A TRAUMATIC BRAIN INJURY IS A HEADACHE IN BOTH MEDICAL AND LEGAL CIRCLES

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ABSTRACT

A traumatic brain injury (TBI) is the most feared consequence of head trauma. The reason is simple—the brain is who we are, our essence, including our memories of past times, how we respond to the environment with its stresses and challenges, and how we dream and plan for the future. A TBI has become one of the hottest and most controversial areas in personal injury litigation. As the result of lawsuits against the National Football League, National Hockey League, and professional wrestling associations, considerable publicity has been generated about the side effects of brain injuries. These injuries, however, are not limited to professional sports, car accidents, and military conflicts. Concussions are a major issue in college and high school sports, and protocols on how to test and treat this type of injury are commonplace.

The brain is the highest of the organs in position, and it is protected by the vault of the head; it has no flesh or blood or refuse. It is the citadel of sense-perception.

—Pliny the Elder (23–79 A.D.)

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I. INTRODUCTION

A traumatic brain injury (TBI) is the most feared consequence of head trauma. The reason is simple—the brain is who we are, our essence, including our memories of past times, how we respond to the environment with its stresses and challenges, and how we dream and plan for the future. Consequently, injury to this vital organ places our very being in jeopardy.

Traumatic brain injury is defined as an injury that occurs when external trauma causes injury to the substance and/or functioning of the brain.[1] A

1. S. Yokobori & R. Bullock, *Pathobiology of Primary Traumatic Brain Injury*, *in* BRAIN INJURY MEDICINE 137–61 (N.D. Zasler et al. eds., 2d ed. 2013).

brain injury may occur from non-traumatic causes as well, such as stroke, cardiac arrest, or infection. Those brain injuries, however, are beyond the scope of this article.

Not every head injury results in a TBI. Likewise, a TBI can occur without direct trauma to the head. A TBI may also occur with head trauma but without disruption of the meninges (coverings of the brain). This is referred to as a closed (non-penetrating, blunt) head trauma. When an external force, such as a projectile, breaches the meninges, this is referred to as an open or penetrating head injury. A TBI can be either a focal or a diffused injury. A focal injury is confined to one part of the brain while a diffuse injury is widespread throughout the brain. Often, a focal injury can produce diffuse injury.

Not only is TBI a personal problem, but a societal one as well—the condition is a pervasive and widespread concern. For example, a 1991 survey in the United States estimated that 1.5 million people had sustained a TBI.[2] Between 3.6 and 5.3 million people in the United States have residual problems as a consequence of TBI.[3] Further, the mortality rate within thirty days after a significant TBI has been reported at 21 percent.[4] Civilian deaths caused by head injury occur in motor vehicle accidents (34 percent), firearm incidents (39 percent), falls (10 percent), and other (17 percent).[5] The estimated cost of TBI in 2010 was $76.5 billion with $11.5 billion attributed to direct medical expenses and $64.8 billion due to indirect costs such as lost productivity.[6] TBI can result from all aspects of life, including motor vehicle accidents, falls, and recreational events.

II. DEGREES OF TBI

Traumatic brain injuries can be wide ranging in severity. The extent of the TBI depends upon the forces involved, the nature of injury, and the extent of the brain damage.[7] Using the Glasgow Coma Scale (GCS), the medical profession often classifies traumatic brain injuries as mild,

2. D.M. Sosin, J.E. Sniezek & D.J. Thurman, *Incidence of Mild and Moderate Brain Injury in the United States, 1991*, 10 BRAIN INJURY 47–54 (1996).

3. V.G. Coronado et al., *Traumatic Brain Injury Epidemiology and Public Health Issues*, *in* BRAIN INJURY MEDICINE, *supra* note 1, at 84–100.

4. B.D. Greenwald, D.M. Burnett & M.A. Miller, *Congenital and Acquired Brain Injury, Brain Injury: Epidemiology and Pathophysiology*, 84 ARCHIVES OF PHYSICAL MED. & REHABILITATION S3-7 (2003).

5. J. Leon-Carrion, M.R. Dominguez-Morales, et al., *Epidemiology of Traumatic Brain Injury and Subarachnoid Hemorrhage*, 8 PITUITARY 3–4 (2005).

6. Coronado et al., *supra* note 3.

7. Greenwald et al., *supra* note 4.

GLASGOW COMA SCALE

Response	Score
Eye opening	
• Opens eyes spontaneously	4
• Opens eyes in response to speech	3
• Opens eyes in response to noxious stimulation	2
• Does not open eyes spontaneously or to stimulation	1
Motor Response	
• Follows commands correctly	6
• Appropriate motor response to painful stimulation	5
• Nonpurposeful response to painful stimulation	4
• Flexes upper/extends lower extremities to painful stimulation	3
• Extends all extremities in response to painful stimulation	2
• No motor response to painful stimulation	1
Verbal Response	
• Oriented to person, place, and time	5
• Responds/converses but is confused	4
• Responds/converses but inappropriate/wrong words	3
• No appropriate verbal response; sounds are incomprehensible	2
• No verbal response	1

moderate, or severe.[8] This scale provides a simple, rapid way to immediately evaluate the clinical state of the patient presenting to the emergency room with a head injury. The GCS uses neurological parameters of verbal, motor, and eye-opening reactions to assess the level of consciousness based upon a scale of 3 to 15. Using the scale, mild TBI has a score of 13–15, moderate TBI a score of 9–12, and severe TBI a score of 8 or less. Concussions are classified as mild.

Multiple episodes of mild head trauma can have a cumulative effect that may also result in serious neurological consequences. For example, an individual who experiences a concussion and has a second one before recovering from the first is prone to development of a second-impact syndrome with brain swelling. This can lead to catastrophic neurologic impairment and even death. In cases of repeated head injuries, for example, with a boxer or professional football player, a form of dementia termed chronic traumatic encephalopathy (CTE) can develop in later years and cause progressive cognitive decline.

8. G. Teasdale et al., *Adding Up the Glasgow Coma Score*, 28 Acta Neurochir Suppl (Wien) 13–16 (1979).

III. MECHANISM OF TBI

When the brain is injured, the organ gets hit with two pathological processes. The first, termed primary injury, is a direct, immediate consequence of the trauma causing disruption of brain function. The second, known as secondary injury, is delayed and is a consequence of a series of resulting biochemical and cellular changes that adversely affect the brain.

A. *Primary Injury*

With a primary injury, there is direct tearing, compression, or stretching of the brain and its blood vessels with injury occurring at the instant of the trauma.[9] The damage can occur at the point of impact, termed a coup injury, or on the opposite side of the brain, termed a contrecoup injury.[10] The contrecoup injury results from the differential momentum of the brain striking the interior of the skull on the opposite side of the head, such as when the moving head strikes a stationary object. In the case of a motor vehicle accident, both types of injury can occur, where, for example, the forehead strikes the windshield (coup injury) and the back of the brain strikes the occipital (back) portion of the skull interior (contrecoup injury).

The biomechanics of brain injury depend upon the concentration of force at the point of impact, the velocity of impact, and the resulting head movements.[11] During impact, the skull is deformed, potentially leading to a skull fracture with the underlying brain injured. While several factors are involved, the key components are the speed of head acceleration and the duration of head impact.[12] Studies have shown that head accelerations during activities of daily living, e.g., hopping off steps, coughing, sneezing, running, and jumping, can deliver up to 12 Gs of force without associated brain injury.[13] By comparison, a boxer's punch with a velocity of over 25 mph can deliver over 71 Gs of force.[14]

9. S. Yokobori & R. Bullock, *supra* note1.

10. N.A. Shaw, *The Neurophysiology of Concussion*, 67 PROGRESS IN NEUROBIOLOGY, 281–344 (2002).

11. D.C. Viano, *Biomechanics of Brain Injury*, *in* BRAIN INJURY MEDICINE, *supra* note 1, at 124–36.

12. *Id.*

13. G-force is a unit of force equal to the force exerted by gravity. *Most concussions deliver 95 g's, neuropsychologist says*, SCIENCEDAILY, June 25, 2010, http://www.sciencedaily.com/releases/2010/06/100624092526.htm.

