FOR THE LOVE OF THE GAME: TRADING MONEY FOR BRAIN CELLS

Hanna Kong

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INTRODUCTION

I knew sooner or later he would get the better of me . . . And I just — I like my face, and I just didn’t want to have it broken.

· Georges Laraque, retired NHL player

Just a few minutes into the second quarter, the San Jose Sharks’ Jody Shelley had no idea that he would not finish the hockey game. Listed at 6’3” and 230 lbs., Shelley was no delicate flower. Even with a tooth knocked out of his jaw, the bloodied

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1 J.D. 2012, University of Notre Dame Law School.
2 John Branch, Derek Boogaard: Blood on the Ice, N.Y. TIMES (Dec. 4, 2011), http://www.nytimes.com/2011/12/05/sports/hockey/derek-boogaard-blood-on-the-ice.html?_r=1&sq=&st=nyt&scp=1&pagewanted=all. (George Laraque stating his reason for retiring as primarily related to Derek Boogard.).
Shelley kept fighting. Yet, with one monstrous punch, Derek “Boogeyman” Boogaard lived up to his fierce National Hockey League reputation as an enforcer, and left Shelley’s helmet crumbled under the force of his bare fist.

Boogaard’s job as an enforcer requires a spectacular level of violence, which fans and players alike expect as an integral part of the sport. Traditionally, teams believe that the threat of violence is the best way to protect the most valuable players against injury. Enforcers, also called “hockey goons,” promise physical retaliation against players who partake in cheap play during a game. Sometimes as part of their job, they resort to violence as a confidence booster for the rest of the team.

During his hockey career, Boogaard suffered the same injuries as a professional fighter. He broke his nose, jaw, hand, and teeth on numerous occasions, and sustained two bulging discs. Instead of complaining of sensitivity to light, he wore...
sunglasses to remedy the issue.\textsuperscript{11} Like the drug-addicted TV character, Dr. Gregory House,\textsuperscript{12} Boogaard devoured pills to help cope with the pain.\textsuperscript{13} Suddenly, friends noticed a dark shift in Boogaard’s personality.\textsuperscript{14} In his final days, the once ferocious enforcer became childlike in his all-consuming loneliness. His January cellphone bill stretched 167 pages, and detailed calls and text messages to people he lost contact with many years ago. His February bill spanned 222 pages, and listed 13,724 text messages.\textsuperscript{15} Derek Boogaard’s behavior resulted from an advanced case of Chronic Traumatic Encephalopathy (CTE), a progressive degenerative brain disease associated with multiple concussions.\textsuperscript{16} Even if he miraculously survived his drug overdose on May 13, 2011, Boogaard would have likely developed middle-aged dementia as his CTE worsened.\textsuperscript{17} At the prime of his career, the twenty-eight-year old Boogaard suffered worse brain damage than the sixteen-year veteran and retired enforcer, Bob Probert.\textsuperscript{18}

This paper examines the feasibility of minimizing, or accommodating, brain injuries in professional sports. Part II provides an overview of the pertinent medical terminology and classifications. Part III notes the current treatment offered to professional athletes and evaluates the medical advances that may change current medical practices. Part IV examines existing state legislation regarding brain injuries, analyzes reactions to state legislation, and discusses proposed solutions to prevent potentially fatal injuries in sports.

\begin{footnotesize}
\begin{enumerate}
\item Id.
\item See generally \textsc{House, M.D.} (Fox Broadcasting Company), http://www.fox.com/house
\item Branch, supra note 1.
\item Id.
\item Id.
\item Id.
\item Id.
\end{enumerate}
\end{footnotesize}
I. BRAIN DAMAGE

There’s no such thing as a minor concussion, and the problem is that once you have one or two, you’re more likely to have a third, fourth or a fifth, and then you are at risk for sustaining cumulative and permanent damage over time.

The brain is a unique organ. You only have one, and you need it for almost everything else.

-Dr. John Leddy

A. Naming the Monster

Doctors cannot agree upon a universally accepted definition of “concussion.” They bicker amongst themselves and interchangeably use medical terminology like mild traumatic brain injury (MTBI) with “concussion,” while others distinguish between the two terms. The need for proper medical labeling of these conditions matters because calling an injury a “concussion” leads to dismissive medical treatment. The diagnosis of a “concussion” instead of “MTBI” often determined whether the

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19 Dr. John L. Leddy serves as the Associate Professor of Clinical Orthopedics and Director of the UB Orthopedics and Sports Medicine Concussion Clinic, and Associate Director of the UB Sports Medicine Institute; David J. Hill, Fourth and One, The UNIV. OF BUFFALO, http://www.buffalo.edu/home/feature_story/the-right-call.html.


21 Zachary Levine, Mild Traumatic Brain Injury, 56 CANADIAN FAMILY PHYSICIAN 658, 658-62 (2010). available at http://www.cfp.ca/content/56/7/658.full.pdf+html. (Concussion: no abnormality is seen with standard neuroimaging. MTBI: cases found to have intracranial lesions. “Therefore, concussion and MTBI are indistinguishable on clinical presentation. Concussion is the term most commonly used when the injury is sports-related.”)

22 Carol A. DeMatteo et al., My Child Doesn’t Have a Brain Injury, He Only Has a Concussion, 125 PEDIATRICS 327,327-34 (2010) available at http://pediatrics.aappublications.org/content/125/2/327.full.pdf+html. (Doctors treated MTBI and a concussion as two separate diagnostic categories despite both being forms of mild brain injury. In a study of children with traumatic brain injury, labeling an injury a concussion strongly predicted earlier discharge from hospital and earlier return to school, independent of Glasgow Coma Scale and the presence of other associated injuries. Investigators questioned whether the concussion label is potentially falsely reassuring to parents, who might understand it to mean lack of serious head or brain injury.)
patient received a longer hospital stay or immediate release.\textsuperscript{23} Unfortunately, neurological tests and procedures, such as CTs, MRIs, and EEGs, cannot reveal the presence or effect of a concussion,\textsuperscript{24} nor can scientific or biological markers highlight the existence of a concussion.\textsuperscript{25}

According to the National Institute of Neurological Disorders and Stroke, a traumatic brain injury (TBI) is one that “occurs when a sudden trauma causes damage to the brain . . . TBI can result from a closed head injury or a penetrating head injury . . . Concussion is the most minor and most common type of TBI.”\textsuperscript{26} The National Football League notes that a “concussion or mild traumatic brain injury is a pathophysiological process affecting the brain induced by direct or indirect biomechanical forces.”\textsuperscript{27} The Sports Legacy Institute prefers defining a concussion as “trauma-induced change in mental status.”\textsuperscript{28} The Center for Disease Control & Prevention (CDC) noted that a “concussion is a type of traumatic brain injury, or TBI, caused by a bump, blow, or jolt to the head that can change the way your brain normally

\textsuperscript{23} Id.

