Head injuries in sport

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Historical perspective

If we define a direct fatality as one occurring directly from participation in the skills of a sport, as opposed to an indirect fatality which is one caused by systemic failure as a result of exertion while participating in a sport, head injury is the most frequent direct cause of death in sport.[1] Furthermore, injury to the head takes on a singular importance when we realise the brain is neither capable of regeneration nor, unlike many other body parts and organs, of transplantation. Every effort must be made to protect the athlete's head as injury can lead to dementia, epilepsy, paralysis, and death.

Starting with President Theodore Roosevelt's threat to ban American football in 1904, injuries from this sport have received more media attention and reports in the medical literature than any other organised sport because none has contributed more fatalities.[2] Starting with the 19 athletes killed or paralysed in 1904 from American football injuries which led to the formation of the National Collegiate Athletic Association (NCAA)[3] as a governing body to establish rules for safer athletic competition, fatalities in American football peaked in 1964 at 30.[4] Between the years 1931 and 1986 at least 819 deaths were directly attributed to American football, most from head injury followed by cervical spinal cord injury.[5] Fatalities in American football from 1973 to 1983 exceeded the deaths in all other competitive sports combined.[2]

Yet, per 100 000 participants American football is not as likely to result in a fatal head injury as horseback riding,[6 7] sky diving,[8 9] or car or motorcycle racing. It has about the same risk of a fatal head injury as gymnastics[10] and ice hockey.[11] Other sports historically shown to have a high rate of head injury includes boxing,[12–14] the martial arts,[15] and rugby football,[16] though a fatal head injury in rugby is rare.[17 18]

Over the last 20 years there has been a dramatic decrease in the most serious head injuries – especially the incidence of subdural haematoma—due to multiple factors including rule changes such as outlawing spear tackling and butt blocking in American football, equipment standards, better conditioning of the neck, and improved on-field medical care. The reduction of the most serious neck injuries, because there presently is no equipment capable of preventing this injury.[19 20]

Injury recognition

Recognition of a head injury is easy if the athlete has loss of consciousness. It is the far more frequent head injuries in which there is no loss of consciousness but rather only a transient loss of alertness that are much more difficult to recognise. More than 90% of all cerebral concussions fall into this most mild category where there has not been a loss of consciousness but rather only a brief period of post-traumatic amnesia or loss of mental alertness.[19] Because the dreaded second impact syndrome can occur after a grade I concussion, just as it can after more serious head injuries, it becomes very important to recognise all grades of concussion.[20 21]

Mechanism of injury

There are three distinct types of stress that can be generated by an acceleration force to the head. The first is compressive; the second is tensile, the opposite of compressive and sometimes called negative pressure; and the third is shearing—a force applied parallel to a surface. Uniform compressive and tensile forces are relatively well tolerated by neural tissue but shearing forces are extremely poorly tolerated.

The cerebrospinal fluid (CSF) that surrounds the brain acts as a protective shock absorber converting focally applied external stress to compressive stress because the fluid follows the contours of the sulci and gyri of the brain and distributes the force in a uniform fashion. The CSF, however, does not totally prevent shearing forces from being transmitted to the brain, especially when rotational forces are being applied to the head. These shearing forces are maximal where rotational gliding is hindered within the brain such as at the dura matter-brain attachments—the rough, irregular surface contacts between the brain and the skull especially prominent in the floor of the frontal and middle fossa.

In understanding how acceleration forces are applied to the brain, it is important to keep in mind Newton's law: force = mass × acceleration, or stated another way, force divided by mass equals acceleration. Therefore, an athlete's head can sustain far greater forces without injury if the neck muscles are

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Table 1 Glasgow coma scale = E + M + V

Eye opening	(E)
Spontaneous	(4)
To speech	(3)
To pain	(2)
No response	(1)
Motor response	(M)
Obeys commands	(6)
Localised pain	(5)
Withdraws from pain	(4)
Decorticate posturing	(3)
Decerebrate posturing	(2)
No response	(1)
Verbal responses	(V)
Oriented	(5)
Confused conversation	(4)
Inappropriate words	(3)
Incomprehensible words	(2)
No response	(1)

coming. In this state, the mass of the head is essentially the mass of the body. In a relaxed state, however, the mass of the head is essentially only its own weight and therefore the same degree of force can impart far greater acceleration.

