



Winter Sports-Associated Traumatic Brain Injuries: Epidemiology and Pathophysiology

David W. Wright, MD

You are volunteering as a team physician at your local community league hockey game. The final game of the season is on the line. JR is a 16-year-old with talent potential for collegiate play. He has been particularly roughed-up during this game taking several hard hits to the face and head. JR approaches the net for a score when two opposing team players hit him from behind and slam him into the boards. He was slightly off balance when his head hit the brick stepwall. He never saw it coming. As he hit the ice his body stiffened. His arms stretched outward for a moment before he roused. Finally, he pulled himself up, shook his head and returned to the bench. No one noticed his difficulties getting up.

The next series, he is helping defend his goal when a group of players charge forward. JR gets slammed into the corner post of the goal along with the goalie. The opposing team scores the goal. When he gets up and skates to the box, he is dazed and has an empty stare. The coach is furious and pulls him aside. You notice a glassy stare as the coach yells at him to pay attention.

You convince the coach to sit the player out for the period. He continues to be dazed. He has no recollection of the score or what the last period entailed. Immediately after the incident, his physical and basic neurological evaluations are normal except for his mini-mental status exam. He is slow to answer, cannot calculate serial sevens, and remembers only 1 word of the five you asked him to remember.

The game is not going well for the home team. Fifteen minutes later the coach is screaming to get him back into play. The player adamantly argues to you that he is fine and feels normal. You decide to reexamine his mini-mental status. He improves his score and is able to remember four out of five words, but still cannot do more than 3 serial sevens and still does not recall any details from the time he was “dinged”.

Key Questions

How big of a problem is sports associated head injury and what is the real impact?

Can sports associated head injury be lethal?

Are there permanent or long-term sequelae of mild head injuries?

How often do head injuries occur?

Who is at highest risk?

Do helmets make a difference?

Background and Significance

How big of a problem is sports associated head injury and what is the real impact?

The problem of sports associated head injury (meaning injury to the brain and/or concussions) has been widely recognized since the early 1900's when President Theodore Roosevelt proclaimed that sporting events, particularly football, were too violent and needed to be made safer.[41] In 1919, there were 53 deaths in football alone. These events lead to the development of the National Collegiate Athletic Association (NCAA) which was charged with regulating sports to enhance the safety of collegiate athletes. However, in the 1960's, there were still 35 deaths attributed to football related injuries, and in 1997 the CDC proclaimed that head injuries in athletes have reached epidemic proportions in the US. [1,41] Despite improved playing conditions (including rules, playing surfaces, education, and equipment), brain injury remains the leading cause of death in sports, accounting for 65-85% of all sports related fatalities. [2, 3] It is estimated that over 800 sports related traumatic brain injuries occur every day in the US, totaling approximately 300,000 per year. [1, 3] Approximately 1 out of 20 athletes will sustain a concussion during play. About 90% of these head injuries are classified as mild, with the other 10 percent moderate and severe.[29] Sixty three thousand mild head injuries occur annually in high school sports alone, [38, 40] and even though classified as mild, these injuries can also result in death or long-term disability.

Can sports associated head injury be lethal?

Intracranial hemorrhage accounts for the majority of lethal brain injuries in sports.[41] Over 800 deaths in boxers have been recorded since 1915. From 1975-1984, there were 132 football players with documented intracranial hemorrhages with an average of 8 deaths per year. More recently, it has been estimated that the number of brain injury related deaths due to football is 5 per year. In high school sports during the period 1982-1996, 374 out of 387 (97%) direct fatalities and catastrophic injuries were associated with football. For high school winter sports (ice hockey, gymnastics, wrestling, swimming, basketball) during the same period, 70 deaths and catastrophic injuries have been reported.[41]

Many of these deaths are due to severe head injuries acquired during play. However, even “mild” head injuries can be devastating and lead to the development of Second Impact Syndrome (SIS). SIS is the most feared consequence of mild TBI and is characterized by rapid neurological demise, brain swelling and death.[27-30, 40] Second impact syndrome was first described by Schneider et al. in 1970. Although the etiology is unknown, young athletes appear to be at much higher risk than their adult counterparts. The majority of victims have been high school athletes between 13 and 18 years of age.[51] The inciting factor appears to be a second impact either during or shortly after an initial mild concussion. Alarming, the second impact may be extremely mild or seemingly inconsequential and may not even be to the head. Approximately 35 deaths have been attributed to SIS in football alone.[41] But SIS is not limited to American football players and has been documented in other sports, including ice hockey and down-hill skiing.[5] Sports associated head injury may have fatal consequences, even in apparently mild cases.

