

Strafford

Presenting a live 90-minute webinar with interactive Q&A

Traumatic Brain Injury Claims: Navigating Litigation Complexities

Assessing TBI Claims, Negotiating Settlements, and
Leveraging Expert Witnesses to Strengthen Your Case

WEDNESDAY, NOVEMBER 14, 2018

1pm Eastern | 12pm Central | 11am Mountain | 10am Pacific

Today's faculty features:

Thomas Crosley, Founder, **Crosley Law**, San Antonio, Texas

Dr. Matthew J. DeGaetano, Program Director and Founder, **Personal Injury Institute**, Dallas

John Jerry Glas, Chair, Civil Litigation Department, **Deutsch Kerrigan**, New Orleans

The audio portion of the conference may be accessed via the telephone or by using your computer's speakers. Please refer to the instructions emailed to registrants for additional information. If you have any questions, please contact **Customer Service at 1-800-926-7926 ext. 1.**

LITIGATING TRAUMATIC BRAIN INJURY CASES: AN OVERVIEW

THOMAS A. CROSELY
Crosley Law Firm, P.C.

Strafford Publications, Inc.

Traumatic Brain Injury Claims Webinar

November 14, 2018

I. Introduction

“Traumatic brain injury” (TBI) has long been an important public health concern. In the last few years, however, there has been ever-increasing public awareness of the devastating effects of even “mild” traumatic brain injury. TBI has been called the “signature injury” of the wars in Afghanistan and Iraq. Many of our U.S. troops who have experienced concussive force blast injuries have developed symptoms attributable to TBI. In many of these cases, no outwardly visible signs of head or brain injury were detected. As a result, the Federal Government has poured a significant amount of funding into research concerning traumatic brain injury, and in particular mild traumatic brain injury.

Because the problems that result from TBI, such as those of thinking and memory, are often not visible, and because limited awareness about TBI among the general public, *mild* traumatic brain injuries have been referred to as the “silent epidemic.” See the Centers for Disease Control (CDC) report on Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations, and Deaths (Langlois, Rutland–Brown and Thomas 2006, T.3). The CDC report underscores that, among the general public, there has long been a lack of awareness about the potential significance of even mild brain injuries. That is changing now, however, due to the increased attention given to the prevention and management of sports concussions, as well as the focus on concussive force blast injuries sustained by U.S. troops during the wars in Iraq and Afghanistan.

On August 29, 2013, the National Football League announced that it reached a \$765 million settlement over concussion-related brain injuries among its retired players, agreeing to compensate victims, pay for medical exams and underwrite research. More than 4,500 former athletes – some suffering from dementia, depression or Alzheimer’s Disease that they blamed on blows to the head – had sued the League, accusing it of concealing the dangers of concussions and failing to adequately protect the players.

On September 23, 2013, the U.S. Department of Defense issued a special report on traumatic brain injury, calling TBI “one of the invisible wounds of war,” and urging the expansion of TBI benefits for veterans, and continued funding of research.

The increase in public awareness of TBI will likely lead to an increase in legal claims alleging traumatic brain injury. Lawyers, clients, and family members of a head injury victim are increasingly likely to recognize behavioral changes as possibly being related to a mild traumatic brain injury. The

result is that there will be an increase in lawsuits where TBI issues will be litigated. Accordingly, it is important for any lawyer regularly handling auto or trucking cases to be familiar with the basics of traumatic brain injury.

It is estimated that there are 1.7 million hospital visits per year related to traumatic brain injury. Over 80 percent of those patients are treated and released from the emergency department within 24 hours. The vast majority of those are commonly referred to as mild TBI (mTBI), or concussion injuries.

Leading causes of TBI include falls, motor vehicle accidents, getting struck by an object, assaults, and in the active duty military population, blast injuries.

II. Defining Traumatic Brain Injury

Traumatic brain injury occurs when an external mechanical force causes brain dysfunction. TBI usually results from a violent blow or jolt to the head or body. TBI is usually classified as mild, moderate, or severe, depending upon the injury characteristics.

A. Glasgow Coma Scale

In the acute trauma phase, the Glasgow Coma Scale, or GCS, is frequently used. GCS is a neurological scale that is used to assess a person’s level of consciousness after head injury. Unfortunately, lawyers tend to either not understand or intentionally misrepresent what a “normal” GCS means. In the typical mTBI case, for example, the defense might be expected to point out that the plaintiff had a normal GCS when evaluated by EMS or in the emergency department. However, a normal GCS does not equate to the lack of a brain injury.

The GCS scale is used by First Aid, EMS, nurses and doctors in acute medical and trauma patients. The scale is composed of three tests: eye, verbal, and motor responses. The lowest possible GCS is a sum of three (indicating deep coma or death), while the highest is 15 (indicating a fully awake person). For example, there are four grades given for the patient’s eye response, ranging from no eye opening (1 point), eye opening in response to pain stimulus (2 points), eye opening to speech (3 points), and eyes opening spontaneously (4 points). For the patient’s verbal response, five grades are given, ranging from none (1 point) to incomprehensible (2 point), inappropriate (3 points), confused (4 points), or oriented (5 points). Similarly, there are six grades given for the patient’s motor response. These primarily gauge conscious or subconscious reactions to pain, with the highest score being given to a person who can obey simple commands.

Generally, under the GCS, brain injury is classified as severe, moderate or minor. Severe is a GCS of less than 9, moderate is a GCS of 9 to 12, and minor is considered to be a GCS of 13 to 15. There is controversy with the use of the GCS, particularly in moderate and mild brain injury cases. This is because the GCS is primarily a tool for managing the patient in the acute setting. In other words, does a neurosurgeon need to be called in? GCS is not predictive of post-acute recovery in mild and moderate TBI.

B. Mild TBI

The term “mild brain injury” can be misleading. The term “mild” usually refers to the severity of the initial trauma that caused the injury. It also may refer to other symptoms within the first 24 hours after injury. The term “mild” is not intended to describe the long-term severity of the consequences of the injury.

The terms mild TBI and concussion are often used interchangeably. Several definitions have been proposed per each term. These definitions are similar, but not identical. The definition of TBI, and mild TBI in particular, tends to vary slightly according to medical specialties. Often, the term brain injury is used synonymously with head injury, which is also interchanged with concussion. These injuries may or may not be associated with neurologic deficits. Of particular importance to lawyers when questioning medical witnesses is the need to be clear on which definition is being used.

1. ACRM Definition

One of the most commonly accepted definitions comes from the American Congress of Rehabilitation Medicine:

A patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function, as manifested by at least one of the following:

- a. Any period of loss of consciousness;
- b. Any loss of memory for events immediately before or after the accident;
- c. Any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused); and

d. Focal neurological deficit(s) that may or may not be transient.

But where the severity of the injury does not exceed the following:

- Loss of consciousness of approximately 30 minutes or less;
- After 30 minutes, an initial Glasgow Coma Scale (GCS) score of 13-15; and
- Post Traumatic Amnesia (PTA) not greater than 24 hours.

See J. Head Trauma Rehabil. 1993; 8(3):86-87. This definition includes the head being struck, the head striking an object, or the brain undergoing an acceleration/deceleration movement (i.e., whiplash) without direct external trauma to the head. It excludes stroke, anoxia, tumor, encephalitis, etc.

According to the ACRM, routine diagnostic tests or neurological evaluations may be normal. “Due to the lack of medical emergency, or the realities of certain medical systems, some patients may not have the above factors medically documented in the acute stage. In such cases, it is appropriate to consider symptomatology that, when linked to a traumatic head injury, can suggest the existence of a mild traumatic brain injury.” *Id.*

2. AAN Definition

Alternatively, the American Academy of Neurology (AAN) defines concussion as “... a trauma-induced alteration in mental status that may or may not involve loss of consciousness.” *Neurology* 1997; 48(3):581-585. According to this definition, “confusion and amnesia are the hallmarks of concussion... [and] may occur immediately after the blow to the head or several minutes later.” *Id.*

In clinical practice, concussion and mTBI are often used synonymously. However, they are distinct terms. Concussion refers to altered function. mTBI describes a pathologic state of brain after the concussive event. Three grades of concussion have been identified according to AAN criteria. The grades are differentiated by duration of altered mental status and any loss of consciousness. Amnesia, although not part of the AAN criteria, is an independent diagnostic indicator of TBI severity, with the loss of memory preceding (retrograde) or following (post-traumatic or

anterograde) injury. A Grade 1 concussion is defined as having altered mental status lasting less than 15 minutes without loss of consciousness. In Grade 2, concussion is altered mental status lasting more than 15 minutes, again without a loss of consciousness. Grade 3 concussion, according to the AAN scale, is characterized by any loss of consciousness.

3. AANS Definition

The American Association of Neurological Surgeons defines concussion as “an injury to the brain that results in temporary loss of normal brain function.” The AANS goes on to say: “It usually is caused by a blow to the head. Cuts or bruises may be present on the head or face, but in many cases, there are no signs of trauma. Many people assume that concussions involve a loss of consciousness, but that is not true. In most cases, a person with a concussion never loses consciousness. See www.aans.org (patient information/conditions/treatments/concussion). The formal AANS medical definition of concussion is: a clinical syndrome characterized by immediate and transient alteration in brain function, including alteration of mental status and level of consciousness, resulting from mechanical force or trauma. *Id.*

C. Moderate TBI

Moderate TBI is usually associated with prolonged loss of consciousness of greater than fifteen minutes but less than 24 hours. Usually there are focal neurologic deficits. These patients usually remain in a hospital setting for several weeks, and are frequently transferred to a rehabilitation hospital once their acute medical crisis has been managed. The majority of survivors of moderate TBI are unable to return to their pre-injury level of function.

D. Severe TBI

Severe TBI occurs when the injury causes the patient to be comatose. Such injury is typically associated with significant neurologic deficits, often with structural lesions revealed by neuroimaging. Examples of this are skull fracture, intracranial hemorrhage, diffuse cerebral edema and related conditions. These patients usually require advanced medical care. Often, a neurosurgeon is brought in to operate, usually to alleviate brain swelling, or to evacuate intracranial hematoma. With severe TBI, recovery is usually prolonged and incomplete, with many of these patients having significantly reduced life expectancy.

III. Brain Imaging Technology

Mild Traumatic Brain Injuries (mTBI) that result in a concussion may not show abnormalities on traditional neuroimaging studies such as the CT and the standard MRI. While structural injuries may have occurred, they are beyond the sensitivity of these modalities. The more advanced neuroimaging techniques include DTI, fMRI, MR spectroscopy (MRS) and PET, each having advantages and disadvantages.

A. Computerized Tomography (CT)

During the initial evaluation of an acute head trauma patient, the primary neuroimaging techniques used are CT or CAT scan. CT scans on the day of the accident may show only damage to bones and not brain tissue. CT scans tell us very little about non-operative brain injury. A CT is a way to detect fractures of the skull and facial bones, accumulations of blood, and swelling or shifting of the brain structures.

1. Pros of CT scans:

- Identifies accumulations of blood
- Swelling, or
- Shifting of the brain structures
- Also, good at seeing fractures
- CT scans are fast
- Available in almost all acute care facilities
- Does a good job of identifying intracranial bleeding/mass effects, such as swelling or shifting of brain structures

2. Cons of CT Scans:

- It is a structural test
- It has poorer resolution than its counter parts
- Has little post-acute value

B. Magnetic Resonance Imaging (MRI)

In contrast to CT, MRI is far superior in detecting smaller lesions that may not involve hemorrhage and evidence of axonal injury. Although MRI is more sensitive than CT in patients with mild traumatic brain injuries, “over two thirds of symptomatic patients who undergo routine MRI show no signs of structural abnormalities.” Flynn F. Memory Impairment After Mild Traumatic Brain Injury. *Continuum Lifelong Learning Neurol* 2010; 16(6): 94.

Through the use of 3 Tesla MRI scanners, the MRI’s higher resolution has a greater relative advantage over CT. The use of the 3 Tesla MRI scanners for clinical diagnosis of mild brain injuries has resulted in the increase in the number of abnormal scans.

Although the use of a 3 Tesla MRI scan is an advancement from the previous use of the 1.5 T, the vast majority of clinical MRI scans are still done using 1.5 T machines. The more expensive 3T machines are found in research settings, and in most cases, a 3T MRI is not ordered, if at all, until a high degree of specificity is sought in the MRI. The increased Tesla results in a higher resolution. A MRI scan uses magnets which allows the molecules in the brain to resonate. The higher the Tesla, the more powerful the magnet. As a result, the scan has the ability to detect smaller things.

C. Functional Magnetic Resonance Imaging (fMRI)

The fMRI provides real-time data on cerebral metabolism during specific cognitive or motor tasks. This non-invasive method demonstrates evidence of neuronal dysfunction by the imaging of regional changes in blood oxygenation patterns.

Studies of sports-related concussions using a fMRI have shown that symptoms of concussions are related to neuronal dysfunction, usually in the absence of a structural brain injury. This technique is a good tool in assisting in concussion management and in monitoring recovery.

D. Diffusion Tensor Imaging (DTI)

DTI, developed from MRI technology, is a technique that creates an image of the pathology in the fiber tracts within the white matter of the brain, even

though individual axons are too small to be seen without a microscope. The pathways can be seen due to the fact that they tend to run with other axons, making a configuration that is large enough to image. Essentially, DTI is a modality for measuring white matter integrity and connectivity.

More specifically DTI assesses the direction of the diffusion of water molecules in the white matter tracts. The organized and specific direction of diffusion in healthy white matter is known as anisotropy. Fractional anisotropy (FA) measures how much local diffusion deviates from isotropic diffusion (lack of directional organization). FA scores correlate with the severity of the injury. Reduced directionality of the diffusion of water is shown by a reduction in FA scores at sites of traumatic axonal shearing injuries, which indicates a loss of microstructural fiber integrity.

A 2008 study found that persons with mild TBI showed increased FA correlated with symptom severity in subjects with normal structural imaging. Difiori JoP, Giza CC. New Techniques in concussion imaging. *Curr Sports Med Rep* 2010; 9(1):35-39. (citing Wilde EA, McCauley SR, Hunter JV, et al. Diffusion tensor imaging of acute mild traumatic brain injury in adolescents. *Neurology*. 2008; 62:1286-95).

Furthermore, other studies have also shown a correlation between DTI and cognitive deficits after a TBI and shown that DTI was able to detect white matter injury even when a 3T MRI was normal. Levin HS, Wilde EA, Chu Z, et al. Diffusion tensor imaging in relation to cognitive and functional outcome of traumatic brain injury in children. *J. Head Trauma Rehabil*. 2008; 23:197-208; Kraus MF, Susmaras T, Caughlin BP, et al. White matter integrity and cognitive in chronic traumatic brain injury: a diffusion tensor imaging study. *Brain*. 2007; 130:2508-19; Niogi SN, Mukherjee P, Gharjar J, et al. Extent of microstructural white matter injury in postconcussive syndrome correlates with impaired cognitive reaction time: a 3T diffusion tensor imaging study of mild traumatic brain injury. *Am. J. Neurodiol*. 2008; 29:967-73.

E. Magnetic Resonance Spectroscopy (MRS)

MRS provides images that illustrate the functional cerebral metabolism and could potentially be used to delineate the physiologic changes seen in concussions. This noninvasive technique uses metabolite data from areas of the brain to provide an

assessment of neurochemical alterations after a brain injury.

The MRS can identify changes in NAA/creatinine and choline/creatinine ratios indicating a brain injury in those who have experienced a TBI, even when no structural injury is visible on a MRI. Another advantage of the MRS is that it may be able to identify metabolic disturbances after a brain injury and in sports related concussions even though clinical symptoms have been resolved.

F. Electroencephalography (EEG)

Electroencephalography (EEG) has been around for one hundred years. The technology is well-known. The brain's electrical charge is maintained by billions of neurons. EEG measures the brain's spontaneous electrical activity over a short period of time, usually 20–40 minutes, as recorded from multiple electrodes placed on the scalp. EEG is frequently used in diagnosing epilepsy, as epileptic activity can create clear abnormalities on a standard EEG study. EEG used to be a first-line method for the diagnosis of tumors, stroke and other focal brain disorders, but this use has decreased with the advent of more modern neuroimaging techniques. Nevertheless, an abnormal EEG, especially when other causes of the abnormal result can be ruled out (e.g., stroke, epilepsy, tumor, etc.), can be useful corroborative evidence of a subtle brain injury.

G. Magnetoencephalography (MEG)

Magnetoencephalography (MEG) is a functional neuroimaging technique for mapping brain activity by recording magnetic fields produced by electrical currents occurring naturally in the brain, using very sensitive magnetometers. Applications of MEG include basic research into perceptual and cognitive brain processes, localizing regions affected by pathology before surgical removal, determining the function of various parts of the brain, and neurofeedback. MEG is useful in the legal setting because it measures the activity, or function, of various regions of the brain. The testing is performed on an awake, alert adult and lasts about thirty minutes. The results are studied by neuroscientists, who identify areas of abnormal slow waves within regions of the brain. Diffuse axonal injury, such as is commonly associated with mild TBI, is one cause of abnormal MEG. Other causes could include stroke, tumor, epilepsy or other organic brain disease. Again, it is essential for other potential causes of abnormal

slow waves to be considered and eliminated in order for MEG evidence to be admissible into evidence in a legal case.

H. Positron Emission Tomography (PET)

PET, through the use of radionuclides with short half-lives injected intravenously crossing the blood-brain barrier and distributed in brain cells, provides images that illustrate the functional cerebral metabolism and could potentially be used to delineate the physiologic changes seen in concussions.

PET scans are used on more severely injured patients and may be used to assess subacute or chronic head injury with cognitive or neurologic deficit.

I. Single photon emission computed tomography (SPECT)

Single photon emission computed tomography (SPECT) is a test that uses a radioactive tracer to detect abnormalities in cerebral blood flow. In general, SPECT is more sensitive than CT and MRI in detecting lesions in TBI patients. However, it is not always clear how abnormalities observed on SPECT correspond to injury. Sometimes abnormalities are seen in SPECT on mild TBI patients with chronic symptoms, even if no structural damage is apparent. Other times the SPECT scan will be normal, yet the patient still suffers from chronic TBI symptoms. Studies have found that decreased blood flow to various parts of the brain correlate with various types of behavior. Because MRI detects lesions missed by SPECT and *vice versa*, a combination of MR and SPECT is sometimes utilized.

IV. Types of Expert Witnesses in a TBI Case

A. Neurologist

A neurologist is a physician with specialized training in diagnosing, treating and managing disorders of the brain and nervous system. Many neurologists also have additional training in one area of neurology such as stroke, epilepsy or movement disorders. Neurologists treat disorders of the nervous system, brain, spinal cord, nerves, muscles and pain. Common neurological disorders include: stroke, Alzheimer's disease, headache, epilepsy, Parkinson's disease, sleep disorders, multiple sclerosis, pain, tremor, brain and spinal cord injuries, brain tumors, peripheral nervous disorders and amyotrophic lateral sclerosis.

The neurologist reviews the patient's health history with special attention to the current condition. The patient then takes a neurological exam. Typically, the exam tests vision, strength, coordination, reflexes and sensation. This information helps the neurologist determine if the problem is in the nervous system. Further tests may be needed to confirm a diagnosis or find a specific treatment. In the typical brain injury case, the neurologist is the one who refers the patient for a neuropsychological examination, and may also order tests, such as MRI or PET scans, as well as manage the patient's medications. If the patient's brain injury results in chronic headaches, or a seizure disorder, the neurologist may be the frontline treater for those conditions as well.