\textsuperscript{24} Although normally useful for neurological and radiological tests, CTs, MRIs, and EEGs are not useful since concussions are metabolic rather than structural injuries. The tests are based upon structural neuroimaging. imPACT Test (tm), http://impacttest.com/about/background; A new imaging technique called High Definition Fiber Tracking (HDFT) has some initial positive results in being able to display the damage from concussions, but given how recent the technology is, further research on its accuracy and clarity is required, http://www.sportsconcussions.org/ibaseline/fantastic-voyage-new-imaging-technique-shows-brain-injury-video.


\textsuperscript{26} National Institute of Neurological Disorders and Stroke, http://www.ninds.nih.gov/.


\textsuperscript{28} \textit{Chronic Traumatic Encephalopathy}, SLI, \textcolor{red}{http://www.sportslegacy.org/cte-concussions/what-is-cte/}. Renowned Concussion Experts Dr. Christopher Nowinski and Dr. Robert Cantu founded the Sports Legacy Institute.
works. Concussions can also occur from a blow to the body that causes the head to move rapidly back and forth.\textsuperscript{29}

Even if there was a definitive understanding of “concussion” in the medical field, health professionals do not agree on a consistent set of symptoms that manifests afterwards.\textsuperscript{30} Understanding the signs of a concussion could mean receiving earlier treatment and care, a significant factor in reducing the “social morbidity and severity of post-concussion symptoms.”\textsuperscript{31} Post-concussive effects can include: attention and concentration problems, memory issues, fatigue, sleep disruption, headaches, dizziness, irritability, anxiety/depression, personality change, apathy, occupational or social decline, sensitivity to light, nausea, slurred speech, disorders of taste and smell, and even psychological adjustment issues.\textsuperscript{32}

Diagnosis and treatment for a concussion largely depend on the highly subjective standards of the athlete’s self-reporting of symptoms and the nature of the incident.\textsuperscript{33} Further, sports medicine physicians, who largely determine whether an athlete returns to his sport to play another game, do not have access to a definitive system of evaluating brain injuries because such a system does not exist.\textsuperscript{34} In one study, researchers used as many as five common evaluations of concussions that included the Automated Neuropsychological Assessment Metrics, Sensory Organization Test, Standardized Assessment of Concussion, and

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Balance Error Scoring System, and Graded Symptom Checklist.35 Yet, physicians use their discretion to issue a “clinical judgment call.”36

The disarray in evaluating whether a concussion occurred carries over to the grading system and what qualifies as a concussion. Despite the mélange of classification systems, the Glasgow Coma Scale (GCS) is one of the most widely used systems.37 Although criticized as overly complex, a GCS score estimates a person’s conscious state and extent of brain injury.38

35 Sonia M. Gysland et al., *The Relationship Between Subconcussive Impacts and Concussion History on Clinical Measures of Neurologic Function in Collegiate Football Players*, Springer (Oct. 13, 2011), http://link.springer.com/article/10.1007/s10439-011-0421-3/fulltext.html. (The Automated Neuropsychological Assessment Metrics (ANAM) battery is a computer-based test consisting of subtests evaluating different neurocognitive functions; The Standardized Assessment of Concussion (SAC) assesses domains commonly affected by mild traumatic brain injury such as orientation, immediate and delayed memory, and concentration; and additionally allows for a neurologic exam and clinical evaluation of symptoms with exertion; The Sensory Organization Test assessed participants’ balance performance during baseline testing administered prior to preseason, as well as following the conclusion of the season; Balance Error Scoring System (BESS) consists of three stances (double, single, and tandem) completed once on a firm surface and repeated on a medium density foam pad for a total of six, 20-s trials; Participants were asked to fill out the Graded Symptom Checklist (GSC), which allowed them to self-report symptoms using a seven-point Likert scale ranging from asymptomatic (0) to mild (1) to severe (6); The Head Impact Telemetry (HIT) System consists of six spring-mounted single axis accelerometers embedded within Riddell VSR-4 and Revolution football helmets used to track the frequency, location, and magnitude of impacts to the players’ helmets.) (internal quotations omitted)

36 Bernstein, supra note 7.

37 Segun T. Dawodu, *Traumatic Brain Injury (TBI) - Definition, Epidemiology, Pathophysiology*, Medscape, http://emedicine.medscape.com/article/326510-overview. (The Glasgow Coma Scale (GCS) defines the severity of a TBI within forty-eight hours of injury based upon the cumulative total of three factors:

1. **Eye opening:** Spontaneous = 4, To speech = 3, To painful stimulation = 2, No response = 1

2. **Motor response:** Follows commands = 6, Makes localizing movements to pain = 5, Makes withdrawal movements to pain = 4, Flexor (decorticate) posturing to pain = 3, Extensor (decerebrate) posturing to pain = 2, No response = 1

3. **Verbal response:** Oriented to person, place, and date = 5, Converses but is disoriented = 4, Says inappropriate words = 3, Says incomprehensible sounds = 2, No response = 1

The severity of TBI according to the GCS score (within 48 h) is as follows: Severe TBI = 3-8, Moderate TBI = 9-12, Mild TBI = 13-15

An examiner scores a response or non-response from each of three categories: eye opening, motor response, and verbal response.\(^3^9\) Next, the examiner adds and assesses the scores from each category.\(^4^0\) Usually, examiners pair the GCS with another system, the Rancho Los Amigos Scale (also known as the Rancho Los Amigos Levels of Cognitive Functioning Scale (LOCF) or Rancho Scale) to gauge subtle emotional or cognitive issues.\(^4^1\) The LOCF

\(^{3^9}\) Id.

\(^{4^0}\) Id.

\(^{4^1}\) *Family Guide to The Rancho Levels of Cognitive Functioning, RANCHO LOS AMIGOS NAT'L REHAB. CTR.* available at http://www.rancho.org/research/bi_cognition.pdf. (The severity of deficit in cognitive functioning can be defined by the Ranchos Los Amigos Scale:

- **Level I**
  No Response. Patient does not respond to external stimuli and appears asleep.

- **Level II**
  Generalized Response. Patient reacts to external stimuli in nonspecific, inconsistent, and non-purposeful manner with stereotypic and limited responses.

- **Level III**
  Localized Response. Patient responds specifically and inconsistently with delays to stimuli, but may follow simple commands for motor action.

- **Level IV**
  Confused, Agitated Response. Patient exhibits bizarre, nonpurposeful, incoherent or inappropriate behaviors, has no short-term recall, attention is short and nonselective.

- **Level V**
  Confused, Inappropriate, Non-agitated Response. Patient gives random, fragmented, and non-purposeful responses to complex or unstructured stimuli. Simple commands are followed consistently, memory and selective attention are impaired, and new information is not retained.

