

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

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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 player¹

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.² Even with a tooth knocked out of his jaw, the bloodied

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¹ 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 Boogaard.)

² *Player Statistics*, NHL, <http://www.nhl.com/ice/player.htm?id=8462535>.

Shelley kept fighting.³ 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.⁴

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.⁵ Traditionally, teams believe that the threat of violence is the best way to protect the most valuable players against injury.⁶ Enforcers, also called “hockey goons,” promise physical retaliation against players who partake in cheap play during a game.⁷ Sometimes as part of their job, they resort to violence as a confidence booster for the rest of the team.⁸

During his hockey career, Boogaard suffered the same injuries as a professional fighter.⁹ He broke his nose, jaw, hand, and teeth on numerous occasions, and sustained two bulging discs.¹⁰ Instead of complaining of sensitivity to light, he wore

³ Michael Russo, *Shelley Will Not Back Down From Boogaard*, STAR TRIBUNE (Jan. 2, 2009), http://www.startribune.com/templates/Print_This_Story?sid=36983239.

⁴ *Id.*

⁵ Branch, *supra* note 1.

⁶ *Id.*

⁷ See generally Elizabeth Merrill, *Derek Boogaard Felt the Pain, Too*, ESPN New York (May 29, 2011, 6:10 PM), <http://sports.espn.go.com/new-york/news/story?id=6598296>. (“Look, you may not appreciate his role; you might not even understand it,” Bernstein said. “But without him, you’re not going to win. You take a kicker in football. You might not think they’re athletes, but you’re not going to win without him.”); See also ROSS BERNSTEIN, *THE CODE: THE UNWRITTEN RULES OF FIGHTING AND RETALIATION IN THE NHL* (2006) (need page number and the edition, and perhaps the editors’ name); see also Russo, *supra* note 3 with explanation that enforcers fight not only based upon real grievances, but sometimes premeditated affairs, to settle simmering disputes, intended to reverse the momentum of a lopsided game, and even a way of proving himself to his team.

⁸ Brent Severyn, *An Enforcer’s Life is a Daily Battle*, SI.COM (Sept. 21, 2011), <http://sportsillustrated.cnn.com/2011/hockey/nhl/09/19/brent.severyn.enforcer.life/>. (An enforcer must also have a feel for how a game is unfolding and continually take stock of his team’s emotional state. Are the guys skating well? Do they seem up? If they need a wake-up call, you fight. If the other team has the emotional edge, you fight. The score also determines when you apply your trade. The minute the other team gets a two-goal lead, it’s time to dust off your knuckles as your coach may put you in to stop your opponents’ surge. Up three or more goals, you get more ice time as you have to be out there to keep the peace.)

⁹ Branch, *supra* note 1.

¹⁰ Elizabeth Merrill, *Derek Boogaard Felt the Pain, Too*, ESPN.COM (May 29, 2011, 6:10 PM), <http://sports.espn.go.com/espn/otl/news/story?id=6598296>.

sunglasses to remedy the issue.¹¹ Like the drug-addicted TV character, Dr. Gregory House,¹² Boogaard devoured pills to help cope with the pain.¹³ Suddenly, friends noticed a dark shift in Boogaard's personality.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.¹⁵ Derek Boogaard's behavior resulted from an advanced case of Chronic Traumatic Encephalopathy (CTE), a progressive degenerative brain disease associated with multiple concussions.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.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.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.

¹¹ *Id.*

¹² *See generally* HOUSE, M.D. (Fox Broadcasting Company), <http://www.fox.com/house>

¹³ Branch, *supra* note 1.

¹⁴ John Branch, *Derek Boogaard: A Brain 'Going Bad'*, N.Y. TIMES (Dec. 5, 2011), http://www.nytimes.com/2011/12/06/sports/hockey/derek-boogaard-a-brain-going-bad.html?_r=0&pagewanted=all.

¹⁵ *Id.*

¹⁶ *Id.*

¹⁷ *Id.*

¹⁸ *Id.*

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 Leddy¹⁹

A. Naming the Monster

Doctors cannot agree upon a universally accepted definition of "concussion."²⁰ They bicker amongst themselves and interchangeably use medical terminology like mild traumatic brain injury (MTBI) with "concussion," while others distinguish between the two terms.²¹ The need for proper medical labeling of these conditions matters because calling an injury a "concussion" leads to dismissive medical treatment.²² The diagnosis of a "concussion" instead of "MTBI" often determined whether the

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.

²⁰ James P. Kelly & Jay Rosenberg, *Diagnosis and Management of Concussion in Sports*, 48 NEUROLOGY 575, 576 (1997). (scientific disagreement on "concussion" and "mild traumatic brain injury.")