B. Neurosurgeon

Insofar as brain injury victims are concerned, a neurosurgeon's role is usually surgery on the brain or skull, often to address emergent bleeding or swelling in the brain that will lead to further damage or death if not immediately addressed. In most brain injury cases, if a neurosurgeon was called in to the ER to operate on the patient, then there is little doubt that the injury was severe and debilitating. Unlike in spine injury cases, where a neurosurgeon's testimony is often vital to establishing that a particular injury or medical condition was caused by a certain traumatic event, there is usually little need for the neurosurgeon's testimony on causation in a brain injury case.

C. Neuropsychologist

A neuropsychologist is a Ph.D. who specializes in studying the relationship between the brain and a person's behavior. When other medical specialists, such as neurosurgeons, psychiatrists, or neurologists, want to assess a patient's brain function after a significant brain trauma, a referral is often made to a neuropsychologist to conduct a "neuropsychological evaluation."

A neuropsychological evaluation is an assessment of how a person's brain is functioning. From this testing, a neuropsychologist will draw conclusions about the structural and functional integrity of a patient's brain. The neuropsychological evaluation involves an interview and the administration of tests. The tests are typically pencil and paper type tests. Some tasks might be self-reports, meaning that they are completed by the patient with assistance from a technician, but the majority of the

tests require administration by a neuropsychologist or trained, skilled psychometrist. Neuropsychological tests (unlike bedside cognitive and behavioral neurologic screens) are standardized, meaning that they are given in the same manner to all patients and scored in a similar manner time after time. An individual's scores on tests are interpreted by comparing their score to that of healthy individuals of a similar demographic background (i.e., of similar age, education, gender, and/or ethnic background) and to expected levels of functioning. In this way, a neuropsychologist can determine whether a particular patient's performance on any given task represents a strength or weakness. Although individual scores are important, the neuropsychologist looks at all of the data from the evaluation to determine a pattern of cognitive strengths and weaknesses and, in turn, to understand more about how the patient's brain is functioning.

Neuropsychological tests evaluate functioning in a number of areas including: intelligence, executive functions (such as planning, abstraction, conceptualization), attention, memory, language, perception, sensorimotor functions, motivation, mood state and emotion, quality of life, and personality styles. The areas addressed in an individual's evaluation are determined by the referral question (what the referring doctor and patient wants to know), patient's complaints and symptoms, and observations made during interview and test administration.

In the typical brain injury case, the neuropsychologist is a vital expert witness. He or she is the one that ties all of the information together, and relates to the jury how the plaintiff's problems are (or are not) the result of a brain injury.

D. Neuroradiologist

Neuroradiologists are physicians who have specialized within the field of radiology. Neuroradiology is the medical subspecialty that deals with the diagnosis and treatment of brain, spinal cord, head and neck, and vascular lesions using x-rays, magnetic fields, radio waves, and ultrasound. Following a four-year radiology residency, the neuroradiologist undergoes two additional years of fellowship training. In legal cases, neuroradiologists are useful expert witnesses, because they can interpret the various diagnostic tests that have been performed on a particular patient. Also, they frequently can order additional testing that may be more sensitive than any testing that was performed previously. The

interpretation of diagnostic imaging tests that reveal the objective evidence of a traumatic brain injury can be compelling expert testimony in trial. Alternatively, if there is nothing to see on a particular image, for example a normal CT or MRI, a neuroradiologist can explain why the test was ordered, and why a “normal” test does not mean there is no brain injury.

E. Physical Medicine and Rehabilitation Physician (PM&R)

The PM&R doctor, also known as a physiatrist, or a rehab doctor, is primarily concerned with restoring functional ability and quality of life to those with physical impairments or disabilities. They commonly treat patients with brain or spine injury. They are also useful when a patient has numerous injuries.

F. Other Therapists

There are many other types of health care providers that play a role in the treatment of the TBI patient. These include psychiatrists, psychologists, counselors, cognitive, occupational or physical therapists, and speech-language pathologists. Lawyers should consider using one or more of these witnesses to fully develop the evidence in a brain injury case. For example, a therapist can describe the patient’s struggles at overcoming day-to-day challenges brought about by the brain injury. Often this type of testimony provides a type of insight that no other healthcare provider can offer.

G. Life Care Planner

Life Care Plans describe the medical condition and ongoing medical requirements of chronically ill or injured individuals, and quantify the cost of supplying them with requisite, medically-related goods and services. The life care planner must review all of the patient’s medical records, and in some cases, conduct a clinical examination or interview of the patient and family, and interact with the medical and health related professional treatment team. The life care planner should also be knowledgeable of the relevant clinical practice guidelines, and the relevant research literature as well as the opinions of consulting team members (physicians, therapists, etc.). The responsibility of the life care planner as expert witness is to explain the disability or injury, project how that injury will affect the patient in the future, and explain how, when, and why specific future cost items will be incurred. The planner must be able to clearly

communicate the nature of a patient's disability, the residual functional limitations, and the effects of the disability throughout the patient's life expectancy.

H. Vocational Rehabilitation Specialist

A vocational rehabilitation specialist is a very useful expert witness in a TBI case. Studies show that survivors of traumatic brain injury – even mild TBI -- have significantly lower rates of employment throughout their work life expectancy. Additionally, the TBI often results in a reduction in educational attainment. In other words, the TBI victim will not finish school, or will not do as well in school as they would have without the injury. Finally, a TBI usually results in a reduction in the types of jobs the survivor can successfully handle, thus limiting the employability and earnings capacity of the injured person. A vocational rehab expert can analyze these issues in a legal case, and offer expert opinions of the loss of earnings capacity, and the costs of job re-training.

I. Educational Consultant

There are many unique issues raised in pediatric brain injury cases that I will not attempt to address in this paper. However, one of them that bears mentioning is the need to hire an educational consultant. This is especially important when the brain injury victim is a school-aged child. An educational consultant with experience in customizing educational plans for children and adolescents who have sustained brain injuries is a critical component of these cases. The cost of these plans should be incorporated into the overall life care plan.

J. Economist

An economic expert is necessary to educate the jury by calculating economic losses that are likely to occur in the future and discounting these sums to today’s dollars. These damages include wages, earning capacity, profits, benefits, stock options, household services, and future medical costs. It is also important to adjust earning capacity losses for personal consumption or taxes. Any case with significant evidence of lost wages or future medical expense justifies having an economist or an accountant as an expert witness.

V. Common Symptoms following Mild TBI

Although most people recover from mild TBI with minimal treatment and rest, there is a distinct percentage that do not recover, and instead develop persistent post-concussive syndrome. Estimates of this group range from 7% to 40%, with most research estimates clustering around 15-20% of the mTBI population. Thus, it is undisputed that mild TBI can result in serious, long-term effects on an individual's cognitive, physical, and psychological function. The focus here will be on persistent post-concussive syndrome, as those are the cases most likely to end up in litigation.

Common symptoms following mild TBI include:

- Headache
- Dizziness
- Malaise
- Fatigue
- Intolerance to noise or light
- Irritability
- Depression
- Anxiety
- Emotional lability
- Impaired concentration
- Memory loss
- Insomnia
- Personality changes

Generally, if a patient has a history of head trauma followed temporally by the onset of three or more of the foregoing symptoms, a diagnosis of post-concussion syndrome can be made. The symptoms generally improve over time and then reach a plateau. However, the medical literature confirms that there can be a high degree of variability in symptomatology from one patient to the next, even when all other factors are similar. Confounding the diagnosis is that there is frequently overlap of symptoms from mild TBI, chronic pain, PTSD, and depression. I recommend that the attorney make a list of the symptoms reported by the client, the family members, and the medical records, and conduct research into each one in an effort to prove on a case-by-case basis that the symptom was brought about by the injury. In some cases, experts will disagree as to the whether a

particular symptom is due to an organic brain injury, or instead due to something else, such as PTSD or depression. In my experience, however, plaintiff and defense experts end up end up agreeing that, regardless of what it is called, the problem arose as a result of the accident that caused the injury in the first place.

VI. Common Defenses in Mild TBI Cases

The main defense in many TBI cases, and especially in mild TBI cases, is what I call the "prove it" defense. In these cases, the plaintiff will usually have no outward signs of injury. They will walk, talk and look like a normal person. The injury is a subtle one that comes to light through details provided either by the plaintiff, his family, friends, co-workers, and his doctors. Herein lies the essential challenge of trying the mild traumatic brain injury case. The plaintiff looks normal, there is no objectively verifiable evidence of lingering injury, and yet the plaintiff claims damages that run into the millions of dollars. How do we know the claim is real?

What follows below is a list of some of the defenses I have encountered in the TBI cases I have litigated.

- Absence of Objectively Verifiable Injury
- Normal CT and MRI Scans
- Lack of Identification of TBI by Other Healthcare Providers
- Mild TBI Symptoms Should Have Resolved Within Three Months
- Lack of Treatment of TBI Symptoms Means No TBI
- Pre-Existing ADHD
- High IQ on Post-Injury Neuropsychological Testing
- Low IQ on Pre-Injury Testing
- Age-Related Changes (If Plaintiff Was Over 60 Years of Age)
- History of Depression
- Prior Psychiatric History
- Prior Drug Use
- History of Alcohol Abuse
- Other Health Problems
- Side Effects from Prescription Medications

- Malingering
- Secondary Gain
- Somatoform Disorder
- Not Organic Brain Injury, Just _____ (fill in the blank – e.g., depression, PTSD, normal for this person, etc.).

In mild brain injury cases, the defense often focuses on the fact that the neuro-imaging of the brain was normal. This is common because, by definition, a mild TBI is one in which there are no visible lesions on the brain. In other words, you expect to find a normal CT and MRI. Just because an injury is not objectively verifiable with a diagnostic test does not mean that it is any less real. The entire field of neuropsychology has developed around this concept. Even defense expert witnesses must agree that the majority of mTBI sufferers have symptoms that are real to them, and are highly correlated with trauma to the brain.

In car and truck wreck cases, the plaintiff's brain may be only one of several parts of the body that were injured in the crash. Frequently in cases involving multiple traumatic injuries, the mild brain injury is overlooked. The focus in the emergency department tends to be on other more visible traumatic injuries. Often, it is not until several weeks later that the patient, his family members, or his doctors realize that there are cognitive and emotional deficits attributable to a TBI. By this time, the same deficits are also reasonably attributable to PTSD or depression resulting from the other traumatic injuries. It can be confusing. These facts frequently lead to a defense based upon the concept that "if the plaintiff's other doctors did not diagnose it, did not observe it, and did not treat it, it must not have been there." In one well known study of TBI in the emergency department, it was documented that 56 percent of mild traumatic brain injuries are not even recognized in the emergency department.

Another common defense is that the plaintiff's mild TBI symptoms should have resolved within three months, because "most people get better." While it is true that most people do get better with little or no treatment, a significant percentage of mTBI victims fall within the "miserable minority." Many studies document that 15 to 20 percent, or even more, of patients develop chronic persistent post concussive syndrome that lasts more than one year following the

injury. In some cases, these symptoms have been documented to be lifelong.

Another common defense involves looking for pre-existing conditions that confound the diagnosis of persistent post concussive syndrome. For example, attention deficit hyperactivity disorder (ADHD), where diagnosed and documented, can muddy the waters somewhat as to whether or not a particular patient's attention deficits were pre-existing, or are instead attributable to a TBI. Also, a history of a major depressive disorder, other psychiatric history, alcohol abuse, or even sporadic drug use can complicate the picture. In these cases, it is important to have a physician, using clinical judgment, review all of the patient's symptoms and history, and provide an opinion based upon reasonable medical probability that takes all of these factors into account.

Another interesting defense centers upon the I.Q. of the plaintiff. If the plaintiff demonstrates a high I.Q. on neuropsychological testing after the injury, then invariably the defense is that the injury did not affect plaintiff's cognitive abilities. However, rarely is it the case that the plaintiff in a mild TBI is asserting a wholesale loss of cognitive function. Rather, the usual claim is that the TBI has impaired some subcomponent of plaintiff's cognition, such as the ability to multitask, or the ability to sustain attention in the presence of distractions. These executive frontal lobe functions are particularly vulnerable to mild traumatic brain injury.

Conversely, in the case of one who has a low I.Q. on testing, especially pre-injury testing, the defense usually invokes the argument that the brain injury has not provided any significant impairment in the plaintiff's day-to-day function. However, in most cases, there are still changes in the plaintiff's ability to navigate the complexities of day-to-day life, mood, irritability, withdrawal, and damage to other relationships.

It can be particularly vexing if the plaintiff is over the age of 60 and suffers a mild traumatic brain injury. At some point, as we age, most of us experience cognitive decline. Frequently, this can be observed on CT or MRI scans that show brain volume loss, or senile atrophy. Many of the symptoms that TBI sufferers complain of are similar to the symptoms that the geriatric population complains of – forgetfulness, distractibility, irritability, mood changes, dizziness, and so on. Therefore, the lawyer must rely heavily on expert witnesses to consider and

rule out other possible causes of the elderly plaintiff's symptom cluster.

Malingering and secondary gain are also frequent defense themes in mild TBI cases. Because the injury is difficult to objectively verify, it lends itself to claims that the plaintiff is faking or exaggerating the injury in order to obtain a monetary settlement. There are entire books written on this subject, with defense-oriented neuropsychologists parsing out certain subgroups of responses from the plaintiff's neuropsychological test battery and then claiming that those responses indicate malingering. This practice is controversial, with a great deal of research, writing, and debate surrounding it.

VII. Ethical Considerations in Settling TBI Cases

Sometimes the question arises: Is an ad litem or court approval necessary to effectuate settlement in a traumatic brain injury case? The answer is, it depends.

An important part of any guardian ad litem's job is to approve any settlement reached of his client's claims. Claims of those being represented by a next friend require court approval to bind the real party in interest, Tex. R. Civ. Pro. 44(2), and guardian ad litem are specifically required to "advise the court whether the settlement is in the party's best interest." Tex. R. Civ. Pro. 173.4(c). *See, generally, Maes v. El Paso Orthopaedic Surgery Gp., P.A.*, 385 S.W.3d 694, 698 (Tex. App. — El Paso 2010, pet. denied).

However, just because a person has suffered a traumatic brain injury does not mean that they must have any settlement they reach approved by an ad litem. If an adult victim of TBI is *compos mentis* and has sufficient capacity to agree to a settlement, and does not have a guardian and is not being represented in the suit by a next friend, no guardian ad litem should be necessary. *See, e.g., In re J.T.H.*, 630 S.W.2d 473, 477 (Tex. App. — San Antonio 1982, no writ) (recognizing that parties must have capacity to contract to enter into a binding settlement). When the adult TBI victim has the capacity to settle, the case does not present the kind of conflict that requires (or even allows) the appointment of an ad litem, and the adult victim should be presumed capable of accepting or rejecting a settlement himself. *See, e.g., Riggins v. Hill*, 14-09-00495-CV, 2011 WL 5248347 at * 8-9 (Tex. App. — Houston [14th Dist.] Nov. 3, 2011, pet. denied) (mem. op.) (rejecting claim that settlement agreement was not binding on adult because of

claimed lack of capacity, even though guardian ad litem had been appointed). This is true, if for no other reason, then Texas law generally presumes that adults have the capacity to enter into a binding contract. *Swink v. City of Dallas*, 36 S.W.2d 222, 224 (Tex. Comm'n App. 1931, hldg. approved); *McKeehan v. McKeehan*, 355 S.W.3d 282, 295 (Tex. App. — Austin 2011, pet. denied).

However, the requirement that the victim have the mental capacity to agree to a settlement raises the corollary -- that a victim lacking the proper capacity cannot enter into a valid settlement, and if he purports to do so the settlement agreement may be avoided. *Kinsel v. Lindsey*, ___ S.W.3d ___, 15-0403, 2017 WL 2324392 at * 5 (Tex. Feb. 16, 2017); *In re Morgan Stanley & Co.*, 293 S.W.3d 182, 193 (Tex. 2009) (orig. proceeding). In some cases, the question of a TBI victim's mental capacity are settled as a matter of law; for example, the appointment of a guardian creates a presumption of the ward's incompetency in other proceedings. *Barker v. Noelke*, 105 S.W.3d 75, 85-86 (Tex. App. — Eastland 2003, pet. denied). However, not all cases are so clear, and defendants in TBI cases may raise the issue of capacity if the question is a close one, as part of ensuring that any settlement made will fully bind all parties. So, what degree of mental capacity must a TBI victim possess to be able to give valid consent to a settlement agreement?

The capacity necessary to consent to a settlement is the same as the capacity necessary to enter into any contract, or sign any document: the party signing the document must appreciate what he is doing, and understand the consequences of his acts and the nature of the business being transacted. *Kinsel* at * 5 (citing *Mandell & Wright v. Thomas*, 441 S.W.2d 841 (Tex. 1969)). The relevant time for making this determination is the time the agreement was made, *Kinsel* at * 5; *Lee v. Lee*, 424 S.W.2d 609, 611 (Tex. 1968), although evidence about what the person understood at other times may be relevant to proving the mental state at the moment required. *Lee*, 424 S.W.2d at 611; *Estate of Grimm*, 180 S.W.3d 602, 606 (Tex. App. — Eastland 2005, no pet.).

Determining whether an adult does or does not have the requisite mental capacity requires considering a number of different factors, not all of which may be present in every case:

- the person’s outward conduct, seen as a manifestation of an inward condition affecting capacity;
- whether there are any pre-existing circumstances that would tend to affect the person’s capacity; and
- the existence of a mental condition, either before or after the document in question was signed, to the extent this allows the court to make an inference regarding his mental capacity at the time.

Texas Capital Bank v. Asche, 05-15-00102-CV, 2017 WL 655923 at * 6 (Tex. App. — Dallas Feb. 17, 2017, n.p.h.) (mem. op.); *Guardianship of Westbo*, 01-14-00705-CV, 2016 WL 262282 at * 3 (Tex. App. — Houston [1st Dist.] Jan. 21, 2016, pet. denied) (mem. op.); *Estate of Robinson*, 140 S.W.3d 782, 793-94 (Tex. App. — Corpus Christi 2004, pet. denied).

Thus, while not necessary in every TBI settlement, if the parties agree then it may be wise to obtain court approval of the parties’ settlement in any serious TBI case, as this would particularly allow the court to enter findings that the adult TBI plaintiff understands the nature of the settlement, which would reduce any possibility, however remote, that the settlement would be subject to be set aside at a later date due to the TBI victim’s lack of capacity.

VIII. Conclusion

“Invisible” brain injuries must be made visible to the judge and jury in order for the plaintiff to win the case. This process begins with the education of the jury about the brain. Then, the jury needs to understand the limits of modern medicine and structural brain imaging tests, such as MRI and CT scan, and why they are frequently normal even in the face of a mild traumatic brain injury. The jury also needs to be taught how the client’s problems are consistent with the brain injury, and are likely the result of the brain injury, as opposed to other causes. Neuropsychological testing is an important component of this proof. So too are cutting edge technologies,

such as MEG or DTI, that can objectively verify the presence of a microscopic brain injury.

Finally, “before and after” witnesses must put everything into perspective, so that the jury understands how the particular injury has affected the quality of life of the plaintiff.

The litigation of traumatic brain injury cases is expensive, time-consuming, and complex. The cases are difficult to evaluate, both for plaintiff’s counsel and defense counsel, because there are so many variables. One never knows how a jury will react to evidence of “invisible” brain injury. The increase in public awareness of brain injuries, especially with sports concussions and military injuries, serves to educate the jury pool, and potentially predispose them to be more accepting of an injury victim’s proof.

Brain injury litigation is filled with paradox. An injury may be “mild” but the effects may be severe. An injury may be “invisible” yet potentially worth millions of dollars. There are vast amounts of published research concerning all aspects of the brain, yet there is much that is still unknown about the human brain and how it functions.

Every case is different and requires an in-depth analysis of the details that make it unique. Just as no two snowflakes are alike, the same is true with brain injury cases.

