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

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By: Jack Hubbard, Samuel D. Hodge, Jr. and Stavroula Kotrotsios

SUMMARY

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. More and more suits are being filed asserting claims for such problems and it is the signature injury of the Afghanistan and Iraq wars. Much publicity has also been generated about the side effects of brain injuries as the result of lawsuits against the NFL, NHL and professional wrestling associations. These injuries, however, are also 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 common place.

The attached article will offer a unique perspective on the topic because it will be an objective analysis of the medical and legal issues involved with traumatic brain injuries. It is written by a board certified neurologist who also has a Ph.D. in anatomy and a lawyer who also teaches anatomy. These authors have been chosen by the American Bar Association to write a definitive work on the topic that will be published in the fall and the book will contain contributions from some of the leading medical experts in the field. This article is based upon one of the chapters in the text.
BIOGRAPHICAL SKETCHES

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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 AD)

INTRODUCTION

A traumatic brain injury (TBI) is the most feared consequence of head trauma.\(^1\) 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 in jeopardy our very being and that for which we strive.

Not only is this a personal problem, but it is a societal one as well for TBI is a pervasive and widespread medical concern. For example, a 1991 survey in the United States estimated that 1.5 million people had sustained a traumatic brain injury.\(^1\) Approximately 3.6-5.3 million people in the United States have some type of residual problems as a consequence of TBI.\(^2\) Further, the mortality rate within thirty days after a significant TBI has been reported at 21%.\(^3\) The breakdown of civilian deaths from head injury is motor vehicle accidents 34%, firearms 39%, falls 10% and other 17%.\(^4\) In contrast, combat mortality due to TBI during the Iraq war climbed to 30-50%.\(^5\) The estimated costs 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.\(^2\)
Traumatic brain injury, as discussed throughout this article, can result from all aspects of life including daily activities (especially motor vehicle accidents and falls), recreational events, work injuries, and violence/combat.

**DEFINITIONS**

As a starting point, it is useful to define certain terms regarding TBI. *Traumatic brain injury* is the result of *external trauma* causing injury to the substance and/or functioning of the brain. A brain injury may occur from non-traumatic causes such as stroke, cardiac arrest, or infection. However, these medical problems are beyond the scope of this article. While head injury is often used interchangeably with traumatic brain injury, one problem may occur without the other. For example, trauma to the more superficial structures of the head may not necessarily cause brain injury. Conversely as in the instance of development of a subdural hematoma in an elderly person who falls or makes a sudden stop without direct head trauma, a brain injury may occur without direct head trauma. It may also occur with head trauma but without disruption of the meninges (coverings of the brain), a condition termed *closed (non-penetrating, blunt) head trauma*. When an external force, such as a projectile breaches the meninges, this situation results in an *open (penetrating) head injury*. Another distinction regarding TBI involves the concepts of focal and diffuse injury. A *focal injury* is one that is confined to a part of the brain such as that which occurs with a bullet entering the brain. A *diffuse injury* is widespread throughout the brain such as that which results with a concussion. Often a focal injury can also produce more diffuse injury as well.
DEGREES OF TBI

Traumatic brain injury occurs in different degrees of severity, an important point when considering symptoms, functioning, and long term prognosis. The extent of the TBI depends upon the forces involved, the nature of injury, and the extent of the brain damage. A useful measure is to divide degrees of TBI into mild, moderate, and severe classifications, using the Glasgow Coma Scale (GCS). This scale provides a simple, rapid way to immediately evaluate the clinical state of the head injured patient presenting to the emergency room. 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 (table 5-1). Using this scale, mild TBI has a score of 13-15, moderate TBI is 9-12, and severe TBI is a GCS score of 8 or less. A concussion is classified as a mild.

Table 5-1
GLASGOW COMA SCALE

<table>
<thead>
<tr>
<th>Response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td></td>
</tr>
<tr>
<td>Opens eyes spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>Opens eyes in response to speech</td>
<td>3</td>
</tr>
<tr>
<td>Opens eyes in response to noxious stimulation</td>
<td>2</td>
</tr>
<tr>
<td>Does not open eyes spontaneously or to stimulation</td>
<td>1</td>
</tr>
<tr>
<td><strong>Motor Response</strong></td>
<td></td>
</tr>
<tr>
<td>Follows commands correctly</td>
<td>6</td>
</tr>
<tr>
<td>Appropriate motor response to painful stimulation</td>
<td>5</td>
</tr>
<tr>
<td>Non-purposeful response to painful stimulation</td>
<td>4</td>
</tr>
<tr>
<td>Flexes upper/extends lower extremities to painful stimulation</td>
<td>3</td>
</tr>
<tr>
<td>Extends all extremities in response to painful stimulation</td>
<td>2</td>
</tr>
<tr>
<td>No motor response to painful stimulation</td>
<td>1</td>
</tr>
</tbody>
</table>

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

In addition, multiple episodes of mild head trauma can have a cumulative effect which may result in serious neurological consequences. For example, a young individual such as a student athlete who experiences a concussion but has a second one before recovering is prone to development of a second-impact syndrome with brain swelling leading to catastrophic neurologic impairment and even death. In cases of multiple head injury such as a boxer or professional football player, a form of dementia termed chronic traumatic encephalopathy (CTE) develops in later years leading to progressive cognitive decline.