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.²³ Unfortunately, neurological tests and procedures, such as CTs, MRIs, and EEGs, cannot reveal the presence or effect of a concussion,²⁴ nor can scientific or biological markers highlight the existence of a concussion.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.”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.”²⁷ The Sports Legacy Institute prefers defining a concussion as “trauma-induced change in mental status.”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

²³ *Id.*

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>.

²⁵ Robert Mitchum, *Consensus Difficult to Find: NFL Conclusions Come Under Attack*, CHI. TRIB. (June 20, 2007), http://articles.chicagotribune.com/2007-06-20/sports/0706191174_1_concussions-brain-cells.

²⁶ National Institute of Neurological Disorders and Stroke, <http://www.ninds.nih.gov/>.

²⁷ Stanley A. Herring et al., *Concussion (Mild Traumatic Brain Injury) and the Team Physician: A Consensus Statement*, 43 MED. & SCI. IN SPORTS & EXERCISE 2412–22 (2011), available at http://journals.lww.com/acsm-msse/Fulltext/2011/12000/Concussion_Mild_Traumatic_Brain_Injury_and_the.24.aspx#.

²⁸ *Chronic Traumatic Encephalopathy*, SLI, <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.”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.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.”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.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.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.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,

²⁹ *Injury Prevention & Control: Traumatic Brain Injury*, CTR. FOR DISEASE CONTROL & PREVENTION, <http://www.cdc.gov/concussion/sports/index.html>.

³⁰ *Heads Up: Concussions Aren’t Part of the Game*, AM. ACAD. OF PHYSICAL MED. AND REHAB., <http://www.aapmr.org/patients/conditions/neurologic/brain/Pages/tbiusa.aspx>. (Dr. Zasler also emphasizes that post-concussive disorders do not all appear alike. Hence with no consistent set of symptoms, there is no single formula to treat every patient).

³¹ D. T. Wade et. al., *Routine follow up after head injury: a second randomised controlled trial*, 65 J. OF NEUROLOGY, NEUROSURGERY & PSYCHIATRY 177, 177-83 (1998).

³² David R. Price & Paul R. Leeshaley, *Defending Claims of Postconcussion Syndrome*, 62 DEF. COUNS. J. 589 (1989); *Concussion: Symptoms*, Mayo Clinic, <http://www.mayoclinic.com/health/concussion/DS00320/DSECTION=symptoms>.

³³ *Id.*

³⁴ William O. Roberts, *Who Plays? Who Sits? Managing Concussions on the Sidelines*, 20 PHYSICIAN SPORTSMED, 66, 66-76 (1992).

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

³⁸ Vivek N. Iyer, Jayawant N. Mandrekar, Richard D. Danielson, Alexander Y. Zubkov, Jennifer L. Elmer, and Eelco F. M. Wijdicks, *Validity of the Four Score Coma*

An examiner scores a response or non-response from each of three categories: eye opening, motor response, and verbal response.³⁹ Next, the examiner adds and assesses the scores from each category.⁴⁰ 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.⁴¹ The LOCF

Scale in the Medical Intensive Care Unit, 84(8)MAYO CLIN. PROC. 694, 694–701 (2009), available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2719522/>.

³⁹ *Id.*

⁴⁰ *Id.*

⁴¹ *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.⁴²

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.⁴³ Typically, the brain recovers over time, depending on the severity of the concussion.⁴⁴ Unfortunately, the brain never returns to its former state, prior to the concussion, because the brain cannot regenerate.⁴⁵

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

⁴² *Id.*

⁴³ Nigel A. Shaw, *Neurophysiology of Concussion: Theoretical Perspectives*, SPRINGER (2006), <http://www.springerlink.com/content/k7xp40310537020r/>. (Couldn't view the document)

⁴⁴ *National Institute of Neurological Disorders and Stroke*, *supra* note 26.

⁴⁵ Robert C. Cantu, *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.

⁴⁶ Barry Willer & John J. Leddy, *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.)

⁴⁷ Annette Sterr, et. al., *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.")

⁴⁸ See, e.g., Ryan C.W. Hall, et al., *Definition, Diagnosis, and Forensic Implications of Postconcussional Syndrome*, 46 PSYCHOSOMATICS, 195, 195-202 (2005), available at http://drryanhall.com/Articles/postconcussion_syndrome.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.⁴⁹ If disturbed, the patient may develop more severe brain injuries such as Second Impact Syndrome (SIS) or CTE. SIS often results in death.⁵⁰ 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.⁵¹ In as little as fifteen seconds, severe cerebral vascular swelling occurs and may result in paralysis, coma, and death.⁵² The mortality rate is 50% while the remaining one-half that survive frequently suffer a permanent disability.⁵³

More common is CTE, a condition caused by repetitive brain trauma.⁵⁴ 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.”).

