Cleveland State Law Review

Volume 66 | Issue 1 Article 6

12-31-2017

Depression: The Often Overlooked Sequela of Head Trauma

Samuel D. Hodge Jr.
Temple University

Jack E. Hubbard

DEPRESSION: THE OFTEN OVERLOOKED SEQUELA OF HEAD TRAUMA

SAMUEL D. HODGE, JR.* & JACK E. HUBBARD**

ABSTRACT

Depression is a common sequela of head trauma. Approximately half of all individuals with a cranial injury will experience depression within the first year, regardless of the severity of the injury. The ailment is characterized clinically as a mood disorder, often associated with intense feelings of sadness. However, depression is more complex than mood disorders, as many mental and bodily complaints—such as insomnia, fatigue, anxiety, appetite changes, aches and pains, and lack of interest in previously enjoyable activities—are associated with depression. These intense feelings, particularly when combined with despair and hopelessness, can lead to suicide, a dreaded potential complication of depression.

Recent studies of traumatic brain injuries have identified specific biochemical and structural factors that can cause depression. From a biochemical standpoint, a brain injury can trigger changes in key neurotransmitters, such as serotonin and dopamine. In addition, head trauma can cause structural damage to certain vulnerable brain circuits linked to depression. Pre-existing issues of depression, substance abuse, and head trauma augment the potential of a depressive mental illness following a traumatic brain injury.

When handling a brain injury claim involving depression, lawyers must understand that being "depressed" is not the same as being "sad." The primary source of depression is *loss,* and those who have sustained a brain injury have experienced the most overwhelming loss of all—the loss of his wellbeing. These cases can be expensive and require a degree of expertise in understanding these problems. Lawyers must obtain records to ascertain the individual's functional status both before and after the traumatic event. Still, defense counsel can take solace in the general treatability of acute depression; most patients react positively to proper care.

This Article will provide a medical analysis of how depression develops following a brain injury. Further, it will offer a legal discussion on depression and brain trauma by examining how lawyers should approach such cases and by reviewing recent court cases on this subject.

CONTENTS

* Samuel D. Hodge, Jr. is a seasoned litigator and professor at Temple University where he teaches both law and anatomy. He has authored more than 150 articles in medical and legal journals and has written six medical/legal texts. He also enjoys an AV preeminent rating and has been named a top lawyer in Pennsylvania on multiple occasions.

** Jack, E. Hubbard, PhD, MD is board certified in both neurology and pain medicine and a former adjunct professor of neurology at the University of Minnesota's School of Medicine. In addition to his medical training, he holds a doctorate in anatomy. Dr. Hubbard has published widely on a number of topics in both medical and legal publications.

"Depression has been called the world's number one public health problem. In fact, depression is so widespread it is considered the common cold of psychiatric disturbances. But there is a grim difference between depression and a cold. Depression can kill you."

- David D. Burns[1]

I. INTRODUCTION

Consider the following names, from the gridiron past, who have provided such enjoyable Sundays during the fall:

2004: Justin Strzelczyk, offensive tackle, Pittsburgh Steelers—killed driving erratically.
2005: Terry Long, offensive lineman, Pittsburgh Steelers—suicide by drinking antifreeze.
2006: Andre Waters, safety, Philadelphia Eagles—self-inflicted gunshot.
2010: Kenny McKinley, wide receiver, Denver Broncos—self-inflicted gunshot.
2011: Dave Duerson, safety, Chicago Bears—self-inflicted gunshot.
2012: Kurt Crain, linebacker, Green Bay Packers—self-inflicted gunshot.

[1] DAVID D. BURNS, FEELING GOOD: THE NEW MOOD THERAPY 9 (1999).

2012: Ray Easterling, safety, Atlanta Falcons—self-inflicted gunshot.
2012: Junior Seau, linebacker, New England Patriots—self-inflicted gunshot.
2012: OJ Murdock, wide receiver, Tennessee Titans—self-inflicted gunshot.
2013: Paul Oliver, safety, San Diego Chargers—self-inflicted gunshot.
2015: Adrian Robinson, Jr, linebacker, Pittsburgh Steelers—suicide by hanging.

The media has widely publicized a seemingly endless stream of professional football players who have committed suicide over the past decade.[2] In the military, suicide is the second leading cause of death among military personnel.[3] These events share a common thread: the connection between head or brain trauma and severe depression. Media attention has focused increasingly upon the issue of multiple head trauma and the resulting psychological consequences within the National Football League ("NFL") and the armed services.[4] This coverage has revealed that traumatic brain injuries ("TBI") are the "signature injury" of the Afghanistan and Iraq conflicts and that TBIs increase the likelihood of suicidal thoughts or deaths by suicide among military veterans.[5]

This Article will discuss depression from both a medical and legal point of view and will examine the intense relationship between head trauma and depression. Part II will focus on the medical implications of depression by considering the definition and description of depression, the causes of depression, and the relationship between head trauma and depression in athletics and the military. The Parts III and IV of the Article will examine how depression is handled from a legal standpoint and will present court cases involving depression and brain trauma.

II. MEDICAL DISCUSSION

A. Depression: Definition/Description

Depression is clinically characterized as a disorder of mood, often associated with intense feelings of sadness.[6] Depression, however, is much more complex. Many mental and bodily complaints accompany it, such as insomnia, fatigue, anxiety, appetite changes, aches, pains, and lack of interest in previously enjoyable activities.[7]

---

[2] Josh Levs et al., *Paul Oliver's Suicide is Latest in a String Among Former NFL Players*, CNN (Sept. 26, 2013), http://www.cnn.com/2013/09/25/us/former-nfl-player-suicide/index.html.

[3] Craig J. Bryan & Tracy A. Clemans, *Repetitive Traumatic Brain Injury, Psychological Symptoms, and Suicide Risk in a Clinical Sample of Deployed Military Personnel*, 70 JAMA PSYCHIATRY 686, 687 (2013).

[4] Sandra Basu, *Army, NFL Team Up in Offensive Against Traumatic Brain Injury*, U.S. MEDICINE (Oct. 11, 2012), http://www.usmedicine.com/agencies/department-of-defense-dod/army-nfl-team-up-in-offensive-against-traumatic-brain-injury/.

[5] Bryan & Clemans, *supra* note 3, at 687.

[6] AM. PSYCHIATRIC ASS'N, DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS: DSM-5 155 (5th ed. 2013) [hereinafter DSM-5].

[7] *Id.* at 160.

These intense feelings, particularly when coupled with feelings of despair or hopelessness, can result in suicide—the worst manifestation of depression.

The scientific community's understanding of depression has evolved over the years from a simple concept to a complex problem with multiple manifestations and nuances. The *Diagnostic and Statistical Manual of Mental Disorders* ("DSM"), published by the American Psychiatric Association ("APA"), categorizes and classifies all types of depression as psychiatric illnesses.[8] First appearing in 1952 as a system for collecting census and psychiatric hospital statistics, the DSM was combined with a War Department Technical Bulletin and served as a system for the classification of mental disorders. The DSM is now the official diagnostic system for mental disorders in the United States.[9] The latest edition, DSM-V, was published on May 18, 2013, the first major edition in twenty years.[10] The DSM mainly focuses on the signs and symptoms of mental disorders, not their underlying causes.[11] The DSM solely deals with psychiatric disorders and thus differs from the International Classification of Diseases ("ICD"),[12] which the World Health Organization ("WHO") maintains.[13] The ICD classifies all known medical conditions, including psychiatric, with the latest ICD-10 edition accepted by member countries in 1994.[14] The United States did not implement ICD-10's latest form—the ICD Clinical Modification ("ICD-10-CM")—until the fall of 2015.[15] Unlike ICD, the DSM is limited to the

[8] *Id.* at 167.

[9] *Diagnostic and Statistical Manual of Mental Disorders*, WIKIPEDIA, https://en.wikipedia.org/wiki/Diagnostic_and_Statistical_Manual_of_Mental_Disorders (last visited Dec. 20, 2016).

[10] DSM-5, *supra* note 6, at 6.

[11] *Id.* at xll.

[12] *Classifications: International Classification of Diseases (ICD)*, WORLD HEALTH ORG., http://www.who.int/classifications/icd/en [https://web.archive.org/web/20140212190115/http://www.who.int/classifications/icd/en/] (last visited Oct. 15, 2017) [hereinafter WORLD HEALTH ORG.] ("The International Classification of Diseases (ICD) is the standard diagnostic tool for epidemiology, health management and clinical purposes"); *International Statistical Classification of Diseases and Related Health Problems*, WIKIPEDIA, https://en.wikipedia.org/wiki/International_Statistical_Classification_of_Diseases_and_Related_Health_Problems (last visited Dec. 20, 2016).

[13] DSM-5, *supra* note 6, at xll; *see generally* WORLD HEALTH ORG., *supra* note 12.

[14] WORLD HEALTH ORG., *supra* note 12 ("ICD-10 was endorsed by the Forty-[T]hird World Health Assembly in May 1990 and came into use in WHO Member States [starting in] 1994"). Although ICD-10 is the latest version, the World Health Organization has already begun drafting ICD-11, scheduled for release in 2018. *Id.*

[15] *International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM)*, CTR. FOR DISEASE CONTROL & PREVENTION, https://www.cdc.gov/nchs/icd/icd10cm.htm#FY%202018%20release%20of%20ICD-10-CM (last visited Oct. 15, 2017); *but see id.* (noting that although ICD-10-CM was not introduced in the United States until October 1, 2015, the United States has been using ICD-9 and ICD-10 for cause of death reporting since 1999).

categorization of psychiatric disorders exclusively, rather than all medical conditions.[16]

DSM-V classifies the following depressive disorders:[17]

- Major depressive disorder,
- Persistent depressive disorder,
- Pre-menstrual dysphoric disorder,
- Disruptive mood dysregulation disorder,
- Substance/medication induced depressive disorder,
- Depressive disorder due to another medical condition,
- Other specified depressive disorder, and
- Unspecified depressive disorder.

The common feature in these forms of depression is the presence of a sad, empty, or irritable mood, associated with somatic[18] and cognitive[19] changes affecting the person's ability to function.[20] These forms differ in duration, timing, and possible cause.[21] DSM-V removed bipolar disorder, also termed manic-depressive disorder, characterized by cyclic, widely varying moods from depression to mania, from DSM-IV's depression category and placed it in DSM-V's chapter on "Bipolar and Related Disorders."[22] Another change since DSM-IV is the addition of Disruptive Mood Dysregulation Disorder as a condition.[23] Disruptive Mood Dysregulation Disorder appears in children up to age eighteen who display persistent irritability and episodes of poorly controlled behavior.[24] The Premenstrual Dysphoric Disorder[25] is also new to DSM-V, occurring in most menstrual cycles just prior to menses and lasting until

---

[16] DSM-5, *supra* note 6, at xll.

[17] *Id.* at 155.

[18] Somatic refers to the body as opposed to the mind. *Somatic*, WIKIPEDIA, https://en.wikipedia.org/wiki/Somatic (last visited Dec. 21, 2016).

[19] This term refers to the mental activities of perception, memory, judgment, and reasoning. *Cognitive*, DICTIONARY.COM, http://www.dictionary.com/browse/cognitive (last visited Dec. 21, 2016).

[20] DSM-5, *supra* note 6, at 155.

[21] *Id.* at 155.

[22] AM. PSYCHIATRIC ASS'N, HIGHLIGHTS OF CHANGES FROM DSM-IV-TR TO DSM-5, 1, 4 (2013).

[23] *Id.*

[24] John M. Grohol, *DSM-5 Changes: Depression and Depressive Disorders*, PSYCH. CENT., http://pro.psychcentral.com/dsm-5-changes-depression-depressive-disorders/004259.html (last visited Dec. 8, 2016).