MECHANISM OF TBI

When the brain has been injured, the organ gets hit with two pathological processes - a kind of a “double-whammy.” The first problem, termed primary injury, is a direct, immediate consequence of the trauma causing disruption of brain function. The second problem, secondary injury, is delayed and is a consequence of a series of resulting biochemical and cellular changes which 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. The damage can occur at the point of impact, termed a coup injury or, the damage can
actually occur on the opposite side of the brain, termed *contrecoup injury*. 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 with the forehead striking the windshield (*coup injury*) and the back of the brain striking 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. During an impact, the skull is deformed which may lead to a skull fracture with the underlying brain injured. While several factors are in play, the key components are the speed of head acceleration and the duration of head impact. Studies have shown that head accelerations during activities of daily living (e.g. hopping off steps, coughing, sneezing, running, jumping) can deliver up to 12 G’s of force without associated brain injury. By comparison, a boxer’s punch with a velocity of over 25 mph can deliver over 71 G’s of force.

**Intracranial Hemorrhage** - The resulting trauma to the brain can produce damage to either the brain substance itself or to the blood vessels going to and from the brain – the arteries and veins. With regard to blood vessel damage, with a focal injury such as the head being 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, termed an *intracerebral hemorrhage* or *hematoma*. An
intracerebral hematoma is differentiated from a contusion in that an intracerebral hematoma is 2 cm in size or larger and does not reach the brain surface.\textsuperscript{6}

Unfortunately, especially in persons on an anticoagulant such as warfarin or an antiplatelet medication such as aspirin, the hematoma may expand, termed delayed traumatic intracerebral hemorrhage.\textsuperscript{6} Such a situation is a neurological emergency for 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 down through the foramen magnum at the base of the skull. When this occurs, the brain is literally pushed down through the opening, a condition termed \textit{brain herniation}. A life-threatening condition, 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 such as with an intraparenchymal bleed or extracerebral hemorrhage discussed below, the person may initially be awake and neurologically intact, termed the \textit{lucid interval}. However, with the expanding hemorrhage, the individual will quickly lapse into a coma.

Bleeding from trauma may also occur outside of the brain but within the skull, termed \textit{extracerebral hemorrhage}, producing equally disastrous results. These types of bleeds are named according to their relationship to one of the three meningeal layers (see Chapter Two) - epidural hematoma, subdural hematoma and subarachnoid bleed. In an \textit{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, tearing the nearby middle meningeal artery. Because this action results in an
arterial bleed, the lucid interval may be very short as 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 flow blood from the brain surface to the superior sagittal sinus. As indicated by its name, subdural bleeding occurs just underneath the dura mater, between the dura mater and arachnoid layer. Because the tearing of a vein causes more of an oozing, being a lower pressure blood vessel compared to arteries, subdural hematomas can be present for hours to days before they cause clinical problems such as headache, drowsiness, or focal neurological symptoms or findings. In the elderly, because of brain atrophy (shrinkage) symptoms may not show up for weeks because of the amount of space available for the blood to accumulate. If they cause significant mass effect as seen on a CT or MRI scan, both epidural and acute subdural hematomas are neurosurgical emergencies to prevent death or permanent neurological impairment. In some cases, such an *acute or subacute* subdural hematoma can turn into a progressively increasing *chronic* subdural hematoma as nearby fluid is slowly absorbed into the mass of blood, causing further expansion and neurological impairment. In the elderly being evaluated for dementia, imaging of the brain is necessary to rule out the presence of a chronic subdural hematoma causing the cognitive decline.

*A subarachnoid hemorrhage* is 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 due to an aneurysm. Head trauma is the
most common cause of subarachnoid bleeding, followed by a ruptured intracranial aneurysm. A complication of subarachnoid bleeding is blockage of cerebrospinal fluid flow; cerebrospinal fluid is produced within the ventricles of the brain and flows outward from the fourth ventricle to the surface of the brain. If this outflow is blocked by the accumulated subarachnoid blood, the condition termed hydrocephalus results with a damming up of spinal fluid within the ventricles, identified on CT or MRI by enlarged ventricles. This condition requires a neurosurgical procedure with 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 head injury. Because of the focal damage from a heavy object or impact with a motor vehicle accident, the person may have localized neurological impairment such as visual loss, paralysis on one side, or speech difficulty. The focal injury may also cause a more wide-spread diffuse injury.

**Microscopic Injury** – Much of the damage in TBI, especially 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 occurs with a concussion, the result may be a *diffuse (traumatic) axonal injury* (DAI) caused by microscopic injury to the axons extending from the neurons.⁶

A *neuron* is the functional cell of the nervous system, consisting of many dendrites, a single cell body and a single axon. Think of a neuron as a microscopic computer chip. The *dendrites* receive the input from other neurons, converts that input in the form of electrical currents which is then processed by the *cell body*. The
cell body 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,
potassium) across the cell membrane of the neuron. An axon is the single process that
extends from the neuronal cell body that 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 (calcium, sodium,
potassium) fluctuations across the axonal membrane. The axon terminates as a
specialized structure termed the synapse. Within the synaptic terminal are synaptic
vesicles which 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 a whitish-appearing insulating material termed
myelin which 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. (See Chapter Two). Consequently, areas
damaged with DAI are those that contain a large amount of white matter such as the
corpus callosum (interconnects the two cerebral hemispheres) and within the core of the brain. A special form of MRI termed diffusion tensor imaging (DTI) is able to identify white matter tracts that are traumatically damaged, revealing injury that is 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, termed glial cells. These glial cells come in different forms and have different functions. Oligodendroglia, for example, lay down the myelin by wrapping around axons much as you would tape around a pipe. Other glial cells are termed astrocytes and microglia. As an aside, it is abnormal divisions of a glial cell that results in a primary brain cancer such as an astrocytoma; neurons within the brain and spinal cord cannot divide and therefore do not become cancerous.

