Fall 2014

Sports Related Traumatic Brain Injuries

Ky Parrott
Otterbein University, ky.parrott@otterbein.edu

Sports Related Traumatic Brain Injuries

Ky Parrott, RN, BSN
Otterbein University, Westerville, Ohio

Sports Related Traumatic Brain Injuries

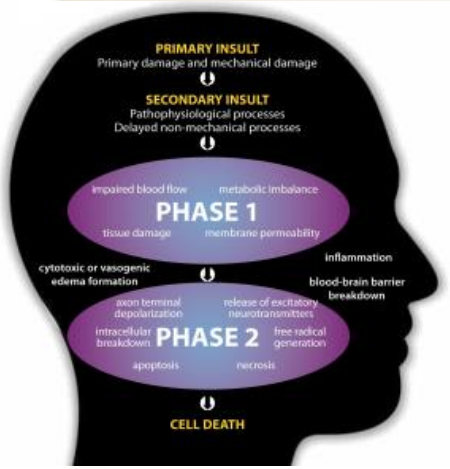
Traumatic Brain Injuries (TBI) are the leading cause of long-term disability and injury death in the United States of America (Dash, Oh, Santiago, & Wade, 2012, p. 108). It is estimated that approximately 75% of all TBI's are sports related. Recently, sports related TBI's have received increased awareness due to the $765 million settlement between 18,000 retired football players who suffer from long-term concussive injuries and the National Football Association (NFL) (Mason, 2013, p. 267). Education regarding the pathophysiology of TBI's is beneficial to understanding what changes the brain is enduring and why it is important to protect the brain, especially in developing years. While professional and college athletes are at a significant risk for traumatic brain injuries, children of all ages are also at risk and should be closely monitored for signs and symptoms of TBI's after receiving any significant blow to the head. These injuries can be detrimental to the individual and their families and it is imperative that Advanced Practice Nurses provide adequate education regarding prevention of sports related TBI's and signs and symptoms to be aware of.

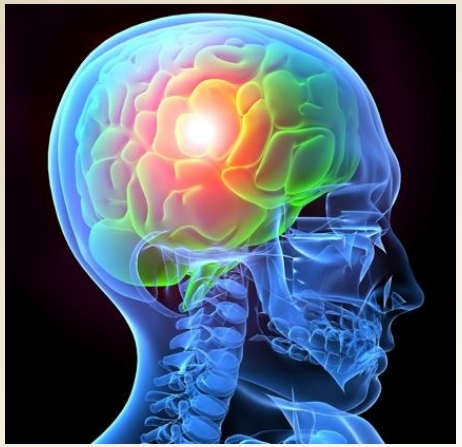
Pathophysiology of Traumatic Brain Injury

Traumatic Brain Injury is an injury to the brain cause by a sudden, significant impact to the cranium by an external force (Leon-Villapalos & McLernon 2012, p. 282). When force is applied to the head the brain moves back and forth causing brain fibers to be stretched, blood vessels to bleed, and inflammation to occur (King, Brughelli, Hume, & Gissane, 2014, p. 449). TBI's have primary and secondary brain injury effects. The primary brain injury is the physical damage to the brain and intracranial contents that is immediately sustained at the time of injury due to the direct contact, acceleration-deceleration, and rotational forces (Farls & Roth, 2000, p. 23). Secondary brain injury refers to the cellular and molecular processes caused by the initial damage to the cerebral tissues associated with the main injury (Tran, 2014, p. 30). Secondary injuries include neuroinflammation, cell death, ionic disturbance, blood-brain barrier disruption, and mitochondrial toxicity (Tran, 2014, p. 30).

This alteration in normal brain functioning, or neurometabolic cascade, requires energy to re-establish homeostasis. However, immediately following a TBI, there is a decrease in cerebral blood flow and oxygen, and ongoing mitochondrial dysfunction, which impairs energy supply and demand (King et al., 2014, p. 452).