[25] Premenstrual dysphoric disorder ("PMDD") is defined as a severe, occasionally disabling expansion of premenstrual syndrome ("PMS"). Jacqueline M. Thielen, *What's the Difference Between Premenstrual Dysphoric Disorder (PMDD) and Premenstrual Syndrome (PMS)? How Is PMDD Treated?*, MAYOCLINIC.ORG, http://www.mayoclinic.org/diseases-conditions/premenstrual-syndrome/expert-answers/pmdd/faq-20058315 (last visited Dec. 20, 2016).

several days post-menses.[26] In addition, the term Persistent Depressive Disorder replaced DSM-IV's category of Dysthymia[27] and describes symptoms of depression that persist for more than two years. Finally, DSM-V removed the "bereavement exclusion"[28] from the diagnosis of major depression, which occurs when a person exhibits the symptoms of major depression within the first two months after the death of a loved one.[29]

The DSM-V manual did not remove or change the "Major Depressive Disorder" category defined in DSM-IV[30] because an epidemiological study of Major Depressive Disorder ("MDD") determined that it is a common problem, is widely distributed in the population, and is usually associated with substantial functional impairment and symptom severity.[31] Researchers also found that in the United States, the lifetime occurrence of MDD is 16.2%, which means that about one in six people will experience MDD during their lifetime.[32] Within the 16.2%, between 2% and 7% of these adults will commit suicide.[33] The prevalence rate of MDD is highest in individuals ranging from eighteen to twenty-nine years old, threefold higher than in individuals sixty years or older.[34] Compared to men, women are roughly two to three times more likely to experience MDD in early adolescence.[35]

Formal, diagnostic descriptions of MDD illustrate some of the symptoms associated with a diagnosis of MDD. The basic DSM-V diagnostic criteria for MDD is as follows:

> A. Five (or more) of the following symptoms have been present during the same two-week period and represent a change from previous functioning;

---

[26] Grohol, *supra* note 24.

[27] Dysthymia is a type of depression that is less severe than major depression. It generally has a longer duration. *Dysthymia*, HARV. HEALTH PUBL'G, http://www.health.harvard.edu/depression/dysthymia (last visited Dec. 8, 2016).

[28] This exclusion was primarily removed because of (1) the lack of sufficiently controlled, clinical studies demonstrating that major depressive syndromes subsequent to bereavement vary in nature, course, or outcome from depression of similar severity in any other context—or from surfacing "out of the blue;" and (2) major depression is a possible fatal problem, with a suicide rate of about four percent. Ronald W. Pies, *The Bereavement Exclusion and DSM-5*: *An Update and Commentary*, 11 INNOVATIONS CLINICAL NEUROSCIENCE 19, 19–20 (2014).

[29] Grohol, *supra* note 24.

[30] *Id.*

[31] Ronald C. Kessler et al., *The Epidemiology of Major Depressive Disorder: Results from the National Comorbidity Survey Replication (NCS-R)*, 289 JAMA 3095, 3095 (2003).

[32] Kelly C. Cukrowicz & Erin K. Poindexter, *Suicide*, *in* 20 THE OXFORD HANDBOOK OF DEPRESSION AND COMORBIDITY 20 (C. Steven Richards & Michael W. O'Hara eds., 2014).

[33] *See* Maria Oquendo et al., *Prospective Studies of Suicidal Behavior in Major Depressive and Bipolar Disorders: What Is the Evidence for Predictive Risk Factors*, 114 ACTA PSYCHIATRICA SCANDINAVIA 151, 151 (2006).

[34] DSM-5, *supra* note 6, at 165.

[35] *Id.*

at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure . . . .[36]

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, and hopeless), or observation made by others (e.g., appears tearful). (Note: In children and adolescents, it can be irritable mood.)[37]
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).[38]
3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (Note: In children, consider failure to make expected weight gain.)[39]
4. Insomnia or hypersomnia nearly every day.[40]
5. Psychomotor agitation or retardation every day (observable by others, not merely subjective feelings of restlessness or being slowed down).[41]
6. Fatigue or loss of energy nearly every day.[42]
7. Feelings of worthlessness or excessive or inappropriate guilt (which maybe delusional) nearly every day (not merely self-reproach or guilt about being sick).[43]
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).[44]
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.[45]

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.[46]

---

[36] *Id.* at 160.

[37] *Id.*

[38] *Id.*

[39] *Id.* at 161.

[40] *Id.*

[41] *Id.*

[42] *Id.*

[43] *Id.*

[44] *Id.*

[45] *Id.*

[46] *Id.*

> C. The episode is not attributable to the physiological effects of a substance or to another medical condition. Note: Criteria A-C represent a major depressive episode.[47]
>
> . . . .
>
> D. The occurrence of the major depressive episode is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders.[48]
>
> E. There has never been a manic episode or a hypomanic episode. Note: This exclusion does not apply if all of the manic-like episodes are substance-induced or are attributable to the physiological effects of another medical condition.
>
> . . . .
>
> *Specify:*
>
> With anxious distress
> With mixed features
> With melancholic features
> With atypical features
> With mood-congruent psychotic features
> With mood-incongruent psychotic features
> With catatonia.
> With peripartum onset
> With seasonal pattern (recurrent episode only).[49]

Thus, MDD is a common psychiatric mood disorder, lasting at least two weeks, characterized by an intense feeling that negatively impacts the person's life.[50] Individuals experiencing this psychologically painful disorder portray many thoughts and feelings including sentiments of worthlessness, anxiety, helplessness, hopeless, poor concentration, poor memory, and sleep disorders.[51] Moreover, depression can present various somatic problems, including headaches, bodily pain, fatigue, appetite changes, and low energy.[52] Problems of depression can even prove fatal, leading to suicide.[53]

---

[47] *Id.* ("Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of the loss.").

[48] *Id.*

[49] *Id.* at 161–62 (internal citations omitted).

[50] *Id.* at 163.

[51] *Id.* at 160.

[52] *Id.*

[53] *Id.* at 164.

B. Causes of Depression

Risks and symptoms associated with depression are multiple and complex, combining biological, genetic, and psychosocial factors; a tendency for depression can also be precipitated by a stressful life event, such as a divorce, bankruptcy, or death of a loved one.[54] In other words, depression can result from a "hardware" or a "software" problem or, more often, a combination of the two difficulties, affecting brain functioning.

1. Biological Basis—A Hardware Problem

As a "hardware" problem, the biological basis of depression presumes an imbalance in the *neurotransmitter substances* within the brain. Neurotransmitters are chemical messengers released from neurons[55] at the synaptic junction[56] that allow one neuron to communicate with other neurons.[57] Scientists consider many neurotransmitters important in depression including serotonin,[58] acetylcholine,[59] norepinephrine,[60] dopamine,[61] glutamate,[62] and gamma-aminobutyric acid

---

[54] *Id.* at 166.

[55] A neuron is the basic cell in the brain. *Neuron*, ENCYCLOPEDIA BRITANNICA ONLINE, https://www.britannica.com/science/neuron (last visited Sept. 10, 2017).

[56] A synapse refers to "the site of transmission of electric nerve impulses between two nerve cells (neurons) or between a neuron and a gland or muscle cell (effector)." *Synapse*, ENCYCLOPEDIA BRITANNICA ONLINE, https://www.britannica.com/science/synapse (last visited Dec. 20, 2016).

[57] Jack E. Hubbard, *Anatomy of the Head and Neck*, *in* HEAD TRAUMA AND BRAIN INJURY FOR LAWYERS 21, 31–32 (Jack E. Hubbard & Samuel D. Hodge eds., 2016) [hereinafter Hubbard, *Anatomy of the Head and Neck*].

[58] Jack E. Hubbard et al., *Outcome Measures of a Chronic Pain Program: A Prospective Statistical Study*, 12 CLINICAL J. PAIN 330, 332 (1996) [hereinafter Hubbard et al., *Outcome Measures*]. Some believe that serotonin is the chemical responsible for maintaining mood balance and that a deficiency of serotonin causes depression. Colette Bouchez, *Serotonin: 9 Questions and Answers*, WEBMD, http://www.webmd.com/depression/features/serotonin#1 (last visited Dec. 20, 2016).

[59] Acetylcholine is one of the most regular chemical messengers found in the central and peripheral nervous systems. It plays an important role in muscle action. Kendra Cherry, *Discovery and Functions of Acetylcholine*, VERYWELL.COM, https://www.verywell.com/what-is-acetylcholine-2794810 (last visited Dec. 20, 2016).

[60] Norepinephrine is comparable to adrenaline. It narrows the blood vessels and increases blood pressure and blood sugar levels. *What Is Norepinephrine?*, DRUGS.COM, https://www.drugs.com/mtm/norepinephrine.html (last visited Dec. 20, 2016).

[61] Dopamine is a neurotransmitter that assists in the regulation of the brain's reward and pleasure centers. *Dopamine*, PSYCHOL. TODAY, https://www.psychologytoday.com/basics/dopamine (last visited Dec. 20, 2016).

[62] Glutamate is a neurotransmitter that forwards signals in the brain and nerves in the body. *What Is Glutamate?*, EVERYDAYHEALTH.COM, http://www.everydayhealth.com/glutamate/guide/ (last visited Dec. 20, 2016).

("GABA").[63] Deficiencies of norepinephrine, dopamine, and serotonin have led to the monoamine hypothesis for the basis of depression.[64] Using this model, pharmacological treatment of depression aims at enhancing these neurotransmitters, such as the selective serotonin reuptake inhibitors (SSRI), including citalopram (Celexa), sertraline (Zoloft), and fluoxetine (Prozac).[65] Certain areas of the brain also appear to be preferentially affected by depression including the *amygdala* (associated with emotions of anger, pleasure, sorrow, and fear—increased activity in depression), *thalamus* (a major sensory relay center that links sensory input to pleasant and unpleasant feelings), and the *hippocampus* (found to be smaller in depressed individuals and plays a major role in forming long-term memory).[66]

Another biological consideration is consideration of underlying medical conditions and the medications used to treat them. A number of chronic medical conditions are known to be associated with depression—an estimated 10% to 14% of all depressions are due to chronic medical conditions.[67] However, whether or not the medical problem leads to depression or causes changes within the brain sufficient to cause depression remains unclear.[68] Examples of the incidence of depression associated with chronic medical illnesses include:

- Heart attack—40% to 65%,
- Cancer—25%,
- Diabetes mellitus—25%, and
- Chronic pain syndrome—30% to 54%.[69]

Thyroid disorders, especially hypothyroidism,[70] can produce symptoms of depression; yet, overt thyroid dysfunction does not appear to be common among depressive

[63] Gamma-Amino Butyric acid is an amino acid that performs like a neurotransmitter in the central nervous system and it retards nerve transmission in the brain. *GABA: Gamma-Amino Butyric Acid*, DENV. NEUROPATHIC CLINIC, http://www.denvernaturopathic.com/news/GABA.html (last visited Dec. 20, 2016); David J. Nutt, *Relationship of Neurotransmitters to the Symptoms of Major Depressive Disorder*, 61 J. CLINICAL PSYCHIATRY 4, 4 (Supp. E1 2008).

[64] Robert M. A. Hirschfeld, *History and Evolution of the Monoamine Hypothesis of Depression*, 61 J. CLINICAL PSYCHIATRY 4, 4 (Supp. 6 2000).

[65] *Id.* at 4–5.

[66] *What Causes Depression?*, HARV. HEALTH PUBL'G, http://www.health.harvard.edu/mind-and-mood/what-causes-depression (last visited Dec. 20, 2016).

[67] *Id.*

[68] *Id.*

[69] *Dealing with Chronic Illnesses and Depression*, WEBMD, http://www.webmd.com/depression/guide/chronic-illnesses-depression#1 (last visited Dec. 20, 2016).