With head trauma, linear and rotational acceleration forces cause the brain tissue to deform, resulting in stretching and compression of the neurons and glial cells.$^{10}$ With disruption of the neuronal membrane, 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.$^{10}$ With axonal stretching greater than 6 mm, the axon can shear off, separating from the neuronal cell body,$^{11}$ resulting in neuronal death or at least 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. An excitatory neurotransmitter, glutamate, which is normally contained within the synaptic vesicles, is released in large amounts paradoxically causing neuronal damage, termed excitotoxicity.\textsuperscript{12} Other lethal events to the neurons include release of inflammatory factors, and free radicals. The blood brain barrier, the important defense mechanism which isolates the brain substance from potential toxins in the blood is broken down. Even with mild TBI such as a concussion, important damaging ionic changes, cerebral blood flow alterations, metabolic depression and axonal injury occur.\textsuperscript{12} In addition to the shearing injury occurring with diffuse axonal injury (DIA) as a primary injury, secondary factors contribute to further axonal damage by a delayed process, termed secondary axotomy.\textsuperscript{12} Once a neuron dies, it is not replaced because nerve cells do not regenerate themselves.

As a result of neuronal death and axonal destruction in large areas of injury, swelling of the brain occurs, termed cerebral edema. Because the brain is enclosed within the skull made of solid bone, 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 can also diminish arterial blood flow to the brain resulting in further brain injury.

In addition to these insults to brain neuronal functioning, certain underlying trauma associated medical conditions may contribute to the secondary injury to the brain. For example, any significant drop in blood pressure (hypotension) such as from blood loss with body trauma diminishes blood flow to the brain and exacerbates the neuronal lethal series of events. Likewise, lack or significantly diminished oxygen (anoxia, hypoxia) such as from a chest wound worsens the brain damage.

Therefore, these secondary injuries, which are most complex and of a cascading nature, may continue to evolve after the initial trauma. As a consequence, they magnify and extend the damage from the primary injury and account for a person’s clinical worsening hours to days after their head trauma.

**PROGNOSIS AFTER A TBI**

How much a person recovers after traumatic brain injury depends primarily upon the nature and severity of the injury as well as the underlying medical health of the injured individual. The spectrum of recovery is quite broad and ranges from no neurological residuals to death. Statistically, long lasting disability occurs in 100% of severe TBI, 66% of moderate TBI and 10% in mild TBI. Mild trauma for the most part does not cause any permanent disability such that the person is able to return to work/school and resume their usual activities. Any residual difficulty might be limited to memory or other cognitive impairments. Approximately 90% of those who experience moderate TBI are able to live independently, but may need assistance with
their work, managing finances, or certain physical problems such as needing a cane or walker. With severe TBI, the consequence may be death or require long-term living care.\textsuperscript{13}

With regard to \textit{severe TBI}, 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.\textsuperscript{14}

In their detailed analysis of the literature, Kothari and Di Tommaso\textsuperscript{13} define outcome following severe brain injury using the following Glasgow Outcome Scale (GOS):

- \textbf{Dead}
- \textbf{Vegetative state} – the body is alive but the brain is unresponsive.
- \textbf{Severe disability} – conscious but totally dependent, not able to live alone more than 24 hours; requires assistance for daily activities.
- \textbf{Moderate disability} - is independent, able to live at home and use public transportation, works in a supported environment.
- \textbf{Good recovery} – mild to no residual deficits so that can resume prior work and social activities; may have minor residual or cognitive difficulty.

After reviewing over a thousand reports published since 1983, these researchers determined the following prognostic correlations after severe TBI:

1. \textit{CT scan} – initial CT scan findings after head injury that are predictive of a worse outcome following head injury include the presence of subarachnoid blood,
epidural or subdural hematoma, or increase in intracranial pressure due to a mass such as brain swelling or blood causing a midline shift.

2. MRI scan – evidence of damage deep within the brain is associated with a worse outcome. If lesions are present on both sides of the brainstem, good recovery is not very likely.

3. Glasgow Coma Scale (GCS) – the initial GCS score is generally not prognostic, although lower GCS scores are usually associated with worse outcome.

4. Length of coma – duration of coma, as measured by the ability to follow commands, is linked to outcome – the longer the coma, the worse the outcome. Coma lasting longer than 4 weeks is usually not associated with a good recovery.

5. Post-traumatic amnesia – amnesia after head injury can occur after the injury (anterograde amnesia) or before the accident (retrograde amnesia). The longer the duration of anterograde amnesia, being unable to form new memories, the worse the outcome. If the amnesia lasts less than 2 months after injury, the individual will not have a severe disability. Amnesia lasting more than 3 months is not likely to result in a good recovery.

6. Age – the age at the time of head injury is a significant prognostic factor with worsening outcome if the individual is older than 65 years of age such that the chances of good recovery is unlikely. Within this older age group, injured individuals with lower GCS scores (e.g. GCS less than 8) are more likely not to have a good recovery.
7. Penetrating injury – in those individuals with a penetrating injury such as a bullet have worse prognosis with a GCS of 8 or less and CT scan findings of damage to both sides of the brain.

In addition to these parameters, work is being done to identify biomarkers, or biochemical substances found in the blood that may provide both diagnostic and prognostic measures of TBI. For example, S100B is a protein that binds calcium which is found highly concentrated within astrocytes. Within minutes after head injury, S100B levels peak within the blood, then decline within several hours later. While this substance is highly sensitive for TBI, it is not specific since elevated blood levels can occur with body trauma but without head trauma. 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 isoenyme BB (CK-BB), myelin basic protein (MBP), glial fibrillary acidic protein (GFAP), fatty acid-binding proteins (FABP), cleaved tau protein, and neuroproteomics. However, none of these biomarkers have been shown to be specific and/or prognostic for cases of TBI.