Injury assessment techniques

In assessing a brain injury, if the athlete is unconsciousness it must be assumed that there has been a neck fracture and the neck must be immobilised. In assessing an athlete with a head injury who is conscious, the level of consciousness or alertness is the most sensitive criterion for both establishing the nature of the head injury and for subsequent follow up. Orientation to person, place, and time should be ascertained. The presence or absence of post-traumatic amnesia, and the ability to retain new information such as the ability to repeat the names of four objects two minutes after having been given them, or the ability to repeat one's assignments with certain plays of the contest should be determined. It is also important to ascertain the presence, absence, and severity of the neurological symptoms such as headaches, lightheadedness, difficulty with balance, coordination, and sensory or motor function. Whereas a complete but brief neurological examination involving cranial nerve, motor, sensory, and reflex testing is appropriate, it is the mental examination and especially the level of consciousness that should be stressed.

Glasgow coma scale

When time permits the use of the Glasgow coma scale (table 1), can be very useful in not only predicting the chances for recovery but also in assessing whether the injured athlete is improving or deteriorating from a given head injury. An initial score of greater than 11 is associated with more than a 90% chance of an essentially complete recovery, whereas an initial score under 5 is associated with more than an 80% chance of death or survival in a vegetative state.

Differential diagnosis

The differential diagnosis with a head injury includes a cerebral concussion, the second syndrome, intracranial haemorrhage, and post-concussion syndrome.

Table 2 Sports with helmets

Ice hockey	0.27
Football	0.25
Men's lacrosse	0.19
Women's softball	0.11

Rate of concussions per 1000 athlete-exposures.[25]

CONCUSSION

Concussion is derived from the Latin *concussus* which means "to shake violently". Initially it was thought to produce only a temporary disturbance of brain function due to neuronal, chemical, or neuroelectrical changes without gross structural damage. We now know that structural damage with loss of brain cells does occur with some concussions. The most common athletic head injury is concussion, with one in five high school American football players suffering one annually.[22] Furthermore, the risk of sustaining a concussion in football is four[22] to six[23] times greater for the player who has sustained a previous concussion. It can occur with direct head trauma in collisions or falls, or may occur without a direct blow to the head when sufficient force is applied to the brain, as in a whiplash injury.[24]

The rates of concussion in some popular sports are listed in tables 2 and 3.[25] Earlier estimates of concussion in American football at all levels put the number at 250 000 per year in this country alone.[26] This number was based on a single survey that found that 20% of high school American football players had sustained some form of concussion[22] and 10% of the college football players sustained concussion in another study.[27] Current estimates of the incidence of concussion in football at all levels suggest that about 100 000 per year may be more accurate (personal communication, Powell J, Medical Sports Systems, Iowa City, Iowa, conducting ongoing surveillance of concussion incidence in NCAA football). No matter whose estimates are used, the dimension of this problem warrants more attention than it has received thus far.

It must be realised that universal agreement on the definition and grading of concussion does not exist.[28–30] This renders the evaluation of epidemiological data extremely difficult. As a neurosurgeon and team physician, I have evaluated many football players who suffered concussion. Most of these injuries were mild and were associated with retrograde amnesia, which is helpful in making the diagnosis, especially in mild cases. I have developed a practical scheme for grading the severity of a concussion based on the duration of unconsciousness and/or posttraumatic amnesia (table 4).[31]

The most mild concussion (grade I) occurs without loss of consciousness and the only neurological deficit is a brief period of confusion or post-traumatic amnesia, by definition lasting less than 30 minutes.

With the moderate (grade II) concussion there is usually a brief period of unconscious-

Table 3 Sports without helmets

Men's soccer	0.25
Women's soccer	0.24
Field hockey	0.20
Wrestling	0.20

Rate of concussions per 1000 athlete-exposures.[25]

Table 4 Severity of concussion

Grade	*Feature*	*Duration of feature*
Grade 1 (mild)	PTA	< 30 minutes
	LOC	None
Grade 2 (moderate)	PTA	> 30 minutes, < 24 hours
	LOC	< 5 minutes
Grade 3 (severe)	PTA	> 24 hours
	LOC	> 5 minutes

PTA, post-traumatic amnesia; LOC, loss of consciousness.

Less commonly, there is no loss of consciousness but only a protracted period of post-traumatic amnesia lasting over 30 minutes but less than 24 hours.

Severe (grade III) concussion occurs with a more protracted period of unconsciousness lasting over five minutes. Rarely, it may occur with a shorter period of unconsciousness, but with a very protracted period of post-traumatic amnesia lasting over 24 hours.