[70] This medical problem is associated with the body's inability to make sufficient thyroid hormones. Because this hormone is designed to "run the body's metabolism," it is logical that people with this problem will have symptoms related to a slow metabolism. James Norman, *Hypothyroidism: Too Little Thyroid Hormone*, ENDOCRINEWEB,

patients.[71] Neurological disorders are also often associated with depression including stroke, multiple sclerosis, Parkinson's disease,[72] amyotrophic lateral sclerosis (ALS—Lou Gehrig's disease),[73] epilepsy,[74] and cluster headaches.[75]

Not only can conditions cause depression, medications can cause depression.[76] Counsel should be cognizant of this and review all medications taken by a claimant, particularly in those individuals who exhibit symptoms of a new onset of depression. The AARP recognizes ten of the major classes of drugs known to cause depression, including:

- *Beta blockers*—used to treat various problems such as hypertension, migraine headaches, and cardiac arrhythmias; examples include propranolol (Inderal) and atenolol (Tenormin);[77]
- *Corticosteroids*—used to treat inflammatory disorders such as rheumatoid arthritis and lupus; examples include prednisone and dexamethasone;[78]
- *Benzodiazepines*—used to treat anxiety, insomnia, muscle spasm; examples include diazepam (Valium) and alprazolam (Xanax);[79]
- *Antiparkinson drugs*—used to treat the symptoms of Parkinson disease; examples include levodopa or carbidopa (Sinemet), and Stalevo;[80]
- *Hormonal preparations*—used to treat various gynecological conditions; examples include estrogen (Premarin);[81]
- *Stimulants*—used to treat excessive daytime sleepiness, narcolepsy, as well as fatigue associated with neurological disorders like multiple

https://www.endocrineweb.com/conditions/thyroid/hypothyroidism-too-little-thyroid-hormone (last visited Dec. 22, 2016).

[71] Konstantinos N. Fountoulakis et al., *Thyroid Function in Clinical Subtypes of Major Depression: An Exploratory Study*, 4 BIOMED CENT. PSYCHIATRY 3, 3–4 (2004).

[72] *Dealing with Chronic Illnesses and Depression*, *supra* note 69.

[73] Judith Rabkin et al., *Cognitive Impairment, Behavioral Impairment, Depression, and Wish to Die in an ALS Cohort*, 87 NEUROLOGY 1320, 1320 (2016).

[74] Genevieve Rayner et al., *Mechanisms of Memory Impairment in Epilepsy Depend on Age at Disease Onset*, 87 NEUROLOGY 1642, 1642 (2016).

[75] Mark A. Louter et al., *Cluster Headaches and Depression*, 87 NEUROLOGY 1899, 1899 (2016).

[76] Armon B. Neel Jr., *10 Types of Medications that Can Make You Feel Depressed*, AARP.ORG, http://www.aarp.org/health/drugs-supplements/info-02-2012/medications-that-can-cause-depression.html (last visited Dec. 20, 2016).

[77] *Id.*

[78] *Id.*

[79] *Id.*

[80] *Id.*

[81] *Id.*

sclerosis; examples include methylphenidate (Ritalin) and modafinil (Provigil);[82]
- *Anticonvulsants*—used to treat seizure disorders, and epilepsy; examples include carbamazepine (Tegretol) and topiramate (Topamax);[83]
- *Antacids/proton pump inhibitors*—used to treat heartburn due to gastroesophageal reflux (GERD); examples include cimetidine (Tagamet) and ranitidine (Zantac);[84] and
- *Anticholinergics*—used to treat intestinal cramping disorders such as irritable bowel syndrome; examples include dicyclomine (Bentyl).[85]

In addition to prescription drugs, substance abuse with recreational or illicit drugs can cause depression.[86] Individuals who abuse alcohol face an increased risk of depression;[87] a recovering alcoholic will face this increased risk for up to two years after withdrawal.[88] Individuals who abuse cannabis and methamphetamine also face an increased risk of depression.[89] The DSM-V lists a number of substances in its Substance/Medication-induced Depressive Disorder category, such as alcohol, phencyclidine, other hallucinogens, inhalants, opioids, sedatives, hypnotics, anxiolytics, amphetamines, other stimulants, cocaine, and other—or unknown—substances.[90]

Finally, in addition to medical conditions, chronic illnesses, medications, and substance abuse, the DSM-V also associates head trauma with depression. This Article will discuss this association in depth *infra*. Iverson presents an interesting theoretical model that analyzes the development of depression following mild traumatic brain injury, which incorporates all of these parameters.[91]

2. Genetics

Somewhat related to the biological basis of depression is *genetics*—the inheritability of the tendency for depression.[92] The chromosomal material which

---

[82] *Id.*

[83] *Id.*

[84] *Id.*

[85] *Id.*

[86] *Cause of Depression*, WEBMD, http://www.webmd.com/depression/guide/causes-depression#1 (last visited Dec. 8, 2016).

[87] Joseph M. Boden & David M. Fergusson, *Alcohol and Depression*, 106 ADDICTION 906, 906 (2011).

[88] George F. Koob, *Animal Models of Drug Dependence: Motivational Perspective*, *in* ADDICTION MEDICINE: SCIENCE AND PRACTICE 333, 342 (Bankole A. Johnson ed., 2011).

[89] Brandon D. L. Marshall & Daniel Werb, *Health Outcomes Associated with Methamphetamine Use Among Young People: A Systematic Review*, 105 ADDICTION 991, 991 (2010).

[90] DSM-5, *supra* note 6, at 176.

[91] Grant L. Iverson et al., *Mild Traumatic Brain Injury*, *in* BRAIN INJURY MEDICINE, PRINCIPLES AND PRACTICE 434–69 (Nathan D. Zasler et al. eds., 2d ed. 2013).

[92] *What Causes Depression?*, *supra* note 66.

makes up our genetic structure controls every biological function. To a certain extent, our inherited genetic make-up also controls mood and behavior.[93] This relationship is most notable with bipolar disorder, where half of patients with this diagnosis also have a relative who is bipolar.[94] Also, a person with a first-degree relative (parent or sibling) with major depression is two to four times more at risk of developing major depression.[95] In one study of 450,000 people, researchers identified seventeen genes associated with a significantly increased risk for developing depression.[96] Other genetic factors impact the likelihood of depression. Supporting the serotonin deficiency basis of depression theory, a variant in a serotonin-transporter gene has also been linked to depression, especially when accompanied by stress.[97]

3. Psychosocial/Environmental—Software Problems

"Software" problems leading to depression are those that any of us can experience. These factors are the psychosocial and environmental stressors that can impact the human psyche to such a negative degree as to cause feelings of hopelessness and sadness.[98] These stressful life events include poverty and social isolation, child abuse (physical, emotional, or sexual), the death of a loved one, the loss of a job, emotional or sexual abuse, divorce, and severe chronic prejudice.[99] Many individuals confronted with these situations do not spiral into major depression, but in those individuals whose biological/genetic makeup render them vulnerable to depression, these stressors can become catalysts leading to psychiatric illness.[100] These individuals do not have sufficient coping skills to rise above these traumatic events in their lives.[101] Further, losses and emotional trauma early in life may leave a person more likely to develop depression later in life.[102] Overall, the causes of depression are complex and often involve life stresses impacting one who is vulnerable because of a predisposing biologically-based matrix that they inherited, developed from underlying medical problems, or both.

---

93 *Id.*

94 *Id.*

95 DSM-5, *supra* note 6, at 166.

96 Dana Dovey, *Genetic Causes of Depression: New Study Finds Major Clues*, MED. DAILY, http://www.medicaldaily.com/genetic-causes-depression-23andme-393465 (last visited Dec. 20, 2016).

97 R. Uher & P. McGuffin, *The Moderation by the Serotonin Transporter Gene of Environmental Adversity in the Etiology of Depression: 2009 Update*, 15 MOLECULAR PSYCHIATRY 18, 21 (2010).

98 Stephen E. Gilman et al., *Psychosocial Stressors and the Prognosis of Major Depression as Test of Axis IV*, 43 PSYCHOL. MED. 303, 304 (2013).

99 Ronald C. Kessler, *The Effects of Stressful Life Events on Depression*, 48 ANN. REV. PSYCHOL. 191, 193, 195–96 (1997).

100 *What Causes Depression*, *supra* note 66.

101 *Id.*

102 *Id.*

C. Head Trauma and Depression

Depression is common after head trauma. Approximately half of all individuals with head trauma experience depression within the first year after injury, and up to two-thirds are affected within seven years of the trauma.[103] Reported prevalence rates of depression after brain injury vary widely from 11% to 77%, with rates in the first three months following mild traumatic brain injury ranging from 12% to 44%.[104] In one study of sixty patients who experienced traumatic brain injury, 26.7% experienced major depression and another 11.7% developed alcohol abuse or dependence.[105] As with all causes of depression, the development of major depression following a head injury is multifactorial, involving an interplay of physical changes in the brain due to the injury, the emotional response to the trauma, and pre-existing factors unrelated to the injury such as prior bouts of depression or an inherited predisposition to depression.[106]

1. Physical Damage to the Brain

Recent studies point to specific biochemical and structural factors that can cause depression in cases of traumatic brain injury ("TBI"). From a biochemical standpoint, a brain injury can cause changes with those neurotransmitters associated with depression, such as serotonin, dopamine, norepinephrine, and acetylcholine.[107] In addition to the biochemical changes that may occur, structural damage to certain brain circuits that are known to play a role in depression may occur due to these circuits' vulnerability during head trauma.[108] These regions include the frontal cortex,[109] temporal cortex,[110] and hippocampus,[111] indicating that the brain regions and circuitry associated with depression are at the greatest risk for damage during a brain injury.[112]

---

[103] Iverson et al., *supra* note 91, at 452.

[104] *Id.* at 451.

[105] Salla Koponen et al., *Axis I and II Psychiatric Disorders After Traumatic Brain Injury: A 30-Year Follow-Up Study*, 159 AM. J. PSYCHIATRY 1315, 1315–17 (2002).

[106] Iverson et al., *supra* note 91, at 451.

[107] Thomas W. McAllister, *Emotional and Behavioral Sequelae of Traumatic Brain Injury*, *in* BRAIN INJURY MEDICINE, PRINCIPLES AND PRACTICE 1034, 1034–35 (Nathan D. Zasler et al. eds., 2d ed. 2013).

[108] *Id.* at 1036.

[109] *Id.* The frontal lobes are thought to be the center for emotional control as well as home to personality. *See generally* CENTRE FOR NEURO SKILLS, *Frontal Lobes*, NEUROSKILLS.COM, http://www.neuroskills.com/brain-injury/frontal-lobes.php (last visited Dec. 21, 2016).

[110] McAllister, *supra* note 107, at 1036. The temporal lobes focus on arranging sensory input, auditory acuity, language, speech production, memory association and formation. *See generally* Regina Bailey, *Temporal Lobes*, THOUGHTCO.COM (Oct. 3, 2017), https://www.thoughtco.com/temporal-lobes-anatomy-373228.

[111] McAllister, *supra* note 107, at 1036. The hippocampus is that part of the brain mainly dealing with memory and spatial navigation. *See generally* Ananya Mandal, *What Is the Hippocampus?*, NEWS-MEDICAL.NET (Aug. 3, 2017), http://www.news-medical.net/health/Hippocampus-What-is-the-Hippocampus.aspx.

[112] McAllister, *supra* note 107, at 1036.