Irresistible Impulses & Disinhibition

Presented By John Jerry Glas
Deutsch, Kerrigan & Stiles, L.L.P.
755 Magazine Street, New Orleans, Louisiana 70130
(504)593-0627; jjglas@dkslaw.com

There are more than 1.7 million estimated traumatic brain injuries every year in the United States. Public awareness and concern about the effects of concussions has never been greater. Sixty years ago, an NFL player “got his bell rung” and was told to “get back out there.” Today, the NFL has mandatory baseline neuropsychological testing, a sideline “concussion assessment” protocol, and strict “return to play” guidelines. Times have changed.

Suddenly, nobody really knows the effects of a concussion, and Terence Moore is writing an article entitled: “It’s Time For Ex-NFL Players To Be Scared, Very Scared.” (AolNews.com, 2/23/11). In a world where everyone can broadcast (blog, tweet, and post), and everything is reported, the blotter of arrests, addictions, commitments, overdoses, and suicides by former football players grows daily. Everyone wants to understand why a player would “do that”, and everyone is asking the same question: should we blame the player or the concussions? The question frames the discussion and marks the times.

In the courtroom, when a plaintiff who sustained a TBI engages in socially inappropriate, impulsive, criminal, or suicidal behavior, it is becoming increasingly common for plaintiff’s counsel to excuse or explain that behavior by claiming the TBI rendered the plaintiff more susceptible to the impulse (disinhibition), or totally unable to resist the impulse (an irresistible impulse). Invariably, counsel will argue that the plaintiff is not responsible for his or her conduct because, but for the TBI-induced disinhibition, plaintiff *could* and *would* have resisted the impulse.

That argument can be as irresistible as the Siren's song. Plaintiff did not behave this way before the TBI, and plaintiff started behaving this way (soon) after the TBI; therefore, the TBI caused the behavior. *Post hoc ergo propter hoc*.

These claims of "irresistible impulses" and "disinhibition" are far more common in criminal cases, where prosecutors and criminal defense attorneys have the benefit of statutes defining the legal concept of insanity, and years of experience handling competency, capacity, and insanity testimony. In many states, testimony and evidence regarding irresistible impulses is strictly excluded from the guilt phase; and Federal Rule of Evidence 704(b) additionally prohibits experts from giving an opinion "as to whether the defendant did or did not have the mental state or condition constituting an element of the crime charged or of a defense thereto." Those advantages do not exist in civil cases.

Every lawyer needs a strategy for evaluating and attacking the allegation that a plaintiff lacks self-control and is not responsible for his or her conduct. The following list of one hundred (100) questions has been designed: (1) to determine whether a plaintiff sustained the type of TBI associated with disinhibition; (2) to determine what frontal lobe impairment exists; (3) to identify alternative causes of disinhibition (i.e., confounding variables); (4) to discover an expert's methodology for determining the existence and cause of disinhibition; (5) to determine how an expert can distinguish between unresisted and irresistible impulses; and (6) to identify facts that prove a plaintiff intentionally and voluntarily engaged in the behavior. Following the actual list, there is a discussion of each question.

Please resist the impulse to read it while watching a football game.

One Hundred Questions:

Location of The Brain Injury

1. Did plaintiff sustain a diffuse brain injury or a focal brain injury?
2. Did plaintiff sustain a focal injury to the frontal lobe?
3. What area (or functional region) of the frontal lobe was damaged?
4. Which frontal subcortical circuits are affected by the injury?
5. What was the nature (severity) of the frontal lobe injury?
6. Did the focal or diffuse brain injury resolve?

Frontal Lobe Functions:

7. Does plaintiff have impaired executive cognitive function?
8. Does plaintiff have impaired behavioral/emotional self-regulation?
9. Does plaintiff have impaired activation regulation?
10. Does plaintiff have impaired meta-cognitive processes?

Frontal Lobe Dysfunction:

11. What cognitive and behavioral impairment has plaintiff demonstrated?
12. Does plaintiff have normal fine movement & strength in arms and hands?
13. Does plaintiff have any frontal release signs?
14. Does plaintiff have impaired spatial orientation?
15. Does plaintiff have normal facial expression?
16. Does plaintiff have Broca's Aphasia?
17. Does plaintiff have apraxia?
18. Does plaintiff perseverate?
19. Does plaintiff have impaired attention?
20. Does plaintiff have emotional lability?
21. Does plaintiff have impaired learning ability?
22. Does plaintiff have impaired working memory?

Neuropsychological Testing:

23. What functions are associated with the frontal [or damaged] lobe?
24. What neuropsychological tests evaluate those functions?
25. How did plaintiff perform on the Wisconsin Card Sorting Test?
26. How did plaintiff perform on the Trail Making Test?
27. How did plaintiff perform on the Stroop Test?
28. How did plaintiff perform on word list learning tasks?
29. What was plaintiff's "working memory" composite score?
30. How did plaintiff perform on the Controlled Oral Word Association Test?
31. How did plaintiff perform on other categorical fluency tests?

One Hundred Questions (continued):

32. How did plaintiff perform on the Digit Span Test?
33. How did plaintiff perform on the Finger Tapping Test?
34. How did plaintiff perform on the Continuous Performance Test?
35. How did the plaintiff perform on the Sustained Attention To Response Test and the Elevator Counting Test?
36. How did plaintiff perform on the Go/No-Go Test?
37. How did plaintiff perform on the Minnesota Impulsive Disorders Interview?
38. How did plaintiff perform on the Behavioral Dyscontrol Scale?
39. How did plaintiff perform on the Zuckerman Sensation-Seeking Scale?
40. How did plaintiff perform on the Barratt Impulsivity Rating Scales?
41. How did plaintiff perform on measures of general and negative affect?
42. How did the neuropsychologist determine plaintiff's pre-morbid ability (and expected performance) on each test?
43. Was plaintiff's performance consistent with pre-morbid functioning?

Daily Functioning:

44. Does plaintiff manage his or her own finances?
45. Did plaintiff make an appointment, arrive on time, & understand the reason for the evaluation?
46. Has plaintiff experienced a change in his ability to smell?
47. Has plaintiff experienced changes in sexual behavior?
48. Has plaintiff experienced outbursts of aggression or rage?
49. Has plaintiff been diagnosed with pseudodepression?
50. Has plaintiff been diagnosed as pseudopsychopathic?

Underlying Mental Illness or Disorder:

51. Has plaintiff been diagnosed with mania (or had manic episodes)?
52. Has plaintiff been diagnosed with bipolar disorder?
53. Has plaintiff been diagnosed with dementia?
54. Has plaintiff been diagnosed with organic personality disorder or intermittent explosive disorder?
55. Does plaintiff have disinhibition syndrome?
56. Has plaintiff been diagnosed with Post-Traumatic Stress Disorder?
57. Has plaintiff been diagnosed with impulse control disorder?
58. Has plaintiff been diagnosed with any mental illness or disorder?
59. What underlying disorder caused plaintiff's disinhibition or the irresistible impulse?
60. Did plaintiff have a "severe mental disease or defect"?

One Hundred Questions (continued):

Drugs, Alcohol & Medications:

- 61.** Does plaintiff have alcohol-related dementia?
- 62.** Did plaintiff have alcohol in his system?
- 63.** Did plaintiff have any drugs in his system?
- 64.** Did alcohol or drugs cause or contribute to plaintiff's disinhibition?
- 65.** What are the side effects of plaintiff's medications?

Cognitive Functioning:

- 66.** What is plaintiff's intelligence quotient (I.Q.)?
- 67.** Was plaintiff conscious when he acted?
- 68.** Does plaintiff recall his behavior?
- 69.** Was plaintiff totally deprived of his understanding?
- 70.** Did plaintiff have a defect of reason?
- 71.** Did plaintiff understand the physical act and its consequences (the nature and quality of the act)?
- 72.** Did plaintiff know the act was against the law (legally wrong)?
- 73.** Did plaintiff believe the act was morally wrong?
- 74.** Could plaintiff distinguish between right and wrong (good and evil)?
- 75.** Could plaintiff recognize reality?
- 76.** Did plaintiff try to hide evidence of his action?
- 77.** Did plaintiff feel guilt after committing the act?
- 78.** Did plaintiff plead guilty?
- 79.** Should a person be held responsible when they know what they are doing is wrong?

Scope & Timing of Expert Testimony:

- 80.** Is the expert qualified to testify as to whether plaintiff suffered from a severe mental illness?
- 81.** Should the expert be allowed to testify that the severe mental illness prevented the defendant from resisting an impulse?
- 82.** When did the expert render a psychiatric diagnosis?

One Hundred Questions (continued):

Volitional Capacity:

83. When did plaintiff first experience the impulse?
84. Did plaintiff resist the impulse for any period of time?
85. Did plaintiff's conduct involve planning and organization (premeditation)?
86. Was the (disinhibited) behavior self-defeating?
87. Was the (disinhibited) behavior self-endangering?
88. Was plaintiff able to conform his conduct to the requirements of law?
89. Did plaintiff physically lose control over his extremities?
90. Did plaintiff lose all ability to control his conduct?
91. Would plaintiff have engaged in the conduct if a policeman was present?
92. Was plaintiff's (involuntary) conduct a product of a mental defect, disease, or illness?
93. Did plaintiff lose the power of his will?
94. When a person is not held responsible for his conduct, can he become more disinhibited?

Mechanism & Causation:

95. How much disinhibition is required to engage in the behavior?
96. Can you say, with a reasonable degree of professional certainty, that the behavior was caused by disinhibition?
97. What evidence proves this underlying disorder or injury can cause (general causation) and did cause (specific causation) disinhibition or irresistible impulses?
98. How did the underlying disorder or injury cause the disinhibition or irresistible impulse (mechanism)?
99. How did you determine plaintiff would have behaved differently before the traumatic brain injury?
100. How can you distinguish between an irresistible impulse and an unresisted impulse?

Location Of The Brain Injury:

Most claims of disinhibition and impulsivity involve focal injuries to the frontal lobe, but plaintiff attorneys and experts will associate disinhibition with focal injuries to other lobes and to diffuse axonal injuries. See *Brewer v. J.B. Transport, Inc.*, 35 So.3d 230, 234 (La. 2010)(traumatic injury to right anterior temporal lobe blamed for Brewer's behavioral dysfunction including "changed personality and disinhibition"); see also *Delores M. v. Southern Farm Bureau Cas. Ins. Co.*, 29 So.3d 654 (La.App. 2 Cir. 1/6/10)(neuropsychologist testified that, as a result of the axonal damage, T.M.G. suffered from disinhibition and inattention resulting in attention deficit disorder."). Always start by trying to eliminate diffuse brain injury, and by determining the specific location and the severity of the focal brain injury.

1. Did plaintiff sustain a diffuse brain injury or a focal brain injury?

A focal brain injury occurs in a specific location; a diffuse brain injury occurs over a more widespread area. Focal brain injuries usually involve focal neurological symptoms. Try to rule out diffuse brain injury.

Diffuse axonal injury (DAI) occurs when a force causes the twisting and shearing of brain tissue, and the subsequent tearing and breaking of axons. DAI prevents electrical impulses from passing down the axon normally, and is called "diffuse" when the shearing force damages axons in many areas of the brain simultaneously. When DAI involves axonal damage to the frontal lobe, a neuropsychologist may testify that the axonal damage caused disinhibition. Try to rule out diffuse axonal brain injury.

2. Did plaintiff sustain a focal injury to the frontal lobe?

Most allegations of disinhibition involve focal injuries to the frontal lobe and claims of frontal lobe dysfunction. See *Small v. Astrue*, 2009 WL 3029737, * 3 (E.D.N.C. 9/22/09)(frontal lobe dysfunction); *Sanders v. Nike, Inc.*, 2004 WL 5504981, at *5 (D.Vt. 2004)("Agitation and disinhibition are neurobehavioral symptoms of prefrontal lobe damage."). Try to rule out a focal injury to the frontal lobe.

There are asymmetrical differences in the frontal lobes. For example, some experts will claim the left frontal lobe is responsible for controlling language related movement, and the right frontal lobe is responsible for non-verbal abilities. Other experts will claim that both frontal lobes are involved in nearly all behavior. Try to limit the area of damage to one side of the frontal lobe.

3. What area (or functional region) of the frontal lobe was damaged?

The frontal lobes form over half the brain volume. Different areas of the frontal lobe have been associated with different functions. Traditional classification systems divide the frontal lobes into four areas: (1) the precentral cortex (the strip immediately anterior to the central or Sylvian fissure); (2) the prefrontal cortex (extending from the frontal poles to the precentral cortex and including the frontal operculum, dorsolateral, and superior mesial regions); (3) the orbitofrontal cortex (including the orbitobasal or ventromedial and the inferior mesial regions); and (4) superior mesial regions (containing, primarily, the anterior cingulate gyrus). Each of these areas has widespread connectivity, but try to limit the number of areas damaged.

Other classification systems divide the frontal lobes into six functional regions: (I) primary motor area; (II) premotor area; (III) frontal eye fields; (IV) dorsolateral prefrontal cortex; (V) orbital and basal areas; and (VI) supplementary motor area and anterior cingulate gyrus area. Try to limit the functional regions damaged.

4. Which frontal subcortical circuits are affected by the injury?

The frontal lobe cortex forms a part of the frontal-subcortical circuits. There are five parallel, separate frontal-subcortical circuits: (1) a motor circuit originating in the motor cortex and pre-motor cortex; (2) an oculomotor unit originating in the frontal eye fields; (3) the dorsolateral prefrontal circuit, which underpins executive functions; (4) the anterior cingulate circuit which underpins motivation; and (5) the orbitofrontal circuit which underpins impulse control and social behavior. Lesions at various sites may have similar effects, but try to limit the circuits affected.

5. What was the nature (severity) of the frontal lobe injury?

All traumatic brain injuries are not the same. Did plaintiff have edema (swelling), hemorrhage (bleeding), or hematoma (pooling of blood)? Was the bleeding and swelling so severe that it caused the shrinking of the ventricles (mass effect) or the shifting of the brain (midline shift)? Did the TBI cause brain herniation? Rule out the more severe injuries.

6. Did the focal or diffuse injury resolve?

The brain heals. Blood is reabsorbed; swelling resolves. Determine whether subsequent diagnostic images (MRI, CT, etc.) show evidence of permanent brain abnormalities, including atrophy, gliosis, and encephalomalacia. Rule out diagnostic evidence of permanent damage.

Frontal Lobe Functions:

Some researchers have identified four functional divisions within the frontal lobes: (1) executive cognitive function; (2) behavioral/emotional self-regulatory function; (3) action regulation; and (4) meta-cognitive processes. Get the expert to identify and limit the impaired frontal lobe functions.

7. Does plaintiff have impaired executive cognitive function?

Executive cognitive functions are involved in the control and direction (e.g. planning, monitoring, activating, switching, inhibiting) of lower level, more automatic functions. The dorsolateral prefrontal cortex (DLPFC) is associated with executive cognitive functions. See Goldman-Rakic, P.S. (1987) Circuitry of primate prefrontal cortex and regulation of behavior by representational memory, In F. Plum, & V. Mountcastle (Eds.), *Handbook of physiology: The nervous system* (Vol. 5, pp. 373-417), Bethesda, MD: American Physiological Society. The dorsolateral prefrontal subcortical circuit is associated with this executive cognitive function.

8. Does plaintiff have impaired behavioral/emotional self-regulation?

The ventral (medial) prefrontal cortex (VPFC) is associated with emotional processing, including the acquisition and reversal of stimulus-reward associations. The VPFC is also associated with behavioral self-regulation required in situations where cognitive analysis, habit, or environmental cues are not sufficient to determine the most adaptive response. The lateral orbital subcortical circuit is associated with this emotional/behavioral function.

9. Does plaintiff have impaired activation regulation?

Disorders of activation and drive are known as apathy or abulia. Disorders of activation have an important impact on self-regulation. The medial anterior cingulate subcortical circuit is associated with activation regulation.

10. Does plaintiff have impaired meta-cognitive processes?

The frontal polar region is associated with the meta-cognitive aspects of human nature: integrative aspects of personality, social cognition, auto-noetic consciousness, and self-awareness.

Frontal Lobe Dysfunction:

Most neuropsychologists are qualified to offer an opinion on the existence of cognitive or behavioral impairment. Determine which functions associated with the damaged area of the brain are impaired. When there is no objective evidence of any focal injury, try to establish the retained frontal lobe functions.

11. What cognitive and behavioral impairment has plaintiff demonstrated?

It is absolutely critical that you identify and investigate every anecdotal report of cognitive and behavioral impairment. Find out every time that plaintiff lost his temper or did something impulsive.

12. Does plaintiff have normal fine movement & strength in arms & hands?

Disturbance of motor function can be characterized by loss of fine movement and strength in the arms, hands, and fingers. See Kuypers, H. (1981) Anatomy of the descending pathways, In V. Brooks, ed. *The Nervous System, Handbook of Physiology*, vol. 2. Baltimore: Williams and Wilkins.

13. Does plaintiff have any frontal release signs?

Frontal release signs are primitive reflexes which are traditionally held to be a sign of disorders that affect the frontal lobes. These reflexes can be elicited in newborns; but, as the brain matures, certain areas (usually within the frontal lobes) exert an inhibitory effect, causing the reflexes to disappear. When disease processes disrupt these inhibitory pathways, the reflex is "released" from its inhibitory shackles and can again be elicited. Signs include: palmar grasp, palmomental reflex, rooting reflex, sucking reflex, snout reflex, and glabellar reflex.

14. Does plaintiff have impaired spatial orientation?

The frontal lobes have been associated with spatial orientation, including the body's orientation in space. Semmes, J., Weinstein, S., Ghent, L., & Teuber, H, (1963) Impaired orientation in personal and extrapersonal space. *Brain*, 86:747-772.

15. Does plaintiff have normal facial expression?

Patients with frontal lobe damage sometimes exhibit little spontaneous facial expression. See Kolb, B., & Milner, B. (1981) Performance of complex arm and facial movements after focal brain lesions. *Neuropsychologia*, 19:505-514.

16. Does plaintiff have Broca's Aphasia (difficulty in speaking)?

Broca's aphasia or "expressive aphasia" is the loss or impairment of the power to use or comprehend words. The Broca's area is in the lower part of the left frontal lobe, and it reportedly controls the motor aspects of speech. Persons with a Broca aphasia can usually understand what words mean, but have trouble performing the motor or output aspects of speech. Depending on the severity of the lesion to Broca's area, the symptoms can range from the mildest type (cortical dysarthria), which involves intact comprehension and the ability to communicate through writing, to a complete loss of speaking out loud. Broca's aphasia has been associated with frontal lobe damage. See Brown, J. (1972) *Aphasia, Apraxia and Agnosia*, Springfield, IL: Charles C. Thomas.

17. Does plaintiff have apraxia?

Apraxia results from an impaired ability to generate the motor programs for speech movements rather than from the disordered transmission of controlling messages to the speech musculature. Apraxia is a planning/programming problem, not a movement problem like dysarthria. Apraxia occurs following damage to Broca's Area, or Brodmann's area 44, which is located on the third gyrus of the left frontal lobe. Thus, apraxia is always the result of a central nervous system lesion. It is a cortical problem, not a motor impulse transmission problem like dysarthria. Types of errors found in apraxic speech, listed from most to least common, include: Repetitions, Additions, Transpositions, Prolongations, Omissions, and Distortions.