In 1991 Kelly *et al*[28] proposed another guideline regarding the severity of concussion in which the most mild concussion (grade I) had no loss of consciousness and no post-traumatic amnesia, but rather just a brief period of disorientation or confusion. A grade II or moderate concussion was one in which there was no loss of consciousness but post-traumatic amnesia was present. In their guideline all athletes rendered unconsciousness were placed in the grade III, or severe, category. While it can be debated that post-traumatic amnesia of over 24 hours may reflect a more severe brain insult than 30 seconds of unconsciousness, both guidelines will prevent the second impact syndrome, as no athlete still symptomatic from a prior head injury is allowed to return to competition.

Today it is recognised that after concussion the ability to process information may be reduced,[32] and the functional impairment may be greater with repeated concussions.[32 33] Furthermore, these studies suggest that the damaging effects of concussion are cumulative. In proportion to the degree to which the motion of the head is accelerated and to which these forces are imparted to the brain, concussion may produce a shearing injury to nerve fibres and neurones.

The late effects of repeated head trauma of concussive or even subconcussive force leads to anatomical patterns of chronic brain injury with correlating signs and symptoms. Martland[34] first introduced the term "punch drunk" (dementia pugilistica) in 1928. Although first described in boxers, this traumatic encephalopathy may occur in anyone subjected to repeated blows to the head from any cause.

The characteristic symptoms and signs of the punch drunk state (table 5) include the dementia with emotional lability, the victim displaying little insight into his deterioration. Speech and thought become progressively slower. Memory deteriorates considerably. There may be mood swings, intense irritability, and sometimes truculence leading to uninhibited violent behaviour. Simple fatuous cheerfulness is, however, the most common prevailing mood, though sometimes there is depression with paranoia. From the clinical standpoint, the neurologist may encounter almost any combination of pyramidal, extrapyramidal, and cerebellar signs. Tremor and dysarthria are two of the most common findings.

Table 5 Dementia pugilistica: areas of brain damage and resultant deficit

Deficit	*Area*
1. Abnormalities of the septum pellucidum and the adjacent periventricular grey matter	Altered affect and memory
2. Cerebellar scarring and nerve cell loss	Slurred speech, loss of balance and coordination
3. Degeneration of the substantia nigra	Tremor
4. The regional occurrence of neurofibrillary tangles	Loss of intellect

Corsellis *et al*[35] described the necropsy findings in the brains of men who had been boxers. They described a characteristic pattern of cerebral change that appeared not only to be the result of boxing but also to underlie many features of the punch drunk syndrome. They documented changes in the middle of the brain, which may shear into two layers or even be shredded by the distortions that follow blows to the head. They found destruction of the limbic system, a portion of the brain that governs emotion and has a role in memory and learning. There was a characteristic loss of cells from the cerebellum, a part of the brain that governs balance and coordination. Finally, there was an unusual microscopic change widespread throughout the brain resembling changes that occur with Alzheimer disease, which causes progressive loss of intelligence, but sufficiently different (neurofibrillary tangles only and no senile plaques) to be regarded as a distinct entity, unique to subjects suffering from blows to the head.

POSTCONCUSSION SYMPTOMS

A second late effect of concussion is the postconcussion syndrome. This syndrome – consisting of headache (especially with exertion), dizziness, fatigue, irritability, and especially impaired memory and concentration— has been reported in football players, but its true incidence is not known. In my experience it is uncommon. The persistence of these symptoms reflect altered neurotransmitter function and usually correlate with the duration of post-traumatic amnesia.[36] When these symptoms persist, the athlete should be evaluated with a computed tomography (CT) scan and neuropsychiatric tests. Return to competition should be deferred until all symptoms have abated and the diagnostic studies are

Intracranial haemorrhage

The leading cause of death from athletic head injury is intracranial haemorrhage. There are four types of haemorrhage, to which the examining trainer or physician must be alert in every instance of head injury. Because all four types of intracranial haemorrhage may be fatal, a rapid and accurate initial assessment, as well as appropriate follow up, is mandatory after an athletic head injury.

