LITIGATING TRAUMATIC BRAIN INJURY CLAIMS

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I. INTRODUCTION.

Social forces over the last decade have driven public awareness (and acceptance) of traumatic brain injuries. (Figures 1 and 2) More particularly, the Gulf Wars with their multitude of IED-related blast injuries, and the NFL players' claims for sports-related head injuries have brought publicity to the nature and devastating long-term effects of insults to the brain. These same forces have driven scientific research and medical advances in the diagnosis, treatment, and understanding of traumatic brain injuries. Public awareness and scientific means of objective verification of these injuries have likewise entered the courtrooms of this country. According to the Centers for Disease Control and Prevention, traumatic brain injury (TBI) is a serious health problem in the United States. Each year, traumatic brain injuries contribute to a substantial number of deaths and cases of permanent disability. Studies have estimated that nearly 1.6 million TBI's occur in the United States every year, resulting in over 50,000 deaths and over 70,000 patients with permanent neurological deficits. This paper is intended to provide a basic framework for personal injury law practitioners to assist in the recognition, evaluation, preparation, and presentation of traumatic brain injury claims. This paper will concentrate particularly on "mild" traumatic brain injury (mTBI) claims.

II. WHAT IS TRAUMATIC BRAIN INJURY?

A. Basic Anatomy of the Brain. (Figure 3).

The brain cerebrum is an organ with the consistency of gelatin that is situated within the skull. The brain is floating in cerebrospinal fluid within the hard skull cavity. Portions of the interior of the skull against which the brain is situated are irregular and sharp. The cerebrum has an outer layer of tissue known as the cerebral cortex. The cerebral cortex surrounds the gray matter. The cerebrum is sectionalized into main regions known as lobes. (Figure 4) The major lobes are the Brain Stem, Cerebellum, Occipital Lobe, Parietal Lobe, Temporal Lobe, and Frontal Lobe. Each of these lobes controls different brain functions. Damage to a particular lobe can cause impairment of functions controlled by it. It is also understood that certain of the lobes are within a network of interconnected structures, and damage to one such structure can affect brain functions controlled by other structures within that network. (Figure 4)

1) The Brain Stem is located in the posterior part of the brain. In humans it is usually described as including the medulla oblongata, pons, and midbrain.[1] The brain stem provides the main motor and sensory innervations to the face and neck via the cranial nerves. This is an extremely important part of the brain as the nerve connections of the motor and sensory systems from the main part of the brain to the rest of the body pass through the brainstem. This includes nerves affecting motor, fine touch, vibration sensation, pain, temperature, itch, and crude touch. The brainstem also plays an important role in the regulation of cardiac and respiratory function. It also regulates the central nervous system, and is pivotal in maintaining consciousness and regulating the sleep cycle. The brainstem also controls many basic functions including heart rate, breathing, sleeping, and eating.
2) The Cerebellum is a region of the brain that plays an important role in motor control. It may also be involved in some cognitive functions such as attention and language, and in regulating fear and pleasure responses.[2] It contributes to coordination, precision, and accurate timing. It receives input from sensory systems of the spinal cord and from other parts of the brain, and integrates these inputs to fine tune motor activity.[3] Damage to the cerebella does not cause paralysis, but instead produces disorders in fine movement, equilibrium, posture, and motor learning. Anatomically, the cerebellum has the appearance of a separate structure attached to the bottom of the brain, tucked underneath the cerebral hemispheres.
3) The two Occipital Lobes are the smallest of four paired lobes in the human cerebral cortex. Located in the rearmost portion of the skull, the occipital lobes are part of the forebrain.[4] The occipital lobe is divided into several functional visual areas. Each visual area contains a full map of the visual world.

[1] Definition of Brainstem, THE FREE DICTIONARY (MEDICAL DICTIONARY ED.), http://medical-dictionary.thefreedictionary.com/brainstem (last visited Apr. 30, 2014).

[2] Uri Wolf et al., *Evaluating the Affective Component of the Cerebellar Cognitive Affective Syndrome*, 21 J. NEUROPSYCHIATRY & CLINICAL NEUROSCIENCE 245 (2009), *available at* http://neuro.psychiatryonline.org/article.aspx?articleid=103781.

[3] Edward J. Fine et. al., *The History of the Development of the Cerebellar Examination*, 22 SEMINARS NEUROLOGY 375 (2002), *available at* https://www.thieme-connect.de/DOI/DOI?10.1055/s-2002-36759.

[4] Daniel L. Schacter, *Psychology* (2nd ed. 2010).

If one occipital lobe is damaged, the result is vision loss from similarly positioned "field cuts" in each eye. Occipital lesions can cause visual hallucinations. Lesions in the parietal-temporal-occipital association area are associated with color agnosia, movement agnosia, and agraphia. Damage to the primary visual cortex, which is located on the surface of the posterior occipital lobe, can cause blindness due to the holes in the visual map on the surface of the visual cortex that result from the lesions.[5]

