NEUR20080

Concussion

**A concussion is an injury to the brain that results in temporary loss of normal brain function. It usually is caused by a blow to the head.**

**The formal medical definition of concussion is a clinical syndrome characterized by immediate and transient alteration in brain function, including alteration of mental status and level of consciousness, resulting from mechanical force or trauma.**

- In most cases there is no external signs of head trauma – person can remain conscience.
- Concussion can affect memory, judgment, reflexes, speech, balance and muscle coordination.
- Concussion are serious and should not be taking lightly a single concussion should not cause permanent damage however if a second one was to occur it doesn't have to be a strong one to have deadly or permanently disabling consequences.

Symptoms

The three principal features of confusion are:

- Inability to maintain a coherent stream of thought
- A disturbance of awareness with heightened distractibility
- Inability to carry out a sequence of goal-directed movements

The following are concussion symptoms:

- Prolonged headache
- Vision disturbances
- Dizziness
- Nausea or vomiting
- Impaired balance
- Confusion
- Memory loss
- Ringing ears
- Difficulty concentrating
- Sensitivity to light
- Loss of smell or taste

The skull protects the brain from penetrating trauma, the cerebrospinal fluid cushions the brain, despite this there is still chances for potential tears if blood vessels, pulling f nerve fibres and bruising of brain substances after sudden blow to the head.

Sometimes the blow can result in microscopic damage to the brain cells without obvious structural damage visible on a CT scan. In severe cases, the brain tissue can begin to swell. Since the brain cannot escape the rigid confines of the skull, severe swelling can compress the brain and its blood vessels, limiting the flow of blood. Without adequate blood flow, the brain does not receive the necessary flow of oxygen and glucose. A stroke can occur. Brain swelling after a concussion has the potential to amplify the severity of the injury.

A blow to the head can cause a more serious initial injury to the brain. A contusion is a bruise of the brain tissue involving bleeding and swelling in the brain. A skull fracture occurs when the bone of the skull breaks. A skull fracture by itself may not necessarily be a serious injury. Sometimes, however, the broken skull bones cause bleeding or other damage by cutting into the brain or its coverings.

A hematoma is a blood clot that collects in or around the brain. If active bleeding persists, hematomas can rapidly enlarge. Like brain swelling, the increasing pressure within the rigid confines of the skull (due to an enlarging blood clot) can cause serious neurological problems, and can even be life-threatening. Some hematomas are surgical emergencies. Hematomas that are small can sometimes go undetected initially, but may cause symptoms and require treatment several days or weeks later.

**Treatment**

The standard treatment for concussion is rest. For headaches, acetaminophen (Tylenol) can be taken. Postconcussive headaches often are resistant to stronger narcotic-based medications.

**Postconcussive Syndrome**

People who suffer a head injury may suffer from side effects that persist for weeks or months. This is known as post-concussive syndrome. Symptoms include memory and concentration problems, mood swings, personality changes, headache, fatigue, dizziness, insomnia and excessive drowsiness. Patients with post-concussive syndrome should avoid activities that put them at risk for a repeated concussion. Athletes should not return to play while experiencing these symptoms. Athletes who suffer repeated concussions should consider ending participation in the sport.

**Second-impact Syndrome**

Second-impact syndrome results from acute, often fatal brain swelling that occurs when a second concussion is sustained before complete recovery from a previous concussion. This is thought to cause vascular congestion and increased intracranial pressure, which can occur very rapidly and may be difficult or impossible to control. The risk of second-impact syndrome is higher in sports such as boxing, football, ice or roller hockey, soccer, baseball, basketball and snow skiing.

The CDC reports an average of 1.5 deaths per year from sports concussions. In most cases, a concussion, usually undiagnosed, had occurred prior to the final on

Studies have shown that repeat concussions lead to severe long-term symptoms and longer recovery times. This suggests recognizing the signs of a concussion early and giving the brain time to fully recover before returning to play is key to long-term brain health.

Most immediate damage from concussions is hard to detect using standard imaging procedures, such as CT scans or MRIs. As a result, scientists have spent the better part of a decade studying animal models, predominately rodents, for clues about the changes that take place in the brain.

When you slam your head, the force rapidly pushes the brain against the interior of the skull. This sudden movement causes brain cells to stretch and tear, altering the electrical and chemical balance critical to cell function and communication (action potentials and graded potentials) . These cells then spring into action, working harder to return to the correct chemical state. This puts the cell in a “highly stressful” state, leaving it unable to function properly or even survive. Cells work so much

harder that they go into an energy crisis causing the axons to swell, compromising the ability of the cells to send signals. Too much swelling and the connections between cells can be permanently lost no action potentials or graded potentials can be generated or transmitted signals never reach the synapses.