- **Level VI**
  Confused, Appropriate Response. Patient gives context appropriate, goal-directed responses, dependent upon external input for direction. There is carry-over for relearned, but not for new tasks, and recent memory problems persist.

- **Level VII**
  Automatic, Appropriate Response. Patient behaves appropriately in familiar settings, performs daily routines automatically, and shows carry-over for new learning at lower than normal rates. Patient initiates social interactions, but judgment remains impaired.

- **Level VIII**
  Purposeful, Appropriate Response. Patient oriented and responds to the environment but abstract reasoning abilities are decreased relative to premorbid levels.
score describes the individual’s unique behavioral characteristics and cognitive deficits in order to design a well-tailored treatment program.\textsuperscript{42} 

\textbf{B. The Monster’s Shadow} 

Concussions signal the beginning of future, more serious neurological, psychological, and emotional complications. When a concussion occurs, brain cells depolarize, which cause neurotransmitters to behave abnormally.\textsuperscript{43} Typically, the brain recovers over time, depending on the severity of the concussion.\textsuperscript{44} Unfortunately, the brain never returns to its former state, prior to the concussion, because the brain cannot regenerate.\textsuperscript{45} 

In some cases, concussive side effects persist for an extensive period of time, a condition known as Post-Concussive Syndrome (PCS).\textsuperscript{46} There is controversy over whether PCS exists and what defines it.\textsuperscript{47} Similar to the disagreement over the definition of concussion, various organizations propose different definitions for PCS.\textsuperscript{48}

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\textsuperscript{42} Id.\textsuperscript{43} Nigel A. Shaw, \textit{Neurophysiology of Concussion: Theoretical Perspectives}, SPRINGER (2006), http://www.springerlink.com/content/k7xp40310537020r/. (Couldn’t view the document)\textsuperscript{44} National Institute of Neurological Disorders and Stroke, supra note 26.\textsuperscript{45} Robert C. Cantu, \textit{Return to Play Guidelines After a Head Injury}, 17(1) CLINICS IN SPORTS MED. 45 (1998), available at http://www.udel.edu/PT/PT%20Clinical%20Services/journalclub/sojc/99_00/oct99/cantu.pdf.\textsuperscript{46} Barry Willer & John J. Leddy, \textit{Management of Concussion and Post-Concussion Syndrome}, 8 CURRENT TREATMENT OPTIONS IN NEUROLOGY 415,417 (2006), available at http://ortholab.buffalo.edu/management%20of%20concussion%20and%20post%20concussion%20syndrome.pdf. (The period of time is not defined because PCS can appear as short as after three weeks or persist for months and years afterwards.)\textsuperscript{47} Annette Sterr, et. al., \textit{Are Mild Head Injuries as Mild as We Think? Neurobehavioral Concomitants of Chronic Post-concussion Syndrome}, BMC NEUROLOGY (2006), http://www.biomedcentral.com/1471-2377/6/7/. (“The clinical validity of these sequelae [PCS] is not undisputed, in part because there is no readily evident physiological damage or deficit that could be made accountable.”)\textsuperscript{48} See, e.g., Ryan C.W. Hall, et al., \textit{Definition, Diagnosis, and Forensic Implications of Postconcussional Syndrome}, 46 PSYCHOSOMATICS, 195, 195-202 (2005), available at http://drryanhall.com/Articles/postconcussional%20syndrome.pdf. (outlining the American Psychiatric Association’s current criteria for postconcussional disorder, and defining a concussion as an “acquired impairment in cognitive functioning, accompanied by specific neurobehavioral symptoms, that occurs as a consequence of a closed head
Until the brain completely heals, it is in an extremely vulnerable state. If disturbed, the patient may develop more severe brain injuries such as Second Impact Syndrome (SIS) or CTE. SIS often results in death. If an athlete suffers a second brain injury after a concussion, the second injury opens holes within the skull, allowing the herniated portions of the brain to squeeze through. In as little as fifteen seconds, severe cerebral vascular swelling occurs and may result in paralysis, coma, and death. The mortality rate is 50% while the remaining one-half that survive frequently suffer a permanent disability.

More common is CTE, a condition caused by repetitive brain trauma. Originally known as dementia pugilistica or less formally as “punch drunk syndrome” from the associated condition seen in boxers, multiple head injuries eventually lead to injury of sufficient severity to produce a significant cerebral concussion."; Randolph W. Evans, The Postconcussion Syndrome and the Sequelae of Mild Head Injury, 10 NEUROLOGIC CLINICS 815 (1992) (defining it as “a condition arising after 'head injury' that produces deficits in three areas of central nervous system functioning: 1) somatic (neurological—usually headache, tendency to become fatigued), 2) psychological (affective change, lack of motivation, anxiety, or emotional ability), and 3) cognitive (impaired memory, attention, and concentration).”; Postconcussional Syndrome, International Statistical Classification of Diseases and Related Health Problems 10th Revision, WORLD HEALTH ORGANIZATION Sec. V, 7.2, available at http://apps.who.int/classifications/icd10/browse/2010/en#F07.2. (defining a concussion as “or as generally as ‘a number of disparate symptoms such as headache, dizziness, fatigue, irritability, difficulty in concentration and performing mental tasks, impairment of memory, insomnia, and reduced tolerance to stress, emotional excitement, or alcohol.”).


50 Matthew Thomas et. al., Epidemiology of Sudden Death in Young, Competitive Athletes Due to Blunt Trauma, 128 OFFICIAL J. OF PEDIATRICS 1, e1 –e9(2011), available at http://pediatrics.aappublications.org/content/128/1/e1.full.pdf+html. (Doctors evaluated 1,827 cases of young athlete fatalities listed in the National Registry of Sudden Death in Athletes from 1980 to 2009. The team determined that 261 football players died from trauma-related injuries, 14 percent of all the fatal injuries, followed head and neck blows which were subsequent to concussions sustained weeks prior).

51 Id.


53 John Whisler, Fighting for Safety, SAN ANTONIO EXPRESS, Feb. 27, 2004, at 1C.

neurodegeneration. Once brain tissue decays, an abnormal protein called tau builds up in a tangle creating a pattern unique only to CTE. Tau protein accumulates in clumps until it starts to disrupt the brain's normal function. Experts estimate changes to the brain begin anywhere from “months, years, or even decades after the last concussion or end of active athletic involvement.” What results is a condition eerily similar to Alzheimer’s disease and results in memory loss, depression, and poor impulse control. Some believe that CTE causes motor neuron diseases similar to amyotrophic lateral sclerosis, commonly known as Lou Gehrig’s disease.