18. Does plaintiff perseverate?

Perseveration is the uncontrolled repetition or continuation of a response, and it has been associated with frontal lobe damage. See Milner, B. (1964), Some effects of frontal lobectomy in man, In Man, J. Warren and K. Akert, eds., *The Frontal Granular Cortex and Behavior*, New York: McGraw-Hill. Perseveration is often evaluated by looking for "errors of perseveration" on the Wisconsin Card Sorting Test. See Milner, B. (1964), Effects of different brain lesions on card sorting, *Archives of Neurology*, 9, 90-100. Older adults, like patients with dorsolateral frontal lobe lesions, have been shown to be progressively susceptible to errors of perseveration on the Wisconsin Card Sorting Test. Ridderinkhof, Richard K., et al, (2002) Perseverative behavior and adaptive control in older adults: performance monitoring, rule induction, and set shifting, *Brain and Cognition*, 49, 382-401.

19. Does plaintiff have impaired attention?

Executive functions also include sustained attention, the inhibition of irrelevant information (distractibility), monitoring of information, and variability in reaction time performance.

20. Does plaintiff have emotional lability?

Emotional lability is a state or condition of excessive emotional reactions and frequent mood changes, and has been associated with frontal lobe injuries. See *In re Succession of Burguières*, 802 So.2d 660 (La.App. 5 Cir. 2000)(“disinhibitions of action and speech and emotional lability, both exhibited by Williams, are symptoms of frontal lobe syndrome.”). It is generally very brief, and can involve uncontrollable crying and inappropriate response (laughing at something sad).

21. Does plaintiff have impaired learning ability?

Frontal lobe damage has been associated with impaired learning ability. Drewe, E. (1975) Go-no-go learning after frontal lobe lesion in humans, *Cortex*, 11:8-16.

22. Does plaintiff have impaired working memory?

Some researchers believe that strategic aspects of encoding and retrieval in certain memory tests, such as word list learning, are examples of executive cognitive functions. There are many different regions of the brain involved in working memory, but the reported role of frontal lobes in working memory is in the manipulation and control of information held on-line. See Baddeley, A. (1986) *Working memory*. Oxford. Clarendon Press. It is interesting to read attempts by experts to explain the association between aggression and loss of memory. See *Liles v. Saffle*, 945 F.2d 333, 338 (CA. 10 (Ok) 1991)(“Because of close proximity of limbic aggressive and memory structures, often the irritative effect which triggers tendencies for explosiveness also impairs memory encoding for the duration of the irritative ictus.”).

Neuropsychological Testing:

Neuropsychologists administer tests to evaluate cognitive and behavioral functioning. Each test is associated with specific cognitive and behavioral functions; each function is associated with a specific area of the brain. Determine how plaintiff performed on those tests commonly associated with the damaged lobe. Establish “normal” performances on tests that evaluate functions associated with the (allegedly) damaged lobe. When there is no objective evidence of any focal injury, try to establish “normal” performances on tests of frontal lobe functions.

23. What cognitive and behavioral functions are associated with the frontal [or the damaged] lobe?

Force plaintiff's expert to identify all cognitive and behavioral functions associated with the damaged lobe. If he leaves any out, then you can use the testimony to impeach.

24. What neuropsychological tests evaluate those cognitive and behavioral functions?

Neuropsychologists can disagree about what functions are associated with specific tests. Do not assume that your expert is correct. Force plaintiff's expert to establish what functions are associated with each test in the battery administered.

25. How did plaintiff perform on the Wisconsin Card Sorting Test?

Frontal lobe damage has been associated with impaired flexibility and problem solving. The Wisconsin Card Sorting Test (WCST) evaluates many different aspects of executive functioning, including performance monitoring, integration of feedback, rule-induction, set-shifting, and suppression of previous sorting rules. Odessa, TX: Psychological Assessment Resources. 1985. When a plaintiff performs well on the WCST, it can be difficult for an expert to explain how a frontal lobe injury was "not severe enough" to impair executive functions, but was "severe enough" to cause disinhibition. Remember that improved performance on a subsequent Wisconsin Card Sorting Test can be the result of practice effect, and be certain to discuss the timing of a subsequent testing with the neuropsychologist.

26. How did plaintiff perform on the Trail Making Test?

The Trail-making test evaluates visual attention and task switching. The task requires a plaintiff to connect-the-dots of 25 consecutive targets on a sheet of paper or computer screen. Two versions are given: A, in which the targets are all numbers (1,2,3, etc.), and B, in which the subject alternates between numbers and letters (1, A, 2, B, etc.). The goal is to finish the test as quickly as possible, and the time taken to complete the test is used as the primary performance metric. The test was initially used for assessing general intelligence (and was part of the Army Individual Test of General Ability), but it has become a common diagnostic tool

because poor performance has been associated with many types of brain impairment, including frontal lobe lesions.

27. How did plaintiff perform on the Stroop Test?

The Stroop effect is a demonstration of the reaction time of a task. When the name of a color (e.g., "blue," "green," or "red") is printed in a color not denoted by the name (e.g., the word "red" printed in blue ink instead of red ink), naming the color of the word takes longer and is more prone to errors than when the color of the ink matches the name of the color. Patients have to control or inhibit themselves from reading the word instead of saying the color. There are different variations of the Stroop Test, and the differences are in the number of subtasks, type and number of stimulus, times for the task, or scoring procedures. EEG and functional neuroimaging studies of the Stroop effect have revealed activation in the frontal lobe, particularly in the cingulate cortex and dorsolateral prefrontal cortex (which have been associated with conflict monitoring and resolution).

28. How did plaintiff perform on word list learning tasks?

Word list learning tasks such as the California Verbal Learning Test (CVLT) can yield measures of basic associative and strategic processes.

29. What was plaintiff's "working memory" composite score?

Wechsler Instruments have added new tasks stressing manipulation and control, and even allow for a separate "working memory" composite score.

30. How did plaintiff perform on the Controlled Oral Word Association Test (COWAT)?

The COWAT requires the plaintiff to produce as many words as possible, in one minute, starting with F, then A, then S. Proper nouns and previously used words with a different suffix are prohibited. See Benton, A. (1968) Differential behavior effects in frontal lobe disease. *Neuropsychologia*. 6:53-60. COWAT evaluates fluency & word retrieval.

31. How did plaintiff perform on other categorical fluency tests?

Other categorical fluency tests include naming animals, fruits and vegetables. See Monsch A, Bondi M, Butters N. (1992) Comparisons of verbal fluency tasks in detection of dementia of the Alzheimer type. *Archives of Neurology* 49:1253-1258. Don't just check the number of items named. Determine whether plaintiff perseverated or uttered any inappropriate or profane responses (disinhibition) during the test.

32. How did plaintiff perform on the Digit Span Test?

Like requiring a plaintiff to recite the days of the week or months of the year backwards, the digit span test requires a plaintiff to retain the task and the information, and then manipulate the information.

33. How did plaintiff perform on the Finger Tapping Test?

The frontal lobe has been associated with motor function, and motor speed as in finger tapping has been listed as a useful test of the primary motor cortex. See Malloy P, Richardson E. (1994) Assessment of frontal lobe functions, *Journal of Neuropsychiatry and Clinical Neurosciences*; 6:358-410. Sensorimotor abilities are tested by asking the patient to touch each finger to the thumb in succession as rapidly as possible.

34. How did plaintiff perform on Continuous Performance Test (CPT)?

The Continuance Performance Test measures vigilance and attention. The right frontal region is important for performance on the CPT, especially when the target complexity is increased.

35. How did the plaintiff perform on the Sustained Attention To Response Test and the Elevator Counting Test?

The Sustained Attention To Response Test and the Elevator Counting Test are tests of sustained attention abilities.

36. How did plaintiff perform on the Go/No-Go Test?

Plaintiff is asked to make a response to one signal (the “Go” signal) and not to respond to another (the “No-Go” signal). The task can be made more demanding by reversing the customary meaning of signals. For example, the plaintiff can be asked to “tap” the knee when the examiner says “stop”, and to “not tap” the knee when the examiner says “go.” See Mallory P, Richardson E. (1994), Assessment of frontal lobe functions, *Journal of Neuropsychiatry and Clinical Neurosciences*; 6: 399-410.

37. How did plaintiff perform on the Minnesota Impulsive Disorders Interview?

The Minnesota Impulsive Disorders Interview is a semistructured clinical interview assessing pathological gambling, trichotillomania, kleptomania, pyromania, intermittent explosive disorder, compulsive buying, and compulsive sexual behavior.

38. How did plaintiff perform on the Behavioral Dyscontrol Scale?

The Behavioral Dyscontrol Scale based on Luria's theory includes tasks assessing both executive cognitive functions and self-regulatory behaviors.

39. How did plaintiff perform on the Zuckerman Sensation-Seeking Scale?

Zuckerman (1994) defined sensation seeking as "...a trait defined by the seeking of varied, novel, complex, and intense sensations and experiences and the willingness to take physical, social, legal, and financial risks for the sake of such experiences." The Sensation Seeking Scale (SSS) is a written questionnaire that has evolved into a multi-dimensional measure, consisting of four interrelated subscales. The subscales, each comprised of ten forced-choice items, include boredom susceptibility, disinhibition, experience seeking, and thrill and adventure seeking. Many studies have linked sensation seeking with risky behavior, including risky driving. You can actually take the test at: <http://www.bbc.co.uk/science/humanbody/mind/surveys/sensation>.

40. How did plaintiff perform on the Barratt Impulsivity Rating Scales?

The Barratt Impulsiveness Scale is a self-report measure of impulsive personality traits. The BIS-11 includes 30 items which may be scored to yield six first-order factors (attention, motor, self-control, cognitive complexity, perseverance, and cognitive instability impulsiveness) and three second-order factors (attentional, motor, and non-planning impulsiveness).

41. How did plaintiff perform on other measures of general affect and negative affect?

Measures of General Affect and Negative Affect include: the PANAS-X Scales, the State-Trait Emotion Measure (STEM), State-Trait Anger Expression Inventory (STAXI), the Anger Rumination Scale (ARS), the State-Trait Anger Scale (STAS), and the Perceived Emotional Appropriateness Rating Scale (PEARS).

42. How did the neuropsychologist determine plaintiff's pre-morbid ability (and expected performance) for each test?

In determining pre-morbid (pre-accident) functioning, neuropsychologists may: (1) employ regression equations; (2) rely on pre-morbid academic records, standardized testing, and employment history; (3) rely on plaintiff's performance on specific neuropsychological tests which should

not have been affected by the injury; and/or (4) rely on clinical interviews of plaintiff, family, and friends (anecdotes). Determine what plaintiff's neuropsychologist relied upon in determining pre-morbid functioning.

43. Was plaintiff's performance consistent with pre-morbid functioning?

Before concluding that an accident caused impairment, a neuropsychologist should first determine the plaintiff's pre-morbid (pre-accident) functioning. Determine whether the neuropsychologist made any effort to determine pre-morbid functioning. Try to establish "expected" performance for each test.

Daily Functioning:

Actions can speak louder than neuropsychological testing. Meet with your neuropsychologist, and find out what daily activities or functions are associated with the (allegedly) damaged lobe, and ask those questions. When there is no objective evidence of any focal injury, try to establish the retained frontal lobe daily functions.

44. Does plaintiff manage his or her own finances?

See Small v. Astrue, 2009 WL 3029737, * 3 (E.D.N.C. 9/22/09) ("In April 2005, Dr. Jones reported that Claimant's frontal lobe dysfunction interfered 'with almost all of his social interactions, ability to manage his finances, and other deficits in terms of disinhibition as well as planning and organizing' and that Claimant's behavioral changes (anger, impulsivity and poor social skills) will make it dangerous to be employed in any job in which he would have to interact with people.").

45. Did plaintiff make an appointment, arrive on time, & understand the reason for the evaluation?

The frontal lobe has been associated with planning and organizing. The plaintiff's ability to make an appointment and arrive on time could be valuable information. Also look at the clinical interview to determine if plaintiff was able to give a comprehensive account of their medical history and understood the reason for the consultation.

46. Has plaintiff experienced a change in his ability to smell?

Anosmia (or hyposmia) is a lack of functioning olfaction or the inability to perceive odors); hyperosmia refers to an increased ability to smell. Anosmia has been associated with the inferior frontal lobes. Yousem DM, et al (1996), Posttraumatic olfactory dysfunction: MR and clinical evaluation, *American Journal of Neuroradiology*, Jun-Jul; 17(6):1171-9.

47. Has plaintiff experienced changes in sexual behavior?

Frontal lobe damage has also been associated with changes in sexual behavior. Some experts believe that orbital frontal damage can cause abnormal sexual behavior, while dorolateral lesions may reduce sexual interest. See Walker, E., & Blumer, D., The localization of sex in the brain, In K.J. Zulch, O. Creutzfeldt, and G. Galbraith, eds., *Cerebral Localization*, Berlin and New York: Springer-Verlag, 1975.

48. Has plaintiff experienced outbursts of aggression or rage?

See *Stokley v. Ryan*, 2009 WL 728492 at *23 (D. Ariz. 2009) (“According to Dr. McKinley, ‘frontal lobe brain deficits, such as those evidence in Mr. Stokley, are and have long been associated with impulsivity, impaired judgment, disinhibition, and sometimes outbursts of aggression or rage growing out of proportion to any precipitating psycho-social stressor.”); See also *State v. Rhomberg*, 516 N.W.2d 803, 804 (Iowa 1994) (“The defense was based on expert testimony that he suffered from frontal lobe brain damage, the symptoms of which is a disinhibition of aggression or rage, along with organic mood disorder.”).

49. Has plaintiff been diagnosed with pseudodepression?

Pseudodepression has been described as a “condition of personality following frontal lobe lesion in which apathy, indifference and a loss of initiative are apparent symptoms but are not accompanied by a sense of depression in the patient.” Researchers have associated Left frontal damage with pseudodepression. See Blumer, D., & Benson, D. (1975), Personality changes with frontal and temporal lobe lesions, In D. Benson and D. Blumer, eds. *Psychiatric Aspects of Neurologic Disease*. New York: Grune & Stratton.

50. Has plaintiff been diagnosed as pseudopsychopathic?

Right frontal damage has been associated with pseudopsychopathic. See Blumer, D., & Benson, D. Personality changes with frontal and temporal lobe lesions. In D. Benson and D. Blumer, eds. *Psychiatric Aspects of Neurologic Disease*. New York: Grune & Stratton, 1975.

Underlying Mental Illness, Defect Or Disorder:

Irresistible Impulses and disinhibition are symptoms. It is easy for an expert to declare that a plaintiff suffers from disinhibition or “must have” experienced an irresistible impulse. Force the expert to identify the specific underlying illness or

disorder which *caused* the disinhibition or irresistible impulse; and rule out as many mental illnesses and disorders as possible.

51. Has plaintiff been diagnosed with mania (or had manic episodes)?

Irresistible Impulses and disinhibition have both been associated with mania and manic episodes. See *O'Neill v. Astrue*, 762 F.Supp.2d 1158,1161 (D.Minn 2011)(“Plaintiff’s manic episodes were typically three days of disinhibition and alcohol craving.”).

52. Has plaintiff been diagnosed with bipolar disorder?

“Patients who have bipolar disorder or manic [sic] may engage in increase sexual activity, may engage in increase use of alcohol and substances. They may engage in gambling activity. They may do that on an impulsive whim. They decide one minute they’re fine, the next minute they want to do something impulsive.” Deposition of psychiatrist, Dr. James W. Thompson, M.D., April 9, 2008, p. 32, lines 2-7.

53. Has plaintiff been diagnosed with dementia?

Dementia has been associated with disinhibition. See *U.S. v. Oestrike*, 2010 WL 3937316, * 2 (E.D. Mich. 10/5/2010)(“... the panel members were in agreement that the Defendant’s dementia caused the behavioral disinhibition which led to the events giving rise to the criminal charges.”).

Frontal lobe dementia has specifically been blamed for disinhibition. See *Kirk v. Woodouse*, 2003 WL 22133847, at *4 (Conn.Super. 2003)(“She was diagnosed as having significant frontal lobe dementia which included perseveration, disinhibition, intrusions, imitation behavior, and utilization behavior.”).

54. Has plaintiff been diagnosed with organic personality disorder or intermittent explosive disorder?

Carpenter v. Johnson, 664 So.2d 1354 (La.App. 1 Cir. 1995)(frontal lobe injury can lead to personality disorder which leads to disinhibition).

55. Does plaintiff have disinhibition syndrome?

Disinhibition Syndrome has been defined as “the loss of ability to discern what is appropriate versus inappropriate behavior in various situations.” *U.S. v. Rothman*, 2009 WL 426282 (S.D.Fla. 2009). Disinhibition syndrome is a sign of frontal lobe impairment. *U.S. v. Rothman*, 2009 WL 426282 (S.D.Fla. 2009)(“Dr. Eisenstein also testified that one of the signs of frontal lobe impairment is the existence of disinhibition syndrome.”)

56. Has plaintiff been diagnosed with Post-Traumatic Stress Disorder?

Haste ex rel. P.M.B. v. Astrue, 2007 WL 3124477, at *2 (S.D. Ind. 2007)(“He suggested the use of Zoloft and noted: ‘It is not uncommon for children to have poor impulse control or even disinhibition following a head injury, particularly if there is a frontal lobe injury.’”); *In re Little*, 2008 WL 142832 (Ca.App. 4 Dist. 2008)(where Abram explains: “There are numerous studies showing that patients with PTSD are more impulsive and prone to violence because of their hypervigilance.”); *People v. Kubrak*, 2001 WL 1221672 (N.Y.Sup. 2001)(“The newest neurological research indicates that people with PTSD don’t use [the] orbitofrontal part of the brain which evaluates primal feelings of fear and modulates behavior.”).

57. Has plaintiff been diagnosed with impulse control disorder?

Impulse control disorders (ICDs) are characterized by urges and behaviors that are excessive or harmful (to oneself or others), and cause significant impairment in social and occupational functioning, as well as legal and financial difficulties. ICDs are relatively common psychiatric conditions. They include: trichotillomania (twisting or pulling hair until it comes off), nail biting, skin picking, kleptomania, pathological gambling, compulsive buying, and compulsive sexual behavior. See Odlaug, BL, Grant JE, (2010) Impulse-control disorders in a college sample: results from self-administered Minnesota Impulse Disorders Interview (MIDI), *Prim Care Companion J Clin Psychiatry*, 12(2). pii:PCC.09m00842. The most common impulse control disorders among psychiatric patients are compulsive buying (9.3%), kleptomania (7.8%), and pathological gambling (6.9%). Grant, JE, Levine L, Kim D, Potenza, MN, (2005) Impulse control disorders in adult psychiatric inpatients, *Am J Psychiatry*, Nov; 162 (11):2184-8.

58. Has plaintiff been diagnosed with any mental illness or disorder?

Don’t forget to ask the open-ended question. See *Pole v. Cole*, 2009 WL 2916426, * 1 (Cal.App.2d Dist., 9/14/09)(“Doctor Askin testified that it was ‘probably safe to assume’ that the mental disorder was an aggravating factor because the mental disorder created disinhibition and lowered appellant’s threshold to commit the offenses.”); see also *Motzkin v. Trustees of Boston University*, 938 F.Supp. 983 (D.Mass 1996)(“To the contrary, he insists that because of a psychological disorder from which he suffers that causes disinhibition...”).

Make certain to specifically ask about development disorders. *See Hough v. Shakopee Public Schools*, 608 F.Supp2d 1087 (D.Minn. 2009)(“Emily exhibited ‘difficulty sustaining attention, disinhibition, impulsivity and mood instability... The evaluator diagnosed her as having pervasive development disorder.”).