14. *Id.*

1. Intracranial Hemorrhage

The resulting trauma to the brain can produce damage to both the brain substance itself and to the blood vessels going to and from the brain. With regard to blood vessel damage, such as a focal injury where the head is struck by a baseball bat, blood vessel damage over the surface of the brain may occur at the point of impact, causing a contusion or bruising of the brain surface. If the trauma is particularly severe, the impact may cause blood vessel rupture deep within the brain. This is referred to as an intracerebral hemorrhage or hematoma. It is differentiated from a contusion by its size. An intracerebral hematoma is two centimeters or larger and does not reach the brain surface.[15] Unfortunately, the hematoma may expand, especially in individuals taking any medication that thins the blood. This is called a delayed traumatic intracerebral hemorrhage,[16] which leads to a neurological emergency because the increasing hematoma exerts a significant mass effect upon the surrounding brain. With the solid cranial vault made of bone, the increasing pressure has nowhere to go except through the foramen magnum at the base of the skull. The brain is literally pushed down through the opening, a condition referred to as brain herniation. Brain herniation causes death because the brainstem structures controlling vital activities such as heart rate and blood pressure are irreversibly damaged. Often with an expanding mass lesion, including an intraparenchymal bleed or extracerebral hemorrhage (discussed below), the person may initially be awake and neurologically intact in what is termed the lucid interval, but quickly lapse into a coma as the hemorrhage continues to expand.

Bleeding from trauma may also occur outside of the brain but within the skull in a process known as extracerebral hemorrhage, producing equally disastrous results. These types of bleeds are named based on their relationship to the three meningeal layers—epidural hematoma, subdural hematoma, and subarachnoid bleed. In an epidural hematoma, bleeding occurs between the inner skull surface and the dura mater, which is the outermost meningeal covering of the brain. This type of bleeding usually results from a blow to the side of the head, causing a fracture of the temporal bone and tearing the nearby middle meningeal artery. Because this results in an arterial bleed, the lucid interval may be short since the uncontrolled arterial bleeding rapidly increases the pressure within the skull and on the brain.

A subdural hematoma is usually caused by rupture of the veins (bridging veins) that carry blood between the brain surface and the superior sagittal sinus. Subdural bleeding occurs just underneath the dura mater

15. Yokobori & Bullock, *supra* note 1.
16. *Id.*

between the dura mater and arachnoid layer. Veins are lower pressure blood vessels compared to the arteries. The tearing of a vein causes more of an oozing—thus, subdural hematomas can be present for hours to days before they cause clinical problems such as headaches, drowsiness, or focal neurological symptoms or findings. In the elderly, brain atrophy may preclude symptoms from developing for weeks because there is more space available for blood to accumulate. Both epidural and acute subdural hematomas are neurosurgical emergencies that may cause permanent neurological impairment or death. In some cases, an acute or subacute subdural hematoma can become a progressively increasing chronic subdural hematoma as nearby fluid is slowly absorbed into the mass of blood, causing further expansion and neurological impairment.

A subarachnoid hemorrhage refers to bleeding into the space between the arachnoid layer and pia mater that results in a more diffuse collection of blood and generally does not require neurosurgical intervention unless an aneurysm occurs. Head trauma is the most common cause of subarachnoid bleeding. Subarachnoid bleeding can block the flow of cerebrospinal fluid, which is produced within the ventricles of the brain and flows outward from the fourth ventricle to the surface of the brain. When this outflow is blocked by accumulated subarachnoid blood, the condition is hydrocephalus. Hydrocephalus, which can be identified on CT or MRI by enlarged ventricles, results from a damming up of spinal fluid within the ventricles. Hydrocephalus is treated with a neurosurgical procedure involving placement of a shunt to drain off the accumulating spinal fluid.

In addition to vascular rupture, focal brain trauma also produces injury at the site of the head injury. Because of the focal damage created by impact from a heavy object or a motor vehicle accident, the injured person may have localized neurological impairment such as loss of vision, paralysis on one side, or speech difficulty. The focal injury may also cause a more widespread diffuse injury.

2. Microscopic Injury

Much of the damage in TBI, particularly in mild cases, occurs at the microscopic level below the imaging ability of CT or MRI scanning. For example, in a diffuse injury such as a concussion, the result may be a diffuse (traumatic) axonal injury (DAI) caused by microscopic injury to the axons extending from the neurons.[17]

The neuron, which consists of many dendrites, a single cell body, and a single axon, is the functional cell of the nervous system. The dendrites receive the input from other neurons and convert that input in the form of electrical currents that then are processed by the cell body. The cell body

17. *Id.*

contains the nucleus and metabolic machinery of the neuron, such as mitochondria, which serve as power sources to run the metabolic functions of the cell. The output from the neuron is carried away from the cell body by a single axon, which fires in response to the changing neuronal membrane electrical currents. The electrical potential changes are the result of transfer of ions—calcium, sodium, and potassium—across the cell membrane of the neuron. An axon is the single process that extends from the neuronal cell body, which reaches out to connect with other neurons within the brain and spinal cord, thereby allowing communication within the nervous system. Thus, the axon carries the electrical output from the neuron to network with the rest of the nervous system. Information is propagated down the axon in the form of an action potential resulting from rapidly changing ionic fluctuations across the axonal membrane. The axon terminates as a specialized structure known as the synapse. Within the synaptic terminal are synaptic vesicles that contain packets of neurochemicals such as serotonin, dopamine, and glutamate. Upon arrival of an action potential, these packets are released into a narrow gap to stimulate receptors on the dendrites or cell body of the next neuron. This whole process is termed synaptic transmission and is critical for the transfer of information and communication by neurons.

Many axons are covered with myelin, a whitish-appearing insulating material that serves to increase the conduction velocity of the axonal action potentials. When thousands of myelinated axons are bundled together, the resulting appearance is pale white and is termed white matter. Areas damaged by DAI contain a large amount of white matter, such as the corpus callosum, which interconnects the two cerebral hemispheres within the core of the brain. A special form of MRI, known as diffusion tensor imaging, can identify white matter tracts that are traumatically damaged and reveal injuries that are not picked up by routine MRI scanning.

In addition to neurons, a second type of cell is found within the brain that has a supportive role to neuronal function. These are called glial cells. Glial cells come in different forms and have different functions. Oligodendroglia, for example, lay down the myelin by wrapping around axons much like tape is wound around a pipe. In fact, it is abnormal division of a glial cell that results in a primary brain cancer. Neurons within the brain and spinal cord cannot divide, however, and therefore do not become cancerous.

When head trauma occurs, linear and rotational acceleration forces cause the brain tissue to deform, resulting in stretching and compression of the neurons and glial cells.[18] With disruption of the neuronal mem-

18. G.L. Iverson et al., *Mild Traumatic Brain Injury*, *in* BRAIN INJURY MEDICINE, *supra* note 1, at 434–69.

brane, abnormal ionic movements across the neuron cell membrane cause neuronal dysfunction and electrical current suppression. In addition, damage occurs to the mitochondria as well as the dendrites and axons. Within axons is a cytoskeleton made of microtubules and neurofilaments. The large amount of calcium flowing into the axon causes damage to this axonal cytoskeleton.[19] When axonal stretching exceeds six millimeters, the axon can shear off, separating from the neuronal cell body,[20] resulting in neuronal death or at least the inability of that cell to communicate with other cells. In addition, dendrites are lost, further reducing the ability of the neuron to communicate with other neurons.

B. *Secondary Injury*

When trauma occurs to the brain, the resulting immediate damage produces a cascade of secondary events, causing even more brain injury and dysfunction. As discussed previously, disruption of the neuronal membranes results in abnormal inflow of calcium and sodium ions into the nerve cell causing impaired function. Glutamate, an excitatory neurotransmitter that is normally contained within the synaptic vesicles, is released in large amounts paradoxically causing neuronal damage called excitotoxicity.[21] Other lethal events to the neurons include release of inflammatory factors and free radicals. The blood-brain barrier, the important defense mechanism that isolates the brain substance from potential toxins in the blood, is broken down. Even with mild TBI, important damaging ionic changes, cerebral blood flow alterations, metabolic depression, and axonal injury occur.[22] In addition to the shearing injury occurring with diffuse axonal injury as a primary injury, secondary factors contribute to further axonal damage by a delayed process, termed secondary axotomy.[23] Once a neuron dies, it cannot be not replaced because nerve cells do not regenerate.