When an individual sustains a head injury, glutamate is released from the presynaptic nerve terminal at an uncontrolled rate, and exitotoxicity occurs, in which neurons become damaged due to persistent glutamate exposure (Tran, 2014, p. 31). After sustaining a head injury, hypoxia occurs, which causes an underproduction of ATP and astrocytes are unable to remove excess glutamate from the extracellular space. The elevated exposure to glutamate leads to the activation of the calcium channel *N*-methyl-d-aspartate (NMDA), triggering mitochondrial dysfunction and intracellular calcium entry (Tran, 2014, p. 31). When intracellular calcium exceeds the excretion rate, calcium is segregated within the mitochondria to maintain intracellular homeostasis. Mitochondria are sensitive to calcium fluctuations and excess calcium stimulates the opening of the mitochondrial permeability transition pore (mPTP) (Tran, 2014, p. 31). This disruption triggers more calcium to invade the mitochondria, which in return causes the mitochondria to swell and burst. When the mPTP is opened, cytochrome c is released and interacts with apoptotic protease-activating factor-1 and forms apoptosome. At the same time, caspase-9 is activated and designates the cells to apoptosis, or regulated cell death (Tran, 2014, p. 32).





Concussion

It is estimated that there are 1.6-3.8 million annual sports related concussions, or mild traumatic brain injuries (mTBI) (King et al., 2014, p. 449). In individuals 15 to 24 years of age, concussions are the second most common form of TBI (King et al., 2014, p. 449). Concussive injuries occur in both men and women and can occur to individuals of all ages. Sports related mild traumatic brain injuries could be attributed to all athletic activities including football, soccer, basketball, horseback riding, hockey, lacrosse, wrestling, softball, and cheerleading.

While considered a "mild" form of a TBI, concussions, especially subsequent concussions, can be detrimental. The severity of the signs and symptoms then determine whether the concussion is mild, moderate, or severe. Common symptoms for a mild TBI include headache, amnesia, confusion, drowsiness, numbness or tingling to extremities, light and noise sensitivity, dizziness, nausea, loss of balance, blurred vision or changes in sleeping patterns (Mason, 2013, p. 269).

Late concussion symptoms include difficulty in school, memory or concentration problems, and increased aggression or a short temper (Mason, 2013, p. 269). These symptoms may last for weeks or months. Individuals who experience multiple concussions are at a significantly higher risk for developing a severe TBI, and will likely show more substantial symptoms for an extended period of time (Mason, 2013, p. 269).

While these symptoms could last from days to months, individuals should be closely observed for the first few days following a mild TBI. Individuals should be monitored for pupillary changes, seizures, vomiting, speech changes, neck pain, worsening headache, loss of consciousness or significant changes in mental status, as these are considered red flags and should be considered emergent situations (Mason, 2013, p. 269). Once an individual has received a concussion they should practice prevention of future mTBI's to minimize long-term complications.

Subsequent Concussions

It is believed that multiple mTBI's can cause long-term brain damage and lifelong emotional and cognitive difficulties. Individuals with repeat concussions have been shown to have an increased risk for on-the-field loss of consciousness, anterograde amnesia, confusion, and higher risk for future concussions (King et al., 2014 p. 458). Those with subsequent concussions have also shown to have a significant decrease in attention span, concentration, visual motor coordination and immediate memory recall (King et al., 2014, p. 458). These long-term effects of concussions have been recently been publicized by the media due to the $765 million settlement between the NFL and retired football players. Schools and professional sports leagues are now taking subsequent concussions more seriously and implementing changes regarding concussion monitoring and the best time to return to play.

Return to Play

Determining when an athlete is ready to return to play is frequently left in the hands of athletic trainers and coaches. While these individuals are not frequently educated regarding significance of mTBI's, we must provide them with evidence-based practice to improve outcomes of those affected by mTBI's. Three commonly utilized forms for concussion evaluation and management are the Acute Concussion Evaluation (ACE), ACE Care Plans, and neuropsychological assessments.

