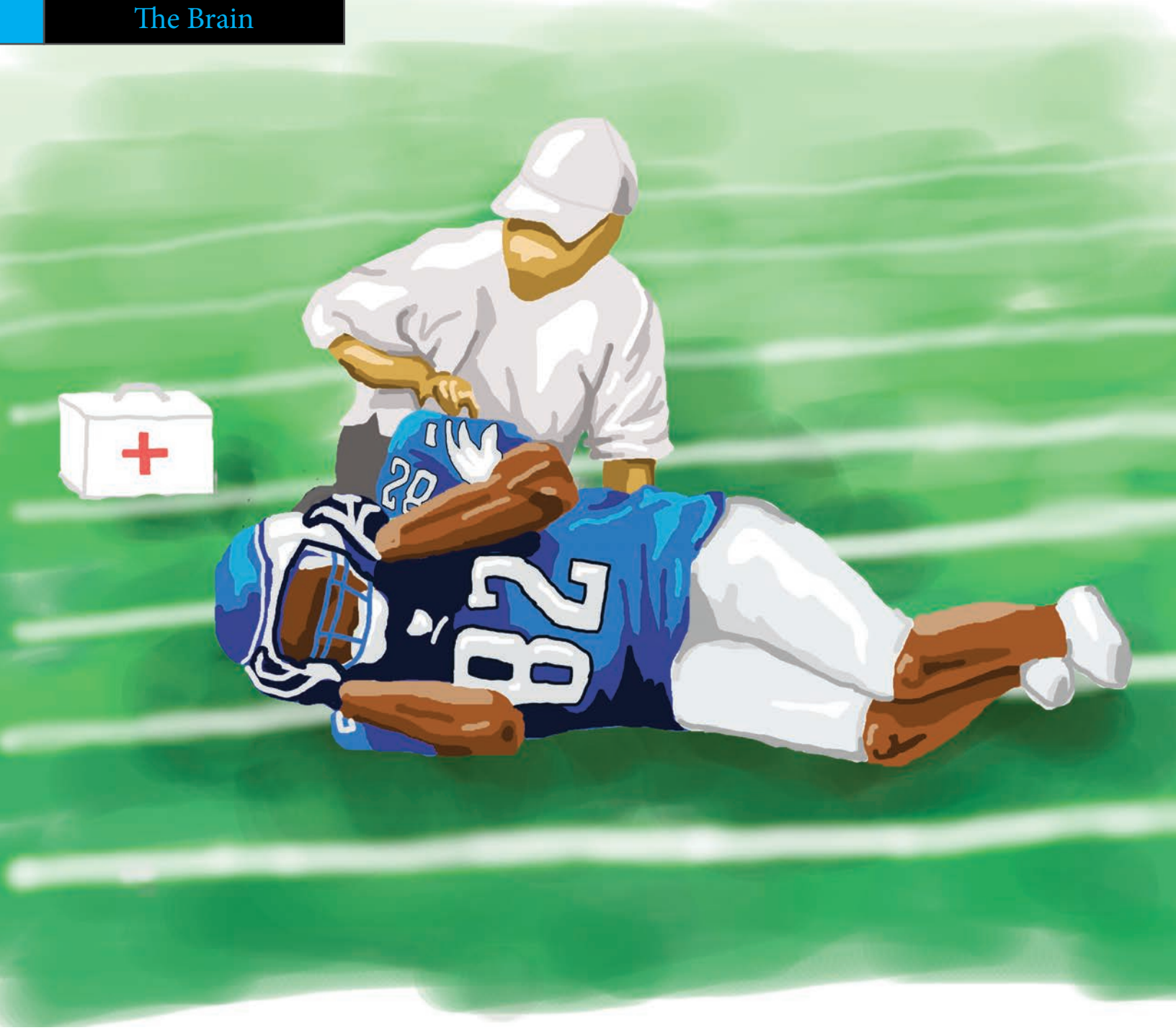
The Synapse: Intercollegiate science magazine

Volume 14 | Issue 1 Article 6

2017

# Get Your Head in The Game: A Call to Action for Proper Treatment of Traumatic Brain Injury

Zoe Swann



# Get Your Head in The Game

*A Call to Action for Proper Treatment of Traumatic Brain Injury*

**Written by Zoe Swann**
**Illustrated by Lydia Newman-Heggie**

How many people do you know with chronic fatigue? How many people do you know who have been in a car crash, or have had a concussion?

There is no debate. Traumatic Brain Injury (TBI) results in endocrine dysfunction far more than previously realized and has life threatening effects that often go unseen.

Brain injury is caused by acceleration and deceleration of brain tissue from motor vehicle, assault, athletic collision, or veteran blast injuries. All of these forms of TBI cause shear strain (where your cells break open), resulting in possible hemorrhage, neural necrosis, network disruptions, and cell death. Injury to the axons of individual neural cells also easily occurs after shear strain.

So how do these injuries occur? If the front of my skull were to hit the dashboard of a car, my brain would hit the back of my skull and then bounce forward due to inertia. This causes the acceleration and deceleration of brain tissue, thus leading to shear strain injuries. After

forming a lesion in the front and back of the brain, hemorrhage and edema occur, which mean blood and water on the brain respectively. This pressure causes cells to rupture and the immune system must come to the rescue, filling the brain and skull with even more fluid. Because pressure means more cell death, more network disconnection occurs, often leading to drastic changes in behavior as parts of the brain can no longer communicate effectively. And all of this can happen easily, even under the mildest of circumstances.

