Second Impact Syndrome

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A controversial term first described by Saunders and Harbaugh[1] in 1984, Second Impact Syndrome (SIS) consists of two events. Typically, it involves an athlete suffering post-concussive symptoms following a head injury.[2] If, within several weeks, the athlete returns to play and sustains a second head injury, diffuse cerebral swelling, brain herniation, and death can occur. SIS can occur with any two events involving head trauma. While rare, it is devastating in that young, healthy patients may die within a few minutes. Emergency physicians should be aware of this syndrome and counsel patients and their parents concerning when to allow an athlete to return to play. Furthermore, we present guidelines for appropriate follow up and evaluation by a specialist when necessary.
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INTRODUCTION

Controversy surrounds Second Impact Syndrome (SIS), a condition so rare that even the frequency of its occurrence is in question. From 1980 to 1993 the National Center for Catastrophic Sports Injury Research in Chapel Hill, NC, identified 35 probable cases among American football players.[2] However, this incidence is called into question by the lack of similar reports from Australian football, despite the high participation rate and a concussive injury rate approximately eight times that of American football. Furthermore, there are no similar reports from the European literature. However, a July 2007 *eMedicine* report summarizing an article in the *American Journal of Sports Medicine* noted:

"A study of American high school and college football players demonstrated 94 catastrophic head injuries (significant intracranial bleeding or edema) over a 13-year period.[3] Of these, only two occurred at the college level. Seventy-one percent of high school players suffering such injuries had a previous concussion in the same season, with 39% playing with residual symptoms."[4]

Ropper and Gorson[5] define concussion as an immediate and transient loss of consciousness accompanied by a brief period of amnesia after a blow to the head. Both anterograde and retrograde amnesia accompany the event.[6] Retrograde amnesia can extend from moments to several days before the head trauma. The extent and period of anterograde memory loss tends to be shorter than retrograde.[5]

SIS is based on rare and mostly disputed cases in which a second mild head injury in children caused massive cerebral edema. McCrory[7] points out that SIS is more likely a condition representing "diffuse cerebral swelling," a consequence of traumatic brain injury with diffuse brain swelling that is well recognized in children. While there is argument over the incidence of SIS, many authors agree that the syndrome is rare.[7,8] While this may be comforting to emergency physicians (EPs), SIS must still be taken seriously as the consequences could be grave.

Epidemiology

The Centers for Disease Control and Prevention estimates about 1.1 million patients with nonfatal traumatic brain injury (TBI) are treated and released from U.S. hospital emergency departments (EDs) annually.[9] An estimated 300,000 TBIs are mild to moderate[10] and some 235,000 require hospitalization.[9] Concussion is not uncommon, affecting about 128 people per 100,000 in the United States yearly. Young children have the highest rates of concussion, with sports and bicycle accidents accounting for the majority of cases in the 5-14 age group. Falls and vehicular accidents are the most common causes of concussion in adults.[5] There is little epidemiological data about SIS. Most of the information comes from case reports or series.[8] The overall incidence of secondary concussion and hence SIS is unknown. One reason for the lack of systematic

epidemiological data comes from the controversy regarding the definition of SIS.[4,5]

Concussion

The word "concussion" comes from the Latin verb *concutere* ("to shake violently").[11] A concussion is defined as an immediate and transient loss of consciousness (LOC) accompanied by a brief period of amnesia after a blow to the head. In post-concussive syndrome, up to three symptoms arise within less than four weeks from the initial LOC.[5] These include headache, dizziness, fatigue, insomnia, irritability and alcohol intolerance, and other symptoms, such as subjective concentration, memory, or intellectual difficulties without neuropsychological evidence of marked impairment.[5] In an article summarizing the Second International Conference on Concussion in Sport (Prague 2004), McCrory et al.[12] differentiate between simple and complex concussions. A simple concussion is an athletic injury that resolves without complications within ten days, whereas a complex one involves persistent symptoms beyond ten days, or additional symptoms of seizures, cognitive impairment or exertional headache or confusion.

The extent of concussive amnesia roughly correlates with the duration of loss of consciousness and the severity of the head injury. Anterograde amnesia is the inability to retain new information. Retrograde amnesia is the inability to remember events preceding a traumatic injury. In some rare cases amnesia can extend backward for several days or longer.[5] Content experts agree that all concussions mandate evaluation by a physician.[12] Repeated brain injuries, including concussions occurring over an extended period of weeks to years, can result in neurologic and cognitive deficit, especially in boxers.[10,13]

Cognitive domain scores are often calculated from executive, attention and memory testing. Newer high technology methods include Diffusion Tensor Imaging (DTI), which is a more recent method of assessing axonal integrity in vivo. DTI characterizes white matter integrity.[14] In a review of 18 cases of young athletes who sustained a second head injury before the first one had resolved Mori et al.[8] found, that ten of the 18 did not lose consciousness. However, after the second traumatic brain injury, eight had a cranial CT scan revealing a subdural hematoma.[8] The symptoms and ED presentation of severe concussion can be identical to SIS. The EP is rarely aware of a preceding concussion days to weeks earlier. Therefore, the evaluation and treatment of these two entities follow the same algorithm.