⁴⁹ *Protect – Watch Your Head*, THE FRANKLIN INST. <http://www.fi.edu/learn/brain/head.html>.

⁵⁰ Matthew Thomas et. al., *Epidemiology of Sudden Death in Young, Competitive Athletes Due to Blunt Trauma*, 128 OFFICIAL J. OF PEDIATRICS 1, e1 –e8(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.*

⁵² Christin C. Donofrio & Rebecca Campbell, *Recognizing Concussion and Treating Postconcussion Syndrome*, J.OF REHAB.MGMT, Sept. 2011, available at http://www.rehabpub.com/issues/articles/2011-09_04.asp.

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

⁵⁴ Bennet I. Omalu, et al., *Chronic Traumatic Encephalopathy in a National Football League Player*, 57 NEUROSURGERY 128 (2005).

neurodegeneration.⁵⁵ Once brain tissue decays, an abnormal protein called tau builds up in a tangle creating a pattern unique only to CTE.⁵⁶ Tau protein accumulates in clumps until it starts to disrupt the brain's normal function.⁵⁷ Experts estimate changes to the brain begin anywhere from “months, years, or even decades after the last concussion or end of active athletic involvement.”⁵⁸ What results is a condition eerily similar to Alzheimer's disease and results in memory loss, depression, and poor impulse control.⁵⁹ Given the perplexing resemblance, CTE diagnoses occur after death.⁶⁰ Some believe that CTE causes motor neuron diseases similar to amyotrophic lateral sclerosis, commonly known as Lou Gehrig's disease.⁶¹

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.⁶² The severity of CTE directly corresponds to the number of traumatic head injuries an athlete sustains and the duration of his athletic

⁵⁵ Shaheen E. Lakhan & Annette Kirchgessner, *Chronic Traumatic Encephalopathy: The Dangers of Getting “Dinged”*, SPRINGERPLUS (March 2012), <http://www.springerplus.com/content/1/1/2>.

⁵⁶ *Often Misdiagnosed as Alzheimer's Disease, Chronic Traumatic Encephalopathy Could Play a Role in Veterans' Dementia*, U.S. MED, <http://www.usmedicine.com/compendium/often-misdiagnosed-as-alzheimers-disease-chronic-traumatic-encephalopathy-could-play-a-role-in-veterans-dementia.html>.

⁵⁷ Terry Zeigler, *Chronic Traumatic Encepholopathy*, SPORTSMED (Feb. 1, 2012), <http://www.sportsmd.com/Articles/id/44.aspx>.

⁵⁸ *Chronic Traumatic Encephalopathy*, *supra* note 28.

⁵⁹ Lakhan & Kirchgessner, *supra* note 55.

⁶⁰ *Boston University Researchers Report NHL Star Rick Martin Had Chronic Traumatic Encephalopathy*, BOSTON UNIVERSITY CENTER FOR THE STUDY OF TRAUMATIC ENCEPHALOPATHY (Oct. 5, 2011), <http://www.bu.edu/cste/news/press-releases/october-5-2011/>

⁶¹ Lakhan & Kirchgessner, *supra* note 55.

⁶² Ann C. McKee et. al., *Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy Following Repetitive Head Injury*, NCBI (Sept. 24, 2012), <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2945234/>; Corsellis JA, Bruton CJ, Freeman-Browne D., *The Aftermath of Boxing*, PSYCHOL MED. 1973, 3, at 270–303, available at <http://www.ncbi.nlm.nih.gov/pubmed/4729191>.(Bad link)

career.⁶³ Even after retirement from the sport, CTE continues to progress and is irreversible.⁶⁴

II. POSSIBILITY OF RECOVERY

“Vita brevis, ars longa, occasiopraeceptis, experimentumpericulosum, iudiciumdifficile.”⁶⁵

A. Current Treatments Offered

Every brain is different.⁶⁶ It is difficult to accurately predict concussion recovery time and/or lessen the physical, emotional, and mental symptoms.⁶⁷ The brain requires rest in order to recover from injury.⁶⁸ The 2008 Zurich guidelines recommend avoiding all stimuli requiring concentration, including television, video games, exercise, text messaging, online browsing, and work.⁶⁹ Although seemingly harmless, the injured brain cannot perform these activities without strain, which worsens symptoms and prevents recovery.⁷⁰ Premature resumption of normal

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.

⁶⁷ Allan H. Ropper & Kenneth C. Gorson, *Concussion*, 356 NEW ENG. J. MED. 166 (2007) available at <http://www.nejm.org/doi/full/10.1056/nejmcp064645>; Although there’s promising recent developments that suggest some measure of predictability in some cases, See also *UPMC Researchers Find Early Indicators May Predict Recovery Time from Sports Concussions*, UPMC (Feb. 27, 2012), <http://www.upmc.com/MediaRelations/NewsReleases/2012/Pages/Early-Indicators-May-Predict-Recovery-Time-Concussions.aspx>.