Are there permanent or long-term sequelae of mild head injuries?

Although most athletes who sustain a mild head injury recover without major sequelae, an unknown number continue to exhibit cognitive dysfunction in memory, attention, and information processing for days, weeks, months, and even years after the event. There is also evidence that these “mild” head injuries lead to permanent deficits. In boxers who have numerous bouts or who have suffered repeated concussions, there is evidence of structural damage, atrophy, and chronic encephalopathy.[18-20], [21] It is estimated that over 90% of professional boxers have sustained a brain injury.[22, 23] Some boxers will develop early onset dementia similar to Alzheimer’s disease; a condition known as “punch drunk syndrome” or dementia pugilistica.[24-27] Even when appropriate time is allowed for the brain to heal, repetitive concussions can result in long-term cognitive deficits. [28-30] Studies of football and boxing athletes who have suffered multiple concussions demonstrate a significant decrease in cognitive performance on neuropsychological tests.[18, 19, 31-37] More recently Collins et al. have confirmed the cumulative effect of concussion. Athletes who have reported previous brain injuries (history of 3 or more previous concussions) exhibit worse symptoms immediately after the injury (LOC, amnesia, confusion) than those who have not previously suffered a head injury.[38]

How often do head injuries occur?

Football is associated with the greatest number of catastrophic injuries compared to all other organized competitive sports; however, both gymnastics and ice hockey are associated with a greater rate of head injury.[Mueller 2001] Ice hockey is a game of high speeds and frequent collisions where the potential for head injury is high.[4] It has one of the highest rates of concussion of any sport (0.27 concussions per 1000 athlete-exposures reported by Cantu in 1996, 1.57 concussions/1000 athlete-exposures in the Canadian Inter-University Athletics Union hockey season 1997-1998, and 2.24 concussions/1000 exposures during game play in the NCAA 1999-2000 season).[4, 5] Concussion has been reported to occur in up to 7% of all ice hockey players in a single season. In comparison, Cantu reported in 1996 the incidence rate in football as 0.25 per 1000 athlete-exposures. New surveillance data from the 1999-2000 NCAA season reports the football rate of concussion during game play may be as high as 4.5 concussions/1000

athlete-exposures. Although at first glance these rates may not seem high, the impact of injury on healthy young athletes who have many productive years ahead of them can translate into significant long-term personal, as well as societal, costs.

As for recreational sports, down hill skiing has the highest rate of catastrophic head injuries, accounting for 32 deaths per year.[5] Approximately 10 million people participate in skiing each winter.[6] Head injury is the most frequent cause of critical injury and the leading cause of death in ski slope accidents.[7, 8] An actual rate of injury per skier-exposure is difficult to calculate and not available for comparison with the organized sports, but in 1997 the number of skiing related head injuries was estimated at 12,700 for the year. Strohecker et al. reported 33 skiers with severe head injury that were admitted to one hospital during a single season.[9] Several high profile skiing deaths, including Sonny Bono, have highlighted the risk of skiing associated TBI.

The number of snowboarding head injuries have increased dramatically with participation from an estimated 1,000 in 1993 to 5,200 in 1997. Beginning snowboarders have a higher incidence of head injury than beginning skiers; lending to the idea that snowboarders at all levels may be at greater risk for head injury than similarly experienced skiers. In one study, the rate of head injury severe enough to prompt an emergency department visit was 6.5 per 100,000 skiers (143 cases / 2years).[10] The number of mild head injuries that did not seek medical attention and were therefore unreported is unknown, but is likely to be very high. Other recreational and competitive winter sports such as tobogganing, acrobatic freestyle skiing, and tubing also account for a significant number of head injuries.[11-15] Overall, head injuries represent about 14% of all skiing and snowboarding injuries for adults and about 22% for children (<16 y.o.).

