in reducing concussion rates has not been shown scientifically.²

Guiding policy for return to sport

- Until **completely** symptom free, concussed athletes should not resume any training or competition
- This should be assessed initially at rest and then after a provocative exercise challenge
- This is recommended to be aerobic exercise, and the athlete should exercise until their heart rate reaches 80% maximum predicted heart rate
- Once the acute concussive symptoms resolve at rest and exercise, a graduated plan of return to low level aerobic training, followed by non-contact drills and finally contact play will allow close monitoring of the development of any adverse symptoms
- Persisting or newly developing symptoms need further follow up and detailed medical evaluation

Although repeated concussive injuries have been proposed as the basis for second impact syndrome, the evidence is not compelling. More likely is that a single impact of any severity may result in this rare complication, but that participation in sport draws attention to incidental concussive injuries

In players who have concussive convulsions, the universally good outcome and absence of structural injury or long term neuropsychological damage reflects the benign nature of these episodes



In sports (such as American football) where there are high speed collisions or falls onto hard surfaces, sport specific helmets can reduce the number of head injuries

Possible methods for preventing concussion

- Helmets
- Mouth guards
- Rule changes and enforcement
- Neck muscle conditioning

Consideration of rule changes (for example, no head checking in ice hockey) and rule enforcement to reduce the head injury rate may be appropriate when a clear cut mechanism is implicated in a particular sport. For most sports, however, head injuries are an accidental by product of normal play, so rule changes and rule enforcement have little effect on rates of head injuries. Nevertheless, the promotion of fair play and respect for opponents are ethical values that should be encouraged in all sports and by sporting associations.

Neck muscle conditioning may be of value in reducing impact forces transmitted to the brain. Biomechanical concepts dictate that the energy from an impacting object is dispersed over the greater mass of an athlete if the head is held rigidly. Although attractive from a theoretical standpoint, little scientific evidence shows the effectiveness of such measures.

Traumatic intracerebral haematoma and contusion

Traumatic intracerebral haematomas are divided into acute and delayed types. Clinical signs and symptoms depend on the size and location of the intracerebral haematoma, as well as the speed of development. In most cases, at least a brief period of confusion or loss of consciousness is reported. Only one third of patients remain lucid throughout their course. Overall cognitive impairment and the speed and quality of recovery are related strongly to coexistent cerebral injury. An intracerebral haematoma that occurs in isolation with a volume <30 ml is compatible with a favourable recovery. Overall, death rates are in the range of 25% to 30%. Medical management of intracerebral haematomas is directed primarily at reducing post-traumatic oedema and cerebral ischaemia.

Subdural haematoma

Subdural haematomas can be the result of non-penetrating or penetrating trauma to the head. In both cases, extravasation of blood into the subdural space is the mechanism for haematoma formation because of stretching and subsequent rupture of bridging cerebral veins. These injuries are typically seen after falls onto hard surfaces or assaults with non-deformable objects rather than low velocity injuries. Clinical signs and symptoms depend on the size and location of the subdural haematoma, as well as the speed of development. In most cases, at least a brief period of confusion or loss of consciousness is reported. Soft tissue injuries usually are seen at the site of impact. Enlargement of the haematoma, or an increase in oedema surrounding it, produces an additional mass effect. Often, the impact produces a coexisting severe brain injury that explains, in large part, the poor outcome of acute subdural haematoma.