⁶⁸ Jon E. Bentz & Edward J. Purzycki, *Concussion: Not so Minor an Injury - Incidence, Pathophysiology, Risks, and Management*, JLGH, <http://www.jlgh.org/Past-Issues/Volume-3---Issue-3/Concussion-Not-so-Minor-an-Injury.aspx>.

⁶⁹ Ross Zafonte, *Diagnosis and Management of Sports-Related Concussion: A 15-Year-Old Athlete with a Concussion*, 306 J. AM. MED. ASS’N., 79 (2011).

⁷⁰ Rosanna C. Sabini & Cara Camiolo Reddy, *Concussion Management and Treatment Considerations in the Adolescent Population*, 38 THE PHYSICIAN AND SPORTS MED. 139 (2010).

activities cause increased psychological distress from decreased efficiency, interpersonal problems, negative feedback, and poor performance.⁷¹ Most injured people recover over time;⁷² however, if symptoms re-emerge, the patient should stop all activities that strain the brain to allow it to rest.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.⁷⁴ Recovery depends on post-concussive symptoms that stem from somatic, cognitive, emotional, and sleep disturbances.⁷⁵ Non-pharmaceutical treatment is first attempted to help with post-concussive syndrome. For somatic symptoms⁷⁶ 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.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.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.⁷⁹ CRT might require lessons of formal problem-solving wherein participants develop a plan to

⁷¹ *Id.*

⁷² *Id.*

⁷³ *Id.*

⁷⁴ *Id.*

⁷⁵ *Id.*

⁷⁶ *Somatic*, MERRIAM WEBSTER, <http://www.merriam-webster.com/dictionary/somatic>.

⁷⁷ Sabini & Reddy, *supra* note 70.

⁷⁸ *Id.* at 78.

⁷⁹ *Id.* at 137.

accomplish a task that requires listing every step necessary to solve it.⁸⁰

In severe cases, physicians turn to pharmacological measures as a last resort.⁸¹ Although the United States Food and Drug Administration has not approved any medicine for post-concussive neurological or psychiatric side effects,⁸² there are approved treatments available for symptoms, which, if used “appropriately and cautiously can improve neurological and functional outcome.”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.⁸⁴ Amantadine improves focus and sustained attention and concentration, orientation, alertness, arousal, processing, time, psychomotor speed, mobility, vocalization, agitation, anxiety and participation in therapy.⁸⁵

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

⁸⁰ *Id.* at 154.

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.)

⁸² *Management of Concussion/mild Traumatic Brain Injury*, Healthquality.va.gov (2009), http://www.healthquality.va.gov/management_of_concussion_mtbi.asp.

⁸³ *Id.*

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

⁸⁵ J. L. Nickels et. al., *Clinical Use of Amantadine in Brain Injury Rehabilitation*, Informahealthcare.com (1994), <http://informahealthcare.com/doi/abs/10.3109/02699059409151025>.

⁸⁶ Francis X. Conidi, *Sports Concussion and the Clinical Neurologist, Part III*, PRACTICAL NEUROLOGY (Feb. 2012), <http://bmctoday.net/practicalneurology/2012/02/article.asp?f=sports-concussion-and-the-clinical-neurologist-part-ii>.

antidepressants.⁸⁸ The most common treatments include citalopram, fluoxetine, sertraline, valproic acid, carbamazepine, or norepinephrine inhibitors.⁸⁹

Sleep dysfunctions overlap and affect other areas of brain function, notably concentration, behavior and emotional well-being.⁹⁰ Sleep studies determine the degree of difficulty in falling or staying asleep;⁹¹ otherwise, the emphasis should be on proper sleep hygiene.⁹² If sleep hygiene measures fail, medication is the next step.⁹³ Some patients use natural, over-the-counter remedies like melatonin and valerian.⁹⁴ Diphenhydramine, a common ingredient in allergy medicine,⁹⁵ also increases sleep while preventing drug addiction and rebound insomnia.⁹⁶ If stronger medication is necessary, then some doctors recommend trazodone.⁹⁷ Doctors avoid prescription sleep medicines such as zolpidem (Ambien) and eszopiclone (Lunesta) because they increase post-concussion symptoms.⁹⁸

B. Medical Advances

Read my lips: no new neurons.

-- Pasko Rakic, Yale University neuroscientist⁹⁹

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.

⁹⁵ *Diphenhydramine*, <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0000704>).

⁹⁶ *Id.*

⁹⁷ Conidi, *supra* note 86.

⁹⁸ *Id.*

⁹⁹ Edwin Kiester & William Kiester, *Birdbrain Breakthrough*, SMITHSONIAN MAGAZINE, June 2002, <http://www.smithsonianmag.com/science-nature/birdbrain.html?c=y&page=1>.