The overall financial toll of sports associated head injury is unknown. The estimated cost for all types of TBI in the US is 56 billion dollars annually.[CDC] There are a few published cost estimates for individual sports, for instance the estimated per-patient costs recorded from one hospital for snowboarding head injuries were \$46,000. The cost to the athlete in terms of lost game time is also unreported in the literature. Although athletes in organized sports are often held from play for presumed head injuries, no detailed data exists on how often they are pulled out or how long they are withheld from play. There have been many publications in the popular press highlighting incidence of head injuries in professional athletes. A number of professional athletes have ended their careers as a result of head injuries, but an exact figure on how many is illusive. The cost to athletes, families, and society in terms of long-term morbidity (permanent or long lasting cognitive deficits, recurrent symptoms, and post concussive syndrome) are also unknown but are likely to be high given the reported incidence of head injury.

Who is at highest risk?

As mentioned above, the highest number of head injuries in organized sports is associated with football, but other organized (ice hockey, gymnastics, soccer) and recreational sports (snow boarding, down hill skiing) also have a high incidence of head injury. Fortunately, in both football and ice hockey, mandatory helmet rules have significantly reduced the number of serious injuries. However, helmet use has not caught on in gymnastics, skiing, snowboarding, and many other sports (see next section).

In addition to the type of sport increasing the risk of injury, younger athletes appear to be at an increased risk for injury and tend to have more serious sequelae. Teenage athletes also have an apparent increased vulnerability to second impact syndrome, a catastrophic head injury that rapidly leads to death. The problem of head injury in young athletes and risk of SIS is exacerbated by the fact that many of these younger athletes are unsupervised or have no access to sideline medical evaluations. In addition, athletes represent the only class of individuals who want, and often demand, to return to play despite just sustaining a head injury. Recent data shows that even with Grade 1 concussions or “Dings”, young athletes have cognitive deficits that do not return to normal for at least 6 days; while impaired cognitively, the athletes remain at increased risk for second impact syndrome or other substantial consequences.[52] At present, it is unknown what neurological and physiological properties put the young brain at an increased risk for injury and morbidity. In addition to physiological vulnerability the young athlete may also practice more risky behaviors and poorer technique due to inexperience with the sport or activity. Combined, these factors place younger athletes at considerable risk for head injuries.

Athletes who have a history of a head injury are also at an increased risk for recurrent injury. There appears to be a six times greater risk for repeat concussion in athletes that have suffered at least one in the past.[39] Collins et al. has shown the cumulative effect of concussion, in that those who have reported previous brain injuries (history of 3 or more previous concussions) exhibit worse symptoms immediately after the injury (LOC, amnesia, confusion) than those who have not had a concussion in the past.[38] Therefore, athletes with a history of concussion are also at an increased risk.

Do helmets make a difference?

The U.S. Consumer Product Safety Commission conducted a special investigation and determined that at least 44% (or ~7,700 annually) of skiing and snowboarding head injuries could be prevented with a helmet. This report also states that for children under 15 years old, 53% of head injuries (2600/4,950 head injuries per year) would have been addressed with helmet use. A survey of the availability and rental practices of helmets at ski resorts showed in 1998-1999 that 19 out of 26 ski resorts surveyed had helmets for rent, but helmets were not considered part of the standard rental package.[16] The survey showed that while 2-38% of skiers and snowboarders rented equipment, less than 1%-8.6% rented helmets.[16] In one study, of the 1,214 patients admitted to a ski resort local hospital, only 3 or .25% reported wearing a helmet.[8] In the subgroup of head injures (350/1,214), only one was wearing a helmet.[8]

Helmet use in football demonstrates the positive impact that helmets can have in contact and high-risk sports. The 1976 prohibition of the use of the head for blocking and tackling and the 1978 (1980 for high schools) NOCSAE football helmet standard have dramatically reduced the frequency of brain and cervical spine injury fatalities in football (Table). [17]

Head and Cervical Spine Fatalities for Collegiate Football [17]

Year	Head Injury		Cervical Spine	
	Frequency	Percent	Frequency	Percent
1945-1954	87	17.2	32	27.3
1955-1964	115	22.8	23	19.7
1965-1974	162	32.1	42	35.9
1975-1984	69	13.7	14	12.0
1985-1994	33	6.5	5	4.3
1995-2004	39	7.7	1	0.8
TOTALS	505	100.0	117	100.0