59. What underlying disorder caused plaintiff’s disinhibition or the irresistible impulse?

Courts have excluded testimony of irresistible impulses when the disorder identified by the expert was untested, unpublished, had no known rate of error, and/or was not generally accepted in the scientific community. *State v. George*, 768 So.2d 748 (La.App. 2 Cir. 2000)(discussing admissibility of testimony regarding Limbic Trigger Reaction); *State v. George*, 92 Wash. App. 80, 960 P.2d 980, 990 (Wash.App. Div. 1, 1998)(“In light of the scientific evidence and the manifestation of the State’s antipodal position, we hold, as a matter of law, that DID meets the Frye standard, but leave to the discretion of a trial court under ER 702 whether expert testimony concerning DID is admissible in a particular case.”); *Daniels v. Henry*, 2007 WL 424441 (N.D. Cal. 2007)(discussing admissibility of BWS for certain purposes), *aff’d* 281 Fed.Appx. 663 (9th Cir. 2008). Determine the legitimacy of the underlying disorder.

60. Did plaintiff have a “severe mental disease or defect”?

“Congress appears to have added the ‘severe mental disease requirement ‘to emphasize that non-psychotic behavior disorders or neuroses such as ‘inadequate personality,’ ‘immature personality,’ or a patter of ‘antisocial tendencies’ do not constitute the defense.’” *U.S. v. Dixon*, 185 F.3d 393, 399 (5th Cir. 8/16/99), *citing* S.Rep. 98-225, *reprinted in* 1984 U.S.C.C.A.N. 3182, 3411.

Drugs, Alcohol & Medication:

Drugs, alcohol, and certain medications can cause disinhibition. Determine whether these are confounding variables in determining causation.

61. Does plaintiff have alcohol-related dementia?

See Landsberg v. Maine Coast Regional Health Facilities, 2009 WL 995177 (D.Me. 2009)(“... there might be ‘dysfunction of the frontal lobes of the brain as a possible cause of disinhibition;’ or that there may be alcohol-related dementia.”).

62. Did plaintiff have alcohol in his system?

Alcohol has been associated with behavioral disinhibition. See *In re Schulz*, 2011 WL 3812563, * 7 (Cal.App. 4 Dist., 2011)(Dr. Robinson testified that Schultz's alcohol and drugs contributed to "behavioral disinhibition and resulted in violent behavior."); See also *Com. V. Gibson*, 19 A.3d 512, 520 (Pa. 5/12/11)("Dr. O'Brien thought that such alcohol-induced disinhibition was a factor in Appellee's actions."); *Mayo v. Henson*, 957 So.2d 318 (La.App. 2 Cir. 2007)("her drinking was inappropriate and led to 'significant disinhibition.'").

63. Did plaintiff have any drugs (controlled dangerous substances) in his system?

See *People v. Stebler*, 2009 WL 522253, at *5 (Cal.App. 2 Dist. 2009)("PCP is a central nervous system depressant and, like alcohol, it causes disinhibition."); *People v. Sandner*, 2007 WL2379612, at *4 (Cal.App. 5 Dist. 2007)(Dr. Weiss opined, 'I don't believe that [appellant] was in total control of the faculties on the date of the incident... I also think that he was experiencing the effects of methamphetamine, some disinhibition, some inability to think clearly as when he would be stable on medications and not taking drugs.').

64. Did alcohol or drugs cause or contribute to plaintiff's disinhibition?

See *In re Commitment of J.N.*, 2008 WL 2511285, at *5 (N.J. Super. A.D. 2008)("Dr. Greenfield opined that J.N. has a good understanding of his deviant cycle, which is significantly tied to the disinhibition of alcohol."); *Stokley v. Ryan*, 2009 WL 728492 (D.Ariz. 2009)("Further, alcohol's disinhibition of brain function would have a cumulative effect on behavior, so that it would take less alcohol to achieve loss of control or emotions in an individual with brain damage and would exacerbate difficulty with cognitive ability."); *Newland v. Hall*, 527 F.3d 1162, 1192 ("Dr. Hyde's affidavit... provides a specific neurological diagnosis, stating that petitioner suffers from 'bilateral frontal lobe dysfunction,' which results in "impaired executive functions, decreased disinhibition, and tendency toward impulsivity,' and that this condition was amplified by excessive alcohol intake."); *State v. Spruill*, 452 S.E.2d 279, 298 (N.C. 1994)("there are interactive effects among them which result in an exacerbation of cognitive intellectual and behavioral deficits. Such as, lower judgment, decreased ability to evaluate, problem solve and consider consequences and heightened disinhibition.").

65. What are the side effects of plaintiff's medications?

Disinhibition and impulsivity have been listed as side effects for certain drugs. Since 1969, courts have heard evidence or argument regarding the ability of various drugs (often in combination with alcohol) to cause “disinhibition.” See *Ranteesi v. Grounds*, 2011 WL 1601575, *2 (E.D. Cal. 4/27/11)(Paxil); *Crumly v. Astrue*, 2010 WL 3023349, *2 (D.Ariz. 7/15/10)(Butalbital); See *Newell v. State*, 49 So.3d 66, fn. 8 (Miss. 12/2/10)(“So he opined that Dr. Hayne would agree that Boyette’s [alprazolam] level of 0.06 was three times the therapeutic level, which could cause disinhibition and aggressive behavior.”); *Venable v. T-Mobile USA, Inc.*, 603 F.Supp.2d (D.Me 2009)(Topomax); *Com v. Atchue*, 2007 WL 1977749, at *10, 22 Mass.L.Rptr. 590 (Benzodiazepines); *Ex parte Martinez*, 195 S.W.3d. 713, 723 (Tex.Crim.App. 2006)(Rohypnol); *Gaines v. State*, 2004 WL 2320367, at *2 (Tex.App. – Fort Worth 2004)(Paxil); *Wiegert v. Goldberg*, 269 Wis.2d 695, 697, 676 N.W.2d 522, 523 (Wis.App. 2004)(Temazepam); *State v. Rutter*, 2002 WL 713246, at *5 (Mo.App. S.D. 2002)(Butalbital); *Miller v. Pfizer Inc.*, 196 F.Supp.2d 1095, 1118 (D.Kan 2002)(Zoloft); *Mulkern v. Cumberland County*, 2001 WL 1519409 (D.Me. 2001)(Klonopin & Xanax); *Cloud v. Pfizer, Inc.*, 198 F.Supp2d 1118, 11124 (D.Ariz 2001)(SSRIs); *State v. Dellinger*, 2001 WL 220186, *10 (Tenn.Crim.App. 2001)(Xanax & Sinequan); *State v. Deangelo*, 2000 WL 264303 (Comm.Super. 2000)(Xanax & Prozac); *Adams v. Rios*, 1996 WL 337108, *1 (Tex.App. – Hous. (14 Dist) 1996)(Halcon); *People v. Duvall*, 9 Cal. 464, 472, 886 P.2d 1252 (Cal. 1995)(Carisoprodol); *Clement v. U.S.*, 980 F.2d 48 (C.A.1 (Me) 1992)(Darvon & Serax); *People v. Hackett*, 237 Cal.Rptr. 281 (Cal.App. 4 Dist. 1990)(Benzodiazepines).

Cognitive Functioning:

Irresistible impulses and disinhibition may be considered volitional issues, but plaintiff’s cognitive ability will always have a direct effect on the determination of those issues. Remember that the insanity defense in some states involves a “cognitive prong” and a “volitional prong.” Establish cognitive functioning and you may succeed in establishing accountability.

66. What is plaintiff’s intelligence quotient (I.Q.)?

Jurors will hold plaintiffs with high I.Q. scores more responsible for their choices and actions, and it should be treated as a factor in determining accountability, but please note that damage to the frontal lobe does not usually affect intelligence (or performance on tests of general intelligence).

67. Was plaintiff conscious when he acted?

Baby steps. Establish that plaintiff was conscious. *See People v. Carr*, 2011 WL 3484947, * 4-5 (Cal.App. 2 Dist., 8/10/11)(“We see no substantial evidence showing that Carr was ‘unconscious’ at the time of the crimes on October 10, 2005.”).

68. Does plaintiff recall his behavior?

Establish that Plaintiff has not been diagnosed with Post-Traumatic amnesia. *See Amaya v. State*, 2011 WL 1529732 (Tex.App. – Houston [1 Dist.], 2011)(“Dr. Pollack testified that Post-Traumatic Amnesia “disrupts rational, goal-directed behavior and causes disinhibition of neurological regulation of behavior.”).

Establish every fact that Plaintiff remembers about incident. *See People v. Carr*, 2011 WL 3484947, * 4-5 (Cal.App. 2 Dist., 8/10/11)(“While Carr testified that he had ‘blacked out’ after the accident and that he could not, at the time of trial, remember events, his remaining testimony showed a momentary blackout only and his pretrial statements to police directly contradicted his testimony in court in that his pretrial statements included a detailed account of the events in October 2005.”); *People v. Halvorsen*, 42 Cal.4th 379, 418 (2007)(“where Supreme Court noted that defendant had a sharp, detailed memory regarding the shootings he committed).

This is a set-up qEstablish Plaintiff has not been diagnosed with Post-Traumatic amnesia. *See Amaya v. State*, 2011 WL 1529732 (Tex.App. – Houston [1 Dist.], 2011)(“Dr. Pollack testified that Post-Traumatic Amnesia “disrupts rational, goal-directed behavior and causes disinhibition of neurological regulation of behavior.”).

69. Was plaintiff totally deprived of his understanding?

In the seventeenth century, Sir Edward Coke did not acknowledge the possibility of irresistible impulses when he outlined the four classes of *non compos mentis*. Concurring with his assessment, Mathew Hale proposed that, as a rule, “where there is a total defect of the understanding there is not free act of the will in the choice of things or actions.” This philosophy was reflected in the 1724 case of *R. v. Arnold* where Judge Tracy insisted with regard to the insanity defense that “it must be a man that is totally deprived of his understanding and memory, and doth not know what he is doing, no more than an infant, than a brute, or a wild beast.”

70. Did plaintiff have a defect of reason?

The 1843 acquittal of Daniel M’Naghten on the ground of insanity prompted the House of Lords to announce the following test: “[t]o establish a defense on the ground of insanity, it must be clearly proved that, at the time of the committing the act, the accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing, or, if he did know it, that he did not know he was doing what was wrong.” See *People v. Schmidt*, 216 N.Y. 324, 110 N.E. 945 (1915), quoting *McNaughton’s Case*, 10 Cl. & F. 200, at 210.

71. Did plaintiff understand the physical act and its consequences (the nature and quality of the act)?

The M’Naghten requirement that an accused have knowledge of the “nature and quality of the act” has been interpreted as a “reference to its physical nature and quality.” See *People v. Schmidt*, 216 N.Y. 324, 110 N.E. 945 (1915) (“There the trial judge (Nott, J.) in a careful and able charge told the jury that the knowledge of the nature and quality of the act has reference to its physical nature and quality, and that knowledge that it is wrong refers to its moral side...”), citing *People v. Purcell*, 214 N.Y. 693, 109 N.E. 1087. Accordingly, establish that the plaintiff understood he was “holding a gun and not a guava melon,” and establish that plaintiff understood the physical consequences of pulling the trigger.

The insanity defense is currently based on 18 U.S.C. § 17, which provides: It is an affirmative defense to a prosecution under any federal statute that, at the time of the commission of the acts constituting the offense, the defendant, as a result of a severe mental disease or defect, was unable to appreciate the nature and quality or the wrongfulness of his acts. Mental disease or defect does not otherwise constitute a defense.”

72. Did plaintiff know that the act was against the law (legally wrong)?

The McNaughton requirement that an accused “know he was doing what was wrong” was discussed by the judges. The judges noted: “If the accused was conscious that the act was one that he ought not to do, and if that act was at the same time contrary to the law of the land, he is punishable...” In Irresistible impulse cases, this question is often asked in terms of the accused’s knowledge of consequences. *State v. Phillis*, 2008 WL 5274861, at *3 (Ohio App. 4 Dist. 2008) (“Although competent to stand trial, a psychologist found that appellant suffered from ‘cognitive impairment’ and ‘disinhibition’ which can cause appellant ‘to behave inappropriately despite knowledge of the consequences.’”).

73. Did plaintiff believe the act was morally wrong?

The M’Naghten requirement that an accused “know he was doing what was wrong” has inspired great debate. That requirement has been interpreted as also requiring an accused to know whether the act is morally wrong. See *People v. Schmidt*, 216 N.Y. 324, 110 N.E. 945 (1915)(“When it is said that a prisoner must, at the time of the alleged criminal act, have sufficient capacity to distinguish between right and wrong with respect to such act, it is implied that he must have sufficient capacity to know whether such act is in violation of the law of God, or of the land, or of both.”)(quoting *Moett v. People*, 85 N.Y. 373, at 380).

74. Could plaintiff distinguish between right and wrong (good and evil)?

The first departure from the wild beast test came in 1812 when the “capacity to distinguish between right and wrong” was put forward as another test. See *People v. Schmidt*, 216 N.Y. 324, 110 N.E. 945 (1915)(citing *Parke’s Case*, *Collinson on Lunacy*, p. 477; *Broler’s Case*, *Id.*, p. 673; *Bellingham’s Case*, *Id.*, p. 636).

75. Could plaintiff recognize reality?

The U.S. Supreme Court has described North Dakota’s insanity defense as “unique” and summarized that test in *See Clark v. Arizona*, 548 U.S. 735, fn. 12, 126 S.Ct. 2209 (2006). That test would involve the question: “was plaintiff’s conduct the result of a loss or serious distortion of the individual’s capacity to recognize reality?” Rule out any reports of hallucinations or delusions.

76. Did plaintiff try to hide evidence of his action (liability or guilt)?

Plaintiff’s conduct after an act can establish knowledge that the act was wrong and the level of cognitive functioning. See *People v. Carr*, 2011 WL 3484947, * 4-5 (Cal.App. 2 Dist., 8/10/11)(“He then knew enough to know how to navigate to his girlfriend’s house, and he knew enough about what had happened to try destroying the evidence – he tried to wipe blood out of Punch’s Mazda.”).

77. Did plaintiff feel guilt after committing the act?

Nobody feels guilty about the act of kicking when that kick is elicited by a doctor tapping below the knee because the patellar reflex is responsible for the kick. Determine whether plaintiff felt guilty about the behavior.

78. Did plaintiff’s plead guilty?

If there was a plea agreement, then obtain a certified copy of that plea agreement and a transcript of the plea in open court to determine what plaintiff admitted. *Essex Ins. Co. v. 7455, Inc.*, 2008 WL 163627, at *2 (D.Or., 2008) (“Thus, the effect of Gee’s plea is an admission that she knowingly attempted – i.e., intentionally engaged in conduct constituting a substantial step toward intentionally or knowingly causing serious physical injury to Montgomery an admission that directly contradicts the notion of ‘irresistible impulse.’”).

79. Should a person be held responsible when they know they are doing wrong?

Force the expert to give an opinion on whether he agrees with the M’Naghten insanity test. *See People v. Hubert* (1897) 119 Cal. 216, 223, 51 P. 329 (“It must be held that, conceding that the act was the offspring of an irresistible impulse, and the impulse was irresistible because of mental disease, still the defendant must be held responsible if he at the time had the requisite knowledge as to the nature and quality of the act, and of its wrongfulness.”).

Scope & Timing Of Expert’s Opinion:

There is a difference between an expert testifying that a plaintiff had a mental illness or frontal lobe dysfunction, and an expert speculating that a plaintiff did not want to throw the punch but lacked self-control (because of the frontal lobe damage) or experienced an irresistible impulse.

80. Is the expert qualified to testify as to whether the plaintiff was suffering from a severe mental illness?

In criminal cases, Federal Rule 704(b) allows experts to testify as to the existence of severe mental illness. *See U.S. v. Dixon*, 185 F.3d 393, 400 (5th Cir. 8/16/99) (“In other words, the ‘mental state or condition’ that constitutes an element of the defense is the inability to appreciate wrongdoing. The ‘severe mental disease’ requirement is subordinate to this overall element and should not be considered a subject prohibited by rule 704(b). An expert is therefore free to testify as to whether the defendant was suffering from a severe mental illness at the time of the criminal conduct; he is prohibited; however, from testifying that this severe mental illness does or does not prevent the defendant from appreciating the wrongfulness of his actions.”).

81. Should the expert be allowed to testify that the severe mental illness prevented the defendant from resisting an impulse?

In criminal cases, Federal Rule 704(b) does not allow an expert witness to give an opinion as to the defendant's ability to appreciate the wrongfulness of his actions. Rule 704(b) provides: "No expert witness testifying with respect to the mental state or condition of a defendant in a criminal case may state an opinion or inference as to whether the defendant did or did not have the mental state or condition constituting an element of the crime charged or of a defense thereto. Such ultimate issues are matters for the trier of fact alone." Similarly, we should challenge under *Daubert* and *Frye* an expert's methodology in determining that an illness or underlying disorder prevented defendant from resisting an impulse (i.e., the plaintiff experienced an "irresistible impulse").

82. When did the expert render a psychiatric diagnosis?

"After-the-fact psychiatric diagnoses are notoriously unreliable." *Vincent v. Heckler*, 739 F.2d 1393, 1395 (9th Cir. 1994), citing *Schauer v. Schweiker*, 675 F.2d 55, 60 n. 5 (2nd Cir. 1982).

Volitional Capacity:

It is easy for an expert to declare that an impulse was irresistible. Force an expert to give an opinion regarding every possible legal articulation of the irresistible impulse test or volitional capacity defense. After obtaining those opinions, go through the standard *Daubert* and *Frye* questions regarding the expert's methodology in reaching those opinions.

83. When did plaintiff first experience the impulse?

How can any expert know the exact moment when a plaintiff first experienced an impulse? If the expert claims that the impulse was irresistible, then plaintiff will have to assume that the impulse was first experienced immediately before the plaintiff's action.

84. Did plaintiff resist the impulse for any period of time?

If a plaintiff can resist an impulse for any period of time, then you will be able to argue that it was not an irresistible impulse and the plaintiff possessed self-control for a period of time. This is the basis for the Policeman At The Elbow classic test for irresistible impulses.

85. Did plaintiff's conduct involve planning and organization (premeditated)?

There is a difference between alleging that plaintiff engaged in a single (“impulsive”) act, and alleging plaintiff (“impulsively”) engaged in hours or days of acts to successfully achieve a desired result. Different states define “premeditated” differently. The question is whether plaintiff’s conduct was characterized by deliberate purpose, previous consideration, some degree of forethought and planning. The amount of time needed for premeditation regarding an act depends on the person and the circumstances. See *People v. Halvorsen*, 42 Cal.4th 379, 416-418 (2007)(where trial court was found to have properly refused unconsciousness instructions where defendant acted in a “complicated and purposive nature,” including driving from one place to another in order to commit violent crimes).

86. Was the (disinhibited) behavior self-defeating?

Jurors are more likely to believe that a plaintiff lacked self-control if the behavior was self-defeating. See *Benyamini v. Schwartz*, 2008 WL 5069917 at *8 (E.D. Cal 2008)(“...self-defeating because they were consistently responded to with physical restraint and restriction of privilege.”).

87. Was the (disinhibited) behavior self-endangering?

Jurors are more likely to believe that a plaintiff lacked self-control if the behavior endangered the plaintiff’s life. See *Benyamini v. Schwartz*, 2008 WL 5069917 at *8 (E.D. Cal 2008)(“...self-endangering when directed, as they often were, against other inmates who were bigger and stronger...”).

88. Was plaintiff able to conform his conduct to the requirements of law?

“A person is not responsible for criminal conduct if at the time of such conduct as a result of mental disease or defect he lacks substantial capacity either to appreciate the criminality of his conduct or to conform his conduct to the requirements of law.” See American Law Institute, Model Penal Code §4.01(1)(p.66)(Proposed Official Draft 1962):

89. Did plaintiff physically lose control over his extremities?

It is easier for a juror to picture an impulsive or involuntary comment than an impulsive or involuntary punch. Tourette syndrome is a disorder of the nervous system characterized by a variable expression of unwanted movements and noises. Force the expert to say that plaintiff’s actions were as involuntary as a Tourette syndrome tic, or the patellar tendon reflex (which doctors test when they tap below knee cap).

