CONCUSSION: AN OVERVIEW AND UPDATE ON CURRENT RESEARCH

OVERVIEW

Concussion is the result of force on the brain. This can either be direct, from a hit to the head, or indirect, from sudden acceleration and deceleration. As the brain is a soft structure, made of billions of thin cells meshed together, it is easily affected by these forces. The force stretches the axons, reducing their flexibility and increasing their risk of tearing. The thinner an axon is the more likely this force will be to break it, or strip it of myelination. This stretching also allows potassium to flood out of the cells, disturbing the electrochemical gradient. To complicate matters further, research suggests that mitochondria may also be damaged by the force of concussion, which disturbs the neuron's ability to produce ATP to re-establish the concentration of potassium on the inside of the cell. The combination of these factors results in short-term concussive symptoms and potentially also long-term structural and functional damage to the brain.

Depending on the area and severity of damage, concussion can present as a number of different symptoms. These symptoms are exacerbated by intoxication, with intoxicated sufferers more likely to score poorly on the Glasgow Coma Scale and be hospitalised for their injuries.

In the case of traumatic accidents, such as car accidents, a concussion can be misattributed as an acute stress response (psychological shock). However, while confusion and amnesia do occur in both conditions, concussion also presents with a number of observable physiological symptoms which are noted below.

SYMPTOMS

The early symptoms of concussion occur minutes to hours after the initial injury and are commonly one or more of the following:

- Headache
- Dizziness
- Disorientation
- Confusion
- Nausea
- Vomiting

Late symptoms occur days to weeks after the initial injury and can cause ongoing issues, the most common presentations are one or more of the following:

- Fatigue
- Headache
- Light-headedness
- Light sensitivity
- Tinnitus
- Disorientation
- Sleep disturbances
- Anxiety
- Depression
- Irritability
- Difficulty remembering
- Difficulty concentrating

The symptoms of confusion experienced by those with concussion should be gone after 24 hours, however other symptoms can last for up to 12 weeks. Health prior to the incident appears to improve recovery outcomes.

While sleeping post-concussion cannot increase the chance of coma, it can obscure the onset of a coma or significant deterioration. Swelling should reduce after six hours, but prior to this the individual should be roused every hour or so to determine whether they are still able to regain consciousness.

IDENTIFYING CONCUSSION

CHILDREN

The Glasgow Coma Scale is a commonly used measure of concussion severity, while the PECARN algorithm is an indicator of whether other complications have occurred which may necessitate a CT scan.

Identification of concussion can also be made via diagnosis of a visual disturbance such as saccadic eye movement disorders or binocular convergence, which occur in around 69% of affected children and adolescents.

Children are particularly at risk post-concussion, and are more greatly impacted by the symptoms of concussion than adults. Concussions during this time period (16 and under) are commonly the result of sport-related injuries.

ADULTS

The severity of a concussion in adults can be determined using the Glasgow Coma Scale. Individuals who have a non-complicated concussion can show any of the symptoms described in GCS 13-15 within 30 minutes after presentation, also loss of consciousness for less than 30 minutes, post-traumatic amnesia for less than 24 hours, any period of confusion or disorientation, or seizures. The NICE scale for individuals 16 and older can be used in conjunction with the GCS in order to determine whether a CT scan is necessary.

ELDERLY

While elderly individuals do not differ significantly from other adults, they are much more likely to experience falls which result in concussion, and so are more likely than others to experience repeat concussions. These individuals are also more likely to be on blood thinning medications, and other medications which may cause complications in the case of concussion or mild traumatic brain injury.

COMPLICATIONS

SWELLING AND BLEEDING

Both swelling and bleeding in the brain can result from trauma related to concussion but, in the case of mild trauma, are difficult to identify in the early stages after the incident. Bleeds are particularly harmful, and occur in around 6—8% of concussions.

The damage caused by both of these issues is exacerbated by normal, or above average temperatures around the brain and skull, and can be minimised by ensuring that the head is kept below average body temperature. Some evidence suggests that this may slow the recovery of concussion symptoms, as it slows the metabolic processes used by neurons to rebalance potassium, but the long term impacts of this are less harmful than the potential complications from brain swelling or bleeding.