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 Gould¹⁰⁰

It all started with a rat.¹⁰¹ Joseph Altman and Gopal Das stumbled upon the scientific discovery of a lifetime: proof that rats could regenerate brain cells.¹⁰² Their 1960's theory challenged the prevailing understanding that the number of brain cells present in a creature at birth remain fixed from birth.¹⁰³ Yet, their colleagues ridiculed them mercilessly.¹⁰⁴ Although a few scientists vindicated their results, the theory of neurogenesis faded into obscurity.¹⁰⁵

In 1981, Fernando Nottebohm expanded upon Altman and Das' original research.¹⁰⁶ Canaries' brains grow and shrink directly in response to their brain functions.¹⁰⁷ During the spring mating season, which requires learning and rehearsing new songs, canary brains create neurons.¹⁰⁸ When canaries do not need to use their musical ability, their brain cells die off.¹⁰⁹ The canary's pattern of growth and shrinkage repeats for the bird's entire life.¹¹⁰

Again, although Nottebohm's novel research confirmed the theory of neurogenesis, other scientists dismissed his findings as aberrations in nature;¹¹¹ rats and canaries were nothing like

¹⁰⁰ Etienne Benson, *Thriving on Complexity*, 33 *MONITOR ON PSYCHOLOGY* 41 (2002), available at <http://www.apa.org/monitor/nov02/thriving.aspx>.

¹⁰¹ Joseph Altman and Gopal Das, *Autoradiographic and Histological Evidence of Postnatal Hippocampal Neurogenesis in Rats*, 124 *J. COMPARATIVE NEUROLOGY* 319 (1965).

¹⁰² *Id.*

¹⁰³ *Id.*

¹⁰⁴ NORMAN DOIDGE, *THE BRAIN THAT CHANGES ITSELF: STORIES OF PERSONAL TRIUMPH FROM THE FRONTIERS OF BRAIN SCIENCE* 250 (2007).

¹⁰⁵ *Id.*

¹⁰⁶ Fernando Nottebohm, *A Brain for All Seasons: Cyclical Anatomical Changes in Song Control Nuclei of the Canary Brain*, 214 *SCIENCE* 1368 (1981).

¹⁰⁷ *Id.*

¹⁰⁸ *Id.*

¹⁰⁹ *Id.*

¹¹⁰ *Id.*

¹¹¹ *Id.*

humans.¹¹² Then in 1998, Elizabeth Gould discovered that mankind's closest relative, the primate, could create new brain cells.¹¹³ In addition, she determined that external environmental factors affect neurogenesis.¹¹⁴ By forcing a monkey who always lived alone into the cage of another monkey, Dr. Gould artificially induced stress.¹¹⁵ After one hour, she removed the intruder monkey from the foreign cage.¹¹⁶ The brief stressful exposure caused fewer new brain cells to develop.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."¹¹⁸

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

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

¹¹² *Id.*

¹¹³ Elizabeth Gould, et al., *Neurogenesis in the Neocortex of Adult Primates*, 286 SCIENCE 548 (1999).

¹¹⁴ Elizabeth Gould, Proliferation of Granule Cell Precursors in the Dentate Gyrus of Adult Monkeys is Diminished By Stress, 95 PROC. NATL. ACAD. SCI. 3168 (1998).

¹¹⁵ *Id.*

¹¹⁶ *Id.*

¹¹⁷ *Id.*

¹¹⁸ Jonah Lehrer, *The Reinvention of the Self*, SEED, February-March 2006, available at http://seedmagazine.com/content/article/the_reinvention_of_the_self.

¹¹⁹ Fred H. Gage and Gerd Kempermann, *New Nerve Cells for Adult Brain*, SCIENTIFIC AM. 280.5 (May 1999).

¹²⁰ *Id.*

¹²¹ *Id.*

¹²² Michael M. Zeineh, et al., *Unfolding the Human Hippocampus With High Resolution Structural and Functional MRI*, THE ANATOMICAL RECORD, Apr. 2001, at 111-120.

¹²³ Kiester & Kiester, *supra* note 99.

¹²⁴ *Id.*

cerebral cortex, where higher thoughts are processed.¹²⁵ Why the hippocampus?¹²⁶ Scientists strive to answer whether new brain cells can grow elsewhere in the brain.¹²⁷ If neurogenesis is possible outside of the hippocampus, how can doctors artificially stimulate growth when the brain sustains injuries or needs repairs?¹²⁸ 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 NHL¹²⁹

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.¹³⁰

¹²⁵ *Id.*

¹²⁶ *Id.*

¹²⁷ Gina Kolata, *Studies Find Brain Grows New Cells*, N.Y. TIMES, Mar. 17, 1998 available at <http://www.nytimes.com/1998/03/17/science/studies-find-brain-grows-new-cells.html?pagewanted=all&src=pm>.