90. Did plaintiff lose all ability to control his conduct?

See Reid v. True, 349 F.3d 788, 803 (C.A. 4 (Va.) 2003)(“Likewise, neither doctor concluded that Reid had totally lost the ability to control his actions, as required by the irresistible impulse test. They merely opined that his ability to do so was impaired.”); *U.S. v. Rothman*, 2009 WL 426282 (S.D.Fla. 2009)(“Dr. Eisenstein acknowledge, however, that the manifestations he saw of the disinhibition syndrome would not change his opinion that Dr. Rothman was capable of manifesting appropriate courtroom behavior.”).

91. Would plaintiff have engaged in the conduct if a policeman was present?

Cecil v. Com., 888 S.W.2d 669, 674 (Ky 1994)(“When asked, Dr. Noonan stated that, in his opinion, Ms. Cecil would not have shot the victim if a police officer had been standing at her elbow (a classic test for the ‘irresistible impulse’ or temporary insanity claim). In our opinion, this was proper cross-examination.”), *citing* Kentucky Rule of Evidence 702

92. Was plaintiff’s (involuntary) conduct a product of a mental defect, disease, or illness?

According to the U.S. Supreme Court, the State of New Hampshire is the only state to adopt the “Product-Of-Mental Illness Test.” *See Clark v. Arizona*, 548 U.S. 735, 752 fn. 12, 126 S.Ct. 2209, 2722 (2006). Make certain to ask if the involuntary conduct was a product of a mental disease or defect. *See Clark v. Arizona*, 548 U.S. 735, fn. 12, 126 S.Ct. 2209 (2006)(discussing the “volitional capacity” or “irresistible Impulse” test).

93. Did plaintiff lose the power of his will?

“In a very recent case, the Supreme Court of New Mexico, recognized the inadequacy of the right-wrong test, and adopted what it called an ‘extension of the M’Naghten Rules.’ Under this extension, lack of knowledge of right and wrong is not essential for acquittal ‘if, by reason of disease of the mind, defendant has been deprived of or lost the power of his will.” *Durham v. United States*, 214 F.2d 862, fn. 32 (D.C. Cir. 1954)(citing *State v. White*, N.M. 270, P.2d 727, 730).

94. When a person is not held responsible for his conduct, can he become more disinhibited?

Consider the effect of telling a plaintiff that he is no longer responsible for his conduct because he has lost the ability to control his actions. Finding that a plaintiff lacks self-control can be a self-fulfilling prophecy. The causal relationship between anonymity and disinhibition is most obvious

with Online Disinhibition Effect. See *U.S. v. Mood*, 741 F.Supp 2d 821 (E.D. Mich., 7/30/10) (“Dervin Mood was significantly impacted and impaired by the ‘Online Disinhibition Effect’ on the Internet, allowing him to take risks and behaviors that he would not have chosen had the situation presented itself in real life.”).

Mechanism & Causation:

The ultimate issue in these cases is whether the underlying disorder or injury caused or contributed to the plaintiff’s conduct. Force the plaintiff to satisfy their burden of proving general causation and specific causation.

95. How much disinhibition is required to engage in the behavior?

This is a great question because there is no unit of measurement for disinhibition or inhibition. It forces the expert to either admit that they do not know or to offer an opinion they reached unscientifically. See *In re Commitment of Grimstead*, 2008 WL 5501164 (Tex.App. Beaumont 2009) (“Dr. Gaines explained that this was relevant to her risk assessment because Grimstead was on marijuana during at least one of the offenses, and ‘sustance abuse is a disinhibitor for Mr. Grimstead, ‘although Gaines further stated she does not think it requires a lot of disinhibition to perform the offenses.”). Force the expert to identify the scientific basis for measuring a person’s capacity for self-control. See *U.S. v. Lyons*, 731 F.2d 243, 248 (C.A. 5 (La) 1984) (“First, as we have mentioned, a majority of psychiatrists now believe that they do not possess sufficient accurate scientific bases for measuring a person’s capacity for self-control or for calibrating the impairment of that capacity.”).

96. Can you say, with a reasonable degree of professional certainty, that the behavior was caused by disinhibition?

Plaintiff must prove: (1) disinhibition caused the conduct; and (2) the underlying disorder or injury caused the disinhibition. *Benyamini v. Schwartz*, 2008 WL 5069917 at *8 (E.D. Cal 2008) (“...violent behavior was not willful bullying but rather true disinhibition resulting from mental illness.”). Don’t skip a step. Make sure the expert can prove the former.

97. What evidence proves this underlying disorder or injury can cause (general causation) and did cause (specific causation) disinhibition or irresistible impulses?

Remember that the plaintiff bears the burden of proving that a disorder or injury is capable of causing disinhibition or irresistible impulses (general causation) and that it *actually did* cause disinhibition or irresistible impulses (specific causation).

98. How did the underlying disorder or injury cause the disinhibition or irresistible impulse (mechanism)?

An expert who is comfortable blaming disinhibition or an irresistible impulse may not be comfortable explaining how a focal frontal lobe injury anatomically, physically and/or chemically caused disinhibition or an irresistible impulse. Force the expert to rule out all confounding variables. *State v. Pith*, 874, N.Y.S.2d 733, 711 (N.Y.Sup. 2008) (“But, as Dr. Frances credibly testified, the instant offense was not caused by cocaine disinhibition it arose from his exhibitionism and voyeurism.”).

99. How did you determine plaintiff would have behaved differently before the accident?

An expert may be able to support an opinion regarding impairment and dysfunction with neuropsychological testing and diagnostic images, but an opinion as to how the plaintiff would have behaved five years earlier (in the absence of a specific injury or disorder) is pure speculation. This opinion will almost always be subjective, based on interviews of family and friends, and difficult to defend with any scientific or reliable methodology. Find out the expert’s methodology for making this specific determination.

100. How can you distinguish between an irresistible impulse and an unresisted impulse?

Force the expert to explain their methodology for distinguishing between an irresistible impulse and an unresisted impulse. “The line between an irresistible impulse and an impulse not resisted is probably no sharper than between twilight and dusk.” *U.S. v. Lyons*, 731 F.2d 243, 248 (C.A. 5 (La) 1984), *quoting* American Psychiatric Association Statement On The Insanity Defense, 11 (1982) [APA Statement. Force the expert to identify the scientific tool that reliably allows experts to diagnose (after the fact) the occurrence of an irresistible impulse. “One need not disbelieve in the existence of Angels in order to conclude that the present state of our knowledge regarding them is not such as to support confident conclusions about how many can dance on the head of a pin. In like vein, it may be that some day tools will be discovered with which reliable conclusions about human volition can be fashioned. It appears to be all but a certainty, however, that despite earlier hopes they do not lie in our hands today.” *U.S. v. Lyons*, 731 F.2d 243, 249 (C.A. 5 (La) 1984).

* * *

Crushin A Concussion: Attacking Claims Of Impairment Following Mild TBI

Presented By John Jerry Glas
Deutsch, Kerrigan & Stiles, L.L.P.
755 Magazine Street, New Orleans, Louisiana 70130
(504)593-0627; jglas@dkslaw.com

There was a time when defense attorneys were satisfied to end their cross-examination by getting a doctor to admit that the plaintiff sustained “just a concussion.” Those days are over. More jurors have heard terrifying stories about concussions; and more experts are willing to testify that concussions cause permanent cognitive and behavioural impairment. Today, when a doctor testifies that a plaintiff sustained a concussion, jurors are left with more questions than answers. The diagnosis marks the beginning, not the end, of the trial.

Jurors may not realize how common concussions have become. An estimated 300,000 Americans lose consciousness from concussions every year, and the total number of concussions could total 3.8 million a year according to the U.S. Centers for Disease Control and Prevention. Because of that frequency, concussions have been well studied, and the recovery period well defined.

It is axiomatic that concussions improve.¹ Most symptoms (usually headaches) manifest in the early weeks;² and those symptoms usually resolve within three months.³ Recovery follows a reasonably consistent pattern, and that pattern has allowed doctors to form a series of mental templates for the expected

¹ See Donald T. Stuss, Ph.D., A Sensible Approach To Mild Traumatic Brain Injury, *Neurology* 1995, Vol. 45, at 1251 (“*Principle 3* is that the symptoms of mild TBI gradually improve.”).

² See Linda J. Carroll, et al, Prognosis For Mild Traumatic Brain Injury: Results Of The WHO Collaborating Centre Task Force On MTBI, *J. Rehabilitation Medicine* 2004; Suppl. 43, p. 102.

³ Carroll, Prognosis For Mild TBI, at 101 (“With respect to other populations [non-athletes], the stronger studies of MTBI, which use appropriate control groups and consider the effects of other non-MTBI factors, generally show resolution of symptoms within weeks or a few months.”); *Id.*, at 101 (“The best evidence consistently suggests there are no MTBI-attributable, objectively measured, cognitive deficits beyond 1-3 months post injury in the majority of cases.”).

results after mild TBI.⁴ When a patient's symptoms do not relate to the severity of the injury, doctors are obligated to consider the role of psychological factors in the "genesis and maintenance of those symptoms."

Not surprisingly, pending litigation is a predictor of persistent symptoms. In 2004, the World Health Organization published the results of their critical review of 428 studies related to prognosis after mild TBI.⁵ After studying recoveries which deviated from the typical pattern, the World Health Organization concluded: "[w]here symptoms persist, compensation/litigation is a factor, but there is little consistent evidence for other predictors."⁶

Every lawyer needs a strategy for attacking claims of permanent cognitive and behavioral impairment following a concussion. The following list of one hundred (100) questions was designed for a concussion case involving no objective evidence of brain damage and no neurological deficits on arrival at the hospital. Not every line of questioning will apply in every concussion case, but the goal should remain the same: (1) establish the lack of force exerted on the brain; (2) explain the lack of injury to the brain; and (3) prove the plaintiff's symptoms are not consistent with the severity of the injury. Teach the jury that the plaintiff's persistent symptoms are an aberration, and the jury will question the cause *and* the existence of those symptoms.

⁴ Stuss, *Sensible Approach*, at 1251 ("Past research and clinical experience allow us to form a series of mental templates for the expected results after mild TBI. Although its course may be much longer than once considered, the recovery does follow a reasonably consistent pattern.") (citations omitted).

⁵ Carroll, *Prognosis For Mild TBI*, at 84 ("Of 428 studies related to prognosis after mild traumatic brain injury, 120 (28%) were accepted after critical review.")

⁶ Carroll, *Prognosis For Mild TBI*, at 101 ("The best evidence consistently suggests there are no MTBI-attributable, objectively measured, cognitive deficits beyond 1-3 months post injury in the majority of cases.")

100 Questions To Ask In A Concussion Case

Mechanism Of Injury:

1. Is the skull rigid?
2. Is the brain surrounded by fluid?
3. Does the brain float inside the rigid skull?
4. If the rigid skull is moving forward and stops abruptly, will the floating brain continue to move forward?
5. If the rigid skull is moving fast enough, and stops abruptly, can the brain strike the inside of the skull vault?
6. Does the inside of the skull vault contain bony ridges?
7. When the brain strikes the bony ridges of the skull vault, can the brain itself be injured?
8. Is a brain injury at the site where the brain first strikes the skull vault called the “coup” injury?
9. If the rigid skull is moving fast enough, and stops abruptly, can the brain bounce off the skull vault, accelerate backwards, and strike the opposite skull vault?
10. Is a brain injury opposite the “coup” injury called the “contrecoup” injury?

Force Of Impact (No Skull Injury):

11. Did plaintiff fracture the weakest bone at the point of impact?
12. Did plaintiff require stitches at the point of impact?
13. Did plaintiff have a laceration or abrasion at the point of impact?
14. Did plaintiff have swelling at the point of impact?
15. Did plaintiff have bruising at the point of impact?
16. Did plaintiff have tenderness at the point of impact?
17. Did plaintiff have any evidence of head trauma at the point of impact?
18. Did plaintiff have Battle’s sign?
19. Did plaintiff have bilateral “Raccoon Eyes”?
20. Did plaintiff identify the head as the location of pain or injury?

Brain Inertia (No Focal Injury):

21. Can striking the skull vault cause a cerebral contusion (bruising)?
22. Can striking the skull vault cause a cerebral laceration (cut)?
23. Can striking the skull vault cause encephalomalacia (loss of brain tissue)?
24. Can striking the skull vault cause cerebral edema (swelling)?
25. Can striking the skull vault cause a subdural hemorrhage (bleeding)?
26. Can striking the skull vault cause a subdural hematoma?
27. Can subdural bleeding increase intracranial pressure?
28. Can bleeding and intracranial pressure cause brain herniation?
29. Can bleeding and intracranial pressure cause midline shift?
30. What diagnostic images were taken of the brain?
31. What is each image capable of visualizing?
32. Did plaintiff have brain shifting (herniation)?
33. Did plaintiff have brain shrinking (mass effect)?

34. Did plaintiff have brain swelling (edema)?
35. Did plaintiff have brain bruising (contusion)?
36. Did plaintiff have brain bleeding (hematoma)?
37. Did any image reveal objective evidence of a contrecoup injury?
38. Did any image reveal objective evidence of a coup injury?
39. Did any image reveal *any* objective evidence of brain damage?

Neck Momentum (No Neck Injury):

40. Can a cervical injury be sustained in this type of accident?
41. Did plaintiff sustain a cervical injury?
42. Did plaintiff report neck pain?

Review Of Symptoms

43. How long did plaintiff remain unconscious?
44. How long did plaintiff remain dazed?
45. When was plaintiff able to communicate?
46. When was plaintiff able to follow commands?
47. Did plaintiff have a 15/15 initial Glasgow Coma Scale Score?
48. Was plaintiff alert & oriented to time, place & person at hospital?
49. Did plaintiff provide an accurate description of the accident?
50. Did plaintiff provide an accurate medical history?
51. Did plaintiff have a seizure?
52. Did plaintiff have nausea or vomiting?
53. Did plaintiff have altered mood or affect?
54. Did plaintiff report a headache?

Evaluation Of 12 Cranial Nerves:

55. Did plaintiff have normal sense of smell?
56. Did plaintiff have normal (same as before) visual acuity?
57. Did plaintiff have normal (equal & round) pupils?
58. Did plaintiff have normal pupillary reaction (equal constriction) to light?
59. Did plaintiff report sensitivity to light?
60. Did plaintiff have normal extra-ocular range of motion?
61. Did plaintiff have normal saccadic function?
62. Did plaintiff have normal accommodation response?
63. Did plaintiff have normal positioning of the upper eyelids?
64. Did plaintiff have normal peripheral vision?
65. Did plaintiff have normal vision (no double vision)?
66. Did plaintiff have normal sensation & pain symmetry?
67. Did plaintiff have normal (symmetric) blink response?
68. Did plaintiff have normal (symmetric) tone in the masseter muscles?
69. Did plaintiff have normal functioning of the Facial Nerve?
70. Did plaintiff have normal sense of taste?
71. Did plaintiff have normal hearing?
72. Did plaintiff report sensitivity to noise?
73. Did plaintiff report ringing in the ears?

74. Did plaintiff have normal gag reflex?
75. Did plaintiff pass the “say aah” test?
76. Did plaintiff have ability to swallow normally?
77. Did plaintiff have a normal voice (not hoarse)?
78. Did plaintiff have normal laryngeal function?
79. Did plaintiff have slurred speech?
80. Did plaintiff have symmetric muscle tone?
81. Did plaintiff have normal tongue strength and control?

Evaluation of Motor Function:

82. Did plaintiff have normal muscle tone?
83. Did plaintiff have normal strength in each muscle group?
84. Did plaintiff have any muscle wasting or atrophy?
85. Did plaintiff have drift?
86. Did patient have normal fine movement control?
87. Did plaintiff have normal upper extremity motor strength?
88. Did plaintiff have normal lower extremity motor strength?
89. Did plaintiff have normal posturing?
90. Did plaintiff have any involuntary movements?
91. Did plaintiff have any fasciculations?

Evaluation of Reflexes

92. Did plaintiff have normal deep tendon reflexes?
93. Did plaintiff have normal plantar response (Babinski’s sign)?
94. Did plaintiff have normal balance (Romberg’s sign)?
95. Did plaintiff have normal finger flexors (Hoffmann’s sign)?

Evaluation of Coordination & Gait

96. Did plaintiff have normal coordination?
97. Did plaintiff have normal gait?

Evaluation of Sensory Functions

98. Did plaintiff have normal tactile sensation?
99. Did plaintiff have normal pain sensation?
100. Did plaintiff have normal vibration sense

Analysis Of 100 Concussion Questions

Mechanism Of Injury

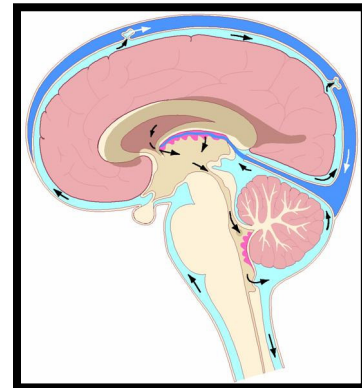
Jurors like bright lines, and bright lines can frame the discussion and define the severity of an injury. In a case involving cervical trauma, the cervical disc either was or was not herniated. In a case involving mild traumatic brain injury, a defense attorney can frame the discussion *and* define the severity of the injury by focusing the jury's attention on whether or not the brain actually struck the inside of the skull vault (cranial vault). That is a bright line that the jury can remember and understand. To draw that bright line, you will have to teach the jury a little (a very little) about what *can* happen to the brain during the traumatic event. Here are ten questions designed to accomplish that goal.

1. Is the skull rigid?

Yes. The cranium is the upper portion of the skull. The eight cranial bones include the frontal, parietal (2), temporal (2), occipital, sphenoid, and ethmoid. These cranial bones are strong but light weight. They are held together by fibrous joints called "sutures," which are held together by "Sharpey's fibres." Sharpey's fibres grow from one cranial bone into the adjacent bone, and bind them in a way that permits very little movement.

2. Is the brain surrounded by fluid?

Yes. The brain is surrounded by cerebrospinal fluid (CSF), which occupies the subarachnoid space and the ventricular system around and inside the brain. CSF is a clear solution containing ions and different substances to serve as an intracerebral transport medium for nutrients, neuroendocrine substances & neurotransmitters. The diagram (right) shows the circulation of CSF.



3. Does the brain float inside the rigid skull?

Yes. ("Kindah, sortah"). The cranium is the upper portion of the skull, and most will agree that the brain basically "floats" in cerebrospinal fluid inside the skull vault (or "cranial vault"). Jurors often remember this imagery of the brain being "cushioned gently by the surrounding spinal fluid;" it can also help jurors focus on what happened to the brain itself.

4. If the rigid skull is moving forward and stops abruptly, will the floating brain continue to move forward?

Yes. Inertia is the resistance of an object to a change in its state of motion. When the skull stops, the brain's inertia keeps it moving forward. Newton's first law of motion states: "An object at rest tends to stay at rest and an object in motion tends to stay in motion with the same speed and in the same direction unless acted upon by an unbalanced force."

5. **If the rigid skull is moving fast enough, and stops abruptly, can the brain strike the inside of the skull vault?**

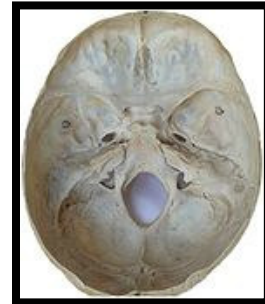
Yes. The brain will strike the inside of the cranial vault. The brain may also rotate along (or rub against) the cranial vault.