An epidural or extradural haematoma is usually the most rapidly progressing intracranial haematoma. It is frequently associated with a fracture of the temporal bone and results from a tear in one of the arteries supplying the covering (dura) of the brain. The haematoma accumulates inside the skull but outside the covering of the brain. Arising from a torn artery, it may progress quite rapidly and reach a fatal size in 30 to 60 minutes. Although this does not always occur, the athlete may have a lucid interval, that is, initially the athlete may regain consciousness after the head trauma and before starting to experience increasing headache and progressive deterioration in the level of consciousness as the clot accumulates and the intracranial pressure increases. This lesion, if present, will almost always declare itself within an hour or two from the time of injury. Usually the brain substance is free from direct injury; thus, if the clot is promptly removed surgically, full recovery is to be expected. Because this lesion is rapidly and universally fatal if missed, all athletes receiving a head injury must be closely and frequently observed during the ensuing several hours, preferably the next 24 hours. This observation should be done at a facility where full neurosurgical services are immediately available.

A subdural haematoma, the second type of intracranial haemorrhage, occurs between the brain surface and the dura. The acute subdural is the leading direct cause of death in athletes.[37] It is thus located under the dura and directly on the brain. It often results from a torn vein running from the surface of the brain to the dura. It may also result from a torn venous sinus or even a small artery on the surface of the brain. With this injury, there is often associated injury to the brain tissue. If a subdural haematoma needs surgery in the first 24 hours, the mortality is high, not because of the clot itself but because of the associated brain damage. With a subdural haematoma that progresses rapidly, the athlete usually does not regain consciousness and immediate neurosurgical evaluation is obviously required. Occasionally, the brain itself will not be injured, and a subdural haematoma may develop slowly over a period of days to weeks. This chronic subdural haematoma, although often associates with headache, may initially cause a variety of very mild, almost imperceptible mental, motor, or sensory signs and symptoms. Since its recognition and removal will lead to full recovery, it must always be suspected in an athlete who has previously sustained a head injury and who, days or weeks later, is not quite right. A computerised axial tomography scan of the

An intracerebral haematoma is the third type of intracranial haemorrhage seen after head trauma. In this instance, the bleeding is into the brain substance itself, usually from a torn artery. It may also result from the rupture of a congenital vascular lesion such as an aneurysm or arteriovenous malformation. Intracerebral haematomas are not usually associated with a lucid interval and may be rapidly progressive. Death occasionally occurs before the injured athlete can be moved to a hospital. Because of the intense reaction such a tragic event precipitates among fellow athletes, family, students, and even the community at large, and because of the inevitable rumours that follow, it is imperative to obtain a complete necropsy in such an even to clarify the causative factors fully. Often the necropsy will reveal a congenital lesion that may indicate that the cause of death was other than presumed and was ultimately unavoidable. Only by such full, factual elucidation will inappropriate feelings of guilt in fellow athletes, friends, and family be assuaged.

A fourth type of intracranial haemorrhage is subarachnoid, confined to the surface of the brain. Following head trauma, such bleeding is the result of disruption of the tiny surface brain vessels and is analogous to a bruise. As with the intracerebral haematoma, there is often brain swelling, and such a haemorrhage can also result from a ruptured cerebral aneurysm or arteriovenous malformation. Because bleeding is superficial, surgery is not usually required unless a congenital vascular anomaly is present.

Post-traumatic seizure

If a seizure occurs in an athlete with a head injury, it is important to log-roll the patient onto his side. By this manoeuvre, any blood or saliva will roll out of the mouth or nose, and the tongue cannot fall back and obstruct the airway. If one has a padded tongue depressor or oral airway, it can be inserted between the teeth. Under no circumstances should one insert one's fingers into the mouth of an athlete who is having a seizure, as a traumatic amputation can easily result from such an unwise manoeuvre. Usually such a traumatic seizure will last only for a minute or two. The athlete will then relax, and transportation to the nearest medical facility can be effected.

Malignant brain oedema syndrome

This condition is found in athletes in the paediatric age range and consists of rapid neurological deterioration from an alert conscious state to coma and sometimes death, minutes to several hours after head trauma.[38 39] Although this sequence in adults almost always is due to an intracranial clot, in children pathology studies show diffuse brain swelling with little or no brain injury.[39] Rather than true cerebral edema, Langfitt and colleagues[40 41] have shown that the diffuse cerebral swelling is the result of a true hyperaemia or vascular engorgement. Prompt recognition is extremely important because there is little initial brain

outcome is secondary to raised intracranial pressure with herniation. Prompt treatment with intubation, hyperventilation, and osmotic agents has helped to reduce the mortality.[42 43]

Second impact syndrome

RECOGNISING THE SYNDROME

What Saunders and Harbaugh called "the second impact syndrome of catastrophic head injury" in 1984[44] was first described by Schneider in 1973.[45] The syndrome occurs when an athlete who sustains a head injury—often a concussion or worse injury, such as cerebral contusion—sustains a second head injury before symptoms associated with the first have cleared.[20 21 46]

Typically, the athlete suffers postconcussional symptoms after the first head injury. These may include visual, motor, or sensory changes and difficulty with thought and memory processes. Before these symptoms resolve—which may take days or weeks—the athlete returns to competition and receives a second blow to the head.