Some doctors believe that CTE has three stages: first, problems with attention, concentration, memory, and confusion; second, more exaggerated behavioral symptoms such as changes in social behavior, erratic behavior, and problems with judgment; and third more severe cognitive deficits and dementia. The severity of CTE directly corresponds to the number of traumatic head injuries an athlete sustains and the duration of his athletic career.

58 Chronic Traumatic Encephalopathy, supra note 28.
59 Lakhan & Kirchgessner, supra note 55.
61 Lakhan & Kirchgessner, supra note 55.
career. Even after retirement from the sport, CTE continues to progress and is irreversible.

II. POSSIBILITY OF RECOVERY

“Vita brevis, ars longa, occasiopraeceps, experimentumpericulosum, iudiciumdifícile.”

A. Current Treatments Offered

Every brain is different. It is difficult to accurately predict concussion recovery time and/or lessen the physical, emotional, and mental symptoms. The brain requires rest in order to recover from injury. The 2008 Zurich guidelines recommend avoiding all stimuli requiring concentration, including television, video games, exercise, text messaging, online browsing, and work. Although seemingly harmless, the injured brain cannot perform these activities without strain, which worsens symptoms and prevents recovery. Premature resumption of normal

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63 McKee, supra note 62.
64 Id.
65 “Life is short, and Art long; the crisis fleeting; experience perilous, and decision difficult.” In his famous book of aphorisms, Hippocrates advises physicians in the practice of their “Art.” Burton Chance, On Hippocrates and the Aphorisms, 2 ANNALS OF MEDICAL HISTORY 31 (1930).
66 CDC delayed recovery based on factors like age; gender; a history of prior concussion; a history of headache or post concussive headache; premorbid developmental disorders; and psychiatric history. Facts About Concussion and Brain Injury: Where to Get Help, www.cdc.gov/concussion/pdf/Facts_about_Concussion_TBI-a.pdf.
activities cause increased psychological distress from decreased efficiency, interpersonal problems, negative feedback, and poor performance.\textsuperscript{71} Most injured people recover over time;\textsuperscript{72} however, if symptoms re-emerge, the patient should stop all activities that strain the brain to allow it to rest.\textsuperscript{73}

For the small percentage of people who continue to suffer from post-concussive syndrome, recovery time reduces the severity of the symptoms and decreases the effects of symptoms on daily life.\textsuperscript{74} Recovery depends on post-concussive symptoms that stem from somatic, cognitive, emotional, and sleep disturbances.\textsuperscript{75} Non-pharmaceutical treatment is first attempted to help with post-concussive syndrome. For somatic symptoms\textsuperscript{76} like “headaches, nausea, vomiting, dizziness, balance difficulty, light and sound sensitivity, numbness, and tingling,” a variety of options exists. For those unable to tolerate light and sound, sunglasses or earplugs minimize sensory perception.\textsuperscript{77} With the wide range of headaches, doctors take particular care to distinguish the cause of headaches. Massage and other physical therapies relieve cervicogenic headaches, caused by tension or stress in the neck. Daily neck exercises relieve pain and increase mobility.

For cognitive symptoms that include “fogginess,” difficulty concentrating, memory deficits, and cognitive fatigue, cognitive rehabilitation therapy (“CRT”) might help. CRT restores, or at least compensates for, the individual’s cognitive abilities by teaching tailored skills-based and process-based techniques.\textsuperscript{78} For example, a person deficient in executive function may be unable to plan, set goals, solve problems, reason, strategize, manage time appropriately, anticipate consequences, multi-task, and demonstrate general awareness.\textsuperscript{79} CRT might require lessons of formal problem-solving wherein participants develop a plan to

\begin{itemize}
\item Id.
\item Id.
\item Id.
\item Id.
\item Id.
\item Id.
\item Sabini & Reddy, supra note 70.
\item Id. at 78.
\item Id. at 137.
\end{itemize}
accomplish a task that requires listing every step necessary to solve it.\textsuperscript{80}

In severe cases, physicians turn to pharmacological measures as a last resort.\textsuperscript{81} Although the United States Food and Drug Administration has not approved any medicine for post-concussive neurological or psychiatric side effects,\textsuperscript{82} there are approved treatments available for symptoms, which, if used “appropriately and cautiously can improve neurological and functional outcome.”\textsuperscript{83}

Doctors prescribe medications sparingly to avoid complicating effects. In such cases, acetaminophen treats most headaches and muscle pains. In extreme cases, doctors prescribe Propranolol, Verapamil, Amitriptyline, Escitalopram (Lexapro), and Sertraline (Zoloft). Cognitive problems do not typically require medication, but in cases of prolonged neurocognitive deficiency, three neurostimulants, Amantadine, Methylphenidate, and Atomoxetine (Strattera), are commonly used.\textsuperscript{84} Amantadine improves focus and sustained attention and concentration, orientation, alertness, arousal, processing, time, psychomotor speed, mobility, vocalization, agitation, anxiety and participation in therapy.\textsuperscript{85}

Doctors employ behavioral therapy before treatment with medication,\textsuperscript{86} which reduces the risk of side effects that cause mood shifts such as irritability, anxiety, sadness, nervousness, personality change, depression, temper or aggression.\textsuperscript{87} Doctors often treat extreme cases of mood disturbances with

\textsuperscript{80} Id. at 154.

\textsuperscript{81} Paul McCrory, Should We Treat Concussion Pharmacologically?, 36 BRIT. J. OF SPORTS MED. 3 (2002) (evidence is only based upon studies of severe brain injury yet pharmacological solutions are proposed for all grades of concussions.)


\textsuperscript{83} Id.

\textsuperscript{84} PEDIATRIC AND ADOLESCENT CONCUSSION: DIAGNOSIS, MANAGEMENT, AND OUTCOMES 111 (Jennifer Niskala Apps & Kevin D. Walter, eds., 2011).


antidepressants. The most common treatments include citalopram, fluoxetine, sertraline, valproic acid, carbamazepine, or norepinephrine inhibitors.

Sleep dysfunctions overlap and affect other areas of brain function, notably concentration, behavior and emotional well-being. Sleep studies determine the degree of difficulty in falling or staying asleep; otherwise, the emphasis should be on proper sleep hygiene. If sleep hygiene measures fail, medication is the next step. Some patients use natural, over-the-counter remedies like melatonin and valerian. Diphenhydramine, a common ingredient in allergy medicine, also increases sleep while preventing drug addiction and rebound insomnia. If stronger medication is necessary, then some doctors recommend trazodone. Doctors avoid prescription sleep medicines such as zolpidem (Ambien) and eszopiclone (Lunesta) because they increase post-concussion symptoms.

B. Medical Advances

Read my lips: no new neurons.