Swelling or cerebral edema of the brain occurs as a result of neuronal death and axonal destruction in large areas of injury. Because the brain is enclosed within the solid bone of the skull, the cerebral edema exerts significant pressure on the brain, resulting in further damage, as well as the life-threatening possibility of brain herniation through the foramen magnum. The resulting increase in pressure on the arteries within the skull

19. *Id.*
20. A.C. Bain et al., *Dynamic Stretch Correlates to Both Morphological Abnormalities and Electrophysiological Impairment in a Model of Traumatic Axonal Injury*, 18 J. NEUROTRAUMA 499–511 (2001).
21. P.M. Kochanek et al., *Pathobiology of Secondary Brain Injury*, *in* BRAIN INJURY MEDICINE, *supra* note 1, at 148–61.
22. *Id.*
23. *Id.*

can also diminish arterial blood flow to the brain resulting in further brain injury.

In addition to these insults to brain neuronal functioning, other trauma-associated medical conditions can contribute to secondary injury to the brain. For example, any significant drop in blood pressure (hypotension) resulting from blood loss with body trauma diminishes blood flow to the brain and exacerbates the neuronal series of events. Likewise, significantly diminished oxygen (hypoxia) or the lack of oxygen (anoxia) worsens the brain damage.

Therefore, these secondary injuries, which are complex and of a cascading nature, often continue to evolve after the initial trauma. As a consequence, they magnify and extend the damage from the primary injury and account for deteriorating clinical signs hours to days after the head trauma.

IV. PROGNOSIS AFTER A TBI

Recovery following a TBI depends upon the nature and severity of the injury, as well as the underlying medical health of the injured individual. The spectrum ranges from the absence of neurological residual effects to death. Long-lasting disability occurs in 100 percent of severe TBIs, 66 percent of moderate TBIs, and 10 percent of mild TBIs.[24] For the most part, mild trauma does not cause any permanent disability, and the person is eventually able to resume normal pre-injury daily activities. Residual difficulty, where it appears, is generally limited to memory or other cognitive impairments.[25] Approximately 90 percent of those who experience moderate TBI are able to live independently, but may need assistance with work, help with managing finances, or require assistive devices such as a cane or a walker to minimize the disabling impact of residual physical problems.[26]

Severe TBI, on the other hand, has more serious consequences. In their detailed analysis of the literature, Kothari and DiTommaso[27] define outcomes following severe brain injury, using the following Glasgow Outcome Scale (GOS):

- Death
- Vegetative state: alive but the brain is unresponsive
- Severe disability: conscious but totally dependent, not able to live alone for more than twenty-four hours, requires assistance for daily activities

24. Leon-Carrion, *supra* note 5.
25. G.L. Iverson et al., *supra* note 18.
26. S. Kothari & C. DiTommaso, *Prognosis After Severe Traumatic Brain Injury: A Practical, Evidence-Based Approach*, *in* BRAIN INJURY MEDICINE, *supra* note 1, at 248–78.
27. *Id.*

- Moderate disability: independent, able to live at home and use public transportation, works in a supported environment
- Good recovery: mild to no residual deficits; able to resume prior work and social activities; minor residual or cognitive difficulty

Predictors of poor outcome include coma at onset; post-traumatic amnesia; subarachnoid or subdural hematoma; evidence of diffuse axonal injury, especially with coma; and medical complications such as significant hypotension, prolonged elevated intracranial pressure, or impaired oxygen intake resulting in hypoxia or anoxia.[28]

After reviewing over a thousand reports published since 1983, Kothari and DiTommaso concluded the following prognostic correlations existed for patients who suffered severe TBI:

1. Initial CT scan findings that are predictive of a worse outcome include the presence of subarachnoid blood, epidural or subdural hematoma, or an increase in intracranial pressure due to a mass, such as brain swelling or blood causing a midline shift.
2. Evidence of damage deep within the brain as shown on MRI scan is associated with a worse outcome. If lesions are present on both sides of the brainstem, good recovery is not very likely.
3. Although the initial Glasgow Coma Scale (GCS) score is generally not prognostic, lower GCS scores are more frequently associated with worse outcomes.
4. The duration of coma, as measured by the ability to follow commands, is linked to outcome. The longer the coma, the worse the outcome. Comas lasting longer than four weeks are usually not associated with a good recovery.
5. Amnesia after head injury can involve an inability to remember following the injury (anterograde amnesia) or an inability to recall things from before the injury (retrograde amnesia). The longer the duration of anterograde amnesia, i.e., the inability to form new memories, the worse the outcome. If the amnesia lasts less than two months after injury, the individual will not have a severe disability. Amnesia lasting more than three months is not likely to result in a good recovery.
6. Age at the time of head injury is a significant prognostic factor with a worse outcome if the individual is older than sixty-five. Within this age group, injured individuals with lower GCS scores (e.g., less than 8) are unlikely to have a good recovery.
7. Individuals who sustain a penetrating injury, such as a bullet wound, have a worse prognosis with a GCS of 8 or less and CT scan findings of damage to both sides of the brain.

28. A.I. Maas, *Moderate and Severe Traumatic Brain Injury in Adults*, 7 LANCET NEUROLOGY 728–41 (2008).

The outcome is much improved in cases of moderate TBI. Risk factors associated with less desirable outcome for moderate TBI patients include lower GCS scores (less than 10), older age, and abnormal CT scans showing edema or blood.[29] Although many individuals who sustain a moderate TBI have what is considered by some to be a good recovery, they often suffer from persistent cognitive and behavioral difficulties.

As to be expected, prognosis in mild TBI is much better and most patients return back to work or school within the first month.[30] In most cases, the symptoms of mild TBI do not involve permanent cognitive, psychological, or psychosocial residual problems.[31] Lasting symptoms may include dizziness, headache, nausea, sensitivity to light and noise, fatigue, sleep disturbance, irritability, and cognitive difficulty. However, individuals with mild TBI may still have persistent symptoms for longer periods, some of which may relate to pre-injury psychological makeup, the development of post-traumatic depression, post-traumatic stress disorder, and possible secondary gain.[32] The prognosis may also be limited by underlying neurological problems, including prior stroke or dementia. Lifestyle factors, such as substance abuse, can also have an impact. Finally, an injured individual's coping abilities, personality, and family and social support affect recovery.

Work is being done to identify biomarkers, or biochemical substances found in the blood, that may provide both diagnostic and prognostic measures of TBI.[33] For example, S100B is a protein that binds calcium that is found highly concentrated within astrocytes. Within minutes after head injury, S100B levels peak within the blood and then decline several hours later. Similarly, neuron specific enolase (NSE) is a neuronal protein released into the blood at the time of brain injury. Other substances of interest include creatine kinase isoenzyme BB (CK-BB), myelin basic protein (MBP), glial fibrillary acidic protein (GFAP), fatty acid-binding proteins (FABP), cleaved tau protein, and neuroproteomics. Unfortunately, to date, none of these biomarkers has been shown to be specific or prognostic in cases of TBI.[34]

V. TREATMENT/MANAGEMENT

There is no specific treatment for TBI. Management of the consequences of head injury is another matter and depends upon the severity of the

29. Kochanek et al., *supra* note 21.
30. G.L. Iverson et al., *supra* note 18.
31. *Id.*
32. *Id.*
33. *Id.*
34. *Id.*

injury and how quickly following the injury the person begins treatment. Management can be divided into three segments—(1) treatment at the time of injury; (2) treatment during the immediate post-traumatic period; and (3) long-term care for chronic problems.