Classification and Pathophysiology

Traumatic brain injury is typically divided into three classifications, (mild, moderate, and severe) depending on the degree of mental depression, as scored by the Glasgow Coma Score (GCS).[42] The most widely recognized definition of concussion, one of the key components of mTBI, is a trauma-induced alteration in mental status. The hallmarks of a concussion are confusion and amnesia.[43] Probably the most detrimental misconception that continues to linger with regards to a concussion is that it must include loss of consciousness (LOC) to be significant. Most mTBIs do not have LOC, and making management decisions based on this fact alone can be dangerous.

The classic pathophysiology of TBI is divided into two parts: primary and secondary injury. The mechanical forces applied to the head and brain and the subsequent injuries associated with the movement of the brain within the cranium make up the primary injury. Preventive measures, such as helmet use, common sense rules of engagement, and other preventative measures can have a significant impact on injury occurrence and severity. Secondary injury refers to two separate categories of events: those such as hypoxemia and hypotension that occur after the injury and left unabated cause a significant increase in morbidity and mortality, and those that occur as a result of a complex array of deleterious biochemical events during the natural progression of tissue injury (i.e. secondary cascade).[53-56] This secondary cascade is set off by the resultant shock wave, surrounding necrotic or dying tissue, and tissue irritants such as extravascular blood.[55, 56] The cascade is initiated by the release of supra-physiological levels of glutamate and kinate into the extracellular and interneuronal spaces. The subsequent activation of ion channels through the NMDA, AMPA, and a variety of other receptors, causes a massive influx of calcium and subsequent ionic destabilization of neurons and glia.[55, 56] The calcium influx activates a number of enzymatic pathways that eventually lead to cell death. The mitochondria of the neuron seem particularly vulnerable to this influx and may be one of the main pathways to cell necrosis and cell suicide (apoptosis).[57] This cascade of events occurs over seconds to days but may be active weeks after the injury. The consequence of this cascade leads to further neuronal loss, expansion of the original injury, and a worsening of the long-term outcome of the patient. How much of this cascade occurs in mTBI and simple concussions is unknown. The etiology and pathophysiology of Second Impact Syndrome (SIS) is not entirely clear. It is thought to involve the loss of neurovascular autoregulation, vascular engorgement, increase blood brain barrier permeability, edema, and uncontrolled intracranial hypertension.[41] The vascular engorgement and edema result in increased pressure within the cranium leading to

herniation and death.[41] The consequences can be rapid, occurring as soon as 2-5 minutes after the second impact or insult.

For mTBI, the lack of obvious signs of injury to the head and brain are of little comfort. Clearly, many individuals continue to have deficits and disabilities after mild head injury. The reason why one individual is likely to recover and another not is unclear and not predictable. Further studies into the basic pathophysiology of minor TBI and mild concussions are needed.