Several schools are recently requiring baseline neuropsychological testing for all athletes prior to sports participation. Neurocognitive testing is used to determine an athlete's pre-injury baseline of cognitive functions including attention, memory, concentration, reaction time, processing speed, and response accuracy (King et al., 2014, p. 458). After sustaining a TBI, recovering athletes must undergo neurocognitive testing until they have regained their baseline cognitive functioning. ACE and Ace Care Plans identify cognitive, physical and emotional symptoms that are able to be tracked over time and then provide an outline for school and home management (King et al., 2014, p. 458). Neuropsychological assessments, ACE, and ACE Care Plans should be used in adjunct with the athletic director or coach's professional opinion to determine the individual's readiness to return to play.

Implications for Nursing Care

Nurses play a substantial role in the prevention and management of concussions. Early diagnosis, management, appropriate referral and education are imperative to prevent concussions and improve patient outcomes. APNs must educate athletes, family members, and coaches regarding risks of sports associated TBI's, prevention strategies, and what to expect and be on alert for after mTBI. APNs should utilize and educate other necessary individuals regarding use of the Acute Concussion Evaluation form, ACE Care Plans, and neuropsychological assessments, as these are all important evaluation strategies used for determining when the individual has returned to baseline cognitive levels and may safely return to play. APN's may also need to educate school officials and family members regarding adjustments of school schedules and work to compensate for the students temporary disruption of cognitive functioning.

Conclusion

Sports related traumatic brain injuries affect an estimated 1.6 to 3.8 million people each year (King et al., 2014, p. 449). While these injuries are frequently overlooked as minor, the long-term consequences can be detrimental to the individual and their families. Those who suffer multiple mTBI's in their athletic career are at significant risk for functional, psychological, and cognitive impairments. Education regarding the pathophysiology behind mTBI's is imperative to the individual understanding the changes evolving in the brain to cause cognitive impairment. Athletes, families, friends, coaches, and teachers should also be educated regarding signs and symptoms of mTBI's, the risks associated with multiple concussions, and prevention strategies for future mTBI's. As Advanced Practice Nurses, we can empower our community with education and resources to adequately diagnose and manage traumatic brain injuries in our athletes.

References

Dash, P., Holcomb, J., Oh, B., Santiago, L., & Wade, C. (2012). A clinical comparison of penetrating and blunt traumatic brain injuries. *Brain Injury*, *26*(2), 107-125. doi:10.3109/02699052.2011.635363

Farls, K., & Roth, P. (2000). Pathophysiology of traumatic brain injury. *Critical Care Nursing Quarterly*, *23*(3), 14-25.

Gioia, G., & Collins, M. (2006). Acute Concussion Evaluation. Retrieved from http://www.cdc.gov/concussion/headsup/pdf/ace-a.pdf

Harvey, H. (2013). Reducing traumatic brain injuries in youth sports: Youth sports traumatic brain injury state laws. *American Journal Of Public Health*, *103*(7), 1249-1254. doi:10.2105/AJPH.2012.301107

King, D., Brughelli, M., Hume, P., & Gissane, C. (2014). Assessment, management and knowledge of sport-related concussion: Systematic review. *Sports Medicine*, *44*(4), 449-471.

Leon-Villapalos, C., & McLernon, S. (2012). The pathophysiology of traumatic brain injury: a case study approach. *British Journal Of Neuroscience Nursing*, *8*(5), 282-287.

Mason, C. (2013). Mild traumatic brain injury in children. *Pediatric Nursing*, *39*(6), 267-282.

Mason, M. (n.d.). Traumatic brain injury basics. *Brainline.org*. Retrieved from http://www.brainline.org/content/2009/06/tbi-basics.html

Nolan, S. (2005). Traumatic brain injury: a review. *Critical Care Nursing Quarterly*, *28*(2), 188-194.

Tran, L. V. (2014). Understanding the pathophysiology of traumatic brain injury and the mechanisms of action of neuroprotective interventions. *Journal Of Trauma Nursing*, *21*(1), 30-35. doi:10.1097/JTN.0000000000000026

OTTERBEIN UNIVERSITY