In cases of moderate TBI, the outcome is much improved. Greater than 90% of individuals with moderate TBI will reach a good recovery or, at worse, have a moderate disability. Risk factors associated with less desirable outcome include lower GCS scores (less than 10), older age and abnormal CT scan showing edema or blood. While many with a moderate TBI have what is considered a good recovery, they may have persistent cognitive and behavioral difficulties.
Prognosis in mild TBI, as expected is much better than with the severe or moderate forms of TBI, with most injured returning back to work or school within the first month.\textsuperscript{10} In most individuals, the symptoms of mild TBI are self-limited without permanent cognitive, psychological or psychosocial residual problems.\textsuperscript{10} These symptoms usually include dizziness, headache, nausea, sensitivity to light and noise, fatigue, sleep disturbance, irritability, and cognitive difficulty. Return back to work can be quite variable with 22-84\% working after the first week, 25-99\% within the first month, 48-100\% three to six months after injury and 46-100\% 1 year after injury. Those with milder injuries (GCS of 15 without loss of consciousness) tend to have a better return to work rate than those with more significant injury (GCS of 13-14 with loss of consciousness).\textsuperscript{10} However, others may have persistent symptoms for longer with multiple contributing factors, some of which may relate to the individual’s pre-injury psychological makeup, the development of posttraumatic depression, posttraumatic stress disorder, and possible secondary gain.\textsuperscript{10}

In addition to age, the pre-injury health of the individual impacts upon the extent of recovery such that underlying neurological problems such as prior stroke or dementia limits the prognosis. Pre-existing life-style factors such as substance abuse as well as other factors such as coping abilities, personality, family and social support also impact upon the outcome.

**TREATMENT/MANAGEMENT**

The short answer is that there is no specific treatment for traumatic brain injury. Management of the consequences of 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 – acute at the time of injury, immediate post-traumatic period, and chronic long-term care.

**Acute** – The approach to an acutely injured person depends upon the severity of trauma. With mild TBI, simple careful observation is all that is needed. With moderate to severe TBI, care is in a hospital intensive care unit (ICU) setting with good medical support for stabilization including vital signs, respiratory status, and neurosurgical involvement if needed for intracranial bleeding and intracranial pressure monitoring. Evaluation of other injuries such as the spine, fractures to the limbs, pelvis, or chest as well as abdominal bleeding should be carried out as a multidisciplinary approach including trauma surgeons and orthopedic surgeons in addition to the neurosurgical support. Management may also include medical treatment of seizures if present as well as generalized body cooling if indicated. While many animal studies have looked at the possibility of a neuroprotective substance to decrease brain injury, none have been found to be effective in humans. In addition to brain function, 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).

**Immediate** – Management of problems within the first one to two weeks immediately following head injury again depend upon the severity of injury. For mild TBI, symptomatic treatment of headaches, dizziness and neck pain may be needed. For
moderate to severe TBI, continued treatment for seizures if indicated, instituting rehabilitation, and symptomatic treatment of headaches, dizziness.

**Chronic** – Long term management may not be needed with mild TBI, or limited to neuropsychological support for memory and cognitive dysfunction. With moderate to severe TBI, the person typically is involved in a brain injury program either on an outpatient basis or inpatient at a rehabilitation hospital. In these cases, a team of professionals is assembled including neurologists, physiatrists, rehabilitation therapists (physical, occupational, speech), and psychologists and/or neuropsychologists for a multi-disciplinary approach. Medical management may be required for continued seizures, behavioral difficulty, or depression.

**PREVENTION**

While many cases of traumatic brain injury cannot be prevented, a number of steps can be taken to mitigate the severity of injury from head trauma due to falls, motor vehicle accidents, firearms, and sports.

With regard to *falls*, this type of injury accounts for an estimated 35% of TBI, and occurs in two major age groups – children four years old or younger and the elderly aged seventy-five years and older.15 Efforts to reduce injury in the young include window guards, safety gates at the top of stairs, softer playground surfaces such as rubber and wood chips, and parental education.16 With regard to the elderly, initiatives such as physical therapy for balance training and strengthening, avoiding over-prescribing of medications that might impair judgment, installation of grab bars
in bathrooms, eliminating throw rugs to prevent tripping, encouraging use of canes or walkers where appropriate, wearing supportive shoes, and providing good lighting.\textsuperscript{16} 

Motor vehicle accidents, a leading cause of TBI, have been addressed by state and federal governments to make the roads safer and improve the incidence and severity of head injury. These legislative initiatives include use of seat belts, airbags, child safety seats, blood alcohol content and roll bars.\textsuperscript{16} Cell phone usage is being targeted by the National Highway Traffic Safety Administration (NHTSA) to reduce driver distraction.\textsuperscript{17} Motorcycle accidents and fatalities have significantly increased over the past 10-20 years due not only to younger riders but also the aging baby boomers seeking to regain their youth.\textsuperscript{16} In states where a helmet is required has led to a significant drop in motorcycle fatalities, TBI-related fatalities, TBI incidence and severity, length of hospitalization with a concomitant drop in cost to society.\textsuperscript{16} Improved roadway design has led to accident reduction such as replacing traditional intersections with a roundabout.\textsuperscript{16} 

A major contributor to traumatic brain injury is from sports and recreational activities, resulting in 1.6-3.8 million TBI per year.\textsuperscript{18} While no athletic activity – contact or non-contact – is without risk, some sports are more prone to head injury, especially multiple times. This topic will be covered in greater detail in Chapters 16-18. 