A. *Acute Treatment*

The approach to an acutely injured person depends upon the severity of trauma. With mild TBI, careful observation is all that is needed. With moderate to severe TBI, care should be provided in a hospital intensive care unit and should include monitoring of vital signs and respiratory status, as well as neurosurgical involvement for intracranial bleeding and intracranial pressure monitoring. Evaluation of other physical injuries should be carried out by a multidisciplinary team that includes trauma specialists and orthopedic surgeons. Proper medical management may also involve treatment of seizures. Because significant brain injury can lead to other organ problems, such as irregularity of heart beat (cardiac arrhythmia) and fluid on the lungs (neurogenic pulmonary edema), consultation with other medical specialists may be appropriate as well. Although many animal studies have looked at the possibility of a neuroprotective substance to reduce brain injury, none has been found to be effective in humans.[35]

B. *Immediate Post-Traumatic Period*

Management of problems within the first one to two weeks immediately following head injury again depends upon the severity of injury. For mild TBI, symptomatic treatment of headaches, dizziness, and neck pain may be needed. Treatment of moderate to severe TBI generally includes continued treatment for conditions such as seizures or cardiac arrhythmia. However, symptomatic treatment must also involve rehabilitation, including physical and occupational therapy, in an attempt to restore pre-injury function as soon as possible.

C. *Chronic Care*

Long-term management may not be needed with mild TBI or may be limited to neuropsychological support for memory and cognitive dysfunction. With moderate to severe TBI, the person typically is involved in a brain injury rehabilitation program, either as an outpatient or an inpatient, often at a specialized rehabilitation hospital or facility. There, a team of multidisciplinary professionals, including neurologists; physiatrists; physical, occupational, and speech therapists; psychologists; and neuropsychologists, is assembled. Medical management often focuses

35. Kothari & DiTommaso, *supra* note 26.

not only on physical problems such as continued seizures or extremity weaknesses, but also on cognitive issues, particularly behavioral difficulties and depression.

VI. PREVENTION

A number of steps can be taken to reduce the severity of injury from head trauma due to falls, motor vehicle accidents, firearms, and sports. Additional studies are ongoing, especially in the area of cumulative trauma, and are likely to lead to the implementation of new and more effective preventive measures.

Falls, which account for an estimated 35 percent of TBI, occur in two major age groups—children under the age of four and those over the age of seventy-five.[36] Methods intended to reduce injury in children include window guards, safety gates at the top of stairs, and softer playground surfaces made of rubber and wood chips rather than concrete and asphalt. The importance of parental education cannot be underestimated.[37] In the case of the elderly, exercise and physical therapy for balance training and strengthening, avoidance of medications that might impair judgment, installation of grab bars in bathrooms, and elimination of throw rugs are among the easiest to implement. Using canes or walkers, if necessary; wearing supportive shoes; and good lighting are also critical.[38]

Traumatic brain injuries caused by motor vehicle accidents have been addressed by governments and manufacturers. Legislative initiatives mandating the installation and use of seat belts, airbags, child safety seats, and roll bars are among a few of the actions taken in recent years.[39] To reduce driver distraction, the National Highway Traffic Safety Administration is now targeting cell phone usage, and texting while driving is illegal in most states.[40] Most states have also passed helmet laws at varying levels in an effort to reduce motorcycle accidents. The use of motorcycle helmets has been shown to reduce motorcycle fatalities, TBI-related fatalities, TBI incidence and severity, and length of hospitalization—with a concomitant drop in cost to society.[41]

36. M. Faul et al., Traumatic Brain Injury in the United States: Emergency Room Visits, Hospitalizations and Deaths 2002–2006 (Ctrs. for Disease Control & Prevention 2010).

37. L.M. Kneer & E.P Elovic, *Primary Prevention*, *in* Brain Injury Medicine, *supra* note 1, at 101–21.

38. *Id.*

39. *Id.*

40. National Highway Traffic Safety Administration, Overview of the National Highway Traffic Safety Administration's Driver Distraction Program (Rep. No. DOT HS 811-299) (2010).

41. *Id.*

Sports and recreational activities, which alone are responsible for between 1.6 million and 3.8 million TBIs a year, are another major contributor.[42] While no athletic activity is without risk, some sports, such as boxing, and recreational activities are more likely to expose participants to the risks of head injury. The increasing use of helmets by downhill skiers and cyclists is evidence that even non-contact sports create dangers for injury to the brain.

American football, for example, is currently the subject of numerous reports on the acute and chronic problems resulting from concussions. In an effort to curb head injuries, the National Football League (NFL) and other organizations at the college and high-school level have instituted regulations that severely penalize on-field activities believed to contribute to head injuries, such as spear tackling and helmet-first contact. Rules are being rewritten to protect specific players, and discussions are ongoing regarding the elimination of the kick-off, a time-honored football tradition, in an effort to reduce concussions.

Soccer also involves risk of TBI for participants.[43] According to a 2011 report, concussions occurred in 3.9 percent of male high school soccer players and 7.4 percent in female players.[44] Head trauma occurs with head-to-head, head-to-body, and goal post collisions during the game, as well as repetitive "headers" when players propel the ball with the head.[45] One improvement to soccer ball construction was to replace the older leather balls with synthetic urethane covered balls.[46] Another proposed approach is the use of protective head gear.[47] Kneer and Elovic suggest that prevention of TBI in soccer needs to include improved training and coaching, strict adherence to guidelines for return to play, careful post-injury medical evaluation, and enforcement of established rules.[48]

In hockey, another sport where head injury is common, most concussions occur during checking, where the player's own body or hockey stick is used to interfere with an opponent's play. One study of youth hockey found that 57 percent of the injuries were due to checking.[49] The American Academy of Pediatrics has recommended that checking not be

42. J.A. Langlois et al., *The Epidemiology and Impact of Traumatic Brain Injury: A Brief Overview*, 21 J. Head Trauma Rehabilitation 375–78 (2006).

43. Brian Cassella, *Heading a Soccer Ball Is Risky Even If Concussions Rare*, Chicago Trib., Apr. 29, 2015, 7:42 AM.

44. A.E. Lincoln et al., *Trends in Concussion Incidence in High School Sports: A Prospective 11-Year Study*, 39 Am. J. Sports Med. 958–63 (2011).

45. Kneer & Elovic, *supra* note 37.

46. *Id.*

47. J.S. Delaney et al., *The Effect of Protective Headgear on Head Injuries and Concussions in Adolescent Football (Soccer) Players*, 42 British J. of Sports Med. 110–15 (2008).

48. Kneer & Elovic, *supra* note 37.

49. S.R. Reid & J.D. Losek, *Factors Associated with Significant Injuries in Youth Ice Hockey Players*, 15 Pediatric Emergency Care 310–13 (1999).

allowed for children under sixteen.[50] At the professional level, the National Hockey League (NHL) has prohibited certain illegal hits such as hitting from behind or "blindsiding."

Boxing and other forms of fighting sports are commonly associated with TBI. In boxing, the effect of a knock-out, an automatic win for the boxer inflicting the knock-out blow, is to cause so much brain trauma that the opponent is rendered unconscious. In these sports, preventative approaches are difficult to implement. Futile efforts to improve the safety of boxing have included changing the weight of the gloves, banning blows to the head, and requiring helmets.[51] Minimally, more attention has been paid to the signs and symptoms of a concussion, and referees can prevent a fight from continuing if a head injury is suspected.[52] Sadly, neither the boxers nor their trainers are generally seen in the ring asking for a fight to be called. Another fighting sport, more recent in popularity, is the mixed martial art (MMA) in which opponents combine hitting (without cushioned gloves), wrestling, and kicking. Interestingly, the concussion rate during MMA matches (3.3 percent) is lower than in boxing matches (11.3 percent).[53]

Cycling is an example of a non-contact sport with a significant incidence of TBI. These injuries usually result from falls from bicycles, with younger children suffering brain injury most often.[54] The use of helmets by cyclists has reduced the risk of brain injury by 88 percent.[55] States that mandate the use of bicycle helmets have seen a decrease in TBI incidence.[56] Other effective preventive measures designed to eliminate bicycle-vehicle collisions include the wearing of conspicuous clothing by cyclists; bicycle-friendly road designs, including bicycle-only lanes; and safety training, especially for children.[57] Training to educate motor vehicle drivers to be aware of bicyclists on the highways is also likely to reduce these accidents.