References

1. *Sports-related recurrent brain injuries--United States*. MMWR - Morbidity & Mortality Weekly Report, 1997. 46(10): p. 224-7.
2. Kelly, J.P. and R.C. Savage, *Concussion in Sports and Return to School Issues Following Concussion*. Brain Injury Source Pediatric Issue, 1999. 3(Summer).
3. Kelly, J.P., *Concussion in sports and recreation*. Seminars in Neurology, 2000. 20(2): p. 165-71.
4. Benson, B.W., et al., *Head and neck injuries among ice hockey players wearing full face shields vs half face shields*. Jama, 1999. 282(24): p. 2328-32.
5. Cantu, R.C., *Head injuries in sports*. British Journal of Sports Medicine, 1996. 30: p. 239-289.
6. Lehman, L.B., *Neurologic injuries from winter sporting accidents. How they happen and how to minimize them*. Postgraduate Medicine, 1986. 80(8): p. 88, 93, 96-8.
7. Furrer, M., et al., *Severe skiing injuries: a retrospective analysis of 361 patients including mechanism of trauma, severity of injury, and mortality*. Journal of Trauma-Injury Infection & Critical Care, 1995. 39(4): p. 737-41.
8. Levy, A.S., et al., *An analysis of head injuries among skiers and snowboarders*. Journal of Trauma-Injury Infection & Critical Care, 2002. 53(4): p. 695-704.
9. Strohecker, J., et al., *Schadel-Hirnverletzungen bei Skifahrern*. Wiener Medizinische Wochenschrift, 1984. 134(1): p. 11-3.
10. Nakaguchi, H., et al., *Snowboard head injury: prospective study in Chino, Nagano, for two seasons from 1995 to 1997.[comment]*. Journal of Trauma-Injury Infection & Critical Care, 1999. 46(6): p. 1066-9.
11. Clancy, W.G., Jr., *Cross-country ski injuries*. Clinics in Sports Medicine, 1982. 1(2): p. 333-8.
12. Ellison, A.E., *Skiing injuries*. JAMA, 1973. 223(8): p. 917-9.
13. Johnson, R.J., et al., *Trends in skiing injuries. Analysis of a 6-year study (1972 to 1978)*. American Journal of Sports Medicine, 1980. 8(2): p. 106-13.
14. Murray, J.J., *Pediatric aspects of Nordic skiing*. Pediatric Clinics of North America, 1982. 29(6): p. 1423-9.
15. Scharplatz, D., K. Thurleman, and F. Enderlin, *Thoracoabdominal trauma in ski accidents*. Injury, 1978. 10(2): p. 86-91.
16. Hennessey, T., et al., *Helmet availability at skiing and snowboarding rental shops. a survey of Colorado ski resort rental practices*. American Journal of Preventive Medicine, 2002. 22(2): p. 110-2.
17. Mueller, F.O. and R. Cantu, *Nineteenth Annual Report Fall 1982 - Spring 2001*. 2002, National Center for Catastrophic Sport Injury Research, University of North Carolina: Chapel Hill. p. 1-24.
18. Bogdanoff, B. and H.M. Natter, *Incidence of cavum septum pellucidum in adults: a sign of boxer's encephalopathy*. Neurology, 1989. 39(7): p. 991-2.
19. Casson, I.R., et al., *Neurological and CT evaluation of knocked-out boxers*. Journal of Neurology, Neurosurgery & Psychiatry, 1982. 45(2): p. 170-4.
20. Lampert, P.W. and J.M. Hardman, *Morphological changes in brains of boxers*. Jama, 1984. 251(20): p. 2676-9.

21. Sironi, V.A., et al., *CT-scan and EEG findings in professional pugilists: early detection of cerebral atrophy in young boxers*. Journal of Neurosurgical Sciences, 1982. 26(3): p. 165-8.
22. Surgeons, A.A.o.N., *Health Resouces*. 1998, Congress of Neurological Surgeons.
23. Brain Injury Association. *Sports and Recreation*. 2001, Brain Injury Association.
24. Friedman, J.H., *Progressive parkinsonism in boxers*. Southern Medical Journal, 1989. 82(5): p. 543-6.
25. Jordan, B.D., *Neurologic aspects of boxing*. Archives of Neurology, 1987. 44(4): p. 453-9.
26. Ryan, A.J., *Intracranial injuries resulting from boxing*. Clinics in Sports Medicine, 1998. 17(1): p. 155-68.
27. Erlanger, D.M., et al., *Neuropsychology of sports-related head injury: Dementia Pugilistica to Post Concussion Syndrome*. Clinical Neuropsychologist, 1999. 13(2): p. 193-209.
28. Kelly, J.P., et al., *Concussion in sports. Guidelines for the prevention of catastrophic outcome*. JAMA, 1991. 266(20): p. 2867-9.
29. Cantu, R.C., *Second-impact syndrome*. Clinics in Sports Medicine, 1998. 17(1): p. 37-44.
30. Cantu, R.C. and R. Voy, *Second-impact syndrome - a risk in any contact sport*. Physician and Sports Medicine, 1995. 23(6): p. 27.
31. Collins, M.W., et al., *Relationship between concussion and neuropsychological performance in college football players*. Jama, 1999. 282(10): p. 964-70.
32. Bowden, S.C. and K.W. Walsh, *Boxing: time for action*. Medical Journal of Australia, 1985. 142(4): p. 282.
33. Casson, I.R., et al., *Brain damage in modern boxers*. Jama, 1984. 251(20): p. 2663-7.
34. Drew, R.H., et al., *Neuropsychological deficits in active licensed professional boxers*. Journal of Clinical Psychology, 1986. 42(3): p. 520-5.
35. Gaetz, M., D. Goodman, and H. Weinberg, *Electrophysiological evidence for the cumulative effects of concussion*. Brain Injury, 2000. 14(12): p. 1077-88.
36. Mrazik, M., et al., *Injury severity and neuropsychological and balance outcomes of four college athletes*. Brain Injury, 2000. 14(10): p. 921-931.
37. Gronwall, D. and P. Wrightson, *Cumulative effect of concussion*. Lancet, 1975. 2(7943): p. 995-7.
38. Collins, M.W., et al., *Cumulative Effects of Concussion in High School Athletes*. Neurosurgery, In Press.
39. Kelly, J.P. and J.H. Rosenberg, *Diagnosis and management of concussion in sports*. Neurology, 1997. 48(3): p. 575-80.
40. Cantu, R.C. and F.O. Mueller, *Catastrophic football injuries: 1977-1998*. Neurosurgery, 2000. 47(3): p. 673-5; discussion 675-7.
41. Bailes, J.E. and R.C. Cantu, *Head injury in athletes*. Neurosurgery, 2001. 48(1): p. 26-45; discussion 45-6.
42. Jennett, B. and M. Bond, *Assessment of outcome after severe brain damage*. Lancet, 1975. 1(7905): p. 480-4.