6. **Does the inside of the skull vault contain bony ridges?**

Yes. The inside of the cranial vault is not smooth. The interior of the skull (right) contains sharp bony ridges that can injure the brain. The following is an excerpt from a deposition of a neuropsychologist in a case where a plaintiff wearing a hard hat struck walked into a steel beam:

“Q. And that part of the brain. . . is the basic area that is associated with the forehead and directly above?

A. Correctly more – and also the region behind the eyes and sinus passages. The inside of the skull vault is not very smooth in that area.” (Dr. Stephen K. Martin, Ph.D. 9/25/07 Deposition)



7. **When the brain strikes the bony ridges of the skull vault, can the brain itself be injured?**

Yes. The brain is vulnerable to trauma. Note: Different experts describe brain tissue *very* differently. Some describe it as being “firm gelatin-like”; others insist it has “the consistency of warm butter.” Be careful.

8. **Is a brain injury at the site where the brain first strikes the skull vault called the “coup” injury?**

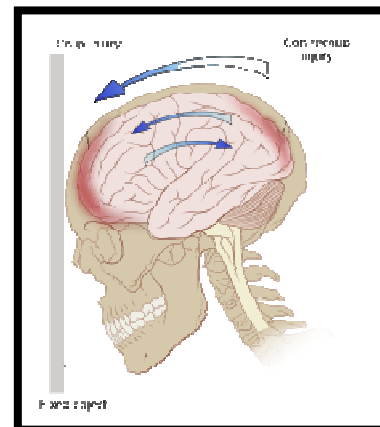
Yes. In a coup injury, the head stops abruptly and the brain collides with the inside of the cranial vault. This type of injury is called a “focal injury,” as opposed to a diffuse injury.

9. **If the skull is moving fast enough, and stops abruptly, can the brain bounce off the skull vault, accelerate backwards, and strike the opposite skull vault?**

Yes. If sufficient speed/force is involved, the brain can experience deceleration forward and then acceleration backwards.

10. **Is a brain injury opposite the “coup” injury called a “contrecoup” injury?**

Yes. A contrecoup injury is a brain injury opposite from the impact. A contrecoup injury occurs when the brain bounces from the point of impact to the opposite side of the skull. It is also a focal injury.



Force Of Impact (No Skull/Skin Injury):

Jurors may not understand complicated calculations of force, but they know that if you hit your head hard enough, you will get a hickey. In most concussion cases, the jury will want to know how “fast” the plaintiff was walking when he struck his head on the steel beam, or how “hard” the plaintiff fell when he struck his head against the ground. In those cases, a defense lawyer can define and limit the amount of force involved in a concussion by reviewing the absence of those injuries at the point of impact. Start by asking about injuries requiring the most force, and end by asking about injuries requiring the least force.

11. Did plaintiff fracture the weakest bone at point of impact?

Identify the weakest bone in the area that struck (or was struck) by the object. Establish that the force of impact was not sufficient to fracture that bone. This can be especially effective line of questioning in cases where an object simultaneously strikes the facial bones.

12. Did plaintiff require stitches at point of impact?

13. Did plaintiff have a laceration or abrasion at point of impact?

14. Did plaintiff have swelling at point of impact?

15. Did plaintiff have bruising at point of impact?

16. Did plaintiff have tenderness at point of impact?

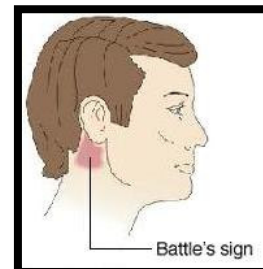
Emergency Room records often include a diagram on which the ER staff is required to record (using specific symbols) whether their physical examination of the plaintiff revealed any lacerations, abrasions, swelling, bruising, point tenderness, or tenderness. In many concussion cases, the patient will sustain no injury to the head or face.

17. Did plaintiff have any evidence of head trauma at point of impact?

Emergency Room records often include a Physical Examination section; and, sometimes, that section includes a box entitled “No evidence of head trauma.” Let the jury know if that box was checked.

18. Did plaintiff have Battle’s sign?

Battle’s sign (“mastoid ecchymosis”) is named after William Henry Battle. It consists of bruising over the mastoid process, a conical prominence projecting from the undersurface of the mastoid process of the temporal bone. It can be an indication of a fracture at the base of the posterior portion of the skull.



19. Did plaintiff have bilateral “Raccoon Eyes”?

It is important to differentiate Raccoon Eyes, which are always bilateral periorbital ecchymoses, from a “black eye” caused by facial trauma. The box for Raccoon Eyes will rarely be checked in ER records because they often develop 2 or 3 days after closed head injury. Raccoon eyes are usually evidence of a basilar skull fracture, and occur when damage (at

the time of fracture) tears the meninges and causes the venous sinuses to bleed into the arachnoid villi and the cranial sinuses.

20. Did plaintiff identify the head as the location of pain or injury?

Emergency Room records often include a section which allows the ER staff to circle the “location of pain/injuries” according to the plaintiff. Always check to see if “head” is circled.

Brain Momentum (No Focal Injury):

When a plaintiff admits that his head did not strike anything, then defense lawyer can define and limit the amount of force involved in a concussion by reviewing the absence of any brain injury at the point where the brain *could have* impacted the cranial vault (*if* sufficient force had been involved). Start by establishing that striking the cranial vault *can* cause each injury, and which injuries the diagnostic image(s) taken of the plaintiff’s brain *can* show. When you have laid the proper foundation, prove that the diagnostic image(s) revealed no objective evidence of any of these injuries (from most severe to least severe).

21. Can striking the skull vault cause a cerebral contusion (bruising)?

Yes. A cerebral contusion is a “bruise of the brain tissue.” It has been described as a heterogenous areas of hemorrhage (bleeding) into the brain parenchyma.

22. Can striking the skull vault cause a cerebral laceration (cut)?

Yes. A cerebral laceration occurs when the tissue of the brain is mechanically cut or torn. The injury is similar to a cerebral contusion, but the pia-arachnoid membranes are torn during a cerebral laceration (but not during a cerebral contusion).

23. Can striking the skull vault cause encephalomalacia (loss of brain tissue)?

Yes. The cerebrum is the large rounded structure of the brain occupying most of the cranial cavity. It is divided into two cerebral hemispheres that are joined at the bottom. It controls and integrates motor, sensory, and higher mental functions, such as thought, reason, emotion, and memory. Striking the skull vault can cause the tearing of brain tissues. Encephalomalacia (or cerebromalacia) refers to the loss of brain tissue, which can be caused by a traumatic brain injury and can be visualized on certain diagnostic images.

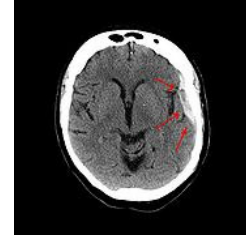
24. Can striking the skull vault cause cerebral edema (swelling)?

Yes. Cerebral edema is an accumulation of fluid in the brain tissue that causes the brain to swell.

25. **Can striking the skull vault cause a subdural hemorrhage (bleeding)?**
Yes. The dura is the outer protective covering of the brain. Whereas epidural bleeding usually results from tears in arteries, subdural bleeding usually results from tears in veins that cross the subdural space.

26. **Can striking the skull vault cause a subdural hematoma?**

Yes. A subdural hematoma is a collection of blood within the meningeal layer of the dura (“on the surface of the brain”). The subdural hematoma in the image (right) is identified by three arrows.

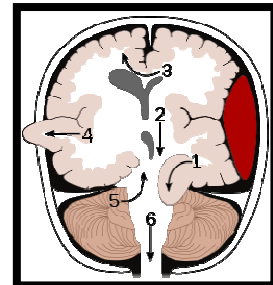


27. **Can bleeding cause increase intracranial pressure?**

Yes. Intracranial Pressure (ICP) is the pressure in the cranium. ICP is maintained in a tight normal range dynamically, through the production and absorption of CSF and pulsates approximately 1mm Hg in a normal healthy adult. Bleeding into the subdural space can increase intracranial pressure in the cranium.

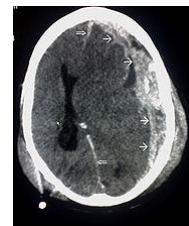
28. **Can bleeding and intracranial pressure cause brain herniation?**

The skull is rigid, and the space between the skull and the brain is small. A subdural hematoma can cause an increase in intracranial pressure. It can have a “mass effect” on the brain, potentially causing brain herniation and/or midline shift. Brain herniation occurs when the brain shifts across structures within the skull, or through the hole called the foramen magnum in the base of the skull (through which the spinal cord connects with the brain). The diagram shows the six types of brain herniation: (1) uncal; (2) central; (3) cingulate; (4) transcalvarial; (5) upward; and (6) tonsillar.



29. **Can bleeding and intracranial pressure cause midline shift?**

Yes. A subdural hematoma or intracranial pressure can cause the brain to shift past its center point (“Midline Shift”). Midline Shift is a measure of ICP; presence of the former is an indication of the latter. Immediate surgery may be indicated if there is midline shift of more than 5mm.



30. What diagnostic images were taken of the brain?

31. What was each image capable of visualizing?

Jurors may be familiar with most diagnostic images (i.e., x-rays, CT scans, MRIs), but they may not understand what each image is and is not capable of revealing. Confirm that diagnostic images were taken of the plaintiff's brain, and then establish what brain injuries each image is capable of visualizing ("Can a CT scan show midline shift..").

32. Did plaintiff have shifting (herniation)?

33. Did plaintiff have shrinking (mass effect)?

34. Did plaintiff have swelling (edema)?

35. Did plaintiff have bruising (contusion)?

36. Did plaintiff have bleeding (hematoma)?

After laying the foundation that sufficient force can cause each injury, establish that plaintiff did NOT sustain any of the injuries. The absence of each injury makes it less likely that the brain struck the cranial vault, and further defines the lack of force involved.

Brain CT Scan:

- NO Shifting
- NO Shrinking
- NO Swelling
- NO Bruising
- NO Bleeding

37. Did plaintiff have any objective evidence of a contra-coup injury?

38. Did plaintiff have any evidence of a coup injury?

39. Did plaintiff have any objective evidence of brain damage?

When the diagnostic images are negative, you can finish this line of questioning by asking these three questions.

Neck Momentum (No Neck Injury):

Jurors perceive the cervical spine as more vulnerable to trauma than the lumbar and/or thoracic spine. If they believe that whiplash is capable of causing a myriad of cervical injuries, then they may have difficulty believing the force involved in an accident could be *insufficient* to cause neck pain, but still be *sufficient* to cause diffuse (invisible) axonal brain injury. In the right case, this line of questioning can be very effective.

40. Can a cervical injury be sustained in this type of accident?

41. Did plaintiff sustain a neck injury?

42. Did plaintiff report neck pain?

These three questions can easily be expanded by identifying specific cervical injuries (i.e., fracture, displacement, herniation, sprain etc). Start by establishing that the type of accident *can* cause each cervical injury, and which injuries the diagnostic image(s) taken of the plaintiff's cervical region *can* show. When you have laid the proper foundation, prove that the diagnostic image(s) revealed no objective evidence of any of these cervical injuries (from most severe to least severe).

Review Of Symptoms:

All concussions are not the same. All mild traumatic brain injuries are not the same. Medical studies will often divide study members into different “severity groups” based on certain significant predictors of outcome. The following questions can help a defense attorney define the severity of the injury. These are questions meant to be answered *before* trial, and asked at trial *only if* the answers are favorable.

43. How long did plaintiff remain unconscious?

Medical studies have reported a dose-response relationship between loss of consciousness and cognitive impairment.⁷ The longer the person experiences loss of consciousness (LOC), the less likely that person will have a full recovery.

Some TBI medical studies divide study members into “severity of injury” groups based on the duration of LOC. When the study divides TBI into three groups (mild, moderate & severe), a concussion will usually meet the criteria for the “mild” TBI group. When the study divides TBI into groups based solely on LOC, a concussion will usually meet the criteria for the least severe group. For example, one study divided members into five “severity groups”:

Group 1:	LOC < 1hr
Group 2:	LOC = 1-23 hr
Group 3:	LOC = 1-6 days
Group 4:	LOC = 7-13 days
Group 5:	LOC = 14-28 days

Concussions are also classified based on LOC. According to the Cantu Guidelines, a Grade I concussion is associated with no LOC. A Grade II concussion is associated with LOC for less than 5 minutes; a Grade III concussion is associated with LOC for more than 5 minutes.

44. How long did plaintiff remain dazed?

45. When was plaintiff able to communicate?

Some plaintiffs will admit that they did not lose consciousness, but insist that they were dazed, disoriented, or confused following the accident. Check the ER records to see if “confusion/disorientation” is checked in the “Neuro/Psych” subsection of the Physical Examination; and determine when plaintiff first spoke and exactly what plaintiff said. The ability to engage in normal conversation is relevant to determining GCS score and cognitive functioning.

⁷ Martin L. Rohling, John E. Meyers, and Scott R. Millis, Neuropsychological Impairment Following Traumatic Brain Injury: A Dose-Response Analysis, *The Clinical Neuropsychologist*, 2003, Vol. 17, No. 3, pp. 289-302, at 289 (stating in the Abstract that “A significant dose-response relationship between loss of consciousness (LOC) and cognitive impairment was found. . .”).

46. When was plaintiff able to follow commands?

Some medical studies divide study members into “severity of injury” groups based on the time to follow commands after the injury (TFC), like “raise your hand” or “stick out your tongue.” When the study divides TBI into groups based solely on TFC, a concussion will usually meet the criteria for the least severe group. For example, one study divided members into six “severity groups”:

- Group 1: TFC < 1hr
- Group 2: TFC = 1-23 hr
- Group 3: TFC = 1-6 days
- Group 4: TFC = 7-13 days
- Group 5: TFC = 14-28 days
- Group 6: TFC > 28 days

47. Did plaintiff have a 15/15 initial Glasgow Coma Scale Score?

The Glasgow Coma Scale (GCS) is the most widely used scoring system for quantifying levels of consciousness following TBI. The GCS requires ER doctors and staff to assess three things: eye opening, motor response and verbal responses. A perfect GCS score is 15/15. In order to receive a 15/15 the plaintiff would have to: (1) demonstrate spontaneous eye movement; (2) have normal motor response; and (3) demonstrate normal conversation.

It is well established that the GCS is used by ER staff because it correlates well with outcome following TBI. A low GCS score more than an hour after an accident can be an indicator that the plaintiff sustained a TBI, and can be a significant predictor of outcome following TBI. The better the GCS score at presentation, the more likely the plaintiff will enjoy a full recovery.

48. Was plaintiff alert & oriented to time, place & person on arrival at the hospital?

In the emergency room, as a part of a mini mental status examination, the plaintiff may be asked whether the plaintiff knows what day it is, where they are, and who they are. If the plaintiff answers the questions correctly, the ER staff will note “AOx3,” which means that the plaintiff was alert and oriented as to time, place and person.

49. Did plaintiff provide an accurate/consistent description of accident?

50. Did plaintiff provide an accurate/consistent medical history?

A traumatic brain injury can cause amnesia, and the plaintiff’s recall can be important in evaluating the severity of the injury. Always check the ER records to determine what details the plaintiff was able to give ER staff about the accident and/or the plaintiff’s medical history. Some ER records

will also require the ER staff to circle whether the plaintiff remembers “impact” and/or “coming to hospital.”

51. Did plaintiff have a seizure?

Approximately 5-10% of individuals with traumatic brain injury experience new onset seizure. The risk of seizure increases with increasing injury severity, depressed skull fracture, intracranial hematoma, and penetrating trauma. The risk is greatest in the first two years after injury and gradually declines thereafter. All types of seizures may occur as a result of trauma, but the most frequent are focal or partial complex seizures. Generalized complex seizures (what are commonly called "grand mal" seizures) occur in approximately 33% of cases. Immediate onset seizures, those that occur immediately or in the first few hours after a brain injury, do not suggest a chronic seizure disorder. Early onset seizures and those which develop within the first 7-8 days after trauma require prophylaxis for up to one year. Spontaneous resolution of seizure activity has been noted in this group.⁸

52. Did plaintiff have nausea or vomiting?

Nausea and vomiting are generally considered “classic” symptoms of a concussion. Most people think that vomiting is controlled by the stomach, but it is actually controlled by an area of the brain which some call the “vomiting center” (yes, seriously). Whatever it is called, that area of the brain initiates the vomiting sequence, which causes the windpipe to close and the abdominal wall and diaphragm muscles to tighten suddenly and forcefully. The brain can initiate the vomiting sequence in response to infection or concussion.

53. Did plaintiff have altered mood or affect?

Plaintiffs often report “changes in personality” following a concussion, but those changes are usually observed or noted days, weeks, or months after the accident. Check the ER records to see if “mood & affect” was checked or circled in the Neuro/Psych subsection. Also confirm that the Plaintiff was not restrained or sedated before discharge from the hospital.

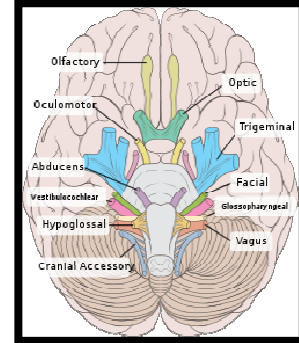
54. Did plaintiff report a headache?

Plaintiffs will almost always report experiencing a headache following a concussion. Find out the severity of the headache (i.e. was it a migraine), the duration of the headache, and whether the headache resolved abruptly or tapered.

⁸ Jay Meythaler, JD,MD, & Tom Novack, PhD, Post Traumatic Seizures Following Head Injury, published by the UAB Traumatic Brain Injury Care System, posted online at <http://main.uab.edu/tbi>.

Examination Of Cranial Nerves:

There are twelve (12) conventionally-recognized cranial nerves, and those cranial nerves emerge directly from the brain stem. In Emergency Room records the cranial nerves will often be abbreviated "CN." Cranial nerve examinations vary. The doctor will detect and interpret the signs during many of the CN examinations; however, during certain neurological examinations, especially of the sensory system, the doctor will rely on the patient to report what he/she is feeling or not feeling. A CN examination will usually include an evaluation of the patient's motor function, reflexes, coordination & gait, and sensory functions. Those aspects are artificially divided below, but only for the sake of organization. Many CN tests will evaluate more than one cranial nerve.



55. Did plaintiff have normal sense of smell?

The olfactory nerve is the 1st cranial nerve. It is composed of sensory fibers, and its sole function is to discern smells. Olfaction depends on the integrity of the olfactory neurons in the roof of the nasal cavity and their connections through the olfactory bulb, tract, and stria to the olfactory cortex of the medial frontal and temporal lobes. To test olfaction, a doctor can present an odorant (concentrated vanilla, peppermint, or coffee extract) to each nostril, and asks the patient to identify each smell.

56. Did plaintiff have normal (same as before) visual acuity?

Visual acuity is the eye's ability to detect fine details and is the quantitative measure of the eye's ability to see an in-focus image at a certain standard. The standard definition of normal visual acuity (20/20) is the ability to resolve a spatial pattern separated by a visual angle of one minute of arc. If the plaintiff can see at a distance of 20 feet an object that can normally be seen at 20 feet, then the plaintiff has 20/20 vision. If the plaintiff can see at 20 feet what a normal person can see at 40 feet, then the plaintiff has 20/40 vision. Visual acuity is often measured with a Snellen chart (see right).