50. Committee on Sports Medicine & Fitness, Am. Acad. of Pediatrics, *Safety In Youth Ice Hockey: The Effects of Body Checking*, 105 PEDIATRICS 657–58 (2000).

51. Kneer & Elovic, *supra* note 37.

52. *Id.*

53. K.M. Ngai et al., *Injury Trends in Sanctioned Mixed Martial Arts Competition: A 5-Year Review from 2002 to 2007*, 42 BRITISH J. OF SPORTS MED. 686–89 (2008).

54. S. Boufous et al., *Cycling Crashes in Children, Adolescents, and Adults—A Comparative Analysis.* 12 Traffic Injury Prevention 244–50 (2011).

55. Kneer & Elovic, *supra* note 37.

56. A. Macpherson & A. Spinks. *Bicycle Helmet Legislation for the Uptake of Helmet Use and Prevention of Head Injuries*, 2008 COCHRANE DATABASE SYS. REV. (3):CD005401.

57. Kneer & Elovic, *supra* note 37.

VII. LEGAL DISCUSSION

Litigation involving traumatic brain injuries has moved to the forefront in legal circles, in part because of the attention generated by the NFL litigation as well as highly publicized damage awards. Because brain injury cases are very complex, they require the skill of a competent attorney with expertise in the area. After all, the brain is the most complex structure in the body and can be injured in any number of ways. Severe brain injury cases will generally involve significant disabilities. Minor injury cases, on the other hand, have unique legal and medical issues due to the subjective nature of many of the complaints. In every case, however, causation is likely to be disputed. The cases discussed below present an overview of some of the litigation involving traumatic brain injuries that has arisen.

One court defined a TBI as "non-degenerative, structural brain damage resulting in residual deficits and disability that have been acquired by external physical injury."[58] This all-encompassing definition covers a large number of reported cases. A survey of court decisions involving some of the injuries examined above follows.

A. *Coup Versus Contrecoup Injury*

The defendant in *State v. Donnelson* was found guilty of manslaughter in the death of his girlfriend, who suffered a coup/contrecoup injury allegedly sustained during an unwitnessed domestic dispute.[59] Numerous medical witnesses testified on both sides of the case.[60] The dispute was over whether the victim died due to a brain injury sustained in a fall or series of falls or because of blows to the head.[61] The testifying physicians agreed that the cause of death was a subdural hemorrhage but they disagreed about how the injury was sustained.[62] Because of this general disagreement among the experts, the defendant contended there was reasonable doubt and, therefore, his conviction should be overturned.[63]

The pathologist testified that the cause of death was "massive bilateral . . . subdural hemorrhage and the complications thereof" and that an injury over the right ear was due to "blunt trauma without pattern."[64] Regarding the coup/contrecoup injury mechanism, he added that when blunt trauma occurs to the head when it is stationary, the injury occurs directly underneath the blow. On the other hand, in a contrecoup injury,

58. State v. Johnson, 218 P.3d 46 (Kan. 2009).
59. 402 N.W.2d 302 (Neb. 1987).
60. *Id.* at 307–08.
61. *Id.* at 309.
62. *Id.* at 307–08.
63. *Id.* at 304.
64. *Id.* at 307.

the injury occurs on the opposite side of the head, indicating that the head was in motion at the time of the trauma.[65] He further explained that where a contrecoup injury is present, there could also be an injury on the same side where the head was impacted, but the main injury would then be the opposite side of the brain.[66] This supported the state's theory that a significant blow to the head was the primary cause of death, rather than a fall.[67] The defendant's conviction was therefore upheld.[68]

In the underlying action to *Galanek v. Wismar*, which involved the plaintiff's suit against her attorney and his firm for spoliation of evidence, the plaintiff landed in the backseat of her car after it was struck by another vehicle when her seat collapsed upon impact.[69] According to the court, the "primary injury she suffered in the accident was a brain injury known as 'contrecoup,' which occurs when the brain bounces off the back and front of the skull as a result of the head being suddenly thrown backward and then rebounding forward."[70] She was found to be totally disabled.[71] In the instant case, the appellate court reversed the trial court's grant of the defendants' motion for nonsuit, noting that the plaintiff lost her products liability claim against the car's manufacturer because her attorneys did not safeguard a critical piece of evidence—her totaled car.[72]

In *Donnellan v. First Student, Inc.*, the plaintiff was hit in the back of the head by either a generator or a power tool that broke through a cargo cage of the van he was driving. The van had been pushed through an intersection and into a ditch by a school bus.[73] The plaintiff's physician testified that he suffered a coup/contrecoup injury.[74] In addition, the physician testified that the plaintiff also suffered from fourth nerve palsy, dystonia, myofascial pain, allodynia, occipital neuralgia, and depression as a result of his brain injury.[75] The injury manifested itself with such symptoms as hypersensitivity to pain, cognitive dysfunction, chronic pain, double vision, headaches, sleeping and mood problems, and a decreased ability to walk.[76] Nevertheless, the plaintiff was still able to work as a construction supervisor with frequent rest periods, and no wage loss claim was made at trial.[77] A verdict of $6 million was upheld

65. *Id.*
66. *Id.*
67. *Id.* at 310.
68. *Id.*
69. 68 Cal. App. 4th 1417, 1420 (1999).
70. *Id.* at 1422.
71. *Id.*
72. *Id.* at 1427
73. 891 N.E.2d 463, 469 (Ill. App. Ct. 2008).
74. *Id.* at 455.
75. *Id.*
76. *Id.*
77. *Id.*

on appeal, even though the plaintiff's economic damages were only $82,000.[78]

B. *Intracranial Hemorrhage*

An intracranial hemorrhage was the subject of a case that involved a worker who slipped on a recently mopped floor at a Coca Cola Bottling Co. plant.[79] Two co-employees observed the worker in a prone position but did not see him fall.[80] After getting up, he continued to work and returned to work the next day for "a short time."[81] The worker died from a cerebral hemorrhage several days after the purported fall.[82] The issue was whether the decedent's injury was caused by a fall or by some other condition, given that there was no evidence of external trauma to the decedent's head.[83] The plaintiff's medical expert testified that someone could hit his head hard enough to cause an intracranial hemorrhage without any external trauma.[84] The medical expert for the defense, however, noted that a person could sustain an intracranial hemorrhage as the result of an abnormality of a blood vessel in the brain that could rupture spontaneously and could not sustain trauma to the head sufficient to injure the blood vessel without having external evidence of a blow to the head.[85] The court ruled in favor of the employer and its insurance carrier, finding that the injury did not arise out of the decedent's employment absent evidence that he struck his head on the floor at work.[86]

C. *Subdural Hematoma*

In *Goetz v. Greater Georgia Life Insurance Co.*, a worker requested long-term disability benefits due to injuries sustained from falling approximately four times and striking his head.[87] The claimant suffered from a subdural hematoma and the defendant employer argued that the claimant's alcoholism contributed to his incoordination and falling.[88] The court, however, found the defendant's decision to deny long-term disability benefits based on the policy's preexisting condition clause was "arbitrary and capricious" because it rested on attenuated extensions of contributing factors to injuries that ERISA itself did not permit.[89]

78. *Id.* at 484.
79. 73 S.E.2d 86, 87 (Ga. Ct. App. 1952).
80. *Id.* at 87.
81. *Id.*
82. *Id.*
83. *Id.*
84. *Id.*
85. *Id.*
86. *Id.* at 87–88.
87. 649 F. Supp. 2d 802, 807 (E.D. Tenn. 2009).
88. *Id.* at 805.
89. *Id.* at 826.