-- Pasko Rakic, Yale University neuroscientist

88 Id. ("SSRIs/NRIs should be avoided secondary to prolonged titration and weaning schedules. They can also be sedating and have a higher risk of suicide in adolescents and late teens.")
89 See Sabini & Reddy, supra note 70.
90 Id.
91 Id.
92 Id. ("A strict sleep schedule should be maintained by going to bed and waking up at the same time every day. The patient should be instructed to get out of bed if unable to sleep within 30 minutes. Napping during the day should also be restricted to 30 minutes. Eating, reading, or watching television while in bed should be avoided. Caffeine should not be consumed in the late afternoon, nor should exercise be performed in the evening, because both can increase wakefulness.")
93 Id.
94 See generally Conidi, supra note 86.
96 Id.
97 Conidi, supra note 86.
98 Id.
I think the fact that there are so many neurons that are produced . . . suggests that they must play some important function, because it wouldn’t make sense for the brain to expend so much energy to make these new cells if they’re not going to be used.

-- Dr. Elizabeth Gould

It all started with a rat. Joseph Altman and Gopal Das stumbled upon the scientific discovery of a lifetime: proof that rats could regenerate brain cells. Their 1960’s theory challenged the prevailing understanding that the number of brain cells present in a creature at birth remain fixed from birth. Yet, their colleagues ridiculed them mercilessly. Although a few scientists vindicated their results, the theory of neurogenesis faded into obscurity.

In 1981, Fernando Nottebohm expanded upon Altman and Das’ original research. Canaries’ brains grow and shrink directly in response to their brain functions. During the spring mating season, which requires learning and rehearsing new songs, canary brains create neurons. When canaries do not need to use their musical ability, their brain cells die off. The canary’s pattern of growth and shrinkage repeats for the bird’s entire life.

Again, although Nottebohm’s novel research confirmed the theory of neurogenesis, other scientists dismissed his findings as aberrations in nature; rats and canaries were nothing like

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102 Id.
103 Id.
105 Id.
107 Id.
108 Id.
109 Id.
110 Id.
111 Id.
humans.\textsuperscript{112} Then in 1998, Elizabeth Gould discovered that mankind’s closest relative, the primate, could create new brain cells.\textsuperscript{113} In addition, she determined that external environmental factors affect neurogenesis.\textsuperscript{114} By forcing a monkey who always lived alone into the cage of another monkey, Dr. Gould artificially induced stress.\textsuperscript{115} After one hour, she removed the intruder monkey from the foreign cage.\textsuperscript{116} The brief stressful exposure caused fewer new brain cells to develop.\textsuperscript{117} Dr. Gould found that if you “[p]ut a primate under stressful conditions, and its brain begins to starve. It stops creating new cells. The cells it already has retreat inwards. The mind is disfigured.”\textsuperscript{118}

Standing on the shoulder of giants, Fred Gage and Gerd Kempermann connected the last link in the theory of neurogenesis to adult humans.\textsuperscript{119} They injected Bromodeoxyuridine (‘BrdU’) into terminally ill cancer patients to monitor tumor growth.\textsuperscript{120} BrdU also highlighted new brain cells that formed after an injection in the dentate gyrus hippocampal area.\textsuperscript{121} The hippocampus assists in the formation of memories, crucial for mental focus and concentration.\textsuperscript{122}

Despite their breakthrough research, many unanswered questions still surround the area of neurogenesis. In adults, neurogenesis predominantly occurs in the hippocampus.\textsuperscript{123} What do the new brain cells do?\textsuperscript{124} It may be that adult neurogenesis does not affect brain function since there is no connection to the

\begin{itemize}
\item \textsuperscript{112} \textit{Id.}
\item \textsuperscript{113} Elizabeth Gould, et al., \textit{Neurogenesis in the Neocortex of Adult Primates}, 286 \textit{SCIENCE} 548 (1999).
\item \textsuperscript{114} Elizabeth Gould, Proliferation of Granule Cell Precursors in the Dentate Gyrus of Adult Monkeys is Diminished By Stress, \textit{95 PROC. NATL. ACAD. SCI.} 3168 (1998).
\item \textsuperscript{115} \textit{Id.}
\item \textsuperscript{116} \textit{Id.}
\item \textsuperscript{117} \textit{Id.}
\item \textsuperscript{118} Jonah Lehrer, \textit{The Reinvention of the Self}, \textit{SEED}, February-March 2006, available at \url{http://seedmagazine.com/content/article/the_reinvention_of_the_self}.
\item \textsuperscript{119} Fred H. Gage and Gerd Kempermann, \textit{New Nerve Cells for Adult Brain}, \textit{SCIENTIFIC AM.} 280.5 (May 1999).
\item \textsuperscript{120} \textit{Id.}
\item \textsuperscript{121} \textit{Id.}
\item \textsuperscript{122} Michael M. Zeineh, et al., \textit{Unfolding the Human Hippocampus With High Resolution Structural and Functional MRI}, \textit{THE ANATOMICAL RECORD}, Apr. 2001, at 111-120.
\item \textsuperscript{123} Kiester & Kiester, \textit{supra} note 99.
\item \textsuperscript{124} \textit{Id.}
\end{itemize}
cerebral cortex, where higher thoughts are processed. Why the hippocampus? Scientists strive to answer whether new brain cells can grow elsewhere in the brain. If neurogenesis is possible outside of the hippocampus, how can doctors artificially stimulate growth when the brain sustains injuries or needs repairs? Hopefully, research will one day answer all of these questions and provide a cure.

III. THE HITS THAT CHANGE THE GAME

There isn’t a lot of data, and the experts who we talked to, who consult with us, think that it’s way premature to be drawing any conclusions at this point . . . Because we’re not sure that any, based on the data we have available, is valid.

- Gary Bettman, Commissioner of the NHL

As Ann McKee explained,

Do we expect 100% of cigarette smokers will develop lung cancer? Do we expect 100% of children who play with matches or even chain saws will get hurt? No. Even if the percentage of affected players is 20%, or 10%, there are still thousands of kids and adults out there, right now, playing football at all levels -who will eventually come down with this devastating and debilitating disorder. And as a doctor and as a mother, I think this calls for immediate action.

125 Id.
126 Id.
128 Id.
129 Branch, supra note 14.
A. Law & Order: Federal & State Legislation

Inspired by Zackery Lystedt’s tragic concussion story, the state of Washington passed the Lystedt Law in 2009. It requires athletes, parents, and coaches to receive education related to concussions, removal of the suspected-concussed athlete from a game or practice with no option to return to play, and health care professional clearance before resuming the sport. As of April 2012, thirty-six states adopted laws on concussions and nine states have pending legislation.

Although the Lystedt Law increases awareness about concussions and sets some guidelines, it is far from comprehensive. The provision requiring a statement of compliance that confirms private, non-profit youth programs will adhere to the school district’s management policies leaves a loophole. If the sports programs use non-school district properties, they receive an exemption from the compliance statement. It is not mandatory that a private sport program’s coaching staff receive education related to concussions, removal of the suspected-concussed athlete from a game or practice with no option to return to play, and health care professional clearance before resuming the sport.