American football, for example, is in the forefront of this topic these days as the press reports the acute and chronic problems resulting from concussions. As early as 1974, reports surfaced that 70\% of football-related deaths were due to TBI, with 75\% of those while tackling.\textsuperscript{19} In an effort to curb head injury during a football game, the
National Football League (NFL) and similar organizations at the college and high school level have instituted rules and regulations regarding the game. These rules regulate and limit such activities as spear tackling, head-to-head contact, and face mask tackling as well as rules protecting specific players such as the kicker and receivers.

Work is ongoing to improve helmet design for use at both the professional and academic levels to withstand impacts from head hits. While this approach seems reasonable, a study out of Mayo Clinic raises some interesting questions. To evaluate the question of long-term outcome after playing football, the authors of this study looked at the incidence of neurodegenerative diseases such as dementia, Parkinson disease or amyotrophic lateral sclerosis (ALS – Lou Gehrig disease) in individuals who had played high school football, comparing this group to males in non-athletic high school activities (band, choir, glee club). More to the point, they chose those who had played from 1946 to 1956 wearing flimsy leather helmets with only a minimal amount of protection. Surprisingly, no differences were found in the incidence of these neurodegenerative disorders comparing the football and non-football groups. The authors discuss the fact that players today are larger and quicker than then and that helmets do not prevent concussions, perhaps providing a false sense of protection.

Soccer, known outside of the United States as football, is also a risk factor for TBI. In a 2011 report in the United States, concussions occurred in 3.9% of male high school soccer players and 7.4% in female players. Head trauma occurs with head-to-head, head-to-body, and goal post collisions during the game as well as repetitive “headers” by propelling the ball with the head. An improvement to soccer ball
construction to aid in their resilience was to replace the older leather balls with synthetic urethane covered balls.\textsuperscript{16} Another proposed approach is through the use of protective head gear.\textsuperscript{22} In their discussion on this topic, Kneer et al. suggest that prevention of TBI in soccer needs to include improved training and coaching, sticking to the guidelines for return to play, careful post-injury medical evaluation, and enforcement of established rules.\textsuperscript{16}

In \textit{hockey}, another sport where head injury is common, most concussions occur with checking, that is, using the player’s own body or hockey stick to interfere with an opponent’s play. For example, a study of youth hockey found that 57\% of the injuries were due to checking.\textsuperscript{23} In fact, the American Academy of Pediatrics has recommended that checking not be allowed in children younger than sixteen years of age.\textsuperscript{24} At the professional level, the National Hockey League (NHL) has prohibited certain illegal hits such as hitting from behind or “blind siding,” resulting in a 75\% decrease in concussions.\textsuperscript{16}

\textit{Boxing} and other forms of fighting sports are commonly associated with traumatic brain injury and, in fact, TBI is the goal of the “sport.” After all, the effect of a knock out is to cause so much brain trauma that the opponent is rendered unconscious. As such, preventive approaches are difficult and, in fact, seem to be a conceptual oxymoron. Futile efforts to improve the safety of boxing have included changing the weight of the gloves, not allowing blows to the head, and requiring helmets.\textsuperscript{16} Other approaches to reduce the long term effect of blows to the head include more attention paid to the signs and symptoms of a concussion and preventing
a boxer from fighting if those findings continue.\textsuperscript{16} A more recent fighting style, the
\textit{mixed martial art} (MMA) is becoming as popular as boxing. In this sport, opponents
combine hitting (without cushioned gloves), wrestling and kicking during the fight.
Interestingly, the concussion rate during MMA matches are lower at 3.3\% than with
boxing matches at 11.3\%.\textsuperscript{25}

\textit{Cycling} is an example of a non-contact sport where the TBI incidence is
significant. These injuries result from falls from bicycles with younger children
suffering brain injury most often.\textsuperscript{26} The use of helmets has significantly improved the
injury statistics, reducing the risk of brain injury by 88\%.\textsuperscript{16} Those states that have
legislation mandating bicycle helmets have seen a decrease in TBI incidence.\textsuperscript{27} Other
effective preventive measures include wearing conspicuous clothing, improved road
design that is bicycle friendly, bicycle lanes, and safety training, especially for
children.\textsuperscript{16}

\textbf{LEGAL DISCUSSION}

Traumatic brain injuries have moved to the forefront in legal circles with
hundreds of articles being devoted to the topic in legal publications. This is due in part
to the attention generated by the litigation involving the National Football League and
the fact that it has become the signature injury in military conflicts. Most states have
also passed legislation to protect young athletes from the consequences of these brain
injuries. For instance, a Google search of “traumatic brain injury lawyers”
demonstrates the lucrative nature of these claims by the number of personal injury
lawyers who promote their talents in handling these problems. Verdicts and
settlements in the millions of dollars are advertised and many of the sites maintained by attorneys provide medical information on traumatic brain injuries. Some even offer free consultations of brain injury claims.

Research fails to uncover any statistics on the number of lawsuits or 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 and advertising by attorneys soliciting these types of cases.

Any attorney can present or defend a traumatic brain injury case but these claims are very complex and require the skill of a competent attorney with experience in the area. After all, the brain is the most complex structure in the body and can be injured in any number of ways. Surprisingly, a severe brain injury may be easier to handle than one involving a minor injury since the later have unique legal/medical issues due to the subjective nature of many of the complaints. This section of the chapter will present an overview of some of the ways that litigation involving a traumatic brain injury has arisen.