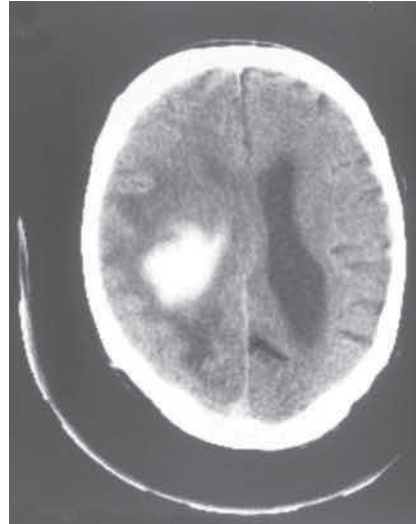
Extradural haematoma

Irrespective of the nature of inciting trauma, a direct blow to the head is essential for extradural haematoma formation. As the skull is deformed by the impact and the adherent dura forcefully detached, haemorrhage may occur into the preformed extradural space. The source of bleeding may be arterial or venous, or both. Haemorrhage from a fracture line may also accumulate to create a mass lesion in the extradural space. The expanding extradural lesion only partially accounts for the neurological morbidity observed with extradural haematomas. Coincident intradural pathology is encountered in up to 50% of cases. In general, sequelae of these lesions dictate the degree of residual functional impairment in patients who survive extradural haematoma. The clinical variability associated with extradural haemorrhage is remarkable. Rarely, extradural haematomas may be asymptomatic; most, however, present with non-specific signs and symptoms referable to an intracranial mass lesion. Alteration in consciousness is a

Education of players, parents, coaches, and other staff plays an important part in ensuring that fair play and respect for opponents are implemented on the field of play

Traumatic intracerebral haematoma

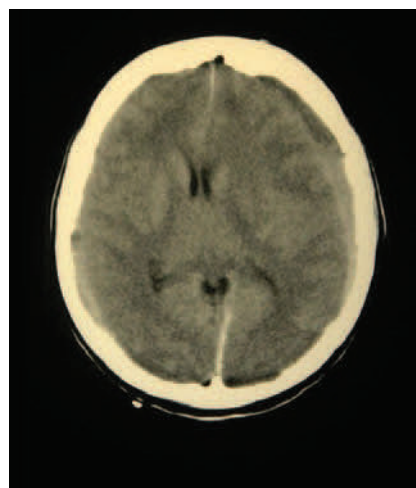
- Acute—occur at the time of the initial head injury
- Delayed—reported to occur as early as six hours after injury to as long as several weeks



Computed tomogram of intracranial bleed

Signs of substantial head trauma that can result in subdural haematomas

- Periorbital and postauricular ecchymoses
- Haemotympanum
- Cerebrospinal fluid otorrhoea or rhinorrhoea
- Facial fractures



Computed tomogram of subdural haematoma

In the supratentorial compartment, haemorrhage from the middle meningeal artery contributes to at least 50% of extradural haematomas; bleeding from the middle meningeal veins accounts for an additional 33%

hallmark manifestation of extradural haematoma. The so-called “lucid interval” occurs in less than one third of patients, and is not a sensitive diagnostic discriminator. The natural course of large traumatic extradural haematomas is dismal if the lesion is unrecognised or untreated. In most cases, the expanding mass lesion precipitates progressive neurological dysfunction. Rapid diagnosis and prompt surgical evacuation afford the best chance for optimising outcome.

Traumatic subarachnoid haemorrhage

Traumatic subarachnoid haemorrhage usually is a consequence of vertebral artery injury—a tear or dissection—although it also may be due to tearing of meningeal vessels. Subarachnoid bleeding typically presents with florid meningeal symptoms such as headache, neck stiffness, and photophobia. The most common initial symptoms in vertebral artery injury are neck pain and occipital headache that may precede the onset of neurological symptoms from a few seconds to several weeks.

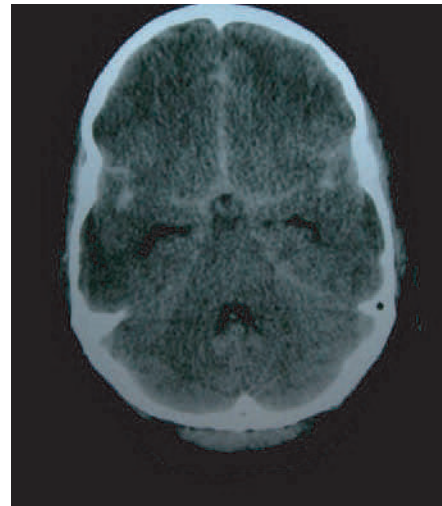
Headache symptoms have been noted in most cases to be ipsilateral to the vascular injury and the pain usually radiates to the temporal region, frontal area, eye, or ear. None of the reported cases had cervical tenderness or objective restriction of neck movement, although subjective exacerbation of pain did occur with neck movement. The outcome of such injuries often is poor.