Furthermore, the record supporting the claimant's chronic alcoholism was "vague, scant, and somewhat speculative."[90]

In *In re MaKenna S.*, the court found that a child suffered from abusive head trauma and her medical records indicated a combination of subdural hematomas and retinal hemorrhages.[91] The child was neurologically devastated and had a poor long-term neurological prognosis.[92] In addition to suffering from global stiffness, the child was likely to be blind and not expected to have receptive or expressive speech or significant cognitive or motor development.[93] The expert concluded that her injuries were highly suspicious for child abuse.[94] In response to a question about differential diagnosis, the expert noted that "the combination of findings [had] a few potential causes, the most common of which is abusive head trauma."[95] She added, however, that "severe accidental head trauma," such as that caused by a high-speed motor vehicle crash or a crush injury, neither of which was reported, might have caused the combination of injuries.[96] Although the medical record revealed an absence of external signs, bruises or contusions, or fractures, this did not exclude abusive head trauma as a diagnosis. The court found the child was injured by at least one episode of abusive head trauma and terminated parental rights.[97]

In *State v. Urbano-Uriostegui*, the court found that the evidence was sufficient to sustain the defendant's conviction for aggravated child abuse because a rational juror could have found that the defendant caused the injuries sustained by the child while in her care.[98] According to the testimony of the child's treating neurosurgeon, who was accepted by the court as an expert in traumatic brain injuries, acute subdural hematomas in children are caused by "nonaccidental trauma, falls from significant heights, and car crashes."[99] He further testified that when he performed surgery on the child's skull and brain, he saw no evidence of a chronic subdural hematoma because "there was no staining of the brain tissue or any of the blood products associated with chronic subdural hematomas.

90. *Id.* at 824.
91. 2011 WL 4447225, at *39 (Conn. Super. Ct. Aug. 31, 2011).
92. *Id.* at *10.
93. *Id.*
94. *Id.*
95. *Id.* at *9.
96. *Id.*
97. *Id.* at *40–41. See also *In re* Clark K., 799 A.2d 1099 (Conn. App. Ct. 2002), where parental rights were terminated when a child when was taken to the hospital and found to have suffered three skull fractures; he had a subdural hematoma and significant swelling on the area of the fractures.
98. State v. Urbano-Uriostegui, 2013 WL 1896931, at *13 (Tenn. Crim. App. May 6, 2013).
99. *Id.* at *4.

Instead, he found active bleeding, blood products associated with acute subdural hematomas, and a ruptured bridging vein."[100] According to a second physician, who was the medical director of the child maltreatment center at the Vanderbilt Children's Hospital, the child "had sustained severe life-threatening head trauma that was inflicted in nature."[101] She categorized the child's subdural hematoma as a shearing injury caused when the brain and the dura are forced apart, adding that the hematoma between the child's skull and scalp could have been caused by a child on his own or by "household accidents."[102] She stated that retinal hemorrhages are almost always caused by inflicted trauma, and the subdural hematoma, the hematoma, and the retinal hemorrhages taken together had "a very[,] very high correlation with inflicted trauma."[103] Following deliberations, the jury found the child's appellant caretaker guilty of aggravated child abuse.[104]

D. *Subarachnoid Hemorrhage*

The plaintiff in *Aetna Life Insurance Co. v. Hale* was taken to the hospital, where she was diagnosed with a subarachnoid hemorrhage, after slipping and falling in her bathroom.[105] The court noted that, "[a]ccording to the evidence, a subarachnoid hemorrhage is a discharge of blood in the arachnoid space, which is between the arachnoid and the next innermost membrane covering the brain."[106] She also had a subdural hematoma, "which is a localized collection of blood, formed at the point of impact, under the outermost membrane covering the brain, the dura."[107] When discovered, the plaintiff was unconscious and "moaning and groaning."[108] Upon examination, the doctor found elevated blood pressure, dilation of the left pupil, and a flaccid left arm.[109] She also had a bruise at the base of her skull, which was not actively bleeding.[110] The plaintiff was denied coverage for injuries under her accident insurance policy because she failed to show that her injuries were caused by accidental means, specifically, that her bathroom fall occurred prior to her ruptured aneurysm.[111]

In *Martinez v. Quarterman*, the defendant admitted to the police that he struck the decedent in the head with sufficient force to knock the

100. *Id.*
101. *Id.* at *6.
102. *Id.*
103. *Id.*
104. *Id.* at *9.
105. 147 S.E.2d 126, 128 (Va. 1966).
106. *Id.* at 128.
107. *Id.*
108. *Id.*
109. *Id.*
110. *Id.*
111. *Id.* at 129–30.

child off the bed and onto the bedroom floor.[112] The decedent was transported to the pediatric intensive care unit at a nearby hospital, where he was diagnosed with TBI and found to be "totally unresponsive to stimulation."[113] He remained on a ventilator and died within twenty-four hours.[114] The autopsy report concluded the following:

> [the deceased] experienced extensive bruising to the occipital (back area) of his scalp, experienced substantial bleeding in both the subdural space between the skull and brain, as well as subarachnoid bleeding between the brain and the thin membrane covering the brain; while no skull fracture was observed, swelling of the brain caused the sagittal suture of the top of his head to separate, [and] his brain injuries suggested an extremely violent impact between the back of his head and a broad, flat surface. . . .[115]

The report found that the extensive nature of the injuries to the decedent's brain could not have been caused by an accidental fall from a bed, but instead, strongly suggested the child's head was violently swung against a hard, unyielding surface.[116] The defendant was found guilty of murder.[117]

In *Philip Morris USA, Inc. v. Mease*, the claimant was discovered lying on the floor at work, "having vomited multiple times and with blood draining from his left ear."[118] He was admitted to the hospital where he was diagnosed as having sustained a "left subdural hematoma with minimal midline shift, as well as a left temporal parietal subarachnoid hemorrhage and left temporal contusions."[119] The parties entered into an agreement to pay benefits, identifying the claimant's injury as "head trauma."[120] The employer denied the doctor's recommendation for increased physical therapy on the grounds that the treatment sought "was not causally related to the compensable injury, was not reasonable or necessary, and was excessive and costly."[121] The court upheld the deputy commissioner's decision, which found that in "the absence of any conflicting advice from another physician, either by record review or personal evaluation, the medical evidence indicates the connection between the need for six days of physical therapy and [claimant's] compensable injuries."[122]

112. 2006 WL 2042958, at *1 (W.D. Tex. July 11, 2006).
113. *Id.*
114. *Id.*
115. *Id.* at *1.
116. *Id.*
117. *Id.* at *1, *15.
118. 745 S.E.2d 155, 159 (Va. Ct. App. 2013).
119. *Id.*
120. *Id.* at 157.
121. *Id.*
122. *Id.* at 157, 160.

E. *Cerebral Edema*

The plaintiff in *Smith v. University of Cincinnati* proved his claim of medical negligence by a preponderance of the evidence.[123] In *Smith*, the decedent had fallen in the kitchen where he had lost consciousness and was bleeding from his skull.[124] He ultimately was transported to University Hospital, where he was found to have a GCS score of 14 or 15, which is "consistent with a mild, rather than moderate or severe, head injury."[125] Despite the fact that "he was unsteady to the point where he needed help walking," the decedent was discharged home, where he died eighteen hours later.[126] The plaintiff's expert testified that the decedent's autopsy revealed "progressive cerebral edema, and that in the final stages of this process, the brain was caused to herniate, or expand outside its normal position, which is nearly always fatal."[127] According to the expert, "the cerebral edema was a progressive process caused both by the meningitis and the trauma to the brain."[128] The expert further opined that the swelling "would have resulted in a headache, confusion, lethargy, somnolence, difficulty ambulating, seizure activity, and, in the final stages, losing consciousness."[129] He concluded that the swelling process ultimately resulted in a "herniation of the brain, whereby the brain protrudes outside the space in which it is normally contained, . . . and that once herniation occurs, respiratory and circulatory functions are diminished, causing cardiac arrest within minutes."[130]

In *Harding v. Noble Taxi Corp*., the plaintiff hit her head against the divider of a taxicab in a motor vehicle accident and was taken by ambulance to the hospital.[131] The doctor failed to conduct a neurological exam and discharged the patient.[132] Subsequently, the plaintiff's condition failed to improve. She continued to vomit blood, discharge clear fluid from her nose, sleep most of the time, and had difficulty walking.[133] Upon referral to a neurologist, she was diagnosed with brain damage.[134] The plaintiff's expert testified that fluid began to accumulate in her brain after the accident, causing it to swell.[135] The fluid was permitted to remain, and the