A traumatic brain injury was defined by one court as “non-degenerative, structural brain damage resulting in residual deficits and disability that have been acquired by external physical injury.” This is an all-encompassing definition that covers a large number of reported cases. Instead of talking about the subject generally, this discussion will present a few court decisions of the various topics that appear in this chapter.
A. Coup v. Contrecoup Injury

In *State v. Weiss*, the Wisconsin Court of Appeals defined a coup and contrecoup injury as: “when the head hits a fixed object the brain hits the skull on that side (a coup injury) and then reverberates inside the skull and hits the other side (contrecoup injury).”

As for trial cases involving this problem, the defendant in *State v. Donnelson*, was found guilty of killing Carolyn Witthoff who suffered a coup/countercoup injury and died from that problem. Dr. Portrfield performed the autopsy and testified for the government that despite the bruises on the right side of decedent’s head, the injury on the left side was a of the brain was a countrecoup injury. He offered that such results are often caused by falls. Regarding the coup/contrecoup injury mechanism, the doctor explaining that when blunt trauma occurs to the head when it is stationary, the injury caused will be directly underneath the blow, whereas in a contrecoup injury, the injury occurs on the opposite side of the head, indicating that the head was in motion at the time trauma. He further explained that where a counterecoup 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.

In *Galanek v. Wismar*, the plaintiff landed in the backseat of her car after it was struck by another vehicle because the seat in which she had been sitting collapsed upon impact. She suffered a contrecoup injury which the court noted may occur 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. Galanek was determined to be completely disabled due to her brain injury.
In Donnellan v. First Student, Inc., the plaintiff was hit in the back of the head by either the generator or a power tool that broke through a cargo cage.\(^8\) The van which the plaintiff was in was then pushed through the intersection and down into a ditch. The plaintiff’s physician testified that Donnellan suffered a coup contrecoup injury, meaning an injury to the brain at the site of impact, the back of plaintiff’s brain, and the opposite side, the front of his brain. In addition, the physician diagnosed him with a fourth nerve palsy, dystonia, myofascial pain, allodynia, occipital neuralgia, and depression. The result of these ailments were hypersensitivity to pain, cognitive dysfunctions, chronic pain, double vision, headaches, sleeping and mood problems and decreased ability to walk. The physician opined that plaintiff’s symptoms will all naturally worsen as plaintiff ages and his body deteriorates, and he concluded that all of these conditions resulted from the traumatic brain injury suffered in the car accident.\(^9\)

B. Intracranial Hemorrhage

An intracranial hemorrhage was the subject of the problem in Butler v. Hartford Acc. & Indem. Co..\(^10\) This case involved a worker who was mopping the floor at a Coca Cola Bottling Company’s plant. Two co-employees with him looked away for one moment and when they looked backed, the worker was in a prone position.\(^11\) He died several days after the fall and it was alleged “that the deceased struck his head in the fall which resulted in an intracranial hemorrhage which caused his death.”\(^12\) The plaintiff’s medical expert testified that someone could hit the head hard enough to cause an intracranial hemorrhage without any external trauma. The medical expert for
the defense, however, noted that an individual can sustain an intracranial hemorrhage as the result of an abnormality of a blood vessel in the brain which vessel could rupture spontaneously and that a person cannot sustain trauma to the head sufficient to cause such an injury to the blood vessel without leaving some outside evidence of a blow to the head.\textsuperscript{13} The court resolved the conflict by ruling against the decedent. No one saw him fall and there was no direct evidence that he struck his head on the floor.\textsuperscript{14}

C. Subdural Hematoma

In \textit{Goetz v. Greater Ga. Life Ins. Co.}, a worker requested long term disability benefits due to injuries sustained from falling approximately four times, striking his head.\textsuperscript{15} The claimant suffered from a subdural hematoma and the defendant employer argued that the claimant’s alcoholism contributing to his incoordination and falling. The court, however, found the defendant’s decision to deny long term disability benefits based on the policy’s pre-existing condition clause was arbitrary and capricious as it rested on attenuated extensions of contributing factors to injuries that ERISA itself did not permit. Furthermore, the record supporting the claimant’s chronic alcoholism was vague, scant, and somewhat speculative.\textsuperscript{16}

\textit{In re Makenna S.}, the court found that a child suffered from abusive head trauma even though the medical records indicated a combination of subdural hematomas and retinal hemorrhages. The child was neurologically devastated and had a poor long-term neurological prognosis. She suffered from global stiffness, was likely to be blind, was not expected to have receptive or expressive speech or significant cognitive or motor development. The expert concluded that Makenna’s injuries were highly
suspicious for child abuse and opined that the combination of Makenna’s injuries might be caused by accidental head trauma in instances that include a high speed motor vehicle crash or a crush injury to the head. The reports revealed an absence of external signs, bruises or contusions, or fractures, however, this did not exclude abusive head trauma as a diagnosis. The court found the child was diagnostic for at least one episode of abusive head trauma, and terminating parental rights of the mother and father thereby adjudicating MaKenna as neglected. 17

In *State v. Urbano-Uriostegui*, the court found that the evidence was sufficient to sustain 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. 18 According to the testimony of Dr. Kyle Weaver, 19 acute subdural hematomas in children are caused by non-accidental trauma, falls from significant heights, and car crashes. Dr. Weaver further testified that he performed surgery on A.B.’s skull and brain where 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. Instead, he found active bleeding, blood products associated with acute subdural hematomas, and a ruptured bridging vein. According to Dr. Piercey’s assessment, A.B. "had sustained severe life-threatening head trauma that was inflicted in nature." 20 Dr. Piercey categorized A.B.’s subdural hematoma as a shearing injury caused when the brain and the dura are forced apart. She stated that the hematoma between A.B.’s skull and scalp could have been caused by "a significant impact" that could not be generated by a child on their own or through "household accidents." 21 Dr. Piercey 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."