43. *Practice parameter: the management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee.[comment].* Neurology, 1997. 48(3): p. 581-5.
44. *Guidelines for assessment and management of sport-related concussion. Canadian Academy of Sport Medicine Concussion Committee.* Clinical Journal of Sport Medicine, 2000. 10(3): p. 209-11.
45. Cantu, R.C., *Guidelines for return to contact sport after a cerebral concussion.* Physician and Sports Medicine, 1986. 14: p. 75-83.
46. Fick, D.S., *Management of concussion in collision sports. Guidelines for the sidelines.* Postgraduate Medicine, 1995. 97(2): p. 53-6.
47. Leblanc, K.E., *Concussions in sports: guidelines for return to competition.* American Family Physician, 1994. 50(4): p. 801-8.
48. Macready, N., *Guidelines issued for sports-related concussion.* Lancet, 1997. 349(9055): p. 857.
49. Roos, R., *Guidelines for managing concussion in sports.* The Physician and Sports Medicine, 1996. 24: p. 67-74.
50. McCrea, M., et al., *Standardized assessment of concussion in football players.* Neurology, 1997. 48(3): p. 586-8.
51. Lovell, M.R., et al., *Grade I or "Ding" Concussions in High School Athletes.* American Journal of Sports Medicine, 2003(In press).
52. Lovell, M.R., et al., *Recovery from mild concussion in high school athletes.* Journal of Neurosurgery, 2003. 98(2): p. 296-301.
53. Brain Trauma Foundation, *Guidelines for the management of severe head injury.* Journal of Neurotrauma, 1996. 13(11): p. 643-5.
54. Brain Trauma Foundation, B., *Management and Prognosis of Severe Traumatic brain Injury. Parts I & II.* Journal of Neurotrauma, 2000. 17(June/July): p. 449-627.
55. McIntosh, T.K., et al., *Neuropathological sequelae of traumatic brain injury: relationship to neurochemical and biomechanical mechanisms.* Lab Invest, 1996. 74(2): p. 315-42.
56. McIntosh, T.K., et al., *Central nervous system resuscitation.* Emergency Medicine Clinics of North America, 1997. 15(3): p. 527-50.
57. Ahmed, S.M., et al., *Stretch-induced injury alters mitochondrial membrane potential and cellular ATP in cultured astrocytes and neurons.* J Neurochem, 2000. 74(5): p. 1951-60.

Outcome of Case

JR's memory deficits seemed to resolve. Under the pressure of the coach, he returned to the game despite your vigorous discouragement. You had no authority to demand he stay out. During the last period, JR was checked from behind. The impact was hard but he did not lose his balance. He continued to play for approximately 5 minutes when suddenly he collapsed on the ice. You found him unresponsive. His pupils were initially responsive to light and equal in size. His vital signs were O₂ sat 99%, BP 130/palp, HR 76, RR 24. When you called for the ambulance to transport him to the ER, you noticed his respirations became slower and more labored, and he seemed to extend his arms. A recheck of his pupils found the left one 4 mm and the right 2 mm. A recheck of the vital signs in the ambulance were O₂ sat 98%, BP 160/palp, HR 55, RR 8.