Pathophysiology of Second Impact Syndrome

A patient who sustains an initial concussion may develop cerebral edema, accounting for loss of consciousness, memory impairment, disorientation and headache. However, the brain's auto regulatory mechanisms compensate for this mechanical and physiologic stress and protect against massive swelling. This is thought to be accomplished by acutely limiting cerebral blood flow, which leads to accumulation of lactate and intracellular acidosis.[15] After the initial phase, a state of altered cerebral metabolism occurs and may last ten days,[16] involving decreased protein synthesis and reduced oxidative capacity.[11] Extensive experimental research suggests that the loss of consciousness after head injuries, the development of secondary brain damage, and the enhanced vulnerability of the brain after an initial insult can be explained largely by characteristic ionic fluxes, acute metabolic changes, and cerebral blood flow alterations that occur immediately after cerebral concussions. Extracellular potassium concentration can increase massively in the brain after concussion, followed by hypermetabolism lasting up to ten days. This makes the brain more vulnerable and susceptible to death after a second sub-lethal insult of even less intensity.[11] Fisher and Vaca[17] hence conclude that when the patient sustains a "second impact," the brain loses its ability to auto regulate intracranial and cerebral perfusion pressures. In severe cases, this may lead to cerebral edema followed by brain herniation. Death has been reported to occur in a matter of two to five minutes, usually without time to stabilize or transport an athlete from the playing field to the ED. This demise can occur far more rapidly than that of an epidural hematoma.[18] Bruce et al.[19] point our that brain swelling in minor head trauma is more significant in small children than in adults. The term "malignant brain edema" has been used to describe this phenomenon. More research in this area is necessary to determine if and when malignant brain edema and SIS are related, or even if they occur by the same process.

Table 1. Data-driven Cantu[4] grading system for concussion

Grade 1 (mild) No loss of consciousness; post-traumatic amnesia* or post-concussion signs or symptoms lasting less than 30 minutes
Grade 2 (moderate) Loss of consciousness lasting less than 1 minute; Post-traumatic amnesia* or post-concussion signs or symptoms lasting longer than 30 minutes but less than 24 hours
Grade 3 (severe) Loss of consciousness lasting more than 1 minute or post-traumatic amnesia* lasting longer than 24 hours; Post-concussion signs or symptoms lasting longer than 7 days

* **Retrograde and anterograde**

Clinical Workup Following Head Trauma

Airway, breathing, and circulation must be assessed and treated following any head trauma seen in the ED

Table 2. Guidelines for the Management of Sport-Related Concussion. These guidelines reflect the latest consensus opinion and are not evidence based. Adapted from the American Academy of Neurology guidelines[24] where newer guidelines are expected to be published in the future. (http://www.aan.com)

Symptoms	First Concussion	Second Concussion
Grade 1: No loss of consciousness, transient confusion, resolution of symptoms and mental abnormalities in <15 min See also Appendix.	Remove from play. Examine at 5-min intervals. May return to play if symptoms disappear and results of mental-function exam return to normal within 15 min	Allow return to play after 1 week if there are no symptoms at rest or with exertion.
Grade 2: as above, but with mental symptoms for >15 min	Remove from play and disallow play for rest of day. Examine for signs of intracranial lesion at sidelines and obtain further exam by a trained person the same day. Allow return to play after 1 week if neurological exam is normal.	Allow return to play after 2-week period of no symptoms at rest or with exertion. Remove from play for season if imaging shows abnormality.
Grade 3: any loss of consciousness	Perform thorough neurological exam in hospital and obtain imaging studies when indicated. Assess neurologic status daily until post-concussive symptoms resolve or stabilize. Remove from play for 1 week if LOC lasts seconds; for 2 weeks if it lasts minutes; must be asymptomatic at rest and with exertion to return to play.	Withhold from play until symptoms have been absent for at least 1 month.