¹²⁸ *Id.*

¹²⁹ Branch, *supra* note 14.

¹³⁰ *Legal Issues Relating to Football Head Injuries: Hearing before the H. Comm. on the Judiciary*, October 28, 2009 (Written Testimony of Ann C. McKee), available at <http://judiciary.house.gov/hearings/pdf/McKee091028.pdf>.

A. Law & Order: Federal & State Legislation

Inspired by Zackery Lystedt's tragic concussion story,¹³¹ the state of Washington passed the Lystedt Law in 2009.¹³² 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.¹³³ As of April 2012, thirty-six states adopted laws on concussions and nine states have pending legislation.¹³⁴

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.¹³⁵ If the sports programs use non-school district properties, they receive an exemption from the compliance statement.¹³⁶ It is not mandatory that a private sport program's coaching staff receive

¹³¹ *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.

¹³² *Lystedt Law Overview*, NFL HEALTH AND SAFETY, <http://nflhealthandsafety.com/zackery-lystedt-law/lystedt-law-overview/>.

¹³³ *Id.*

¹³⁴ *Traumatic Brain Injury Legislation*, NATIONAL CONFERENCE OF STATE LEGISLATURES, <http://www.ncsl.org/issues-research/health/traumatic-brain-injury-legislation.aspx>.

¹³⁵ Marie-France Wilson, *Young Athletes at Risk: Preventing and Managing Consequences of Sports Concussions in Young Athletes and the Related Legal Issues*. 21 MARQ. SPORTS L. REV. 241, 285 (2010).

¹³⁶ *Id.*

concussion education ordinarily mandatory for the public school district's coaches.¹³⁷ Furthermore, there are no sanctions for non-compliance.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.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.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.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.¹⁴² Even well-intending physicians fail to accurately assess a player's status after an apparent concussion and make return-to-play decisions.¹⁴³

Currently, no federal laws exist to force the remaining five states without legislation¹⁴⁴ to adhere to concussion laws.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."¹⁴⁶ Unfortunately, the ConTACT bill died in Congress.¹⁴⁷ A similar bill titled the Protecting Student Athletes from Concussions Act of

¹³⁷ *Id.*

¹³⁸ *Id.*

¹³⁹ *Id.*

¹⁴⁰ *Id.*

¹⁴¹ *Id.*

¹⁴² *Id.*

¹⁴³ *Id.*

¹⁴⁴ *Id.* (Nevada, Montana, Arkansas, Michigan, and Georgia).

¹⁴⁵ H.R. 1347 Concussion Treatment and Care Tools Act of 2009, <http://www.opencongress.org/bill/111-h1347/show>.

¹⁴⁶ *Id.*

¹⁴⁷ Concussion Treatment and Care Tools Act of 2009, GOVTRACK.US, <http://www.govtrack.us/congress/bills/111/s2840>.

2010 proposed that public school districts create their own concussion management and treatment plans.¹⁴⁸ This bill also failed.¹⁴⁹

The closest thing to federal intervention is the “Heads Up” toolkit from the federally funded CDC.¹⁵⁰ Although not mandatory, the “Heads Up” toolkit educates physicians about the severity of concussions.¹⁵¹ Recipients of the “Heads Up” toolkit were less likely to immediately return concussed athletes to practice and games.¹⁵²

B. Reaction to State Legislation

A year after Lystedt’s Law, the National Football League (“NFL”) released new rules to minimize head injuries.¹⁵³ 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,¹⁵⁴ (2) once a player loses his helmet, the play stops,¹⁵⁵ and, (3) during field-goal and extra-point attempts, the defense cannot situate any player directly across from the snapper.¹⁵⁶ The NFL also created stricter return-to-play guidelines for concussed football players that required a consultation with an independent neurologist.¹⁵⁷

In 2011, perhaps in response to litigation by former NFL players,¹⁵⁸ the NFL further increased football player safety by

¹⁴⁸ H.R. 6172, 111th Cong. (2nd Sess. 2010).

¹⁴⁹ *Id.*

¹⁵⁰ Bryan Toporek, *CDC to Create National Youth-Concussion Guidelines*, EDUCATION WEEK (Sept. 27, 2011), http://blogs.edweek.org/edweek/schooled_in_sports/2011/09/cdc_to_create_national_youth_concussion_guidelines.html.

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.*

¹⁵³ New NFL Rules Designed to Limit Head Injuries, NFL.COM, <http://www.nfl.com/news/story/09000d5d81990bdf/article/new-nfl-rules-designed-to-limit-head-injuries>.