123. Smith v. Univ. of Cincinnati, Case No. 2008-11389 (Ohio Cl. Ct. Feb. 21, 2012), https://cases.justia.com/ohio/court-of-claims/2008-11389.pdf?ts=1344957822.
124. *Id.* ¶ 3.
125. *Id.* ¶ 7.
126. *Id.* ¶ 14.
127. *Id.* ¶ 20.
128. *Id.* ¶ 23.
129. *Id.*
130. *Id.*
131. 182 A.D.2d 365 (N.Y. App. Div. 1992).
132. *Id.* at 366–67.
133. *Id.* at 367.
134. *Id.*
135. *Id.* at 367–68.

swelling led to the death of the nerve cells in the brain.[136] The expert opined that had the plaintiff received "timely and proper treatment," 80 percent of her injuries could have been prevented.[137]

F. *Traumatic Brain Injury*

1. Minor

From a legal point of view, minor traumatic brain injuries are very troublesome because of the lack of an objective test to prove the injury. Therefore, the cases have had varying results.

In *Bennett v. Richmond*, the plaintiff sued the employer of a truck driver for injuries he sustained to his neck and back in an automobile collision.[138] As a result of the accident, the plaintiff suffered from "chronic headaches, a loss of cognitive efficiency, difficulties in information processing, and some adverse personality changes."[139] The court admitted the neuropsychogical testimony of a psychologist, who concluded that Richmond had suffered a mild to moderate TBI.[140] He explained that in a whiplash case, "the head is forced either forward or backward or somehow side to side. . . . The brain, which is the consistency of Jello, moves at a different pace than the hard shell of the skull . . . and the brain tissues are dragged across those bony protuberances."[141] He added "that kind of motion to the head caused damage to the connections between the cells of the brain through the axons. They were sufficiently messed up to provide him with these processing problems [that manifested within two years]."[142] Richmond died in February 2011.

The only issue before the Indiana Supreme Court was whether "the trial court abused its discretion when it permitted a psychologist to testify on behalf of a plaintiff in a personal injury case as to the cause of a brain injury."[143] The Indiana Court of Appeals held that

> a psychologist who is not a medical doctor but is otherwise qualified under Rule 702 to offer expert testimony as to the existence and evaluation of a brain injury is not qualified to offer his or her opinion as its cause without demonstrating some medical expertise in determining the etiology of brain injuries.[144]

136. *Id.* at 368.
137. *Id.*
138. 960 N.E.2d 782, 784 (Ind. 2012).
139. *Id.* at 788.
140. *Id.* at 786.
141. *Id.*
142. *Id.* at 788.
143. *Id.* at 784.
144. *Id.* at 788.

Reversing the appellate decision, the supreme court held that the appellate court "imposed more stringent requirements on [the expert witness] than are required under Rule 702," adding that Rule 702 requires that witnesses have "knowledge, skill, experience, training, or education in order to be qualified as an expert, and in fact, only one of these characteristics is necessary."[145]

In Myers v. Private Investigations & Counter Intelligence, Inc., when a physician evaluated the claimant on behalf of the employer, the worker complained of headaches.[146] After taking a history, performing a physical examination, and reviewing the treatment records and diagnostic tests, the doctor concluded that the claimant sustained a minor traumatic head injury with post-concussive headache syndrome, a whiplash injury, and possible occipital neuralgia.[147] He also thought that the claimant suffered from depression, but deferred that diagnosis to a specialist.[148] He found no evidence that the accident produced a permanent impairment, noting that the mild concussion would have resolved and permitted a return to work in four to six weeks.[149] He did not think that further treatment was necessary.[150]

In *Boynton v. Allstate*, the plaintiff, then nineteen years old, was in a motor vehicle accident in which his head struck the steering wheel.[151] The plaintiff's doctors made a diagnosis of a minor TBI. After the accident, the plaintiff experienced headaches, dizziness, vision problems, and loss of concentration and memory. He also became irritable, violent, and abusive and experienced suicidal and homicidal thoughts. Over the years, he continued to have difficulties related to the injury and sought treatment in rehabilitation facilities on multiple occasions. The court reviewed the decision to pay rehabilitation costs and concluded that reasonable jurors could find that the accommodations and services provided by the rehabilitation center were necessary for the "care, recovery, or rehabilitation" and that such treatment is related to the injury the plaintiff suffered in the motor vehicle accident.[152]

2. Moderate

At issue in *Berner v. Carnival Corp.* was whether a biomedical engineer was qualified to conclude "that the energy on [the plaintiff's] head upon striking

145. *Id.* at 789.
146. 2008 WL 4552949, at *1 (Ky. Ct. App. Oct. 10, 2008), *aff'd*, 2009 WL 1830774 (Ky. June 25, 2009).
147. *Id.* at *2.
148. *Id.* at *1.
149. *Id.* at *2.
150. *Id.*
151. 2008 WL 4414235 (Mich. Ct. App. Sept. 30, 2008).
152. *Id.*

the floor was sufficient to have caused his mild to moderate traumatic brain injury."[153] The plaintiff had been attacked by two passengers on a cruise ship, where he was punched, lost consciousness, and fell to the floor.[154] He suffered considerable injuries to his eye, face, and skull, and alleged that he suffered a TBI.[155] The court noted that the expert was not called upon to "give an opinion whether [the plaintiff] suffered a brain injury. Rather, she may give an opinion about the energy involved and whether the energy is sufficient to have caused an injury of the type [the plaintiff] alleges to have suffered."[156]

In *Pennsylvania Uninsured Employers Guaranty Fund. v. Workers' Compensation Appeal Board*, the claimant fell approximately twelve feet while working and struck his head.[157] The claimant's physician testified that during the worker's first visit he complained of "significant pressure headaches behind his left eye, blurred vision, fatigue, short-term memory problems, and episodes of losing consciousness."[158] The physician's final diagnosis was that the worker sustained a moderate TBI in a work-related accident, and that he was unable to return to work as a laborer as of the date of his last examination.[159] The court granted the claimant's petition and directed that the claimant receive disability compensation.[160]

3. Severe Traumatic Brain Injuries

The decedent in *Durham v. State*, suffered a severe TBI as a result of abuse.[161] In *Durham*, the chief of pediatric neurosurgery described the various aspects of TBI and causation as follows: "'Diffuse axonal injury' is trauma where the gray matter and white matter planes in the brain are caused by external forces to slide 'out of kilter.'"[162] The physician explained that the "degree of injury depends on the magnitude of force used. The types of external forces that can cause a diffuse axonal injury in a child include car accidents, falls off a bike, riding a roller coaster, being forcibly struck, and being forcefully banged against a wall."[163] Upon reviewing the decedent's autopsy report, the doctor found a "subscalpular hemorrhage, subdural and subarachnoid hemorrhaging, swollen brain,

153. 632 F. Supp. 2d 1208, 1209–10 (S.D. Fla. 2009).
154. *Id.* at 1209.
155. *Id.*
156. *Id.* at 1213.
157. 85 A.3d 1109, 1111 (Pa. Commw. Ct. 2014).
158. *Id.*
159. *Id.* at 1112.
160. *Id.* at 1117.
161. 2005 WL 2787550 (Tex. App. Oct. 26, 2005).
162. *Id.* at *7.
163. *Id.*

and hemorrhage in the ligaments or soft tissue at the base of the skull on the left side."[164] The decedent also had blood in the subdural space in the thoracic spinal canal.[165] In the medical expert's opinion, the child suffered an impact to the skull and had a skull fracture because she had "widenings along the occipital suture lines of the posterior skull plates."[166] He concluded that the decedent was brain-dead as a result of TBI, specifically a blow to the back left portion of the head.[167]