HYPOXIA, HYPERCARBIA, HYPOTENSION

Hypoxia, hypercarbia, and hypotension have all been associated with worsened outcomes for patients who have had a concussion or mild-TBI. These issues can be identified early during treatment, and must be treated quickly in order to avoid unnecessary complications.

ACUTE ALCOHOL INTOXICATION

Between 30-50% of adult patients who sustain a concussion will have been inebriated at the time of the incident. Traumatic brain injury combined with alcohol can induce symptoms of severe intoxication, and can skew diagnosis. The neuroinflammation which occurs during concussion and mTBI resolves much more slowly in these individuals, which can result in further complications.

While patients who experience AAI have, on average, better treatment outcomes after concussion they tend to be identified with lower than expected GCS scores, and are much more likely to be hospitalised.

CURRENT RESEARCH INTO THE EFFECTS OF CONCUSSION ON THE BRAIN

There are two types of axonal injury which occur as a result of concussion: micro-tearing, and diffuse axonal injury.

Diffuse axonal injury, or DAI, is a misleading name as the damage is not diffuse, but rather stems from one or more focal regions. This damage tends to be concentrated around the central white matter tracts of the brain: the corpus callosum, internal capsules, cerebellar peduncles and brainstem. DAI is believed to be caused by damage to axonal transport as a result of acceleration. This leads to swelling of the axon, and then detachment of the axon from those points of swelling.

Micro-tearing is incomplete tearing of the axonal plasma membrane, which damages the structural integrity of the axon but does not result in axonal detachment.

Diffusion tensor imaging (DTI), which is a scanning technique that uses the pattern of water diffusion throughout the brain to identify fibre tracts, has confirmed that white matter tracts are significantly damaged as a result of concussion via both DAI and micro-tearing. DTI scans also point to reduced structural integrity of axons. Differences have been detected relating to how the concussion is triggered, for instance sports-related concussions tend to result in damage to the axonal tracts which extend from the front of the brain to the back of the brain, whereas blast-related concussions received by military servicemen tend to have diffuse axonal damage with multiple focal areas of injury.

Structural damage, however, is not the only damage caused by concussion. Resting state fMRI studies have demonstrated changes in the functional connectivity of the brains of people with concussion. These changes persist even after symptoms have resolved. Functional connectivity is the ability of the brain to use two or more physically separated regions for the same task. This is achieved by having tracts of axons connecting these different regions of the brain in order to allow them to communicate with one another effectively. Damage to the axons which allow for this functional connection can lessen the connectivity of the regions and impact neurological performance.

Individuals who did not experience lingering symptoms of concussion, or who had been believed to have recovered from concussion, showed no changes to functional connectivity during rest but presented with deficits after physical exertion. This suggests that even after symptoms have resolved underlying physical damage remains.

Repeated concussions, in particular those which occur in contact sports, have been associated with disorders such as post-concussive syndrome and chronic traumatic encephalopathy.

Post-concussive syndrome is the more common of the two to be observed in non-sport playing individuals, and is the persistence of concussion symptoms. Children and the elderly are particularly at risk of developing this syndrome, though it is unclear why. Evidence suggests that the functional connectivity of individuals with persistent post-concussive syndrome (lasting 6 months or longer) is significantly reduced even when compared to the functional connectivity of individuals with recent concussion.

Chronic traumatic encephalopathy occurs almost exclusively in soldiers and professional contact-sportsmen. The individual develops concussion-like symptoms as well as issues related to regulating mood, memory deficits, and problems with verbal communication. Autopsies have revealed a build-up of the protein Tau in the brains of these individuals, in some cases, resulting in severe dementia.

Concussion, though a primarily subjective illness, is much better understood now than it was 20 years ago. The ramifications of concussion, especially repeat concussion, are still up for debate but evidence suggests that these events result in observable damage to the brain and as such should be taken seriously.