The second blow may be remarkably minor, perhaps only involving a blow to the chest that jerks the athlete's head and indirectly imparts accelerative forces to the brain. Affected athletes may appear stunned but usually do not lose consciousness and often complete the play. They usually remain on their feet for 15 seconds to a minute or so but seem dazed, like someone suffering from a grade I concussion without loss of consciousness. Often affected athletes remain on the playing field or walk off under their own power.

What happens in the next 15 seconds to several minutes sets this syndrome apart from a concussion or even a subdural haematoma. Usually within seconds to minutes of the second impact, the athlete—conscious yet stunned—quite precipitously collapses to the ground, semicomatose with rapidly dilating pupils, loss of eye movement, and evidence of respiratory failure.

The pathophysiology of second impact syndrome is thought to involve a loss of autoregulation of the brain's blood supply. This loss of autoregulation leads to vascular engorgement within the cranium, which in turn markedly increases intracranial pressure and leads to herniation either of the medial surface (uncus) of the temporal lobe or lobes below the tentorium or of the cerebellar tonsils through the foramen magnum. Animal research has shown that vascular engorgement of the brain after a mild head injury is difficult if not impossible to control.[47 48] The usual time from second impact to brainstem failure is rapid, taking two to five minutes. Once brain herniation and brainstem compromise occur, ocular involvement and respiratory failure precipitously ensue. Demise occurs far more rapidly than usually seen with an epidural haematoma.

Magnetic resonance imaging (MRI) and CT scan are the neuroimaging studies most likely to demonstrate the second impact syndrome. While MRI is the more sensitive to traumatic CT scan is usually adequate to show bleeding or midline shifts of the brain requiring neurosurgical intervention. This is important because CT scanning is cheaper, more widely available, and more quickly performed than MRI.

INCIDENCE

While the precise incidence per 100 000 participants is not known because the precise population at risk in unknown, nonetheless the second impact syndrome is more common than previous reports have suggested. Between 1980 and 1993, the National Center for Catastrophic Sports Injury Research in Chapel Hill, North Carolina, USA, identified 35 probable cases among American football players alone. Necropsy or surgery and MRI findings confirmed 17 of these cases. An additional 18 cases, though not conclusively documented with necropsy findings, most probably are cases of second impact syndrome. Careful scrutiny excluded this diagnosis in 22 of 57 cases originally suspected.[20]

Second impact syndrome is not confined to American football players. Head injury reports of athletes in other sports almost certainly represent the syndrome but do not label it as such. Fekete, for example, described a 16 year old high school hockey player who fell during a game, striking the back of his head on the ice.[11] The boy lost consciousness and afterward complained of unsteadiness and headaches. While playing in the next game four days later, he was checked forcibly and again fell striking his left temple on the ice. His pupils rapidly became fixed and dilated, and he died within two hours while in transit to a neurosurgical facility. Necropsy revealed contusions of several days' duration, an oedematous brain with a thin layer of subdural and subarachnoid haemorrhage, and bilateral herniation of the cerebellar tonsils into the foramen magnum. Though Fekete did not use the label "second impact syndrome", the clinical course and necropsy findings in this case are consistent with the syndrome.

Other cases include an 18 year old male downhill skier described by McQuillen *et al*,[46] who remains in a persistent vegetative state, and a 17 year old football player described by Kelly *et al* who died.[28] Such cases indicate that the brain is vulnerable to accelerative forces in a variety of contact and collision sports. Therefore, physicians who cover athletic events, especially those in which head trauma is likely, must understand the second impact syndrome and be prepared to initiate emergency treatment.

PREVENTION IS PRIMARY

For a catastrophic condition that has a mortality rate approaching 50% and a morbidity rate nearing 100%, prevention takes on the utmost importance. An athlete who is symptomatic from a head injury *must not* participate in contact or collision sports until all cerebral

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