Depending the injury mechanism, a particularly vulnerable area of the brain is the hypothalamic-pituitary-axis. Part of the endocrine system, it communicates via the pituitary with glands all over the body by way of hormones. These hormones are produced and secreted by several glands in the body and are extremely susceptible to disruption of the system. Each hormone contributes to a myriad of disorders after TBI, such as growth, stress, sex dysfunction, and even diabetes insipidus.

The hypothalamus in the forebrain is responsible for sending neural input to the pituitary gland below it, which is encased in the bony sella turcica. For spatial reference, a pituitary tumor can be surgically accessed by going directly up the nose by drilling through the sella bone. After TBI, the bony sella may fracture, injuring the pituitary gland. If the pituitary, "the master gland," is injured, dysfunction occurs. A study

> 1.5 million people sustain a TBI each year, and only 230,000 patients, or approximately a sixth, survive.

published 2015 in the *Indian Journal of Endocrinology and Metabolism* found that 80 percent of TBI patients had sella abnormalities. The number of people diagnosed with endocrine dysfunction is nowhere near that, meaning that a significant portion of the TBI population is going without proper diagnosis of any endocrine dysfunction and subsequent treatment.

Often, endocrine dysfunction is missed entirely or misdiagnosed as PTSD or post-concussive disorder. This misdiagnosis can result in life threatening or quality-of-life threatening conditions. For example, hypogonadism—or sex hormone dysfunction—may not be apparent in a pre-pubescent injured child, who then by puberty will be very difficult to treat. Children also produce more growth hormones than adults, so they are prone to experience undiagnosed stunted growth after TBI.

It is estimated by the CDC that 1.5 million people sustain a TBI each year, and only 230,000 patients, or approximately one-sixth, survive. The prevalence of pituitary dysfunction is diagnosed at the rate of about 27 to 47 percent regardless of injury severity. However, it likely occurs at a much higher rate of 70 percent in TBI patients. This means that millions of people each year are living without a proper diagnosis.

A high population of people who experience traumatic brain injuries are athletes. Athletic institutions, such as the NFL, have been subject to concern and scrutiny after the discovery of CTE (chronic traumatic encephalopathy), which occurs after several mild, repeated injuries and is related to Alzheimer's Disease. Many athletes, veterans, and victims of assault who have sustained concussions have later developed symptoms of Alzheimer's. These symptoms often include memory loss, behavior changes, aggression, and substance abuse.

There have been over 200 post-mortem confirmed cases of CTE in professional athletes. Football players are particularly susceptible. Although symptoms begin in a person's 40s or 50s, if this person experienced a head impact before the age of 12, they are much more likely to have severe symptoms and a severe prognosis. These athletes also face an increased risk of suicide and substance abuse. For these reasons, research into this disorder should be recognized by organizations like the NFL as worthy of funding. This is an essential step in ensuring the safety of their players.

Interestingly, sex hormones, like progesterone and estrogen, might have neuroprotective effects by regulating fluid balance,

> If this person experienced a head impact before the age of 12, they are more likely to have severe symptoms and a severe prognosis

inflammation, stroke, and edema. One solution to improve the prognosis of many athletes may be to consider hormone therapy. However, professional athletes may be reluctant to take sexy hormones typically associated with femaleness.

People serving in the military are also highly susceptible to TBI; in fact, it is these days considered "the injury" of the military. Veterans most often suffer blast injuries from explosions, either due to the initial pressure wave or flying bodies and shrapnel. Opioids are often used for pain management, which has led to the rising opioid epidemic. This epidemic has major implications for endocrine dysfunction after TBI.

Following primary mechanical and secondary injury, stress and medication can be a major factor in endocrine dysfunction post-injury. Opioids are relevant to several models, as civilians are often bedridden or hospitalized after a moderate to severe injury, and veterans suffering a blast injury are often in stress-inducing situations both prior to and directly following the injury event. Additionally, overuse of opioids or phenobarbital in the ICU may aggravate secondary injury to the pituitary and cause adrenal insufficiency, resulting in low cortisol levels. Without cortisol, the body cannot respond normally to stress, such as common infections that occur during hospitalization.

What are we doing about the misdiagnoses and treatment in all of these various populations who experience TBI? Unfortunately, pituitary dysfunction is still not on the radar of many health care professionals. But, by understanding how endocrine dysfunction manifests in different age groups and different models of injury, we may be able to produce an acute treatment and help those with immediate injuries and chronic symptoms.

If you're interested in learning more about the basics of traumatic injuries, see "Explosions and Blast Injuries: A Primer for Clinicians" from the Department of Health and Human Services, available on the CDC Injury Prevention website. If you'd like to read more about problems with the pituitary gland as a consequence of brain injuries, see Eva Fernandez-Rodriguez's July 2017 article "Hypopituitarism Following Traumatic Brain Injury: Determining Factors for Diagnosis" in *Frontiers in Endocrinology.*