An interesting study of brain MRI morphometry (automated measurements), which compared patients with TBI and non-TBI depression, revealed that both groups of patients had diminished tissue volume in the same parts of the brain—the left rostral anterior cingulate[113] and the bilateral orbitofrontal cortex.[114]

2. Emotional Response to Injury

Depression can arise as a psychological reaction to the injuries sustained, whether real or imagined, and the injuries can impact the person's ability to function. The person may be experiencing monetary loss by not being able to work; social isolation by not interacting with friends and family; marital discord; or perceived general loss of function through pain and/or cognitive difficulties. Enduring chronic headaches and neck pain are common symptoms that can result from head trauma and provoke associative depression.[115] Persistent pain restricting activities may bring about a sense of victimization as the result of the head injury, particularly when the person perceives him or herself as without fault.[116] This response is prevalent particularly when the victim had a prior history of physical, sexual, or emotional abuse, or a combination of the three.[117] Closely tied in with victimization is a sense of loss of control of one's life, particularly when trying to work through "the system."[118] Working through the system includes maneuvering through multiple physician and specialty evaluations without a clear medical diagnosis, legal entanglements, the consequences of adverse independent medical evaluations ("IME"), and an insurer's delay or refusal to provide a benefit.[119] Moreover, the possibility of secondary gain, either consciously or subconsciously realized, cannot be ignored.[120] For example, one group of researchers found that workers' compensation-injured participants, in a multidisciplinary pain program receiving compensation, did not respond as well as those not receiving compensation.[121]

---

[113] This anatomical region is "a central station for processing top-down and bottom-up stimuli and assigning appropriate control to other areas in the brain." *Anterior Cingulate Cortex*, WIKIPEDIA, https://en.wikipedia.org/wiki/Anterior_cingulate_cortex (last visited Dec. 21, 2016).

[114] Anne Hudak et al., *Brain Morphometry Changes and Depressive Symptoms After Traumatic Brain Injury*, 191 PSYCHIATRY RES: NEUROIMAGING 160, 160–64 (2011).

[115] Hubbard et al., *Outcome Measures*, *supra* note 58, at 330, 335.

[116] *Id.* at 335.

[117] Robert L. Karol et al., *Physical, Emotional, and Sexual Abuse Among Pain Patients and Health Care Providers: Implications for Psychologists in Multidisciplinary Pain Treatment Centers*, 23 PROF. PSYCHOL. RES. PRAC. 480, 483 (1992).

[118] Hubbard et al., *Outcome Measures*, *supra* note 58, at 336.

[119] *Id.*

[120] John W. Burns et al., *Association Between Workers' Compensation and Outcome Following Multidisciplinary Treatment for Chronic Pain: Roles of Mediators and Moderators*, 11 CLINICAL. J. PAIN 94, 94–95 (1995).

[121] *Id.*

3. Pre-Existing Factors

A pre-existing history of depression also affects the development of depression after head trauma. McAllister said, "[p]re-injury depression and other psychiatric disorders including substance abuse greatly increase the risk of post-injury depression."[122] Whereas, Iverson and his coworkers observed that individuals who have a TBI have higher rates of pre-injury psychiatric disorders such as depression and substance abuse.[123] The number of prior episodes of depression is also predictive of the probability of developing depression after head trauma.[124] Other pre-injury factors placing the TBI patient at risk for post-traumatic depression include other mood and anxiety disorders, psychosocial dysfunction, and alcohol abuse.[125]

In a study reporting that 33% of ninety-one patients developed major depression within the first year after TBI, researchers found that those individuals who developed depression were more likely to have a prior history of mood and anxiety disorders as well as a history of exhibiting aggressive behavior.[126] Those with post-TBI depression also struggled with problem solving, cognitive flexibility, and social interactions.[127] Another study that analyzed depression and suicidal behavior resulting from mild traumatic brain injury reported that the TBI depressed patients were more likely to be male, to have a history of substance abuse, to be more aggressive and hostile, and to attempt suicide or display suicidal ideation.[128]

Thus, the cause of depression following brain trauma does not have one simple explanation, but rather is the result of multiple interrelated factors acting in unison to produce depression. As Iverson stated:

> From an etiological perspective, the manifestation of depression likely represents the cumulative effect of multiple variables, not all of which are operative in any given case, such as genetics, adverse life events in childhood, mental health history, current life stress, activity restrictions and reduced exposure to positive reinforcements in daily life, general medical problems, chronic pain, insomnia, chronic headaches, a co-occurring anxiety disorder (e.g., PTSD), substance abuse, and macroscopic or microstructural damage to the brain.[129]

---

122 McAllister, *supra* note 107, at 1040.

123 Iverson et al., *supra* note 91, at 451.

124 *Id.*

125 Jonathan M. Silver et al., *Depression and Cognitive Complaints Following Mild Traumatic Brain Injury*, 166 AM. J. PSYCHIATRY 653, 657 (2009).

126 Ricardo E. Jorge et al., *Major Depression Following Traumatic Brain Injury*, 61 ARCHIVE GEN. PSYCHIATRY 42, 48 (2004).

127 *Id.*

128 Maria A. Oquendo et al., *Suicidal Behavior and Mild Traumatic Brain Injury in Major Depression*, 192 J. NERVOUS MENTAL DISORDERS 430, 430–32 (2004).

129 Iverson et al., *supra* note 91, at 451.

D. Causes of Head Trauma

Risk of head trauma is ever-present for anyone who can walk or ride in some type of vehicle. In the civilian population, the causes for TBI include motor vehicle accidents (45%), falls (30%), work related accidents (10%), recreational accidents (10%), and assault (5%).[130] According to the Center for Disease Control and Prevention ("CDC"), falls are the leading cause of TBIs, greatest in children under four years old and in adults seventy-five years old or older.[131] The CDC estimates that between 1.6 and 3.8 million concussions occur annually in the United States, accounting for 5% to 9% of all sports injuries, with 30% occurring in individuals between the ages of five and nineteen years old.[132] In one study, those individuals particularly at risk for a TBI include:

- Young, single individuals;[133]
- Low income individuals, especially minorities;[134]
- Individuals with a prior history of substance abuse;[135] and
- Individuals with a prior history of previous TBI.[136]

These, along with many other studies, show that head trauma is pervasive. Head trauma also has significant medical, economic, and legal ramifications.[137] The two most studied causes of head trauma, particularly as related to depression, are athletics and the military.

1. Athletics

Practically every athlete, whether school-aged or professional, is prone to head trauma. An estimated 3.8 million sports-related concussions—considered mild TBIs—occur annually in the United States.[138] In high school and collegiate sports, the highest incidence of concussion occurs among players in men's collegiate football (3.02/1000 games), followed by men's collegiate ice hockey (1.96/1000 games), and

---

[130] Bryan Jennett & Ralph Frankowski, *The Epidemiology of Head Injury*, HANDBOOK CLINICAL NEUROLOGY 1, 10–11 (1990).

[131] CTR. FOR DISEASE CONTROL & PREVENTION, TRAUMATIC BRAIN INJURY IN THE UNITED STATES: EMERGENCY DEPARTMENT VISITS, HOSPITALIZATIONS AND DEATHS 2002-2006 (2010), https://www.cdc.gov/traumaticbraininjury/pdf/blue_book.pdf.

[132] Luke M. Gessel et al., *Concussions Among United States High School and Collegiate Athletes*, 42 J. ATHLETIC TRAINING 495, 495 (2007).

[133] John E. Hubbard, *Head Trauma and Brain Injury: An Overview*, *in* HEAD TRAUMA AND BRAIN INJURY FOR LAWYERS 3, 4 (Jack E. Hubbard & Samuel D. Hodge eds., 2016) [hereinafter Hubbard, *Head Trauma and Brain Injury: An Overview*].

[134] *Id.*

[135] *Id.*

[136] Kelly S. Tieves et al., *The Epidemiology of Traumatic Brain Injury in Wisconsin, 2001*, 104 WIS. MED. J. 22, 22 (2005).

[137] Hubbard, *Head Trauma and Brain Injury: An Overview*, *supra* note 133, at 4.

[138] Christopher G. Giza et al., *Summary of Evidence-Based Guideline Update: Evaluation and Management of Concussion in Sports*, 80 NEUROLOGY 2250, 2251 (2013).

women's collegiate soccer (1.8/1000 games).[139] Conversely, the lowest incidence of concussion occurs among players in women's high school baseball and softball (0.04/1000 games).[140] Overall, American football has become the sport most closely associated with brain injury.[141] A study of professional NFL players from 1996 to 2001 revealed that 0.41 concussions occurred per game, with quarterbacks at the greatest risk, followed by wide receivers, tight ends, and defensive secondary players.[142]

The incidence of depression is significantly increased among football players, leading the media frequently to report on suicides of NFL and former NFL players. In a study of retired NFL players, 40% reported mild to moderate symptoms of depression as compared to an age-matched control group of 15%, a rate nearly three times higher than that of the general population.[143] Common symptoms reported in this study included feelings of sadness, worthlessness, self-criticism, and suicidal thoughts, as well as problems with concentration, changes in appetite, loss of energy, sleep pattern changes, and decreased interest in sex.[144] Furthermore, the same study demonstrated a close correlation between the number of concussions and the depth or severity of the depression.[145]

In addition to the psychological impact of depression, athletes subjected to multiple head traumas, such as football players and boxers, may develop a progressive neurological disorder termed *chronic traumatic encephalopathy* ("CTE").[146] Bennet Omalu, M.D., a pathologist in the Allegheny Coroner's Office in Pittsburgh, first described this condition that gained public attention in the 2015 movie *Concussion* starring Will Smith.[147] Dr. Omalu's paper[148] described autopsy results detailing abnormalities present in the brain of Mike Webster, a former Pittsburgh Steeler lineman, finding abnormal concentrations of the tau protein,[149] now the hallmark of

---

[139] *Id.*

[140] *Id.*

[141] Elliot Pellman et al., *Concussion in Professional Football: Epidemiological Features of Game Injuries and Review of the Literature—Part 3*, 54 NEUROSURGERY 81, 81 (2004).

[142] *Id.*

[143] Nyaz Didehbani et al., *Depressive Symptoms and Concussions in Aging Retired NFL Players*, 28 ARCHIVES CLINICAL NEUROPSYCHOLOGY 418, 422 (2013).

[144] *Id.* at 421–22.

[145] *Id.* at 418.

[146] *CTE Resources*, CONCUSSION LEGACY FOUND., https://concussionfoundation.org/CTE-resources/what-is-CTE (last visited Sept. 11, 2017).

[147] *The Real Story Behind 'Concussion'*, BIOGRAPHY.COM, https://www.biography.com/news/concussion-movie-true-story (last visited Sept. 11, 2017).

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

[149] Tau protein is protein that acts as a structural stabilizer for neuronal axons. *See generally What Are Tau Proteins?*, NEWS-MEDICAL.NET, https://www.news-medical.net/life-sciences/What-are-Tau-Proteins.aspx (last visited Sept. 26, 2017).

CTE.[150] Many professional football players have had similar brain abnormalities, like Chicago Bears' safety Dave Duerson, who committed suicide in 2011.[151] Notably, most of the football players mentioned in the Introduction, *supra*, had CTE at the time of their autopsies.[152] In addition to progressive cognitive decline, CTE is associated with severe depression and suicidal ideation.[153]

2. Military

Experts consider TBIs to be the signature injury of present day military conflict.[154] From 2000 to 2012, a total of 244,217 TBIs occurred in the military, with 76.8% classified as mild, 16.6% as moderate, and 1% as severe.[155] Explosions with a blast injury is the leading cause of TBIs for active military personnel on the battlefield.[156] In one study of combat-related brain injuries, 45.2% were caused by direct blast, 35.7% occurred inside a structure or vehicle with a nearby blast, 9.5% were caused by vehicular crashes, and 9.5% were caused by combat-related falls.[157] In the Iraq and Afghanistan conflicts, most blast injuries resulted from improvised explosive devices ("IED"), landmines, rocket propelled grenades ("RPG"), and other similar explosives.[158]

Troops who experience a TBI are more likely to develop depression.[159] A study of service members returning from Iraq found that head injury with loss of consciousness was independently associated with the development of major depression.[160] In a similar study of troops returning from Iraq, 31.3% of the service members with mild TBIs tested positive for depression.[161] Also of concern is the increasing risk of suicide

---

[150] Jack E. Hubbard, *Delayed Neurological Complications After Head Trauma*, *in* HEAD TRAUMA AND BRAIN INJURY FOR LAWYERS, 170, 182 (Jack E. Hubbard & Samuel D. Hodge eds., 2016).