Because you appropriately suspected secondary impact syndrome and Cushing's response (due to increased intracranial pressure), you intubated JR using the rapid sequence technique and hyperventilated him. Normal saline was started at KVO through a large bore IV. No other drugs were available in the ambulance. JR was rushed to the emergency department. In transport he began having seizure activity and was given 5 mg of diazepam. Evaluation in the ER included proper placement of the airway, oxygenation at 95%, and repeat exam and vital signs. The repeat VS were: O₂ sat 99%, BP 210/70, HR 45, RR 16 (ventilated). JR was still extending his arms and had a GCS of 3t (E1VtM2). Mannitol was initiated and Neurosurgery was consulted immediately. JR was whisked to the CT scanner where diffuse cerebral edema, slit-like ventricles, and mild uncal herniation were seen on the CT monitor. He was transferred to the neuro intensive care unit and an intracranial pressure monitor was inserted. The monitor consistently displayed ICP's in the 40-50's despite mannitol, sedation, and barbiturates. JR subsequently died later that evening.

Annotated Bibliography

1. Lovell, M.R., et al., *Grade 1 or "Ding" Concussions in High School Athletes*. *American Journal of Sports Medicine*, 2003(In press).

The authors found that cognitive deficits from minor concussions (Grade 1 on AAN guidelines) persist longer than once thought and that return to play within 15 minutes of recovery may not be justified. Thirty-six hours after a mild concussion in high school athletes, there was a significant decline in memory and an increase in self reported symptoms.

2. Lovell, M.R., et al., *Recovery from mild concussion in high school athletes*. *Journal of Neurosurgery*, 2003. 98(2): p. 296-301.

This study evaluated memory dysfunction and symptoms of high school students after "mild" concussion. The study used computerized neuropsychological testing to detect sustained cognitive deficits. Statistically significant differences were seen in the pretest versus post-concussive neuropsychological tests up to 7 days post injury. The study also found that longer periods of on-the-field mental status abnormalities resulted in more profound and longer lasting neuropsychological tests differences. The results suggest that deficits from mild concussion persist much longer than previously thought and that caution should be taken in returning players to a game. The results also shows that the more substantial the abnormalities on the sidelines, the more long lasting the cognitive deficits.

3. Levy, A.S., et al., *An analysis of head injuries among skiers and snowboarders*. *Journal of Trauma-Injury Infection & Critical Care*, 2002. 53(4): p. 695-704.

This study reviewed the epidemiology of skiing and snowboarding related head injuries presenting to a level I trauma center near a ski resort during the period of 1982-1998. The study found that 350 skiers and snowboarders were admitted to the trauma center. This report does not assess or discuss the number of concussions that occurred and did not seek medical attention. Of the admitted patients, 69% were classified and mild concussion and 14% were severe head injuries. Collision verses tree was the most common mechanism (47%). Mortality of skier-tree head injuries was 7.2%. Given that the most common source of severe and mild head injury were direct impact, the authors suggest that helmets would have reduced the incidence of concussion and death.

4. Mueller, F.O. and R. Cantu, *Nineteenth Annual Report Fall 1982 - Spring 2001*. 2002, National Center for Catastrophic Sport Injury Research, University of North Carolina: Chapel Hill. p. 1-24.

This is an annual report of catastrophic sports injuries maintained by the National Center for Catastrophic Sport Injury Research at the University of North Carolina. The report outlines the epidemiology of all catastrophic injuries including head injury. The winter sport with the highest incidence of injury was ice hockey.

5. Hackam, D.J., M. Kreller, and R.H. Pearl, *Snow-related recreational injuries in children: assessment of morbidity and management strategies.[comment]*. *Journal of Pediatric Surgery*, 1999. 34(1): p. 65-8; discussion 69.

The aim of this study was to investigate the causes, clinical course, and costs of snow-related (skiing, snowboarding, tobogganing) injuries in children. The authors reviewed 147 consecutive admissions of snow-related injuries. Head injury was the most common injury. Snowboard accidents were the most common mechanism. The estimated per-patient costs were \$46,000.