The Lystedt Law: A Concussion Survivor’s Journey, www.cdc.gov/media/subtopic/matte/pdf/031210-Zack-story.pdf. Zack Lystedt’s story emphasizes why ‘shaking it off’ puts players at risk for serious injury. Zack, a gifted athlete who played both offense and defense on his junior high school football team, was injured at 13 when his head struck the ground after tackling an opponent. A video of the game shows Zack lying on the ground with his hands clutching both sides of his helmet. The official called a time out, and Zack was sidelined for just three plays before half-time. After a hard-played 2nd half, Zack collapsed on the field and was airlifted to Harborview Medical Center where he underwent emergency life-saving surgery to remove the left and right side of his skull to relieve the pressure from his injured and swelling brain. He experienced numerous strokes, seven days on a ventilator and three months in a coma before he awoke to his parents and a new reality. Prematurely returning to the game had resulted in the battle for Zack’s young life, including four weeks in a nursing home, two months in a children’s hospital for rehabilitation, nine months before he spoke his first word, thirteen months before he moved a leg or an arm, and twenty months on a feeding tube. It would be nearly three years before Zack would stand, with assistance, on his own two feet.


Id.


Id.
concussion education ordinarily mandatory for the public school district’s coaches.\textsuperscript{137} Furthermore, there are no sanctions for non-compliance.\textsuperscript{138}

Even more troubling, the Lystedt Law lumps licensed physicians, athletic trainers, and nurse practitioners together with others that determine whether an injured athlete can return to practice or a game.\textsuperscript{139} Despite the licensing requirement that health care providers become familiar with the evaluation and management of concussions, the difference between physicians and athletic trainers is obvious.\textsuperscript{140} Physicians are professionals licensed in the study of medicine, unlike athletic trainers who may not receive training regarding concussions and their symptoms. Some concern about the limitation on liability for injuries associated with return to play decisions by non-school district physicians may be valid.\textsuperscript{141} Since every brain is unique, the lack of a baseline test for each athlete at the start of every sports season adds to the confusion.\textsuperscript{142} Even well-intending physicians fail to accurately assess a player’s status after an apparent concussion and make return-to-play decisions.\textsuperscript{143}

Currently, no federal laws exist to force the remaining five states without legislation\textsuperscript{144} to adhere to concussion laws.\textsuperscript{145} The proposed Concussion Treatment and Care Tools Act of 2009 ("ConTACT") would establish concussion management guidelines for “the prevention, identification, treatment, and management of concussions in school-aged children, including standards for student athletes to return to play after a concussion.”\textsuperscript{146} Unfortunately, the ConTACT bill died in Congress.\textsuperscript{147} A similar bill titled the Protecting Student Athletes from Concussions Act of

\textsuperscript{137} Id.
\textsuperscript{138} Id.
\textsuperscript{139} Id.
\textsuperscript{140} Id.
\textsuperscript{141} Id.
\textsuperscript{142} Id.
\textsuperscript{143} Id.
\textsuperscript{144} Id. (Nevada, Montana, Arkansas, Michigan, and Georgia).
\textsuperscript{146} Id.
2010 proposed that public school districts create their own concussion management and treatment plans. This bill also failed.

The closest thing to federal intervention is the “Heads Up” toolkit from the federally funded CDC. Although not mandatory, the “Heads Up” toolkit educates physicians about the severity of concussions. Recipients of the “Heads Up” toolkit were less likely to immediately return concussed athletes to practice and games.

B. Reaction to State Legislation

A year after Lystedt’s Law, the National Football League (“NFL”) released new rules to minimize head injuries. The new rules stated that (1) no player may launch himself from the ground to strike others in the head or neck with his helmet; once a player loses his helmet, the play stops, and, (3) during field-goal and extra-point attempts, the defense cannot situate any player directly across from the snapper. The NFL also created stricter return-to-play guidelines for concussed football players that required a consultation with an independent neurologist.

In 2011, perhaps in response to litigation by former NFL players, the NFL further increased football player safety by

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148 H.R. 6172, 111th Cong. (2nd Sess. 2010).
149 Id.
151 Sara P. Chrisman, Physician Concussion Knowledge and the Effect of Mailing the CDC’s “Heads Up” Toolkit 50(11) CLINICAL PEDIATRICS 1031 (Nov. 2011).
152 Id.
154 Id.
155 Id.
156 Id.
157 Id.
enacting additional rules.\textsuperscript{159} The NFL increased defenseless neck and head launching penalties to fifteen yards and created rules allowing officials to remove the offender from the game.\textsuperscript{160} In addition, team fines for too many illegal hits,\textsuperscript{161} coupled with the fact that kick-offs now take place at the 35-yard line instead of the 30-yard line help limit contact.\textsuperscript{162} Also, coverage teams can only have 5-yards of run-up instead of 15-yards.\textsuperscript{163}

Although not mandatory, the NFL distributed a strongly worded memo with the “Madden Rule”\textsuperscript{164} that influenced current practices. The coach must remove any player suspected of suffering a concussion, even if not formally diagnosed.\textsuperscript{165} A medical staff member must observe the player to determine whether immediate hospitalization is necessary and the player cannot return to the field under any circumstances.\textsuperscript{166} Following the NFL’s lead, the National Collegiate Athletic Association (“NCAA”) changed its rules to favor player safety.\textsuperscript{167} Much like the NFL, it too faces class action suits from former football players.\textsuperscript{168} Since August 2010, the NCAA required each university to develop its own concussion management plan.\textsuperscript{169} In response, the NCAA adopted new rules: kickoff and touchback starting lines moved, loss of helmet stops play, no blocking below the waist, shield-blocking scheme on punting plays, and increased protection to

\textsuperscript{160} Id.
\textsuperscript{161} Id.
\textsuperscript{162} Id.
\textsuperscript{163} Id.
\textsuperscript{165} Id.
\textsuperscript{166} Id.
\textsuperscript{167} See generally, NCAA History, http://www.ncaa.org/wps/wcm/connect/public/ncaa/about+the+ncaa/who+we+are/about+the+ncaa/history.
They also moved the kickoff to the thirty-five yard line. To limit the running start that kicking teams have during the play, the NCAA shortened the distance of kicking to five yards from the thirty-five yard line. Also, the NCAA hopes to encourage more touchbacks by moving the touchback distance on free kicks to the twenty-five yard line.

If a player loses his helmet, he must leave the game and cannot participate in the next play. Offensive players who are not in motion can block below the waist legally without restriction. Otherwise, no one may block below the waist. In cases of shield blocking, the receiving team cannot jump over blockers unless the player jumps straight up or between two players. Kick returners must complete a catch before the kicking team can make contact.