[151] *Id.*

[152] *Id.* at 183.

[153] Robert A. Stern et al., *Clinical Presentation of Chronic Traumatic Encephalopathy*, 81 NEUROLOGY 1122, 1126 (2013).

[154] Hubbard, *Head Trauma and Brain Injury: An Overview*, *supra* note 133, at 3, 4.

[155] *Id.*

[156] *Id.*; *see also* Howard R. Champion et al., *Injuries from Explosions: Physics, Biophysics, Pathology, and Required Research Focus*, 66 J. TRAUMA 1468, 1472 (2009).

[157] Matthew Kozminski, *Combat-Related Post-Traumatic Headaches: Diagnosis, Mechanisms of Injury, and Challenges to Treatment*, 9 J. AM. OSTEOPATHIC ASS'N 514, 515 (2010).

[158] Josh Duckworth et al., *Traumatic Brain Injury in the Military*, *in* HEAD TRAUMA AND BRAIN INJURY FOR LAWYERS 436, 437 (Jack E. Hubbard & Samuel D. Hodge eds., 2016).

[159] Charles Hoge et al., *Mild Traumatic Brain Injury in U.S. Soldiers Returning from Iraq*, 358 NEW ENG. J. MED. 453, 453–62 (2008).

[160] *Id.* at 457.

[161] Jennifer Vasterling & Molly Reis Franz, *Neuropsychological Outcomes of Mild Traumatic Brain Injury, Post-Traumatic Stress Disorder and Depression in Iraq-Deployed US Army Soldiers*, 201 BRITISH J. PSYCHIATRY 186, 186 (2012).

among combat veterans who sustained brain injuries. For instance, 161 combat personnel in one study reported a direct correlation between the number of head injuries they sustained and the risk for suicide.[162] Furthermore, nearly 22% of troops who experienced more than one TBI in their lifetime reported suicidal ideations compared to the 6% of service personnel who had only one TBI; those with no history of TBI reported no suicidal thoughts.[163] Brain injuries that occurred earlier in life, such as in school sports, further compounded the problem and demonstrably impacted results.[164] The implication is that these earlier injuries created a pre-existing vulnerability to depression that subsequent combat-related head injuries compounded.[165]

Depression is causally linked to TBI, regardless of the root cause of the head injury.[166] Because injuries from sports-related activities and military actions can be closely monitored in these defined populations, investigations into these two areas have provided important insights into both how depression develops and potential risk factors.[167]

III. LEGAL DISCUSSION

Depression is common following a head injury, in varying degrees of severity.[168] The extent that depression contributes to a long-term disability is unknown, but research shows that depression is one of several psychiatric problems that may follow a TBI.[169] This fact is well-known, largely because of highly publicized cases of professional athletes who have committed suicide following repeated blows to the head.[170] Traditionally, plaintiff's counsel overlooks psychological injuries,[171] but all personal injury or disability claims involving brain trauma should be investigated for this complication.

---

[162] Bryan & Clemans, *supra* note 3, at 689–90.

[163] *Id.* at 686–91.

[164] *Id.* at 687.

[165] *Id.* at 690.

[166] *Id.* at 686.

[167] *Id.*

[168] AGENCY FOR HEALTHCARE RESEARCH & QUALITY, TRAUMATIC BRAIN INJURY AND DEPRESSION (2011), https://ahrq-ehc-application.s3.amazonaws.com/media/pdf/depression-brain-injury_research.pdf.

[169] *Id.*

[170] Junior Seau has one of the most well-known stories of a professional athlete who killed himself as the result of chronic traumatic encephalopathy. *9 Football Players Killed By Brain Trauma*, CARE2.COM, http://www.care2.com/causes/9-football-players-killed-by-brain-trauma.html (last visited Dec. 7, 2016).

[171] Jerry Von Talge, *Major Depressive Disorder*, 26 AM. JUR. PROOF OF FACTS 3d § 1 at 1 (2017).

A. Proving the Case

In handling a claim involving depression, one must understand that being "depressed" is not the same as being "sad."[172] The primary source of depression is *loss,* and those who have sustained a brain injury have experienced the most overwhelming loss of all—the loss of their wellbeing. Part II of this Article detailed numerous symptoms of depression at length, which include sleep disturbance, loss of self-confidence, suicidal thoughts, loss of appetite, loss of sexual function, the inability to concentrate, and the resultant loss of cognitive abilities.[173] Even those with mild traumatic brain injuries can suffer from depression and functional disability.[174]

The bible in mental health is the Diagnostic and Statistical Manual of Mental Disorders,[175] taken from DSM-IV. The latest, updated DSM-V categories, discussed *supra*, are:

- Major depressive disorder, single episode;[176]
- Major depressive disorder, recurrent;[177]
- Dysthymic disorder;[178] and
- Depressive disorder not otherwise specified.[179]

These classifications, however, are merely definitional; thus, counsel must be cognizant that the classifications are not treated as diagnostic of the origins of a mental health issue.[180]

B. Witnesses to Testify at Trial

The most obvious witness to help prove or disprove depression is a mental health expert—such as a psychologist, psychiatrist, counselor, or therapist—who has treated the patient.[181] These professionals have known the claimant over a period of time, and they are usually in a superior position to talk about the claimant's mental health.[182]

---

[172] Elizabeth Emmett, *Depression: Research Sheds Light on a Dark Illness*, 31 ARK. L. 4, 4 (1996).

[173] MARTIN BLINDER, PSYCHIATRY IN THE EVERYDAY PRACTICE OF LAW, § 5:16, at 188 (4th ed. 2016).

[174] *Id*. at 189.

[175] Currently in its fifth edition, the Diagnostic and Statistical Manual of Mental Disorders is the accepted classification of mental disorders utilized by mental health professionals in this country. It used for patient diagnosis and treatment, and is important for collecting and communicating accurate public health statistics. *DSM IV*, PSYWEB.COM, http://www.psyweb.com/DSM_IV/jsp/dsm_iv.jsp (last visited Dec. 8, 2016).

[176] Talge, *supra* note 171, at 7.

[177] *Id.*

[178] *Id.*

[179] *Id.* at 8.

[180] *Id.* at 66.

[181] *Id.* at 8.

[182] *Id.* at 66.

Moreover, asking independent mental health experts to examine the same claimant and to express an opinion is allegedly more detached and objective.[183] Therefore, these individuals serve both a therapeutic and forensic role.[184]

Certain ethical constraints are present whenever such a witness is testifying. Judicial proceedings are adversarial, but the mental health professional should not take sides or advocate for one party over another.[185] In this regard, the American Psychological Association's Ethical Principles of Psychologists and Code of Conduct provide that "psychologists seek to promote accuracy, honesty, and truthfulness in science, teaching, and practice of psychology."[186]

Using a patient's treating mental health professional as the expert does have a drawback. The doctor or therapist usually accepts the accuracy of the patient's narrative. Unlike the adversarial legal system, a core tenant in medicine is to trust the patient. Mental health professionals are not trained to constantly scrutinize their patients' stories for falsehoods; if the patient's story is plausible, counsel will carry that narrative into court as a fact by the expert even though it may be inaccurate or misleading.[187] Consequently, opposing counsel frequently challenges a mental health professional's testimony on cross-examination.[188]

Counsel also should not overlook using lay witnesses to prove psychological damages in a brain injury case. This strategy should start early in the case by meeting with claimants and perhaps their significant others. This is important to establish a relationship of trust, to better understand the client's issues, and to grasp the severity of the claimant's depression.[189] The rationale is that clients have a vast amount of information that counsel can glean by spending time with them.[190] Counsel should spend additional time with the clients' significant others, if applicable, because they can share facts that claimants—having sustained head trauma—are unable to recall or convey or are too embarrassed to discuss.[191]

Friends, neighbors, co-workers, and relatives can also be important witnesses; they may be able to provide evidence of a long-standing relationship with the claimant and be able to talk about the changes in the claimant's personality over time as the result

---

[183] Albert Drukteinis, *Treating vs. Expert Psychologist as Witness*, NEW ENG. PSYCHODIAGNOSTICS, http://www.psychlaw.com/LibraryFiles/PsychologistWitness.html (last visited Dec. 8, 2016).

[184] *Id.*

[185] *Id.*

[186] Michael S. Cardwell, *The Psychological Forensic Expert Witness: Ethical Considerations*, THE FORENSIC EXAMINER, http://www.theforensicexaminer.com/2014/Psychforensic.php (last visited Dec. 8, 2016).

[187] Drukteinis, *supra* note 183.

[188] *Id.*

[189] Richard M. Jurewicz, *Traumatic Brain Injury—A Plaintiff's Perspective*, *in* HEAD TRAUMA AND BRAIN INJURY FOR LAWYERS 505, 509 (Jack E. Hubbard & Samuel D. Hodge eds., 2016).

[190] *Id.* at 508.

[191] *Id.* at 509.

of the depressive state.[192] These witnesses rarely have medical training, so they often can explain the mental changes in terms that juries can easily understand.[193]

Counsel should also consider asking the client to maintain a journal of how they feel. Journals are especially important when the client struggles to remember details. This diary can assist counsel in learning about experiences or difficulties that might be helpful at trial for anecdotal testimony.[194] However, counsel must note that the client's depression may prevent him from expending the energy to record events on a regular basis.[195]

C. Strategies for Defense Counsel

On the other hand, defense counsel can take solace in the fact that acute depression is a treatable illness in many people, and most patients will react positively to proper care.[196] The bulk of depressive incidents are self-limiting and should abate without any particular intervention.[197] For example, an untreated occurrence of depression typically takes six to thirteen months to resolve; yet, treatment can accelerate this timetable to approximately six months.[198]

In cases where a claimant's depression is chronic, the defense needs to obtain all of the necessary records to properly investigate the claimant's background. Because depression is usually a secondary manifestation of a brain injury, counsel needs to investigate both the underlying brain trauma and the psychological symptoms. The logical starting point is to obtain the claimant's medical, psychological, and employment records; these records should be from both before and after the brain injury.[199] Counsel should also explore whether the claimant had a history of any pre-existing psychological problems or medical conditions that could cause a psychological manifestation. Other records that may prove beneficial include those from school, the military, if applicable, IQ tests, and SAT scores.

The defense should always be mindful of the possibility of malingering.[200] The American Psychiatric Association's Diagnostic and Statistical Manual buttresses this

---

[192] *Id.* at 510–11.

[193] *Id.* at 509.

[194] *Id.* at 512.

[195] VETERANS AFFAIRS SAN DIEGO HEALTHCARE SYS., *Traumatic Brain Injury: A Guide for Patients* 6, https://www.mentalhealth.va.gov/docs/tbi.pdf.

[196] Talge, *supra* note 171, at 47.

[197] *Id.* at 49.

[198] *Id.* § 36.

[199] *Id.* at 37–38; Scott M. Salter & Allison J. Adams, *TBI Neuropsychological Testing: A Defense Perspective*, *in* HEAD TRAUMA AND BRAIN INJURY FOR LAWYERS 529, 529–30 (Jack E. Hubbard & Samuel D. Hodge eds., 2016).

[200] Steve Rubenzer, *Personal Injury Settings: Malingering Psychiatric Disorders and Cognitive Impairment*, 47 No. 4 DRI FOR DEF. (DRI) 18, at 1 (April 2005); *see generally* H. W. Lebourgeois III, *Malingering: Key Points in Assessment*, PSYCHIATRIC TIMES (Apr. 15, 2007), http://www.psychiatrictimes.com/forensic-psychiatry/malingering-key-points-assessment ("*DSM-IV-TR* defines malingering as the "intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives such as avoiding military

point by making abundantly clear that "malingering should be ruled out in those situations in which financial remuneration, benefit eligibility, and forensic determinations play a role."[201] The problem is that the mental health professional may not know that the claimant has such motives, frequently does not question the chance of malingering, and usually fails to possess the education or knowledge to check for malingering even if they believe it is present.[202] As a result, counsel should always cross-examine the expert concerning whether the witness considered and checked for malingering.