Following deliberations, the jury found the appellant caretaker of A.B. guilty of aggravated child abuse.22

D. Subarchnoid Hemorrhage

The plaintiff in Aetna Life Ins. Co. v. Hale, was taken to the hospital after slipping and falling, where she was diagnosed with a subarachnoid hemorrhage. According 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. He 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. When discovered, the plaintiff was unconscious, "moaning and groaning". Upon examination, the doctor found elevated blood pressure, dilation of the left pupil and her left arm was flaccid. Additionally the plaintiff had a bruise at the base of her skull, which was not actively bleeding. 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, she failed to show that her bathroom fall occurred prior to her ruptured aneurysm.

In Martinez v. Quarterman, the defendant admitted to the police that he struck the decedent in the head with sufficient force to knock the child off the bed and on to the bedroom floor.23 As a result, he was found guilty of murder. The facts show that the decedent was transported to pediatric intensive care unit at University Hospital,
where he was diagnosed with traumatic brain injury, and found to be "totally unresponsive to stimulation." He remained on a ventilator, and died within 24 hours. The autopsy report concluded that the deceased experienced (1) a 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, (2) while no skull fracture was observed, swelling of the brain caused the sagittal suture of the top of his head to separate, and (3) the decedent's brain injuries suggested an extremely violent impact between the back of his head and a broad, flat surface. The report found that the extensive nature of the injuries to decedent's brain could not have been caused by an accidental fall from a bed but, instead, strongly suggested Anthony's head was violently swung against a hard, unyielding surface.

In *Philip Morris USA, Inc. v. Mease*, an accident report indicated that the claimant was discovered lying on the floor having vomited multiple times and with blood draining from his left ear. 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." The parties entered into an agreement to pay benefits, identifying the claimant's injury as "head trauma." Upon the doctor's recommendation for increased physical therapy, the employer rejected the request and defended 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. The court upheld the deputy commissioners decision which found that "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.”

E. Cerebral Edema

Plaintiff proved his claim of medical negligence by a preponderance of the evidence in *Smith v. Univ. of Cincinnati*. In this case, the decedent had fallen in the kitchen where he had lost consciousness and was bleeding from his skull. The plaintiff’s expert testified that the decedent’s autopsy revealed a fractured skull and contamination of the material lining of the brain, which resulted in a meningitis infection. According to the expert, the cerebral edema was a progressive process caused both by the meningitis and the trauma to the brain. The expert further explained that the swelling would have resulted in a headache, confusion, lethargy, somnolence, difficulty ambulating, seizure activity, and, in the final stages, going comatose. The expert 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.

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. The doctor failed to conduct a neurological exam, and discharged the patient. 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. She then consulted her family physician who referred her to a neurologist who diagnosed her
with brain damage. The plaintiff’s expert testified that fluid began to accumulate in the plaintiff’s brain after the accident, causing it to swell. The fluid was permitted to remain, and the swelling led to the death of the nerve cells in the brain. He added that had the plaintiff received timely and proper treatment, 80% of her injuries could have been prevented.32

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 Richmond sustained to his neck and back in an automobile collision.33 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. The court admitted the neuropsychological testimony of a psychologist who concluded that Richmond had suffered a mild to moderate traumatic brain injury. He explained how the process occurs in a whiplash case, “when 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”.34 He explained that the plaintiff suffered from a diffuse axonal injury, “…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]."  

In Myers v. Private Investigations & Counter Intelligence, Inc., a physician evaluated the claimant on behalf of the employer, at which time the worker complained of headaches. 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. He also thought that the claimant suffered from depression but deferred that diagnosis to a specialist. 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. He did not think that further treatment was necessary.

In Boynton v. Allstate, the plaintiff, then 19 years old, was in a motor vehicle accident in which his head struck the steering wheel. Plaintiff’s doctors made a diagnosis of a minor traumatic brain injury. After the accident, the plaintiff experienced headaches, dizziness, vision problems, and loss of concentration and memory. Plaintiff also became irritable, violent, 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 reviewing the decision to pay rehabilitation costs and concluded that, reasonable jurors could find that the accommodations and services provided by the rehabilitation
center are measures necessary for the "care, recovery, or rehabilitation", and that such
treatment is related to the injury plaintiff caused by the motor vehicle accident.39

2. Moderate

The following are a few cases involving moderate traumatic brain injuries. In
Berner v. Carnival Corp., the plaintiff was attacked by two passengers on a cruise ship,
where he was first punched, lost consciousness and fell to the floor.40 Berner suffered
considerable injuries to his eye, face, and skull, and alleged that he suffered a traumatic
brain injury. The plaintiff's expert witness testified that the Williams may testify that
the "energy on Berner's head upon striking the floor was sufficient to have caused a
mild to moderate traumatic brain injury".41

fell approximately twelve feet while working and struck his head.42 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 that he also experienced episodes of losing consciousness. The
physician's final diagnosis was that the worker sustained a moderate traumatic brain
injury in a work-related accident and that he was unable to return to work as a laborer
as of the date of the last examination. The court granted claimant’s petition and
directed that claimant receive his disability compensation.43

3. Severe Traumatic Brain Injuries
The decedent in *Durham v. State*, suffered a severe traumatic brain injury as a result of abuse. In that case, the chief of pediatric neurosurgery at a hospital, described the various aspects of traumatic brain injury 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." 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. Upon reviewing decedent’s autopsy report, he found a subscalpular hemorrhage, subdural and subarachnoid hemorrhaging, swollen brain, and hemorrhage in the ligaments or soft tissue at the base of the skull on the left side. The decedent also had blood in the subdural space in the thoracic spinal canal. In the medical expert’s opinion, the child suffered an impact to the skull and had a skull fracture because she had widening along the occipital suture lines of the posterior skull plates. He concluded that the decedent was brain dead as a result of traumatic brain injury, specifically a blow to the back left portion of the head.