¹⁵⁴ *Id.*

¹⁵⁵ *Id.*

¹⁵⁶ *Id.*

¹⁵⁷ *Id.*

¹⁵⁸ David Ariosto, *Former NFL Players Sue League Over Head Injuries*, CNN.COM (Dec. 22, 2011), http://articles.cnn.com/2011-12-22/us/us_sport-nfl-concussion-lawsuits_1_concussions-head-trauma-player-safety?_s=PM:US.

enacting additional rules.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.¹⁶⁰ In addition, team fines for too many illegal hits,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.¹⁶² Also, coverage teams can only have 5-yards of run-up instead of 15-yards,¹⁶³

Although not mandatory, the NFL distributed a strongly worded memo with the “Madden Rule¹⁶⁴ that influenced current practices. The coach must remove any player suspected of suffering a concussion, even if not formally diagnosed.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.¹⁶⁶ Following the NFL’s lead, the National Collegiate Athletic Association (“NCAA”) changed its rules to favor player safety.¹⁶⁷ Much like the NFL, it too faces class action suits from former football players.¹⁶⁸ Since August 2010, the NCAA required each university to develop its own concussion management plan.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

¹⁵⁹ *2011 Rule Changes*, NFL.COM (July 25, 2011), <http://www.nfl.com/videos/nfl-network-total-access/09000d5d820b36ee/2011-rule-changes>.

¹⁶⁰ *Id.*

¹⁶¹ *Id.*

¹⁶² *Id.*

¹⁶³ *Id.*

¹⁶⁴ NFL Concussion Guidelines, Hunt Batjer, MD and Richard Ellenbogen, MD, <http://nflhealthandsafety.files.wordpress.com/2011/06/nfl-hns-memo-2011v2.pdf>.

¹⁶⁵ *Id.*

¹⁶⁶ *Id.*

¹⁶⁷ *See generally*, NCAA History, <http://www.ncaa.org/wps/wcm/connect/public/ncaa/about+the+ncaa/who+we+are/about+the+ncaa+history>.

¹⁶⁸ George Vecsey, *College Athletes Move Concussions Into the Courtroom*, N.Y. TIMES(Nov. 29, 2011) *available at* <http://www.nytimes.com/2011/11/30/sports/ncaafotball/college-players-move-concussions-issue-into-the-courtroom.html?pagewanted=all>.

¹⁶⁹ Concussions, NCAA.COM, <http://www.ncaa.org/wps/wcm/connect/public/ncaa/health+and+safety/concussion+home+page/concussion+landing+page>.

kick returners.¹⁷⁰ They also moved the kickoff to the thirty-five yard line.¹⁷¹ 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.¹⁷² Also, the NCAA hopes to encourage more touchbacks by moving the touchback distance on free kicks to the twenty-five yard line.¹⁷³

If a player loses his helmet, he must leave the game and cannot participate in the next play.¹⁷⁴ Offensive players who are not in motion can block below the waist legally without restriction.¹⁷⁵ Otherwise, no one may block below the waist.¹⁷⁶ In cases of shield blocking, the receiving team cannot jump over blockers unless the player jumps straight up or between two players.¹⁷⁷ Kick returners must complete a catch before the kicking team can make contact.¹⁷⁸

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,¹⁷⁹ the National Federation of State High School Associations (“NFSH”) implemented similar rule changes.¹⁸⁰ 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.¹⁸¹

¹⁷⁰ Greg Johnson, *Playing Rules Oversight Panel Approves Rules Changes in Football, Other Sports*, NCAA.ORG (Feb. 24, 2012), <http://www.ncaa.org/wps/wcm/connect/public/NCAA/Resources/Latest+News/2012/February/Playing+Rules+Oversight+Panel+approves+rules+changes+in+football,+other+sports#.T0ewIgx7e7g.twitter>.

¹⁷¹ *Id.*

¹⁷² *Id.*

¹⁷³ *Id.*

¹⁷⁴ *Id.*

¹⁷⁵ *Id.*

¹⁷⁶ *Id.*

¹⁷⁷ *Id.*

¹⁷⁸ *Id.*

¹⁷⁹ Sergio R. Russo Buzzini and Kevin M. Guskiewicz, *Sport-related Concussion in the Young Athlete*, 18 CURRENT OPINION PEDIATRICS 376, 377 (2006).

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.¹⁸² Such an action even has precedence in history.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.¹⁸⁴ On average, helmets reduce concussions by thirty-three percent.¹⁸⁵ Some simple measures like ensuring the helmet has a tight, snug fit on players reduces the risk of brain injury.¹⁸⁶ Most players do not wear their helmets properly, evidenced by their easy removal at the sidelines.¹⁸⁷ No helmet can completely prevent brain injury, even if the fit is perfect.¹⁸⁸ However, helmets reduce skull fractures and hematomas.¹⁸⁹ Similarly, mouth guards effectively prevent dental injuries.¹⁹⁰ It is unlikely that mouth guards will prevent concussions, however, since the thickest mouth guard absorbs only 2% of the blow.¹⁹¹

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

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+act+1424+scotland&hl=en&sa=X&ei=dh6cUL7XDInwyQHSvIHAAG&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 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"

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).