D. Skilled Litigators, Mediators, and Judges Examine the Link Between Traumatic Brain Injuries and Depression

Discussed *supra* in the medical section of this Article, depression is a complex issue that can be caused by a variety of factors other than a brain injury.[203] Contributing factors include problems such as physical or sexual abuse, certain medications, a death in the family, and a major event such as a divorce or substance abuse.[204] Chronic illness can also cause depression including diabetes, heart disease, arthritis, lupus, kidney disease, and hypothyroidism.[205] Seasonal changes and smoking also have been linked to depression.[206] Thus, counsel needs to obtain the person's records to search for other possible causes for the mental illness.

Cases concerning a traumatic brain injury and depression require a large amount of investigation—which makes litigating them very expensive.[207] These cases are also time consuming and complicated. In this section, attorneys, mediators, and judges who have handled traumatic brain injury cases involving depression provide insight into how they approach these cases.

For instance, plaintiff's attorney J.B. Dilsheimer, Esq., stated, "[d]epression is a common, often expected side effect of a traumatic brain injury. The consequences of depression can be just as devastating as the TBI itself."[208] Dilsheimer went on to add,

---

duty, avoiding work, obtaining financial compensation, evading criminal prosecution, or obtaining drugs").

[201] Rubenzer, *supra* note 200, at 1.

[202] *Id.*

[203] *Cause of Depression*, *supra* note 86.

[204] *Id.*

[205] *Id.*

[206] Allanah Dykes, *Instagram's New Feature Is a Mental Health Game-Changer*, HEALTH.COM (Nov. 2, 2016), http://www.health.com/depression/new-instagram-feature-mental-health; Mayo Clinic Staff, *Seasonal Affective Disorder (SAD)*, MAYOCLINIC.ORG (Sept. 12, 2014), http://www.mayoclinic.org/diseases-conditions/seasonal-affective-disorder/basics/definition/con-20021047; Krisha McCoy, *Smoking and Depression*, EVERYDAYHEALTH.COM (July 6, 2012), https://www.everydayhealth.com/depression/smoking-and-depression.aspx.

[207] NEWSOME MELTON, *Case Value of Traumatic Brain Injuries*, BRAINANDSPINALCORD.ORG, http://www.brainandspinalcord.org/case-value-traumatic-brain-injuries/ (last visited Aug. 22, 2017).

[208] Mr. Dilsheimer is a plaintiffs' attorney with Saltz Mongeluzzi Barrett & Bendesky, P.C. and handles complex personal injury cases. His comments are based upon an email exchange

> [t]hat depression [as] a side effect of a TBI is completely understandable. Those who suffer this type of injury can no longer perform a myriad of tasks taken for granted pre-injury and may no longer be living pain free. Chronic headaches, inability to multi-task, light and noise sensitivity, short term memory loss, word retrieval problems, loss of sexual desire, dizziness, and so on can all lead to chronic frustration, irritability, and depression.[209]

Richard M. Jurewicz, Esq., who has written on the topic of brain trauma and subsequent depression said, "[t]here is plenty of medical literature available that supports the proposition that a major stressor in life, such as an accident with significant injuries, can trigger depression."[210] When asked how he would prove the relationship between a brain injury and depression, he noted:

> First, I would review the records of the primary care physician to ascertain if the claimant has a pre-accident history of mental illnesses. After all, someone with a serious bout of depression will usually go to the family doctor for evaluation, to obtain medication for the condition or to obtain a referral to a mental health specialist for clinical management. If there is no documented record of a prior history of depression, this "negative evidence" has a significant value to support causation. Secondly, someone with true depression cannot hide or fake it as their affect will be very "flat," something that is easily noticeable by family, and close friends and perhaps co-workers. So, I would canvass [sic] people that know the injured person well and can provide non-clinical proof of this condition.[211]

Clifford Rieders, Esq., offered more insight on the connection between depression and brain trauma. Rieders stated, "[t]he connection between a traumatic brain injury and depression is a known quantity. From a practical point of view, depression always seems to follow head trauma and that has been vindicated by the work of many neuropsychologists that we have utilized in a litigation context."[212]

---

with the authors on December 12, 2016. E-mail from J.B. Dilsheimer, Att'y, Saltz Mongeluzzi Barrett & Bendesky, P.C., to Samuel D. Hodge, Jr. & Jack E. Hubbard (Dec. 12, 2016) (on file with authors) [hereinafter Dilsheimer E-mail]; *see generally J.B. Dilsheimer*, SALTZ MONGELUZZI BARRETT & BENDESKY, P.C., http://www.smbb.com/attorneys/jb-dilsheimer (last visited Aug. 25, 2017).

[209] Dilsheimer E-mail, *supra*, note 208.

[210] Richard M. Jurewicz, Esquire, is a top-rated lawyer and senior partner with Galf and Berger. His comments are based upon an email exchange with the authors on December 12, 2016. E-mail from Richard M. Jurewicz, Senior Partner, Galf and Berger, Samuel D. Hodge, Jr. & Jack E. Hubbard (Dec. 12, 2016) (on file with authors) [hereinafter Jurewicz E-mail]; *see generally Richard M. Jurewicz*, GALF BERGER LLP, http://www.galfandberger.com/attorneys/richard-jurewicz/ (last visited Aug. 25, 2017).

[211] Jurewicz E-mail, *supra* note 210.

[212] Clifford Rieders, Esquire, is the past President of the Pennsylvania Association for Justice, founding member of Pennsylvania's Patient Safety Authority, and Chair of the Pennsylvania Bar Association's Health Care Law Committee. His comments are based upon an e-mail exchange with the authors on December 12, 2016. E-mail from Clifford A. Reiders, Chair of the Pa. B. Ass'n Health Care Law Comm., Partner, Rieders, Travis, Humphrey, Waters & Dohrmann, to Samuel D. Hodge, Jr. & Jack E. Hubbard (Dec. 12, 2016) (on file with authors);

Attorneys recognize the complexity of trying a TBI and depression case; these unique cases lend themselves to unique litigation tactics. Anthony Baratta, Esq., an attorney from Philadelphia, provided advice about trying a TBI and depression case.[213] While the medical, psychological, and legal complexities of these cases may seem overwhelming to lay jurors, Baratta noted, "I believe in explaining these issues to a jury. It is important that the legal principle of taking your victim as you find them is imperative and must be discussed up front."[214] Baratta suggested that attorneys should remain focused on the effects that TBI and its accompanying depression has on the claimant's life, stating "TBI and depression are labels which help categorize a problem so it can be diagnosed, treated and then properly compensated. Whatever the label that is used, it is the effect on the injured person's life which must be evaluated and judged."[215] On a final note, Baratta provided counsel with the following specific suggestions to consider when trying a TBI and depression case:

1. Counsel must prove the old normal through witnesses, including medical providers, who knew the person before the incident occurred;[216]
2. Counsel must explain what happens to the brain in a TBI;[217]
3. Counsel can use neuropsychological testing to help demonstrate a baseline of the brain before and after the injury;[218]
4. Counsel should rely on psychologists to help explain why symptoms of a TBI mix with the challenges of life to create anxiety and depression;[219] and
5. Counsel should carefully select lay witnesses to describe both the person before and after the event.[220]

While the above attorneys provide valuable advice on how lawyers should handle the adversarial task of either bringing or defending a TBI and depression case, judges and mediators offer similarly valuable advice because they occupy a more neutral role in the proceedings. The Honorable Richard B. Klein, a mediator and former judge,

---

see generally Clifford A. Reiders, RIEDERS, TRAVIS, HUMPHREY, WATERS, & DOHRMANN, http://www.riederstravis.com/attorneys/clifford-a-rieders/ (last visited Aug. 25, 2017).

213 Anthony J. Baratta, Esquire, is a founding member of the firm Baratta, Russell, and Baratta, which is located in the suburbs of Philadelphia. His comments are based upon an e-mail exchange with the authors on December 13, 2016. E-mail from Anthony J. Baratta, Founding Member, Baratta, Russell & Baratta, to Samuel D. Hodge, Jr. & Jack E. Hubbard (Dec. 13, 2016) (on file with authors) [hereinafter Baratta E-mail]; *see generally Anthony J. Baratta*, BARATTA, RUSSELL, & BARATTA, https://www.barattarussell.com/about-us/meet-our-lawyers/anthony-j-baratta/ (last visited Aug. 25, 2017).

214 Baratta E-mail, *supra* note 213.

215 *Id.*

216 *Id.*

217 *Id.*

218 *Id.*

219 *Id.*

220 *Id.*

explained how he determines whether a claimant has raised a credible claim for depression following a traumatic brain injury. Judge Klein said,

> to some extent I use the traditional tools in evaluating whether or not there is a credible claim for depression following a traumatic brain injury. I look at the qualifications of the experts as I would in any case where there is a battle of the experts. As a mediator, I generally will have the opportunity to hear from the injured party and make judgments as to credibility.[221]

In addition to evaluating the qualifications of the experts, Judge Klein provided further insight into how he approaches a TBI and depression case from the bench:

> One of the first things to assess is the nature of the brain injury. How did it occur and should it be classified as mild, moderate or severe? It is normal for one who has a significant brain injury that has disrupted his or her life to feel sad because of the problems that ensue. The question, therefore, becomes whether it rises beyond the normal to be classified as serious depression. The symptoms the experts will be asked to evaluate include feeling sad, lacking energy, not enjoying daily life, trouble sleeping, feelings of guilt or worthlessness and others. The judge or mediator can look at the evidence presented to see if these are real complaints.[222]

The prevalence of depression is not lost on Judge Klein, who noted that "[i]t should not be overlooked that people suffer from depression even without physical injury, and the experts say the number is about one in ten."[223] However, Judge Klein recognized the strong correlation between TBIs and depression and is aware that "the experts also opine that two to three times as many people with a traumatic brain injury develop depression."[224] Judge Klein then suggested that other physical effects of the TBI, apart from the depression, are important to keep in mind, specifically whether "the person has headaches, ringing in the ears, mood changes, memory problems, etc." following the injury.[225] Additionally, Judge Klein said it is important "to ascertain whether the claimant had counseling or was put on antidepressants."[226] If the claimant was put on antidepressants following the TBI, Judge Klein finds it important to ask what the results were and whether "the person ha[d] side effects from the medication"[227]

---

[221] Judge Klein was both a trial and appellate court judge in Pennsylvania and currently serves as a mediator with the *Director of Dispute Resolution Institute.* E-mail from Judge Richard B. Klein, Former Judge, Mediator, Disp. Resol. Inst., to Samuel D. Hodge, Jr. & Jack E. Hubbard (Dec. 13, 2016) (on file with authors) [hereinafter Klein E-mail]; *see generally Judge Richard B. Klein (Ret.)*, DISP. RESOL. INST., http://adrdri.com/member/judge-richard-b-klein-ret/ (last visited Aug. 25, 2017).

[222] Klein E-mail, *supra* note 221.