It is believed amongst researchers that most of the changes to the neurons are temporary and rest cab aid with returning cell to its normal internal chemistry. However experimental studies suggest that some of the damage is permanent, potentially reflected by persisting cognitive dysfunction in some cases of single concussion. Moreover, experiencing multiple concussions before the brain has had time to fully heal can lead to more significant and potentially long-term changes in the brain.

In recent years, scientists have found signs of a neurodegenerative disease called chronic traumatic encephalopathy (CTE) in the brains of deceased athletes who either experienced multiple concussions or played positions where they were exposed to frequent impacts. Symptoms of the disease include increased irritability and impulsivity early on and dementia in later stages.

**NEUROCHEMISTRY OF CONCUSSION**

Damage to brain tissue resulting from movement of the brain within the skull, as may occur with a head impact, initiates a cascade of molecular events that disrupt normal brain cell function. In this section the molecular processes that have been shown to characterize brain injuries are discussed in the context of age and sex. It should be noted that much of what is currently known about these processes is drawn from research involving animals and subjects sustaining moderate to severe brain injuries.

**ionic Flux and Neurotransmitter Release**

TBI induces immediate changes in brain neurochemistry. Normally, significant cellular energy is used to keep ions distributed across the plasma membranes in such a way so as to maintain a membrane potential between –40 and –80 millivolts (mVs). Studies primarily involving rodents show that an indiscriminate efflux of potassium and glutamate and an influx of calcium follow immediately after a concussive fluid percussion brain injury. The magnitude of the potassium rise in the extracellular space increases with injury severity as much as a fivefold increase observed at 1.5 minutes post injury. These rapid increases resolve after approximately 2.5 minutes in mild injuries and within 6 minutes after more severe injuries. Increases in extracellular glutamate concentrations showed a similar time-course following TBI in the adult rat. The release of excitatory amino acids following severe brain injury in humans has been observed with microdialysis probes.

Research involving humans also provides evidence for neurotransmitter release after concussive injuries. In a study involving 12 concussed athletes (mean age 22.5 years) and 12 non-concussed athletes, they used (1)H magnetic resonance spectroscopy ((1)H-MR) to noninvasively study acute metabolic changes post injury. The concussed athletes showed more symptoms and a significant decrease in N-acetylaspartylglutamic acid (NAA) in the primary motor cortex between 1 and 6 days post injury as compared with the non-concussed the controls. The findings of decreased glutamate are in contrast to the changes seen in studies involving animals and more severe TBI, but the time-course of changes was not examined in this study. Scans collected between 1 and 6 days post injury were averaged and may not reflect the same time-course of changes observed in other studies. The animal studies were conducted immediately after impact, and the clinical studies were conducted 1

to 6 days post injury. There is currently little research that examines the time-course of these changes over longer periods and at different injury severities.

Neurochemical cascade observed after moderate traumatic brain injuries. Many of these events are believed to be involved to a lesser extent following mild and concussive brain injuries. eg the indiscriminate release of glutamate and other neurotransmitters

Collectively, the animal studies of moderate brain injuries suggest that such injuries cause a release of signaling molecules which results in a disruption of the ionic balance across the cell membrane immediately after the injury event. Unless these ions are pumped back to the correct side of the cell membranes, the brain cells will not be able to be activated again. While it is thought that these events occur to a lesser degree or for a shorter duration following milder or concussive injuries, human studies examining acute neurochemical changes following concussive injuries in youth as well as adults or that address sex differences in response are lacking.

**Metabolic Cascade After Traumatic Brain Injury: Glucose and Cerebral Blood Flow**

The energy required to resolve the disruption of the neurochemical environment seen after injury triggers an immediate increase in brain glucose uptake or CMRglc. The immediate release of glutamate after impact plays a significant role in this transient increase in glucose uptake. In an adult rat, CMRglc rates increase 30 to 46 percent immediately after moderate fluid percussion impact and remain elevated for 30 minutes. The early increase in cerebral glucose uptake following brain injury is thought to reflect the increased energy demands associated with reestablishing ionic homeostasis and maintaining neuronal membrane potential. While [18F]-fluorodeoxyglucose positron emission tomography (FDG-PET) studies have shown increased glucose uptake following severe TBI in humans, no studies have examined this immediate increase in glucose uptake after mild or concussive brain injuries.

TBI-induced increases in cerebral glucose uptake are followed by a reduction of adenosine triphosphate (ATP) levels and prolonged CMRglc depression. This post-injury decrease in CMRglc has been established as a hallmark response after TBI. It is a response that is observed in animals following experimental fluid percussion that results in concussive injuries and cortical contusion) and that is also observed in humans who have sustained severe TBI. On the other hand, there is no evidence of these changes from studies involving humans who have sustained concussions.

**Further reading paper:**

**2Neuroscience, Biomechanics, and Risks of Concussion in the Developing Brain.**

**https://www.ncbi.nlm.nih.gov/books/NBK185339/**