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.

¹⁸⁶ Gregg Easterbrook, *Virginia Tech Helmet Research Crucial*, ESPN.COM (July 19, 2011), http://sports.espn.go.com/espn/page2/story?page=easterbrook-110719_virginia_tech_helmet_study&sportCat=nfl.

¹⁸⁷ *Id.*

¹⁸⁸ *Id.*

¹⁸⁹ *OK Dad, I'm Ready for ... Basketball*, SPORTSCONCUSSIONS.ORG, <http://www.sportsconcussions.org/ibaseline/ok-dad-im-ready-forbasketball.html>.

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.

¹⁹¹ *Id.*

Conversely, improved equipment may result in more aggressive behavior, thus, increasing the probability of head injury.¹⁹² Experts believe that “coaching reforms and stricter officiating will do more to reduce concussion incidence than any type of headgear.”¹⁹³ 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.¹⁹⁴ Praising helmet-to-helmet contact unnecessarily glamorizes the risk of concussions without mentioning any of the side-effects.¹⁹⁵

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.¹⁹⁶ He must pass the physical exam next season in order to receive payment.¹⁹⁷ 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.¹⁹⁸ Some teams restructure the contract in their favor knowing that employment opportunities are unlikely elsewhere with a brain injury.¹⁹⁹

Another viable option is extending sport seasons. The current industry standard of one “bye week”²⁰⁰ is insufficient. If the NFL mandated that a player could not play for two consecutive weeks,

¹⁹² Paul McCrory et al., *Can We Manage Sport Related Concussions in Children the Same as in Adults?*, 38 BRIT. J. SPORTS MED. 516, 517 (2004).

¹⁹³ Easterbrook, *supra* note 186.

¹⁹⁴ *Unexpected Youth Football Impact Data*, SPORTS CONCUSSIONS, <http://www.sportsconcussions.org/ibase/line/unexpected-youth-football-impact-data-pbs-video.html>.

¹⁹⁵ *Id.*

¹⁹⁶ *NFL Collective Bargaining Agreement 2006-2012*, art. XLIV, § 9 at 251, <http://images.nflplayers.com/mediaResources/files/PDFs/General/NFL%20COLLECTIVE%20BARGAINING%20AGREEMENT%202006%20-%202012.pdf>.

¹⁹⁷ *Id.*

¹⁹⁸ *Id.* at 41.

¹⁹⁹ E.M. Swift, *One Big Headache*, SPORTS ILLUSTRATED, Feb. 12, 2007, at 22.

²⁰⁰ Bill Simmons, *Breaking Down the NFL’s Bye Week*, ESPN.COM, <http://sports.espn.go.com/espn/page2/story?page=simmons nfl2010/byeweek&sportCat=nfl> (Nov. 15, 2012).

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.²⁰¹ 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

²⁰¹ *Sports Industry Statistical Overview*, PLUNKETT RESEARCH, <http://www.plunkettresearch.com/sports-recreation-leisure-market-research/industry-statistics>.

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 B-3. Physical Symptoms – TREATMENT

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

APPENDIX 2:

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

* 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.²⁰² The possible good news is tempered by some evidence that there is no neuroprotective effect and may lead to higher mortality rates.²⁰³

Progesterone:

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

Erythropoietin:

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

²⁰² Matthew T. Neal, Jonathan L. Wilson, Wesley Hsu, Alexander K Powers, *Concussions: What a Neurosurgeon Should Know About Current Scientific Evidence and Management Strategies*, SURGICAL NEUROLOGY INT’L, 2012, 3:16, available at <http://www.surgicalneurologyint.com/text.asp?2012/3/1/16/92930>.

²⁰³ *Id.*

²⁰⁴ *Id.*

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.

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.

Kinins, 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.”²⁰⁷ 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 nitron (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.²⁰⁸

²⁰⁸ Laura Cofsky, *Penn Med Study Shows Ingredient in Muscle Milk May Treat Concussions*, *The Daily Pennsylvanian*, Jan. 22, 2012, http://thedp.com/index.php/article/2012/01/penn_med_study_shows_muscle_milk_may_treat_concussions; See also *Amino Acids may restore Concussion's Chemical Imbalance*, (last updated Sept. 9, 2012) <http://www.sportsconcussions.org/ibaseline/amino-acids-may-restore-concussions-chemical-imbalance-3.html>.