[223] *Id.*

[224] *Id.*

[225] *Id.*

[226] *Id.*

[227] *Id.*

The Honorable A. Michael Snyder also discussed what he looks for in deciding whether a claimant's depression is believable and related to the trauma:

> There are no magic words that do it. I look to see if the individual had been treated for depression before the trauma or not. If there was no treatment before, then the logical conclusion is that the depression is related. If there was treatment before, did the frequency change, or medications change after the trauma. If the individual had been treated before, but had stopped treatment before the trauma, and treatment again resumes, I would normally assume that the new treatment was related. Of course, one looks to see whether the treating psychiatrist or psychologist will provide a report indicating causal connection, but that is just too easy.[228]

In addition to considering whether the claimant was ever treated for depression before the TBI, Judge Snyder found consideration of how the claimant's behavior has changed post-TBI to be important.[229] For instance,

> [i]f the individual was able to work or relate to the world before the trauma but can't do so after, again the conclusion is normally that the depression is related. If there was prior baseline psychiatric testing, not neuropsychological testing, I'd like to see the difference if the testing is done after the trauma.[230]

Mediator Harris Bock offered a contrary view that the physical impact of the injury on the brain could partially cause a claimant's post-TBI depression.[231] Bock opined that "depression does not occur as a result of the insult causing the traumatic brain injury. However, the emotional and cognitive problems that flow from a bona fide TBI can in fact be the legal cause of the resulting depression."[232]

IV. COURT CASES

Depression is usually not the primary focus of a brain trauma case. Most of the time, it is a secondary complication.[233] An analysis of the court decisions in brain

---

[228] Judge Synder is a former Worker's Compensation Judge in Pennsylvania and is currently a mediator with the Dispute Resolution Institute. E-mail from Judge A. Michael Snyder, Former Judge, Mediator, Disp. Resol. Inst., to Samuel D. Hodge, Jr. & Jack E. Hubbard (Dec. 13, 2016) (on file with authors) [hereinafter Snyder E-mail]; *see generally Judge A. Michael Snyder*, DISP. RESOL. INST., http://adrdri.com/member/judge-a-michael-snyder-ret/ (last visited Aug. 25, 2017).

[229] Snyder E-mail, *supra* note 228.

[230] *Id.*

[231] Harris Bock, Esquire, is the *Director of Dispute Resolution Institute* and a recognized leader in alternative dispute resolution. His comments are based upon an e-mail exchange with the authors on December 12, 2016. E-mail from Harris Bock, Esq., Director, Disp. Resol. Inst., to Samuel D. Hodge, Jr. & Jack E. Hubbard (Dec. 13, 2016) (on file with authors) [hereinafter Harris E-mail]; *see generally Harris T. Bock, Esq.*, DISP. RESOL. INST., http://adrdri.com/member/harris-t-bock-esq/ (last visited Aug. 25, 2017).

[232] Harris E-mail, *supra* note 231.

[233] *E.g.*, Morales v. Peake, No. 07-1717, 2009 U.S. App. Vet. Claims LEXIS 26, at *2–3 (Vet. App. Jan. 15, 2009).

trauma cases suggests that claimants seeking to establish the connection between their depression and their brain injuries must appear credible to the court and must successfully prove the causal link between their brain injuries and their depression.[234] The claimant's past health has also been a fertile area of interest.[235] This section will offer an overview of a few cases in which depression was at issue.

A. Insurance

TBCI, P.C. v. State Farm Mutual Automobile Insurance Company involved a no-fault dispute over medical expenses of $62,747 for care rendered to the insured claimant following a car accident.[236] The claimant alleged that he sustained a traumatic brain injury that caused major depression.[237] The treating doctor noted that the patient exhibited a reduced energy level, fatigue, slowed motor function, and isolation from family interaction.[238] A subsequent examination showed additional symptoms of hypersensitivity to light, problems with depth perception, tinnitus, and a numbness and tingling sensation.[239] The defense produced a number of experts who opined that the claimant did not sustain a closed head injury.[240] The court ruled in favor of State Farm, finding State Farm's experts to be more credible.[241] The court noted that the emergency responders found the claimant to be alert and conscious when they arrived on the scene and that he had a normal Glasgow Coma score.[242] Therefore, the court found it questionable that the claimant's experts relied on a history of unconsciousness, as related by the claimant, in establishing the diagnosis of a closed head injury.[243] Evidence also demonstrated that the patient made a poor effort in testing. The court opined that it is impossible for a person with a mild traumatic brain injury to have long-term memory problems; instead, the individual should only have a short-term memory issue.[244] The court further pointed out that the claimant's doctor admitted that "the neuropsychological profile was difficult to interpret due to the relatively uniformed depression across a wide range of abilities."[245]

---

234 *E.g.*, Marks v. 84 Lumber Co., 939 So. 2d 723 (La. Ct. App. 2006).

235 *See, e.g.*, Dillon v. Auto-Owners Ins., No. 14-cv-00246-LTB-MJW, 2014 U.S. Dist. LEXIS 141799, 2014 WL 4976315, at *2 (D. Colo. Oct. 6, 2014); White v. Prudential Ins., 908 F. Supp. 2d 618 (E.D. Pa. 2012); TBCI PC v. State Farm Mut. Auto. Ins., No. 277260, 2008 Mich. App. LEXIS 2712, 2008 WL 4367530, at *6 (Mich. Ct. App. Sept. 25, 2008).

236 *TBCI PC*, No. 277260, at *3.

237 *Id.*

238 *Id.*

239 *Id.*

240 *Id.* at *4.

241 *Id.*

242 *What is the Glasgow Coma Scale?*, GLASGOW COMA SCALE, http://www.glasgowcomascale.org/what-is-gcs/ (last visited Aug. 22, 2017).

243 *TBCI PC*, No. 277260, at *6.

244 *Id.* at *9.

245 *Id.* at *6.

The importance of independent medical examinations ("IME") to corroborate or disprove the link between a TBI and depression was at issue in *Dillon v. Auto-Owners Insurance Company*.[246] Specifically, this case involved a claim for underinsured motorist benefits. The court considered a motion to compel IMEs with a either a neurologist or a psychiatrist in addition to a neuropsychologist.[247] The claimant argued that she suffered from depression and anxiety as the result of a motor vehicle accident.[248] She also alleged cognitive and neurological deficits resulting from a traumatic brain injury.[249] The court granted the motion ordering the IMEs because there was "good cause" to show that the IMEs were needed to determine the extent of the claimant's depression, anxiety, and emotional distress.[250]

White v. Prudential Insurance Company of America involved a claim for long term disability as the result of a rollover car accident.[251] The claimant asserted that he suffered a traumatic brain injury, while Prudential Insurance maintained that he suffered from mental problems unrelated to the accident.[252] Under Prudential's Policy, if the disability is solely related to mental illness, then the benefits are limited to twenty-four months.[253] The facts revealed that before the accident, the claimant was the chief financial officer of a company, where he had to perform complex tasks under pressure.[254] A week after the accident, he lost his job and refused to work in any capacity while undergoing treatment.[255] After exhausting all of his benefits, he applied for long term disability; this claim was denied after a treating physician noted that the claimant was "alert and oriented, could sit, stand, and walk without physical limitations."[256] The doctor also noted that the claimant had no objective evidence of a traumatic brain injury.[257] The claimant appealed, and Prudential reversed its position and granted him long term benefits.[258] Subsequently, Prudential terminated the benefits questioning the etiology of the cognitive impairments and claimed that the claimant's problem was due to depression, which therefore precluded him from long term benefits under the twenty-four month mental illness limitation period.[259] In the end, the court disagreed with Prudential and stated that several doctors noted that his

---

246 Dillon v. Auto-Owners Ins., No. 14-cv-00246-LTB-MJW, 2014 U.S. Dist. LEXIS 141799, 2014 WL 4976315, at *1 (D. Colo. Oct. 6, 2014).

247 *Id.*

248 *Id.*

249 *Id.* at *1.

250 *Id.* at *3.

251 White v. Prudential Ins., 908 F. Supp. 2d 618 (E.D. Pa. 2012).

252 *Id.*

253 *Id.*

254 *Id.* at 621.

255 *Id.* at 623.

256 *Id.*

257 *Id.*

258 *Id.*

259 *Id.* at 624.

disability was from head trauma that caused the depression and other difficulties.[260] Therefore, the depression was a secondary effect of his injury and was caused by the accident.[261]

Laird v. Metlife and Accident Insurance Company also involved a claim under a long term disability policy stemming from injuries following a motor vehicle accident.[262] The claimant argued that he was completely disabled and could not continue his employment because of a traumatic brain injury that constituted an organic brain disease.[263] This problem caused a mood disorder and depression which continued to worsen.[264] A neuropsychologist assessed the claimant's "neurocognitive and adjustment status secondary to cognitive problems from depression and a mild traumatic brain injury sustained in a [motor vehicle accident] four months ago."[265] The defense, however, asserted that the problems were more related to depression than the TBI.[266] The administrator pointed to a twenty-four month limitation period for benefits in the policy but noted the exception for organic brain disease.[267] In this case, the defense asserted that there was no evidence of an organic brain disease and the diagnosis of a traumatic brain injury was not based upon any "persuasive clinical or radiological evidence."[268] Therefore, the plaintiff was not entitled to any additional benefits beyond the twenty-four month period.[269] The court upheld this determination and did not find that the administrator acted in an arbitrary or capricious manner.[270] Nothing in the plan defined organic brain disease but the defendant offered thorough and well-reasoned evidence as to why the disability was due to depression and not an organic brain injury.[271]

B. Torts

This issue also comes up frequently in tortious litigation. *Donnellan v. First Student, Inc.* dealt with a rear-end motor vehicle accident in which the plaintiff was hit in the back of the head by either a generator or a power tool that broke through a cargo cage.[272] The plaintiff was thirty-one years old and had years of consultations,

---

260 *Id.* at 628.

261 *Id.* at 620, 639.

262 Laird v. Metlife & Accident Ins., No. 08–13075, 2009 U.S. Dist. LEXIS 72201, 2009 WL 2496491 at *2 (E.D. Mich. Aug. 17, 2009).

263 *Id.* at *2.

264 *Id.* at *6.

265 *Id.* at *7.

266 *Id.* at *8.

267 *Id.* at *10.

268 *Id.* at *13.

269 *Id.* at *19.

270 *Id.* at *21.

271 *Id.* at *19.

272 Donnellan v. First Student, Inc., 891 N.E.2d 463 (Ill. App. Ct. 2008).

treatments, and physical therapy.[273] He continued to suffer from headaches, pain, sleep disturbances, memory loss, and vision problems.[274] Donnellan was diagnosed with a traumatic brain injury that triggered his depression.[275] The jury found in favor of the claimant and returned a $6 million dollar verdict, which the appellate court later upheld.[276] The case is noteworthy because a SPECT Scan[277] diagnosed the brain injury, and the court held a Frye hearing to determine the test's admissibility.[278]

In *Green v. K-Mart Corporation*, the plaintiff was a patron in a K-Mart store when she was hit in the head by falling crawfish platters.[279] Green was diagnosed with a concussion that led to chronic pain and major depression.[280] An examination revealed that the depression had taken over her life, that she had psychomotor retardation, and that she was guarded and withdrawn.[281] A clinical social worker met with the plaintiff multiple times and noted that her condition had also affected her children, who also exhibited signs of depression.[282] An expert for the defense opined that the plaintiff suffered from undifferentiated schizophrenia and that the depression was related to this mental illness.[283] The jury resolved the conflicting testimony in favor of the plaintiff and noted that none of her psychological problems were present before the incident.[284] The court awarded her $500,000 in general damages and $3,458,453 in future medical expenses.[285]

Simon v. State Farm Mutual Insurance Company involved a plaintiff who was injured in a significant motor vehicle accident in which her car had to be cut in two before she could be extricated.[286] Her treating physician testified that there was a strong indication of a traumatic brain injury and a basal skull fracture.[287] As a result, she developed major depression and occasional thoughts of suicide requiring hospitalization.[288] Her experts noted that the traumatic brain injuries she sustained in

---

[273] *Id.* at 466, 469.

[274] *Id.* at 469.

[275] *Id.* at 472.

[276] *Id.* at 473, 484.

[277] Mayo Clinic Staff, *SPECT Scan*, MAYOCLINIC.ORG (Dec. 23, 2016), http://www.mayoclinic.org/tests-procedures/spect-scan/home/ovc-20303153.

[278] *Donnellan*, 891 N.E.2d at 468.

[279] Green v. K-Mart Corp., 849 So. 2d 814 (La. Ct. App. 2003).

[280] *Id.* at 826.

[281] *Id.*

[282] *Id.* at 827.

[283] *Id.* at 829.

[284] *Id.* at 830.