In *State v. Hudson*, the defendant's conviction was upheld in a case where the victim suffered from a severe traumatic brain injury as a result of repeated beatings with a wooden softball bat to the head.[168] When the victim was found, he was unconscious and in a coma.[169] The victim had sustained a "large laceration to the left frontal part of his scalp, exposing the bone; a laceration just to the right top of his scalp; and a large entry wound to the back of his head."[170] He also sustained multiple fractures to the back section of his skull.[171] Upon admission to the hospital, his condition allowed for "a 30 percent chance of death and the patient was at risk of developing pneumonia, infection, abnormal bone formation, hydrocephalus (a buildup of pressure in the brain), and seizures."[172] Moreover, as a result of his extensive brain injury, he was also at risk for impaired swallowing.[173] The victim died four months after the incident.[174]

The plaintiff in *Saint v. United States* suffered a severe TBI as a result of a collision between a tractor-trailer owned by the U.S. Postal Service and a Lincoln Towncar driven by a twenty-year-old.[175] When the plaintiff was first transported to Nassau County Medical Center, he was comatose, which, according to an expert witness for the defense, "is a cardinal manifestation of a severe closed head injury."[176] He then suffered from a generalized seizure in the emergency room.[177] He was diagnosed with a "severe closed head brain injury, including a subdural hematoma, and unconsciousness for a lengthy period and seizures. He also sustained the sequella of

164. *Id.* at *8.
165. *Id.*
166. *Id.*
167. *Id.*
168. 680 N.W.2d 603, 606 (Neb. 2004).
169. *Id.*
170. *Id.*
171. *Id.*
172. *Id.*
173. *Id.* at 607.
174. *Id.* at 608.
175. 483 F. Supp. 2d 267, 269 (E.D.N.Y. 2007).
176. *Id.* at 288.
177. *Id.*

brain damage, including vision problems and post-traumatic stress disorders."[178] He remained severely limited in both physical and cognitive abilities, and he suffered from "depression, anxiety, and feelings of hostility."[179] The discharge summary revealed that he was diagnosed with a TBI with "cerebral edema, loss of consciousness, and seizures."[180]

Although the plaintiff's mother testified that her son would require constant supervision, his treating physician stated that "he does not require 24 hour supervision, but needs supervision on an intermittent basis," adding that "there is no credible evidence that he will need professional paid supervision in the future."[181] The court also noted its concern that the plaintiff had not appeared, if only for a short time, during the trial, noting that his "appearance would have assisted the Court in resolving some of the disputed damages issues, such as (1) his present condition; (2) his future ability to be employed; and (3) his need for constant or even intermittent supervision."[182]

G. *Penetrating Head Injury*

In *Jiles v. Barnhart*, an administrative law judge found that a plaintiff suffered from "severe" impairment following "gunshot wound injuries, headaches, seizure disorder, cognitive disorder, and dysthymia."[183] The plaintiff reportedly suffered a penetrating head injury when he was shot in the eye during a party.[184] Subsequently, he suffered from "seizures, headaches, memory problems, depth perception, and irritability."[185] The plaintiff's expert testified that he was capable of "understanding and carrying out instructions, but *would likely have difficulty remembering them, due to short term memory problems*."[186] The expert also explained that although interactions with co-workers would "probably be adequate, *the plaintiff does not seem capable of coping with the demands of the normal work environment due to cognitive limitations*."[187] The court reversed the decision of the Social Security Administration and remanded the case with instruction that the plaintiff be awarded the benefits claimed.[188]

178. *Id.* at 292.
179. *Id.* at 282.
180. *Id.*
181. *Id.* at 286.
182. *Id.* at 292.
183. 2006 WL 4402937, at *2 (N.D. Ala. Sept. 11, 2006).
184. *Id.* at *3.
185. *Id.*
186. *Id.*
187. *Id.*
188. *Id.* at *6.

H. *Encephalopathy*

Nagell v. United States involved a former army officer who had been found guilty of robbing a bank and sought a new trial on the basis of "newly discovered crucial evidence" concerning several head injuries, the most serious of which was received in a plane crash that occurred while he was on active duty.[189] As the Fifth Circuit noted during the first appeal, "from the outset, the defendant in many ways manifested the most unusual behavior."[190] Four expert witnesses testified during the intial trial; acknowledging that Nagell displayed some "psychological abnormalities," they testified that he was capable of distinguishing right from wrong.[191] The defendant denied that he had ever received psychiatric treatment.[192]

One expert witness, who had treated Nagell after one of the military incidents ten years before the bank robbery, testified during the appeal that "he had apparently suffered a fracture through the base of his brain, which injured the underside of the brain, and not only damaged the brain but some of the cranial nerves coming off the brain."[193] Based on his experience with Nagell and other military personnel with TBIs, he wrote a monograph on patients who deny their illness after a brain injury,[194] a behavior that Nagell had exhibited during his trial. He opined that Nagell "was disassociated with reality at the time of the incident [and that the robbery] was directly related to his mental illness, [and that he saw] the act was an alternative to suicide.[195] The Fifth Circuit overturned the trial court's decision.

Former NFL player Gene Atkins filed suit in *Atkins v. Bell*, seeking more generous disability benefits under the defendant's retirement plan.[196] An expert on brain trauma sustained by athletes, including chronic traumatic encephalopathy suffered by former NFL players,[197] opined that Atkins suffered from severe post-concussion syndrome and was "probably beyond that into early traumatic encephalopathy."[198] He also concluded that Atkins was unable to work indefinitely due to a "demented mental status."[199] An independent neurologist who evaluated Atkins found that he suffered from impairments of cognitive dysfunction,

189. Nagell v. United States, 354 F.2d 441 (5th Cir. 1966), *appeal after remand*, 392 F.2d 934 (5th Cir. 1968).
190. *Nagell*, 354 F.2d at 444.
191. *Nagell*, 392 F.2d at 936.
192. *Nagell*, 354 F.2d at 445.
193. *Id.* at 447.
194. *Id.*
195. *Id.*
196. 694 F.3d 557, 559 (5th Cir. 2012).
197. Atkins v. Bell, 694 F.3d 557, 562–63 (5th Cir. 2012).
198. *Id.*
199. *Id.* at 563.

depression, chronic and post-concussion headaches.[200] The neurologist concluded that the chronic and post-concussion headaches resulted from football, but that the other two impairments were only partially the result of football.[201] The Fifth Circuit affirmed the lower court's decision after viewing the doctors' opinions in the aggregate and finding no conclusive result regarding whether the player was or was not totally disabled, and if he was, whether his disability arose from football-related activities.[202]

VIII. CONCLUSION

A traumatic brain injury can have devastating consequences. After all, the brain is our body's control center. Not only is this a personal problem, but it is a societal one as well because TBI is a pervasive and widespread concern. This insult to the brain can result from all aspects of life—daily activities, motor vehicle accidents, falls, recreational events, work injuries, violence, and combat.

The mechanism of injury is usually the result of external trauma causing injury to the substance or functioning of the brain. Although "head injury" is often used interchangeably with "traumatic brain injury," one problem may occur without the other and TBI can be classified as closed head trauma, an open head injury, or a diffuse injury. How much a person recovers after a TBI depends primarily upon the nature and severity of the injury as well as his underlying medical health. The spectrum of recovery is quite broad and ranges from the absence of neurological residuals to death.

There is no specific treatment for these brain injuries. Management of the consequences of a head injury is another matter and depends upon the severity and time frame in which the person is being seen. As such, the management can be divided into three segments—(1) acute at the time of injury, (2) immediate post-traumatic period, and (3) chronic long-term care.

Traumatic brain injuries have moved to the forefront in the legal arena, in part due to the litigation involving the National Football League. Our research failed to uncover any statistics on the number of claims currently being advanced on behalf of those alleging traumatic brain injuries. Common sense, however, suggests that their number is on the rise because of the increasing public awareness on the topic.

200. *Id.*
201. *Id.*
202. *Id.* at 569, 571.

As this article demonstrates, TBI claims are very complex and require the skill of a competent attorney with experience in the area. This article has attempted to provide counsel with a primer on the medical and legal implications of this type of injury.