In *State v. Hudson*, the defendant’s conviction was upheld where the victim suffered from a severe traumatic brain injury as a result of repeated beatings with a wooden softball bat to the head. When the victim was found, he was unconscious and in a coma. 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. Rodriguez also sustained multiple fractures to the back section of his skull. Upon admission to the hospital, his condition allowed for only 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. Moreover, as a result of the victim’s extensive brain injury, the patient was also at risk for impaired swallowing.\textsuperscript{48}

The plaintiff in \textit{Saint v. United States}, suffered a severe traumatic brain injury as a result of a motor vehicle accident.\textsuperscript{49} When he was first transported to Nassau County Medical Center, he was comatose, which is a cardinal manifestation of a severe closed head injury. The patient then suffered from a generalized seizure in the emergency room. 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 brain damage including vision problems and post-traumatic stress disorders. He remained severely limited in both physical and cognitive abilities, and he suffered from depression, anxiety, and feelings of hostility. The discharge summary revealed that he was diagnosed with a traumatic brain injury with cerebral edema, loss of consciousness and seizures.\textsuperscript{50}

G. Penetrating Head Injury

In \textit{Jiles v. Barnhart}, the ALJ found that the plaintiff suffered from the "severe" impairment of status post-gunshot wound injuries, headaches, seizure disorder, cognitive disorder, and dysthymia.\textsuperscript{51} The plaintiff reportedly suffered a penetrating head injury when he was shot in the eye while attending a party. Subsequently, he suffered from seizures, headaches, memory problems, depth perception, and irritability. The plaintiff’s expert testified that the plaintiff was capable of
understanding, and carrying out instructions, but would likely have difficulty remembering them, due to short term memory problems. The expert also explained that although interactions with coworkers would probably be adequate, the plaintiff does not seem capable of coping with the demands of the normal work environment due to cognitive limitations. The Social Security Administration reversed and remanded the case to the Commissioner with instruction that the plaintiff be awarded the benefits claimed.\textsuperscript{52}

\textbf{H. Chronic Traumatic Encephalitis}

Chronic Traumatic Encephalitis or CTE is starting to become more recognized in the legal community and cases are finding their way into the courts. The following is a sample.

\textit{Nagell v. United States} involved an army officer who sustained several head injuries while on active military duty, the most severe being a head injury received in a plane crash.\textsuperscript{53} The record is replete with expert testimony regarding the claimant’s mental condition which experts characterized as CTE. \textsuperscript{54} These experts explained that CTE caused by trauma, persisting for a long period after the initial injury, is manifested chiefly by behavioral disturbances in certain areas, namely the area of social and emotional behavior, rather than the ability to use language, to calculate and carry on the ordinary thinking processes of everyday life.\textsuperscript{55} One doctor testified that plaintiff was suffering "traumatic encephalopathy," a mental disease caused by an injury or trauma, incurred in the airplane accident, to the subcortical, or interior
aspect of the brain. The symptoms are: paranoia, suicidal preoccupations, confabulations, tendency toward projection, impaired judgment, and lack of contact with reality. The disease was manifested in plaintiff by emotional instability, passive-aggressive behavior and paranoid trends. As a result, the court concluded that the officer was entitled to payment of military disability pension from the time of his resignation.

Former NFL player, Gene Atkins, filed suit in *Atkins v. Bell* seeking more generous disability benefits under the defendant’s retirement plan. Dr. Robert Cantu, an expert on brain trauma sustained by athletics, including chronic traumatic encephalopathy suffered by former NFL players, opined that Atkins suffered from severe post-concussion syndrome and was "probably beyond that into early traumatic encephalopathy". Dr. Cantu also concluded that Atkins was unable to work indefinitely due to a "demented mental status." Dr. Gordon, an independent neurologist who evaluated Atkins, found that the plaintiff suffered from impairments of: (1) cognitive dysfunction; (2) depression; and (3) chronic and post-concussion headaches. Gordon concluded that the chronic and post-concussion headaches resulted from football, but that the other two impairments were only partially the result of football. The court affirmed the lower court’s decision, viewing the doctors’ opinions in the aggregate, there was no conclusive result regarding whether the player was or was not totally disabled, and if he was, whether his disability arose from football activities.

**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 for TBI is a pervasive and widespread medical concern. This insult to the brain can result from all aspects of life including 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 and/or functioning of the brain. While head injury is often used interchangeably with traumatic brain injury, one problem may occur without the other and it 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 the underlying medical health of the injured individual. The spectrum of recovery is quite broad and ranges from no 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 – acute at the time of injury, immediate post-traumatic period, and chronic long-term care.

Traumatic brain injuries have moved to the forefront in the legal arena. This is partially due to the litigation involving the National Football League and the fact that it has become the signature injury in military conflicts. Research fails 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 and advertising by attorneys soliciting these types of cases.

Any attorney can present or defend a traumatic brain injury case but these 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.

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1 This article is derived from materials that the authors have written for a book on traumatic brain injuries that will be published by the American Bar Association in 2015.


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9 *Id.*


12 *Id.*

13 *Id.*

14 *Id.*

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17 See also: In re Clark K., supra, 70 Conn.App. 665 where there was a termination of parental rights when was taken to the hospital and found to have suffered three fractures to the skull. The child had a subdural hematoma and significant swelling on the area of the fractures.


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