Diffuse cerebral oedema

The injured brain has long been known to swell in the cranial cavity. The increase in intracerebral volume—whatever its cause or nature—eventually will be associated with an increase in intracranial pressure. In the first few hours and days after a severe head injury, intracranial pressure often is raised because of alterations in the volume of the cerebrovascular bed, whereas brain swelling after this time is because of an increase in water content of the brain tissue. Downward displacement of the cerebrum because of increased intracranial pressure results in compression of the midbrain through the tentorial notch or coning. Treatment for elevated intracranial pressure because of oedema relies on several methods; however, the outcome generally is poor and reflects the initiating cause of the oedema, as well as the variable response to treatment.



Computed tomogram of an acute extradural haematoma



Computed tomogram of a subarachnoid haemorrhage

Management of traumatic brain injury

Acute management

The management of an acute brain injury largely follows the approach for concussion. The so-called primary and secondary survey of injuries, although popular in the literature about trauma, does not reflect current practice in sports medicine.

Vital signs must be recorded after an injury. Abnormalities may reflect brain stem dysfunction. Although head injury produces several types of respiratory patterns, an acute rise in intracranial pressure with central herniation usually manifests rising blood pressure and falling pulse rate (the Cushing response). Hypotension rarely is because of brain injury, except as a terminal event, and alternate sources for the drop in blood pressure should be aggressively sought and treated. This point cannot be underestimated, as cerebral hypotension and hypoxia are the main determinants of outcome after brain injury and are easily treatable.

A neurological examination should be performed, including measurement of the score on the Glasgow coma scale. Findings of this examination must be recorded, so that an overall trend in improving or deteriorating mental function can be documented clearly and objectively. In addition, palpation of the skull, which is quick and simple, should be a component of every physical examination in patients with head trauma.

Anecdotal evidence shows that massive traumatic cerebral oedema, documented by computed tomography, occurs within 20 minutes of cerebral injury

The history of the injury often gives important clues to its nature. This often needs an eyewitness account; in the case of professional sport, videotape analysis may be available

The importance of the initial neurological examination is that it serves as a reference against which other serial neurological examinations may be compared

When time permits, a more thorough physical examination should be performed to exclude coexistent injuries or detect signs of skull injury. Restlessness is a frequent accompaniment of brain injury or cerebral hypoxia, and it may be confused with a belligerent patient who is presumably intoxicated. If the patient is unconscious but restless, attention should be given to the possibility of increased cerebral hypoxia, a distended bladder, painful wounds, or tight casts. Only these are definitely ruled out should drug treatment be considered.

Investigations in head trauma

A number of indications exist for emergent cranial computed tomography in the initial evaluation of patients with head injuries. Evaluation by computed tomography should start as soon as the patient is haemodynamically stable and once immediately life threatening injuries have been dealt with. As the incidence of delayed formation of extradural haematoma after head trauma is substantial, any deterioration in the neurological examination warrants prompt evaluation by computed tomography, even if a previous study was normal.

The role of magnetic resonance imaging in the evaluation of acute head trauma is limited. Compared with computed tomography, magnetic resonance imaging is time consuming, expensive, and less sensitive to acute haemorrhage and associated bony injury. Moreover, access to critically ill patients is restricted during lengthy periods of image acquisition, and the strong magnetic fields generated by the scanner need the use of non-ferromagnetic resuscitative equipment.

In the initial assessment of patients with head injuries, other more traditional diagnostic tools have largely been supplanted by computed tomography of the cranium. Plain skull radiographs are inexpensive and easily obtained, and often demonstrate fractures in patients with extradural haemorrhage. However, the predictive value of such films is poor, as one finding is not requisite for the other. Lateral shift of the pineal gland, indicative of hemispheric mass effect, is a non-specific and highly variable finding.

Management of acute post-traumatic seizures

As described previously, impact seizures or convulsive convulsions are rare but well recognised sequelae of head impacts. These occur within seconds of injury, are not epileptic, and require no specific management beyond the treatment of the underlying concussive injury.

In contrast, post-traumatic epilepsy may occur and is more common as the severity of brain injury increases. A convulsing patient is at increased risk of hypoxia, with resultant exacerbation of the underlying brain injury. Maintenance of cerebral oxygenation and perfusion pressure (blood pressure) is critical in the management of such patients.

Non-brain head injury

As well as the various brain injuries described above, sports doctors should be familiar with the various soft tissue, bony, ocular, and other injuries that may occur to the head. Soft tissue injuries (such as contusions) and sense organ injuries (such as eye injuries) are discussed in chapters 5 and 6.