57. Did plaintiff have normal (equal & round) pupils?

58. Did plaintiff have normal pupillary reaction (equal constriction) to light?

The oculomotor nerve is the 3rd. An examination of pupillary function includes inspecting the pupils for equal size (1mm or less of difference may be normal), regular shape, and reactivity to light. To test pupillary reaction, the doctor can use the swinging flashlight test. Normally, both pupils will constrict when the first pupil is exposed to light. Normally, as

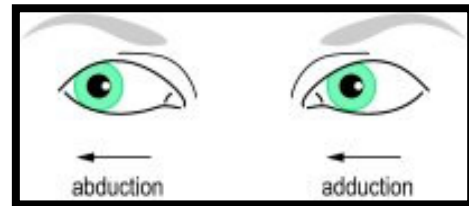
the light is being moved from the first pupil toward the second pupil, both pupils will begin to dilate; and, when the light reaches the second pupil, both pupils will constrict again. In hospital records, this examination may be abbreviated PERRL, which stands for Pupils Equal, Round, Reactive (or Responds To Light).

59. Did plaintiff report sensitivity to light?

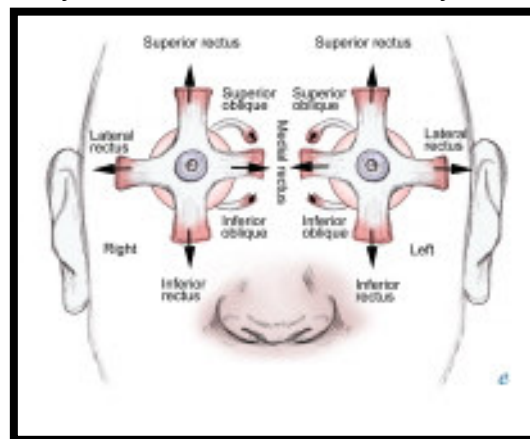
Photophobia is not a morbid fear of light; it is the experience of discomfort or pain to the eyes due to light exposure. When too much light enters the eyes, the light causes over stimulation of the photoreceptors in the retina, and excessive electrical impulses to the optic nerve. Damage to the eye (i.e., corneal abrasion) can allow too much light to enter. Damage to the pupil's ability to constrict equally (i.e., damage to oculomotor nerve) can also allow too much light to enter. See question *supra* regarding normal constriction.

60. Did plaintiff have normal extra-ocular range of motion?

The “follow my finger test” requires a patient to follow the doctor’s finger as it moves through the six principal positions of gaze (in an “H” pattern). The test involves adduction (rotation of the eye toward midline) and abduction (outward rotation of the eye away from midline). The test can reveal problems with the 2nd Cranial Nerve (Optic Nerve), the 4th Cranial Nerve (Trochlear Nerve) or the 6th Cranial Nerve (Abducens) Nerve.



The Optic nerve contains special sensory afferent fibers that convey visual information from the retina to the occipital lobe via the visual pathway. The extra-ocular muscles are the six muscles that control the movements of the eye. To test slow tracking or “pursuits,” a doctor can use the “follow my finger test.”



The Trochlear Nerve supplies somatic efferent motor fibers that innervate the superior oblique muscle. To test the superior oblique muscle (and isolate the trochlear nerve), the doctor can move a finger downward during the “H” pattern.

The Abducens Nerve supplies somatic efferent motor fibers to the lateral rectus muscle, which functions to abduct the eye. To test the lateral

rectus muscle (isolate the Abducens Nerve), the doctor can move a finger horizontally during the “H” pattern.

61. Did plaintiff have normal saccadic function?

The eyes do not move continuously over a line of text; they make short rapid movements (“saccades”) intermingled with short stops (“fixations”). To evaluate saccades, the doctor can have the patient move his/her eyes quickly to a target at the far right, left, top and bottom. If the eyes are unable to “jump” from one place to another, it may impair the patient’s reading ability and other skills.

62. Did plaintiff have normal accommodation response?

The extra-ocular muscles are responsible for accommodation. To test accommodation, the doctor may hold a finger about 4 inches from the patient’s nose and then moving that finger toward the patient. If the eyes can maintain focus on the finger, then the eyes have exhibited a normal accommodation response. In hospital records, this examination may be included with the pupil examination and abbreviated as the “A” in “PERRLA” (Pupils Equal, Round, Reactive (or Responds To Light), & Accommodation).

63. Did plaintiff have normal positioning of the upper eyelids?

Ptosis is an abnormally low position (drooping) of the upper eyelid. Ptosis can be caused by damage to the muscles that raise the eyelid (levator & Müller’s muscles) or by damage to the 3rd Cranial Nerve (Oculomotor Nerve) which controls this muscle.



64. Did plaintiff have normal peripheral vision?

To test the visual fields, the doctor can perform confrontation field testing in which each eye is tested separately to assess the extent of the peripheral field. During that test, the doctor covers one of the patient’s eyes, and tells the patient to fixate the uncovered eye on the doctor. The doctor then tells the patient to count the number of fingers that are briefly flashed in each of the four quadrants.

65. Did plaintiff have normal vision (no double vision)?

Diplopia is commonly known as “double vision.” It is the simultaneous perception of two images of a single object. These images may be displaced horizontally, vertically, or diagonally (i.e., both vertically & horizontally) in relation to each other. Temporary diplopia can be caused by a concussion. Loss of the 4th Cranial Nerve (Trochlear Nerve) can cause diplopia with compensating head tilt. Loss of the 6th Cranial Nerve

(Abducens Nerve) can elicit complaints of horizontal diplopia and may cause patients to appear esotropic (where one or both eyes turn inward).

66. Did plaintiff have normal sensation and pain symmetry?

The trigeminal nerve is the 5th cranial nerve. It supplies both sensory and motor fibers to the face and periorbital area. The afferent sensory fibers separate into three division and carry touch, pressure, pain, and temperature sense from the oral and nasal cavities, and the face. To test the sensory portion of the trigeminal nerve, the doctor can touch one side of the forehead with a tissue, touch the opposite side of the forehead with a tissue, and ask the patient (whose eyes are closed) to compare sensations. A sharp object can be used in the same manner when testing for pain symmetry. The test is then repeated on the cheek and jaw line to assess the second and third divisions.

67. Did plaintiff have normal (symmetric) blink response?

An additional test used to evaluate the trigeminal nerve is the corneal reflex test. To evaluate the corneal reflex, the doctor can gently touch each cornea with a cotton wisp and observes any asymmetries in the blink response. This tests both the sensory portion of the 5th Cranial Nerve (Trigeminal Nerve) and the motor portion of the 7th Cranial Nerve (Facial Nerve), which is responsible for lid closure.

68. Did plaintiff have normal (symmetric) tone in the masseter muscles?

To test the motor component of the 5th Cranial Nerve (Trigeminal Nerve), the doctor can feel and compare the tone of the masseter muscles during jaw clench. The doctor asks the patient open his/her mouth and resist the examiner's attempt to close it. If there is weakness of the pterygoids, the jaw will deviate towards the side of the weakness.

69. Did plaintiff have normal functioning of the Facial Nerve?

The Facial Nerve is the 7th Cranial Nerve. It supplies efferent nerve motor innervation to the muscles of facial expression, and carries sensory afferent fibers from the anterior two thirds of the tongue for taste. To test the motor division of the Facial Nerve, the doctor can ask a patient to wrinkle the forehead and checks for asymmetry. The doctor can then ask the patient to shut the eyes tightly while the doctor attempts to open them, checking for any weakness on one side. The doctor may also have the patient show his/her teeth or smile, and compare the nasolabial folds on either side of the patient's face.

70. Did plaintiff have normal sense of taste?

To test the sensory fibers of the Facial Nerve, the doctor can apply sugar, salt, or lemon juice on a cotton swab to the lateral aspect of each side of the tongue and ask the patient identify the taste. Taste is often tested only when specific pathology of the facial nerve is suspected.

71. Did plaintiff have normal hearing?

The Vestibulocochlear Nerve is the 8th cranial nerve. It carries two special sensory afferent fibers, one for audition (hearing) and one for vestibular function (balance). Damage to the 8th Cranial Nerve can lead to hearing loss, dizziness, loss of balance, tinnitus, and deafness. To test the cochlear division, the doctor can screen for auditory acuity. To test auditory acuity, the doctor can lightly rub fingers together next to each of the patient's ears and comparing the left and right side responses.

Weber Test: The Webber test consists of pacing a vibrating tuning fork on the middle of the forehead and asking if the patient feels or hears it best on one side or the other. The normal patient will say that it is the same on both sides. The patient with unilateral neurosensory hearing loss will hear it best in the normal ear, and the patient with unilateral conductive hearing loss will hear it best in the abnormal ear. The tuning fork is struck and placed in the middle of the patient's forehead. The patient compares the loudness on both sides.

Rinne Test: The Rinne test consists of comparing bone conduction, assessed by placing the tuning fork on the mastoid process behind the ear, versus air conduction, assessed by holding the tuning fork in the air near the front of the ear. Normally, air conduction volume is greater than bone conduction sound volume. For neurosensory hearing loss, air conduction volume is still greater than bone conduction, but for conduction hearing loss, bone conduction sound volume will be greater than air conduction volume. A tuning fork is held against the mastoid process until it can no longer be heard. It is then brought to the ear to evaluate the patient's response.

72. Did plaintiff report sensitivity to noise?

Hyperacusis (also spelled "hyperacousis") is a condition of reduced tolerance to auditory stimuli. A person with hyperacusis may experience ambient noises (i.e. dog barking, dishwasher purring) as inner ear pain or pressure. Hyperacusis is usually caused by damage to the inner ear or the auditory nerve, but it can occur as a cerebral processing disorder (i.e. as a result of the brain's perception of the sound). A doctor can use the Johnson's Hyperacusis Quotient to measure its severity.



73. Did plaintiff report "ringing" in the ears?

Tinnitus is the perception of sound within the human ear in the absence of corresponding external sound. It is usually described as a ringing sound, but it can take the form of a high pitched whining, buzzing, hissing,

screaming, humming, tinging or whistling sound. It can be intermittent or continuous. To quantitatively measure tinnitus, a doctor can play sample sounds of known amplitude, and decreasing the amplitude until the tinnitus becomes audible. The tinnitus will always be equal to or less than the sample noises heard by the patient.

74. Did plaintiff have a normal gag reflex?

The gag reflex tests both the sensory & motor components of the 9th Cranial Nerve (Glossopharyngeal Nerve) and the 10th Cranial Nerve (Vagus Nerve). To test the involuntary gag reflex, the doctor can stroke the back of the pharynx with a tongue depressor and watches the elevation of the palate (as well as causing the patient to gag).

75. Did plaintiff pass the “say aah” test?

To test the motor division of the 9th Cranial Nerve (Glossopharyngeal Nerve) & the 10th Cranial Nerve (Vagus Nerve), the doctor can ask the patient to say “ahh” or “kah.” The palate and uvula will normally elevate symmetrically without deviation. Paralysis of the 9th nerve can cause a pulling of the uvula to the unaffected side.

76. Did plaintiff have the ability to swallow normally?

77. Did plaintiff have a normal voice (not hoarse)?

78. Did plaintiff have normal laryngeal function?

The Vagus Nerve is the 10th Cranial Nerve. It carries sensory afferent fibers from the larynx, trachea, esophagus, pharynx, and abdominal viscera. It also sends efferent motor fibers to the pharynx, tongue, thoracic and abdominal viscera and the larynx. Testing of the vagus nerve is performed by the gag reflex and the “ahh” test. A unilateral lesion affecting the vagus nerve can produce hoarseness and difficulty swallowing due to a loss of laryngeal function.

79. Did plaintiff have normal speech (no slurred speech)?

“Slurred speech” is abnormal speech in which words are not enunciated clearly or completely but are run together or partially eliminated. There are many causes of slurred speech, but it is associated with post-concussion syndrome.

80. Did plaintiff have symmetric muscle tone?

The Accessory Nerve is the 11th Cranial Nerve. It carries efferent motor fibers to innervate the sternomastoid and trapezius muscles. To test the Accessory Nerve, the doctor can ask the patient to shrug the shoulders (trapezius muscles) and turn the head (sternomastoid muscles) against resistance. While the patient is turning the head, the doctor palpates the sternocleidomastoid muscles. The muscle tone on both sides is compared.

81. Did plaintiff have normal tongue strength and control?

The Hypoglossal Nerve is the 12th Cranial Nerve. It supplies efferent motor fibers to the muscles of the tongue. To test the hypoglossal nerve, the doctor can ask the patient to stick out their tongue and move it side to side. If there is unilateral weakness, the protruded tongue will deviate toward the side of the weakness. Further testing includes moving the tongue right to left against resistance, or having the patient say “la, la, la.”

Evaluation Of Motor Function:

82. Did patient have normal muscle tone?

83. Did plaintiff have normal strength in each muscle group?

84. Did plaintiff have any muscle wasting or hypertrophy?

Doctor may test the muscle strength of each muscle group and record it in a systematic fashion. To determine muscle tone, the doctor can ask the patient to relax, and then passively move each limb at several joints to evaluate any resistance or rigidity that might be present.

85. Did patient have drift?

To test for drift, the doctor can ask a patient to close her/his eyes and extend both arms to the front with palms up. The doctor then observes the patient’s arms to determine if one or both drift downward to side.

86. Did patient have normal fine movement control?

To test fine movement control, a doctor can ask a patient to make rapid hand movements or tap a foot rapidly.

87. Did plaintiff have normal upper extremity motor strength?

To test upper extremity motor strength, the doctor can ask a patient to raise both arms in front of them while the doctor provides resistance. The doctor then records any weakness of one limb when compared to the contralateral limb.

88. Did plaintiff have normal lower extremity motor strength?

To test lower extremity motor strength, the doctor can ask a patient to flex and extend both legs in front of them while the doctor provides resistance. The doctor then records any weakness of one limb when compared to the contralateral limb.

89. Did plaintiff have normal posturing?

Abnormal posturing is an involuntary flexion or extension of the arms and legs. It occurs when one set of muscles becomes incapacitated while the opposing set is not, and an external stimulus (such as pain) causes the working set of muscles to contract. It can be caused by conditions that lead to large increases in intracranial pressure, and typically indicates severe brain damage.

90. Did plaintiff have any involuntary movements?

91. Did plaintiff have any fasciculations?

A complete neurological examination should include observation of any twitches or involuntary movements. Fasciculations are quivering movements caused by firing of muscle motor units.

Evaluation Of Reflexes:

92. Did plaintiff have normal deep tendon reflexes?

In a normal person, when a muscle tendon is tapped briskly, the muscle immediately contracts due to a two-neuron reflex arc involving the spinal or brainstem segment that innervates the muscle. To test deep tendon reflexes, a doctor can perform the patellar tendon (knee jerk) test. When the doctor strikes the patellar tendon with a reflex hammer, the it should be possible to feel the quadriceps contract and the knee extend. The deep tendon reflexes are typically graded as follows:

- 0 = no response
- 1+ = a slight but definitely present response
- 2+ = a brisk response
- 3+ = a very brisk response
- 4+ = a tap elicits a repeating reflex (clonus)

Whether the 1 + and 3 + responses are normal depends on what they were before the accident (i.e., the patient's reflex history), what the other reflexes are, and analysis of associated findings such as muscle tone, muscle strength, or other evidence of disease. Asymmetry of reflexes suggests abnormality.

93. Did plaintiff have normal plantar response (Babinski's sign)?

To test plantar response, a doctor can try to elicit the Babinski response. There are different methods, including stroking the sole (the plantar surface of the foot) firmly with a thumb from back to front along the outside edge. There are three possible responses:



- Flexor: the toes curve inward and the foot everts; this is the response seen in healthy adults (aka a "negative" Babinski)
- Indifferent: there is no response.
- Extensor: the hallux dorsiflexes and the other toes fan out - the "positive Babinski's sign" indicating damage to the central nervous system.

Babinski's sign is associated with upper motor neuron lesions anywhere along the corticospinal tract. **Hoffmann's Note:** It may not be possible to elicit Babinski's sign if there is severe weakness of the toe extensors.


94. Did plaintiff have normal balance (Romberg's sign)?

Balance comes from the combination of several neurological systems, namely proprioception, vestibular input, and vision. If any two of these systems are working, then the plaintiff should be able to demonstrate a fair degree of balance. To test balance, a doctor can ask the patient to stand with heels and toes together; to close their eyes, and to balance. The doctor will observe for one minute. If the plaintiff loses balance (sways or falls) while the eyes are closed, then the Romberg's test is positive.

4 stages

- ▶ 1st: Stand straight with feet and arm close together
- ▶ 2nd: With the eye closed
- ▶ 3rd: With the eyes reopened
- ▶ 4th: With the eyes stay open and arm outstretched

Warning
If the patient falls on to the ground
So are you

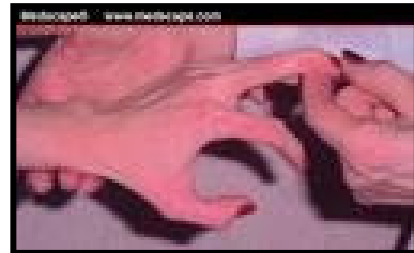


	Sensory	Cerebellar
1 st	No sway	Sway
2 nd	↑↑ sway	Same
3 rd	↓ Sway	Same
4 th	Same	↓ Sway

95. Did plaintiff have normal finger flexor reflexes (Hoffmann's sign)?

There is no precise hand equivalent for the plantar response, however, finger flexor reflexes can help demonstrate hyperreflexia in the upper extremities. To test finger flexor reflexes, a doctor can tap gently on the palm with the reflex hammer. Alternatively, heightened reflexes can be demonstrated by the presence of Hoffmann's sign.

To elicit Hoffmann's sign, a doctor can hold the patient's middle finger loosely and flick the fingernail downward, causing the finger to rebound slightly into extension. If the thumb flexes and adducts in response, Hoffmann's sign is present. Hoffmann's sign (heightened finger flexor reflexes) suggest an upper motor neuron lesion affecting the hands.



Evaluation Of Coordination & Gait:

96. Did plaintiff have normal coordination?

The cerebellum coordinates muscle actions to produce organized activities such as walking. To test coordination, the doctor can ask the patient to perform rapidly alternating and point-to-point movements; ask the patient to place hands on thighs and then rapidly turn the hands over and lift them off the thighs; and, holding an index finger at arms length

from the patient, ask the patient to touch the patient's nose and then the doctor's finger. This is repeated with patient's eyes open and then with them closed. Nose to finger touching is an example of a point-to-point movement. A patient with a disorder of the cerebellum tends to overshoot the target.

97. Did plaintiff have normal gait (no ataxic gait)?

To test a patient's gait, a doctor can ask the patient to walk across the room. The doctor then watches for normal posture and coordinated arms movements. The doctor can ask the patient to walk heel to toe (tandem gait) across room, to walk on their toes (to test for plantar flexion weakness), and to walk on their heels (to test for dorsiflexion weakness). An ataxic gait is an unsteady, uncoordinated walk, employing a wide base and the feet thrown out.

Evaluation Of Sensory Functions:

98. Did plaintiff have normal tactile sensation?

To test a patient's tactile sensation, a doctor can ask the patient to close her/his eyes, and then touch the patient's fingers and toes lightly with a tissue. The doctor can then ask the patient to identify when they feel the stroke of the tissue.

99. Did plaintiff have normal pain sensation?

To test a patient's pain sensation, the doctor can ask the patient to close his/her eyes, and then touch the patient on the fingers and hand with a safety pin. The doctor alternates the sharp tip with the blunt end to determine whether the patient can tell the difference between sharp and dull sensations. This test may be repeated on the toes.

100. Did plaintiff have normal vibration sense?

To test a patient's vibration sense, the doctor can strike a tuning fork and place it over the base of the nail bed on the patient's index finger. The doctor can then place a finger under the patient's finger to feel the vibration, and ask the patient to identify when they (both) no longer feel the vibration. The doctor will test each side of the body for each extremity and make a comparison. A significant finding during testing is a marked decrease in sensitivity.