Given that younger athletes are more vulnerable to concussions, less likely to have medical oversight during practice and games, and more likely to have longer periods of recovery and suffer worse post-concussive effects if injured, the National Federation of State High School Associations (“NFSH”) implemented similar rule changes. NFSH justifies mandatory time-outs if helmets are lost as an incentive for schools to fit players properly with helmets while discouraging players from tampering with the fit.

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171 Id.

172 Id.

173 Id.

174 Id.

175 Id.

176 Id.

177 Id.

178 Id.


180 High School Football Players Must Sit Out One Play When Helmet Comes Off, (Feb. 9, 2012),http://www.nfhs.org/content.aspx?id=6588

181 Id.
C. Solutions

What is the best way to avoid a concussion? Don’t get hurt. Banning sports all together would eliminate the second leading cause of concussions.\textsuperscript{182} Such an action even has precedence in history.\textsuperscript{183} Yet, few would advocate for universal application. For this reason, other solutions will be the focus of this section.

Improvement to athletic equipment, particularly helmets and mouth guards, are thought to be crucial in preventing concussions.\textsuperscript{184} On average, helmets reduce concussions by thirty-three percent.\textsuperscript{185} Some simple measures like ensuring the helmet has a tight, snug fit on players reduces the risk of brain injury.\textsuperscript{186} Most players do not wear their helmets properly, evidenced by their easy removal at the sidelines.\textsuperscript{187} No helmet can completely prevent brain injury, even if the fit is perfect.\textsuperscript{188} However, helmets reduce skull fractures and hematomas.\textsuperscript{189} Similarly, mouth guards effectively prevent dental injuries.\textsuperscript{190} It is unlikely that mouth guards will prevent concussions, however, since the thickest mouth guard absorbs only 2\% of the blow.\textsuperscript{191}

\textsuperscript{182} Mallika Marar, et al., Epidemiology of Concussions Among United States High School Athletes in 20 Sports, 40 AM. J. SPORTS MED. 747 (2012).

\textsuperscript{183} Herbert Willis, A Synopsis of Scottish History: or Historical Note-book, 61 (1885). http://books.google.com/books?id=Dj4IAAAAQAAJ&pg=PA61&dq=football%20act%201424%20scotland&hl=en&sa=X&ei=dh6cUL7XDInwyQHSvHAAg&ved=0CDwQ6AEwBA#v=onepage&q=football%20act%201424%20scotland&f=false (11/12/12). “is statute and the king forbids that no man at the football under the pain of id to be raised to th the lord the land as oft as he be tainted or to the sheriff of the land or ministers if the lords will not punish such trespassers”

\textsuperscript{184} Alexander N. Hecht, Legal and Ethical Aspects of Sports-Related Concussions: The Merril Hoge Story, 12 SETON HALL J. SPORTS L. 17, 59 (2002).

\textsuperscript{185} Mickey Collins, Examining Concussion Rates and Return to Play in High School Football Players Wearing Newer Helmet Technology: A Three-Year Prospective Cohort Study, Vol. 58 No. 2 NEUROSURGERY (Feb. 2006), 275-86.


\textsuperscript{187} Id.

\textsuperscript{188} Id.


\textsuperscript{190} David C. Viano, Chris Withnall, and Michael Wonnacott. Effect of Mouthguards on Head Responses and Mandible Forces in Football Helmet Impacts, Vol. 4 No. 1 ANNALS OF BIOMEDICAL ENGINEERING, Jan. 2012, 47.

\textsuperscript{191} Id.
Conversely, improved equipment may result in more aggressive behavior, thus, increasing the probability of head injury. Experts believe that “coaching reforms and stricter officiating will do more to reduce concussion incidence than any type of headgear.” Coaches hold enormous sway over the management of their players with their ability to change starting positions in the lineup. Fewer contact drills and practices reduce the risk of concussion indirectly by lessening the effect of accumulated concussive hits. Praising helmet-to-helmet contact unnecessarily glamorizes the risk of concussions without mentioning any of the side-effects.

Changing the power position of the players encourages transparency and honesty about injuries. As it stands, NFL players have few incentives to admit to their brain injuries. For example, an injured NFL player’s contract guarantees payment only for the season of the injury. He must pass the physical exam next season in order to receive payment. Even assuming the injured player plays through the pain in order to pass the qualifying physical exam, the NFL has the power to terminate contracts at will if the team can find another player to replace him. Some teams restructure the contract in their favor knowing that employment opportunities are unlikely elsewhere with a brain injury.

Another viable option is extending sport seasons. The current industry standard of one “bye week” is insufficient. If the NFL mandated that a player could not play for two consecutive weeks,
player self-reporting issues for minor concussions would disappear. Players would have more time to rest and recuperate from all injuries. Coaches would not use the obligatory rest week for more strenuous practices because they could not use those players in the next game. Opportunities for injuries decrease.

From a monetary perspective, the advantages of extending the sport season outweigh any possible disadvantages. Having one week on and one week off would force teams to cultivate a larger talent base. Replacing injured starters would not be as difficult; backbenchers would be more accustomed to playing. Games would be more unpredictable. TV contract revenue would increase. Fans would still be able to follow their teams weekly. Fewer injured players translate to less money spent on healthcare and retirement. Unlike the current “bye week” system, every team would have a “bye week” at the same time and be evenly matched. The proposed solution of bi-weekly game time is radical. Rule changes like lifting roster limits would be required. Fans may not have the patience to follow a much longer season. They may be loyal to certain players, not teams.

CONCLUSION

The sports industry is a $422 billion dollar factory. When everyone from the coaches to the team physicians has an economic stake in the outcome, business mixes with health. This problem compounds as physicians disagree amongst themselves on the fundamentals of concussions: definitions, diagnoses, and treatments. With so much uncertainty in medicine, the NFL cannot effectively impose a legal regime to guide players and the NFL. Perhaps this explains why no federal legislation exists or why the State of Washington’s Lystedt Law is so ambiguous and weak. Without laws on concussions and return to play, it seems unlikely that the NFL and the players will change current concussion practices.

There are alternatives to current prevention methods, treatments and rehabilitation programs. These alternatives, such

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as helmet safety and stricter rules regarding concussions, can significantly improve a player’s chance of recovery or at least reduce the likelihood of severe damage. Despite the existence of these alternatives, players, sports organizations and coaches fail to reduce the occurrence and severity of concussions because they each have overlapping interests in their sport: fame, money, and packed stadiums.