LOC, loss of consciousness.

or in the prehospital setting. Even in the absence of hard signs suggesting cervical spine or spinal cord injury, such as posterior midline pain or tenderness, paresthesias, extremity weakness or depressed consciousness, injury should be assumed and immobilization and radiography routine. A thorough history is imperative and must be taken from the patient, if conversant, or from a witness. Important information includes mechanism of injury, loss of consciousness, previous concussions, seizure history, transient weakness or paresthesias, difficulty walking, bladder or bowel incontinence, and alcohol or drug use. A thorough general physical exam should be performed with special attention to the neurological exam.

Diagnostic Imaging

If there is suspicion of a serious structural brain injury, immediate computed tomography (CT) of the head is needed.[20] CT is usually easier to obtain and more sensitive than magnetic resonance imaging (MRI) to detect acute intracranial bleeding and identify surgically reversible injury.[21] The CT scan should be reviewed for intraparenchymal, extra-axial, intraventricular or subarachnoid bleeding, diffuse cerebral swelling manifested by loss of the grey-white junction, and midline shift. In case of an anatomic abnormality, a neurosurgeon should be consulted.

Treatment

As stated above, true SIS involves brain herniation and death within minutes. Therefore, the odds of seeing acute SIS in the ED are unlikely. If suspected, the patient should be immediately stabilized with special emphasis on airway management, and neurosurgery consulted. Cantu[22] also recommends rapid intubation and mannitol to minimize morbidity. This has not been tested and is based on expert opinion. The use of mannitol in the treatment of intracranial pressure (ICP) shows a small beneficial effect. However, there are insufficient data on the effectiveness of prehospital administration of mannitol.[23] In the management of increased ICP the intubated patient should maintain normocapnic at about 30 mm Hg.[24] The recent literature suggests that hypertonic saline is evolving as a real alternative to mannitol or may be used in otherwise refractory intracranial hypertension. Safety data on hypertonic saline in the treatment of intracranial hypertension are very limited, and the efficacy and duration of ICP-lowering are difficult to predict.[25]

Prevention

Any athlete who still shows signs of concussion should not be allowed to return to play. Such signs include fatigue, headache, disorientation, nausea, vomiting, feeling "in a fog" or "slowed down," as well as other differences from a patient's baseline.[18] If there are any doubts about the severity of injury, the patient or athlete should not be allowed to resume play.

The difficulty lies in deciding the appropriate return to play when the athlete is completely asymptomatic. Parents, teachers and the coach must observe the athlete closely.

High school athletes and those with scholarship possibilities, especially, will try to convince parents and coaches that they feel fine, in order to resume play. There are differences of opinion as to when it is appropriate for a post-concussive patient to resume play. Cantu[18] suggests a grading system to rate the severity of concussions, ranging from Grade 1 through Grade 3 (Table 1)**.** Three widely referenced clinical guidelines advise on the timing and level of participation after a first concussion.[3,4,5] Unfortunately, none are evidence based, and their recommendations differ widely. Some even discuss a stepwise approach to return to play. The EP should focus on the initial stabilization, imaging and the exclusion of a life-threatening or surgically-treatable lesion. The EP should also ensure appropriate outpatient follow up by a neurosurgeon or a sports physician who has expertise with concussive symptoms. Although information on SIS is present in the medical literature, its description in 1984 may leave some non-specialists unaware of the potential danger. Currently the guidelines suggested by the American Academy of Neurology[26] listed in Table 2 are the most widely disseminated and used.

The most sensible approach to prevention is to prevent the first concussion. For soccer, Kangaroo Soccer Headgear, similar to that used in martial arts, is primarily intended to provide protection for children and youth players. Coverage extends around the head, including the ears. The headgear is intended to protect against player-to-player impacts.[27] These headgear models did provide measurable benefit during head-to-head impacts, but not necessarily with ball impact. Experts advise wearing a helmet during high-impact contact sports and preventing or mitigating especially head-to-head contact.

CONCLUSION

More meaningful research is needed to investigate SIS. In light of its rarity, research should focus on observational study of the incidence of primary concussion in similar systems that do and do not employ head protection. Research should be aimed at unifying an algorithm for the work up and treatment of concussions and potential predisposition for SIS, as well as developing guidelines for return to play. At this point, given the limited information on this topic, the prevention of high impacts during sport is the goal for young athletes. When in doubt after a severe concussion, the athletes should not resume play. "When in doubt, sit them out."[11]

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Appendix. Testing Guidelines for the Management of Sport-Related Concussion.[24] Testing includes:

- orientation
- repetition of digit strings
- recall of word list at zero and five minutes
- recall of recent game events, recall of current events
- finger-to-nose and tandem-gait tests
- Romberg test
- provocative testing for symptoms with a 40-yard sprint
- five push-ups
- five sit-ups
- five knee bends