[285] *Id.* at 832.

[286] Simon v. State Farm Mut. Auto. Ins., 43 So. 3d 990 (La. Ct. App. 2010).

[287] *Id.* at 994.

[288] *Id.*

the accident were the cause of these problems.[289] The defense did not offer any medical evidence to rebut these assertions; rather, it relied upon the testimony of a neuropsychologist who claimed that the plaintiff was malingering.[290] The jury found in favor of the plaintiff, but awarded nothing for disability and only $3,500 for loss of earning capacity.[291] This was found to be reversible error and the appellate court conducted a de novo review.[292] The reviewing court noted that none of the presented evidence contradicted the plaintiff's need for medical care, diagnostic testing, and medication that she required to function every day.[293] While the claimant may have suffered from depression before the accident, she was a different person following the collision.[294] The court also determined that depression increases pain and pain increases depression in a vicious cycle.[295] Evidence demonstrated that the plaintiff had more than three bouts of depression since the accident and, thus, that she had a 90% chance of having life-long problems with depression.[296] Therefore, the court readjusted the award and gave her $234,415 for future medicals, $250,000 in general damages, and $350,000 in loss of earning capacity.[297]

Fairchild v. United States involved a claim under the Federal Tort Claims Act.[298] The plaintiff was involved in a clear liability accident with a cargo truck owned by the government.[299] She sustained an alleged closed head injury and organic delusional depression.[300] Since the accident, numerous witnesses stated that she suffered serious emotional problems including memory loss, sleepiness, and the inability to handle stress.[301] The plaintiff also heard voices and demonstrated other forms of psychotic behavior.[302] The expert for the defense asserted that the plaintiff suffered no type of organic brain damage and that her brain injury was not documented and inconsistent with the information reviewed.[303] The court sided with the plaintiff and noted that the evidence was convincing on the question of whether the claimant struck her head in the accident—evidenced by her broken teeth and the vehicle's damaged windshield.[304]

---

289 *Id.*

290 *Id.*

291 *Id.* at 997.

292 *Id.*

293 *Id.* at 999.

294 *Id.* at 1001.

295 *Id.* at 1000.

296 *Id.*

297 *Id.* at 1006.

298 Fairchild v. United States, 769 F. Supp. 964 (W.D. La. 1991).

299 *Id.* at 965.

300 *Id.* at 966.

301 *Id.*

302 *Id.*

303 *Id.* at 967.

304 *Id.*

Also, the numerous tests conducted by the plaintiff's experts supported the diagnosis of a traumatic brain injury. The court accepted the testimony of the plaintiff's experts who argued that the motor vehicle accident was the stimulus that caused the brain injury and related psychotic disorder.[305] As a result, the court awarded her $490,568 to fund her future lost wages, over $1 million in future medical expenses, and $250,000 for general damages.[306]

In *Davis v. William & Jeanette Victory*, a forty-four-year-old woman was hit on the head by a ladder while walking along the sidewalk.[307] The plaintiff alleged that she sustained a brain injury and depression.[308] Evidence showed that she worked two years following the accident and then took a leave of absence as the result of depression.[309] The defendant asserted that because the plaintiff only suffered from depression and had no permanent brain injury, her inability to work was unrelated to the event.[310] The defendant alleged that the plaintiff was waiting for the compensation from her case in order to start a business.[311] Although the plaintiff demanded $2.5 million, and the jury only awarded her $27,500.[312]

C. Social Security

In *Faucett v. Astrue,* the plaintiff alleged a disability as the result of a subarachnoid hemorrhage, a traumatic brain injury, and depression.[313] The facts demonstrated that the claimant was involved in a motorcycle accident and lost consciousness.[314] Following discharge from the hospital, he was diagnosed with a closed head injury, memory loss, and headaches.[315] He was seen again in the emergency room several months later after having rage outbursts.[316] A psychologist examined Faucett at the request of a state agency, and Faucett stated that he could not work and had difficulty controlling his anger.[317] He was diagnosed with major depressive and cognitive disorders secondary to a traumatic brain injury.[318] A state psychologist reviewed the claimant's medical records and opined that the plaintiff could work in a job that did

---

[305] *Id.*

[306] *Id.* at 968.

[307] Davis v. Victory, [2015] 96 N.W. Pers. Inj. Litig. Rep. (Jury Verdicts N.W., Inc.) 388, at *1, 2 (Wash. Super. Ct. Aug. 14, 1996).

[308] *Id.*

[309] *Id.*

[310] *Id.*

[311] *Id.*

[312] *Id.*

[313] Faucett v. Astrue, No. 3:10-3093-JFA-JRM, 2012 U.S. Dist. LEXIS 39648, 2012 WL 988057, at *1 (D.S.C. Feb. 21, 2012).

[314] *Id.* at *3.

[315] *Id.*

[316] *Id.* at *4.

[317] *Id.*

[318] *Id.*

not involve interaction with the public.[319] The administrative law judge (ALJ) found the claimant was disabled as of his fifty-fifth birthday rather than on the date of the much earlier accident.[320] The plaintiff appealed and claimed that the ALJ did not properly consider his long term cognitive, behavioral, and depressive symptoms that the traumatic brain injury caused.[321] The court on appeal disagreed and found that the ALJ reasonably accounted for the limitations from the injury, limited his work to a job that did not require contact with the public, and found him disabled at the age of fifty-five because the Social Security Grids had a different standard at that age.[322] The court further noted that the Commissioner's findings must be upheld even in view of conflicting evidence if substantial evidence supports the decision.[323]

D. Worker's Compensation

Felipe v. Department of Labor and Industries involved a worker's compensation claim in which the plaintiff fell off a ladder at work.[324] He developed headaches following the injury and was diagnosed with major depression.[325] The claimant went to a psychologist who then referred him to a brain injury specialist.[326] The brain injury specialist thought that the claimant had sustained a traumatic brain injury.[327] The claimant alleged that he suffered at least four headaches a day, vertigo, and light-headedness.[328] His diagnosis was changed to a TBI with uncontrolled major depression.[329] A doctor hired by the Department of Labor admitted that the plaintiff sustained a closed head injury, but opined that it had resolved.[330] The claim was denied and an appeal followed.[331] The claimant asserted that he should not have to prove his injury by objective evidence.[332] The claimant analogized his claim to one involving a psychiatric disability because the symptoms cannot be measured objectively.[333] The court agreed that it was error to require an objective finding for the diagnosis.[334]

---

319 *Id.*

320 *Id.* at *6.

321 *Id.*

322 *Id.*

323 *Id.* at *11.

324 Felipe v. Dep't of Labor & Indus., 318 P.3d 205, 206 (Wash. Ct. App. 2016).

325 *Id.* at 207.

326 *Id.*

327 *Id.*

328 *Id.*

329 *Id.*

330 *Id.*

331 *Id.*

332 *Id.* at 208.

333 *Id.* at 210.

334 *Id.* at 211.

In *Paul Johnson Plastering v. Johnson*, the claimant asserted that he suffered a work related brain injury that resulted in a permanent total disability.[335] The plaintiff was working on dry wall stilts when he fell and sustained various injuries including a wrist fracture.[336] A couple of months later, he complained of vision problems and depression.[337] He tried to work but could not do his job.[338] The plaintiff was referred to a psychologist who diagnosed him with a major depression disorder which "could [have been] triggered" by his fall.[339] He was granted temporary disability benefits which continued for eight years.[340] At the expiration of that time, the worker filed an application for permanent disability.[341] The defense contested the request and produced experts who opined that the plaintiff did not suffer a traumatic brain injury.[342] The deputy commissioner denied compensation claiming that the initial application—filed eight years earlier—did not identify a brain injury so that such a claim was time barred.[343] The court held that if the brain injury was caused by the depression which originated from the original wrist injury, then the brain injury is not compensable.[344] Accordingly, his claim was denied.[345]

Dawson v. Jaflo, Inc. involved a worker's compensation claim by a tree trimmer who was injured when a branch fell and stuck him.[346] The employee claimed that he sustained a concussion, neck sprain, and shoulder injury.[347] During the course of treatment, he went to a neuropsychologist for an evaluation of the extent of his memory loss and mood disorder.[348] A psychological evaluation found that he had a mildly impaired memory and moderately impaired concentration, along with self-reported severe symptoms of anxiety and depression.[349] Another doctor opined that the claimant had a depressive and anxiety disorder related to post-concussion syndrome and made an assessment of a 20% whole person impairment of which he apportioned 5% for any pre-existing condition that may have existed.[350] Another

---

335 Paul Johnson Plastering v. Johnson, 576 S.E.2d 447, 448 (Va. 2003).

336 *Id.*

337 *Id.* at 449.

338 *Id.*

339 *Id.*

340 *Id.*

341 *Id.*

342 *Id.* at 450.

343 *Id.*

344 *Id.* at 452.

345 *Id.*

346 Dawson v. Jaflo, Inc., No. 16-0063, 2016 W. Va. LEXIS 968, 2016 WL 6962334, at *1 (W. Va. Nov. 29, 2016).

347 *Id.*

348 *Id.*

349 *Id.*

350 *Id.*

doctor opined that the worker had a somatic symptom disorder with predominant pain.[351] That physician then stated that the somatic pain disorder was excluded as compensable by the worker's compensation statute and signified the presence of a pre-existing psychological impairment that is not compensable.[352] The ALJ found that the condition demonstrated a pre-existing psychological impairment that was not compensable and noted that the claimant exaggerated his psychological symptoms and memory loss.[353] The appellate court later upheld this ruling.[354]

V. CONCLUSION

Regardless of cause, depression after a head injury occurs frequently, usually within the first year.[355] Considered a disorder of mood, depression is complex with multiple symptoms such as extreme sadness, hopelessness, lack of self-worth, anxiety, and various somatic issues including fatigue, appetite changes, and sleep pattern changes.[356] These problems may become so painful to the claimant that they lead to suicidal behavior and ideation.[357] The diagnostic criteria for depression, as well as the different types of depression, are classified in the Diagnostic and Statistical Manual of Mental Disorders (DSM) published by the American Psychiatric Association—currently in its fifth edition (DSM-V).[358]

The causes of depression vary, but can include biological changes, medical conditions, medications, substance abuse, genetics, psychosocial and environmental stress, family support, and head trauma.[359] The risk for the development of depression in any one individual depends upon the interplay of all of these factors. Pre-existing issues of depression, substance abuse, and head trauma further increase the potential for a depressive mental illness following a TBI.[360] Investigations into TBI in athletics and the military have provided new understanding of the interrelationships of TBI with depression development.

Handling a brain injury case involving depression can be expensive and requires a degree of expertise in understanding these types of problems. The parties must obtain many records to ascertain the claimant's functional status—both before and after the traumatic event. Witness selection is also important. Not only must parties call the appropriate medical and psychological experts to discuss causation, but they must call lay witnesses as well, playing an equally important role. Parties should use friends, neighbors, co-workers, and relatives, if appropriate, to establish a long-standing

---

351 *Id.* at *2.

352 *Id.*

353 *Id.*

354 *Id.* at *3.

355 Ricardo E. Jorge & David B. Arciniegas, *Mood Disorders After TBI*, 37 PSYCHIATRIC CLINICS N. AM. 13–29 (2014).

356 *Id.*

357 *Id.*

358 DSM-5, *supra* note 6.

359 *Id.* at 166.

360 *Id.* at 181.

relationship with the claimant so they can talk about the claimant's personality changes as the result of the depressive state. The defense should remember that in most cases, depression is generally a treatable illness, and patients will react positively to proper care.[361]

If the depression is chronic, the defense should comb through the records to see if any pre-existing psychological problems or medical conditions exist that could be the cause of the psychological manifestations. Counsel should also be mindful of the possibility of malingering and be prepared to cross-examine the witnesses about this possibility.

[361] MENTAL HEALTH AM., DEPRESSION: A TREATABLE ILLNESS (FACT SHEET) (2004).