APPENDIX 1:

<table>
<thead>
<tr>
<th>Common Symptoms Following Concussion/mTBI</th>
<th>Pharmacologic Treatment</th>
<th>Non-Pharmacologic Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headaches</td>
<td>- Non narcotic pain meds</td>
<td>- Sleep hygiene education</td>
</tr>
<tr>
<td></td>
<td>- NSAIDs</td>
<td>- Physical therapy</td>
</tr>
<tr>
<td></td>
<td>- Triptans (migraine type)</td>
<td>- Relaxation</td>
</tr>
<tr>
<td>Feeling dizzy</td>
<td>- Antibiotics, decongestants for infections and fluid</td>
<td>-</td>
</tr>
<tr>
<td>Loss of balance</td>
<td>-</td>
<td>- Physical therapy</td>
</tr>
<tr>
<td>Poor coordination</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Nausea</td>
<td>- Antiemetics</td>
<td>- Sleep hygiene education</td>
</tr>
<tr>
<td>Change in appetite</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Sleep disturbances</td>
<td>- Sleep Medications</td>
<td>- Sleep hygiene education</td>
</tr>
<tr>
<td>- Difficulty falling or staying asleep (insomnia)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Vision problems</td>
<td>-</td>
<td>- Sleep hygiene education</td>
</tr>
<tr>
<td>- Blurring</td>
<td>-</td>
<td>- Light desensitization</td>
</tr>
<tr>
<td>- Trouble seeing</td>
<td>-</td>
<td>- Sunglasses</td>
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<tr>
<td>- Sensitivity to light</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Hearing difficulty</td>
<td>-</td>
<td>- Environmental Modifications</td>
</tr>
<tr>
<td>- Sensitivity to noise</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
### APPENDIX 2:

<table>
<thead>
<tr>
<th>Common Symptoms Following Concussion/mTBI</th>
<th>Job Review</th>
<th>Pharmacologic Treatment</th>
<th>Non-Pharmacologic Treatment</th>
<th>Referral after failed response to initial intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>✓</td>
<td>Stimulant*</td>
<td>Reassurance</td>
<td>- Mental Health</td>
</tr>
<tr>
<td>- Loss of energy</td>
<td></td>
<td></td>
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<tr>
<td>- Getting tired easily</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Cognitive difficulties</td>
<td>✓</td>
<td>SSRI Stimulant*</td>
<td>Encourage regular scheduled aerobic exercise</td>
<td>- TBI specialist for cognitive rehabilitation or mental health</td>
</tr>
<tr>
<td>- Concentration</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>- Memory</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>- Decision-making</td>
<td></td>
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</tr>
<tr>
<td>Feeling anxious</td>
<td>✓</td>
<td>Anxiolytic (short term) SSRI</td>
<td>Activity restriction adjustment</td>
<td>- Mental Health</td>
</tr>
<tr>
<td>Emotional difficulties</td>
<td>✓</td>
<td>Anti epileptics SSRI</td>
<td>Sleep hygiene Education Sleep study</td>
<td>- Social support</td>
</tr>
<tr>
<td>- Feeling depressed</td>
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<tr>
<td>- Irritability</td>
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<tr>
<td>- Poor frustration tolerance</td>
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</table>

* Consider in the specialty care setting after ruling out a sleep disorder
APPENDIX 3:

With the growing attention on the dangers of concussions, more research into resolving post-concussive side-effects are underway. Among the more promising research:

Magnesium:
Essential for optimal functioning of many organ systems. In animal studies, magnesium demonstrates antidepressant properties and makes animals less susceptible to neuronal cell death.\textsuperscript{202} The possible good news is tempered by some evidence that there is no neuroprotective effect and may lead to higher mortality rates.\textsuperscript{203}

Progesterone:
It may “decrease oxidative stress by reducing membrane lipid peroxidation, reduce BBB disruption, and ameliorate the brain’s inflammatory response.”\textsuperscript{204} Currently, the Progesterone for the Treatment of Traumatic Brain Injury (ProTECT\textsuperscript{TM} III) clinical trials are in progress, at phase III.\textsuperscript{205}

Erythropoietin:
It may improve neurological functioning. Clinical trial of the Erythropoietin in Traumatic Brain Injury (EPO-TBI) in progress.\textsuperscript{206}


\textsuperscript{203} Id.

\textsuperscript{204} Id.

\textsuperscript{205} Id. Phase III means “The drug or treatment is given to large groups of people to confirm its effectiveness, monitor side effects, compare it to commonly used treatments, and collect information that will allow the drug or treatment to be used safely.” http://www.nlm.nih.gov/services/ctphases.html; see also University of Pittsburgh which clarifies that this stage allows additional information about the effectiveness on clinical outcomes and evaluate the overall risk-benefit ratio in a demographically diverse sample, available at http://www.clinicalresearch.pitt.edu/docs/comparison_of_clinical_trial_phases.pdf.

\textsuperscript{206} Id.
N-type calcium channel antagonist SNX-111 or Ziconotide:
May reduce calcium accumulation in the cortex and white matter structures and partially restore mitochondrial function. However, a clinical trial terminated because of increased mortality in the treatment group. Different N-type calcium channel antagonists are currently being investigated as alternative.

Kinis, which include substance P and neurokinin A:
NK1 receptor, receptor for substance P, may improve motor and cognitive neurologic prognosis.

Minocycline:
Thought to be an effective antioxidant and reduces inflammation. The study, A Safety and Feasibility of Minocycline in the Treatment of Traumatic Brain Injury, is currently underway.

Cyclosporin:
Might “improve mitochondrial functioning, block free radical production, and inhibit calcium accumulation.” Animal data demonstrate positive therapeutic benefits. Study design variability of study makes results difficult to interpret.

Toll-like receptors:
Key component of the brain’s immune system. Interest in manipulating receptors to reduce the neuroinflammation.

Nicotinamide, a soluble B-group vitamin:
Animal studies demonstrate reduced cortical damage, inflammation, and behavioral disruption. Cytoflavin, a drug containing nicotinamide, improves behavioral and cognitive symptoms in cases of mild brain injury.

Omega-3 essential fatty acids:

207 Id.
May “improve the blood flow, reduce the toxic effects of glutamate, and stabilize membranes.” Animal studies suggest that fatty acids can be supplemented and still protect the brain. Some authors have recommended supplementation with high-dose fish oil in the 2- to 4-g/day range for patients suffering from PCS.

Vitamin E:
Animal study shows neuroprotective effects by reducing lipid peroxidation levels.

Antioxidant, α-lipoic acid:
Animal studies show neuroprotective effects by “reducing inflammatory markers, preserving BBB permeability, and reducing brain edema.”

Resveratrol, which is a polyphenol:
Its antioxidant properties improve behavioral outcome in rats.

α-phenyl-N-tert-butyl nitrone (PBN):
Antioxidant effect and reduces brain inflammation in animal studies.

Zinc protoporphyrin:
Reduces brain edema and BBB permeability in an animal study.

Branched chain amino acids:
May provide a faster healing method by re-balancing levels of potassium, glucose, and calcium.\(^\text{208}\)