Scalp wounds, although dramatic in appearance, usually heal well if good wound management is followed. Blood loss from scalp wounds may be extensive, particularly in children, but it rarely causes shock. In the case of severed large vessels (for example, superficial temporal artery), the arterial bleeder should be located, clamped, and ligated. The wound always should be inspected carefully and inside the laceration should

Indications for emergent neuroimaging

- History of loss of consciousness
 - Depressed level of consciousness
 - Focal neurological deficit
 - Deteriorating neurological status
 - Skull fracture
 - Progressive or severe headache
 - Persistent nausea or vomiting
 - Post-traumatic seizure
 - Mechanism of injury that suggests high risk of intracranial haemorrhage
 - Examination obscured by alcohol, drugs, metabolic derangement, or post-ictal state
 - Patient inaccessibility for serial neurological examinations
 - Coagulopathy and other high risk medical conditions
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Computed tomogram showing brain swelling

Magnetic resonance imaging is best suited to defining associated parenchymal injuries after the acute event

Drugs used to treat post-traumatic epilepsy

- Intravenous phenytoin (or fosphenytoin) usually is the drug of choice in this situation, because a loading dose can be administered intravenously to rapidly achieve therapeutic concentrations and because phenytoin does not impair consciousness
 - Benzodiazepines (such as lorazepam and clonazepam) can be used for acute treatment of post-traumatic seizures, but they will produce at least transient impairment of consciousness
 - Phenytoin, none of the benzodiazepines, and no other antiepileptic drug has been shown to be effective for preventing development of post-traumatic epilepsy
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be palpated with a sterile glove for signs of an underlying skull fracture. All cases that involve open fractures or depressed skull fractures should have a neurosurgical consultation. The wound should be irrigated with copious amounts of saline before it is closed. Any debris, including hair, must be removed from the wound. Primary repair should be accomplished with the use of a sterile technique.

The scalp and soft tissues of the head and neck are extremely well vascularised, and this often results in copious bleeding if these structures are injured. The doctor should adopt universal precautions against blood borne infections such as hepatitis B virus, hepatitis C virus, and HIV.

Summary

Traumatic head injury represents one of the most common types of injury in sporting situations. Although most such injuries are mild concussions that recover without long term sequelae, the potential exists for more severe brain injury that may have a catastrophic outcome. Sports medicine doctors should be familiar with the clinical signs and symptoms of such injuries and have a clear, well practiced management algorithm.

The photograph of the racing car crash is courtesy of Getty Images.

Further reading

- Jennett B, Bond M. Assessment of outcome after severe brain damage: a practical scale. *Lancet* 1975;1:480-4
 - Hovda D, Lee S, Smith M, von Stuck S, Bergschneider M, Kelly D, et al. The neurochemical and metabolic cascade following brain injury: moving from animal models to man. *J Neurotrauma* 1995;12:903-6
 - McCrory PR, Berkovic SF. Second impact syndrome. *Neurology* 1998;50:677-83
 - McCrory PR, Bladin PF, Berkovic SF. Retrospective study of concussive convulsions in elite Australian rules and rugby league footballers: phenomenology, aetiology, and outcome. *BMJ* 1997;314:171-4
 - Finch C, McIntosh A, McCrory P. What do under 15 year old schoolboy rugby union players think about protective headgear? *Br J Sports Med* 2001;35:89-95
-

- 1 Jennett B. Epidemiology of head injury. *J Neurol Neurosurg Psych* 1996;60:362-9.
- 2 Johnston K, McCrory P, Mohtadi N, Meeuwisse W. Evidence based review of sport-related concussion—clinical science. *Clin J Sport Med* 2001;11:150-60.
- 3 Aubry M, Cantu R, Dvorak J, Graf-Baumann T, Johnston K, Kelly J et al. Summary and agreement statement of the first International Conference on Concussion in Sport, Vienna 2001. *Br J Sports Med* 2002;36:6-10.
- 4 Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. *Clin J Sport Med* 1995;5:32-5.
- 5 McCreagh M, Kelly J, Randolph C, Kluge J, Bartolic E, Finn G et al. Standardised assessment of concussion (SAC): On site mental status evaluation of the athlete. *J Head Trauma Rehab* 1998;13:27-36.