6. Cantu, R.C., *Second-impact syndrome*. *Clinics in Sports Medicine*, 1998. 17(1): p. 37-44.

The author reviewed the evidence for, and suggests the etiology of second impact syndrome (SIS). The article details five individual cases. The pathophysiology is discussed. Schneider first described second impact syndrome in 1973. The syndrome is characterized by rapid cerebral edema after a second impact prior to recovery from the first. The athlete may be stunned but usually does not lose consciousness and may continue to play for the next few minutes but then rapidly deteriorates.

7. Cantu, R.C., *Head injuries in sports*. *British Journal of Sports Medicine*, 1996. 30: p. 239-289.

This is an excellent review of head injury in sports by one of the most respected experts in the field. The article reviews the history, mechanisms, assessment, and differential diagnosis of concussion in athletes. The article also reviews the pathophysiology of concussion and secondary complications such as post-concussive syndrome, post-traumatic seizures, malignant brain edema, diffuse axonal injury, and second impact syndrome.

8. Furrer, M., et al., *Severe skiing injuries: a retrospective analysis of 361 patients including mechanism of trauma, severity of injury, and mortality*. *Journal of Trauma-Injury Infection & Critical Care*, 1995. 39(4): p. 737-41.

This article reviewed all skiing accidents that required hospital admission from 1984 to 1992 at a major trauma center located near ski resorts(n=2,053). Three hundred sixty one cases were classified as severe. Mortality increased over the time period from 2% to 7%.

Questions

1. What organized winter sport has the highest rate of head injuries?
 - a) Football
 - b) Ice hockey
 - c) Gymnastics
 - d) Basketball

2. What is the percent of athletes who will sustain a head injury during their sports career?
 - a) 1%
 - b) 5%
 - c) 10%
 - d) 20%

3. All of the following are long term sequelae of mild head injuries except
 - a) increased attention
 - b) chronic headaches
 - c) increased memory deficits
 - d) personality changes
 - e) delayed response time

4. What factor(s) is/are important for increased risk for head injuries in athletes?
 - a) Age
 - b) Previous concussion
 - c) Experience with the sport
 - d) Type of sport
 - e) all of the above

5. The highest incidence of head injuries in recreational winter sports occurs in:
 - a) tobogganing
 - b) snow boarding
 - c) skating
 - d) down hill skiing
 - e) tubing

6. All of the following a part of the deleterious secondary cascade except:
 - a) glutamate release into the intercellular space
 - b) activation of NMDA, AMPA and other receptors
 - c) influx of calcium into cells
 - d) activation of proteases, lipid peroxidases, and phospholipases
 - e) activation of antiapoptotic enzymes.

Answers

1. Answer c

Gymnastics actually has the highest rate of head injuries for NCAA classified winter sports. Although football accounts for the highest number of head injuries in all sports, it is not considered a winter sport and has a lower rate (head injuries per athlete exposure) than does gymnastics and ice hockey.

2. Answer b

One out of 20 athletes will sustain a head injury during their tenure in sports.

3. Answer a

Decreased attention span, memory deficits, prolonged response time, headaches, dizziness and balance difficulties and even personality changes are all findings associated with long term sequelae from mild TBI.

4. Answer e

All of the above. Younger athletes are at an increased risk for head injury due to underlying pathophysiological reasons as well as skill level. Previous concussion seems to increase the risk of a second sports related head injury. Certain sports have higher incidences of head injury than others, particularly the high contact sports.

5. Answer d

Down hill skiing is responsible for the highest incidence of head injuries in recreational winter sports. Acrobatic skiing has a higher rate of head injury than standard down hill skiing, followed by the lowest rate with cross country skiing.

6. Answer e

Activation of NMDA and AMPA receptors by excessive glutamate release causes an influx of a pathologically high level of calcium and the activation of calcium dependent proteolytic enzymes. The subsequent phosphorylation and activation of lipases and other enzymes begin to degrade the cell from the inside out. Apoptosis, activated by caspase 3 and other factors, is also initiated. Antiapoptotic enzymes may be released, but these would be considered good as opposed to deleterious.