4) The Parietal Lobe is one of the four major lobes of the cerebral cortex in the brain. The parietal lobe is positioned above (superior to) the occipital lobe and behind (posterior to) the frontal lobe and central sulcus. The parietal lobe plays important roles in integrating sensory information from various parts of the body, knowledge of numbers and their relations,[6] and in the manipulation of objects. Its function also includes processing information relating to the sense of touch.[7] Portions of the parietal lobe are involved with visuospatial processing. Damage to the right hemisphere of this lobe results in the loss of imagery, visualization of spatial relationships, and neglect of left-side space and left side of the body. Even drawings may be neglected on the left side. Damage to the left hemisphere of this lobe will result in problems in mathematics, long reading, writing, and understanding symbols.
   The parietal association cortex enables individuals to read, write, and solve mathematical problems.
5) The Temporal Lobe is one of the four major lobes of the cerebral cortex in the brain. The temporal lobe is located beneath the lateral fissure on both cerebral hemispheres of the brain.[8] The medial temporal lobe consists of structures that are vital for long-term memory. The hippocampus is critical for memory formation, and the surrounding medial temporal cortex is currently theorized to be critical for memory storage.[9] Individuals who suffer from medial temporal lobe damage have a difficult time recalling visual stimuli. This is due to the inability to connect the visual stimuli perceived to the visual processing and interpretation centers.[10]
6) The Frontal Lobes are considered our emotional control center and home to our personality. There is no other part of the brain where lesions can cause such a wide variety of symptoms (Kolb &Wishaw, 1990). The frontal lobes are involved in motor function, problem solving, spontaneity, memory, language, initiation, judgment, impulse control, and social and sexual behavior. The frontal lobes are extremely vulnerable to injury due to their location at the front of the cranium, proximity to the sphenoid wing, and their large size. MRI studies have shown that the frontal area is the most common region of injury following mild to moderate traumatic brain injury.[11] The left frontal lobe is involved in controlling language related movement, whereas the right frontal lobe plays a role in non-verbal abilities. Some researchers emphasize that this rule is not absolute and that with many people, both lobes are involved in nearly all behavior. Another area often associated with frontal lobe damage is that of "behavioral spontaneity." In 1981, Kolb & Milner found that individuals with frontal damage displayed fewer spontaneous facial movements, spoke fewer words (left frontal lesions) or spoke excessively (right frontal lesions).[12] One of the most common effects of frontal damage can be a dramatic change in social behavior. A person's personality can undergo significant changes after an injury to the frontal lobes, especially when both lobes are involved. There are some differences in the left versus right

[5] Neil R. Carlson et al., *Psychology: The Science of Behavior* (7th ed. 2010).
[6] Sarah-Jayne Blakemore & Uta Frith, *The Learning Brain: Lessons for Education* (2005).
[7] Wilder Penfield & Theodore Rasmussen, *The Cerebral Cortex of a Man: A Clinical Study of Localization of Function* (1950).
[8] Description of Temporal Lobe, RICE UNIVERSITY, http://www.ruf.rice.edu/~lngbrain/cglidden/temporal.html (last visited May 5, 2014).
[9] Edward E. Smith & Stephen M. Kosslyn, *Cognitive Psychology: Mind and Brain* (2007).
[10] Yoni Pertzov et al., *Binding Deficits in Memory Following Medial Temporal Lobe Damage in Patients With Voltage-Gated Potassium Channel Complex Antibody-Associated Limbic Encephalitis*, 136 BRAIN J. NEUROLOGY 2474 (2013), *available at* http://brain.oxfordjournals.org/content/136/8/2474.full.
[11] Harvey S. Levin et al., *Magnetic Resonance Imaging and Computerized Tomography in Relation to the Neurobehavioral Sequelae of Mild and Moderate Head Injuries*, 67 J. NEUROSURGERY 703 (1987).
[12] Bryan Kolb & Brenda Milner, *Performance of Complex Arm and Facial Movements After Focal Brain Lesions*, 19 NEUROPSYCHOLOGIA 505 (1981)..

> frontal lobes in this area. Left frontal damage usually manifests as pseudodepression and right frontal damage as pseduopyschopathic.[13] Sexual behavior can also be affected by frontal lesions. Orbital front damage can introduce abnormal sexual behavior, while dorolateral lesions may reduce sexual interest.[14]

B. Basic Neuro-Cellular Anatomy.

The brain is comprised of billions of cells. The basic cell is the neuron (Figure 5). It is estimated that the brain contains approximately 20 billion neurons. The neuron has a supporting cast of cells called glial cells. The neurons conduct electrochemical impulses that transmit information in the brain and throughout the central nervous system. Neurons are comprised of the cell nucleus with multiple branching dendrites that receive information from other neurons, and the axon that carries the electrical nerve impulses for transmission to connecting neurons. Neurons are very small and are typically measured in microns (1/10,000 of a mm). The average length of an axon is 1,000 microns and the average diameter is only a few microns. Axonal bundles, however, can have lengths up to 30 centimeters. Neurons communicate via the synapse (Figure 6) located at the tips of the axons and produce and house neurotransmitters which, when released, interface with dendrites of adjacent neurons through electrochemical reactions. "A single neuron may have direct synaptic contact with thousands of other neurons and thereby be involved with an almost unfathomable multiplicity and complexity of functioning synapses underlying behavior and cognition at any given moment." (Lezak, Neuropsychological Assessment, 5th Ed., p. 44.) This explains why an interruption of the functioning of a few neurons can produce significant changes in brain function. (Izhikovich and Edelman, 2008.)

C. Definitions and Classification of Traumatic Brain Injury.

Traumatic brain injuries have been defined and classified by various medical organizations to assist medical practitioners in diagnosing such injuries. The definition of TBI has not been consistent and tends to vary according to specialties and circumstances. Generally, a traumatic brain injury is a non-degenerative, non-congenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions, with an associated diminished or altered state of consciousness. It has also been defined as "an alteration in brain function, or other evidence of brain pathology, caused by an external force." TBI's are often classified by use of the Glasgow Coma Scale as severe (GCS 3-8), moderate (GCS 9-12), or mild (GCS 13-15).

1. The Glasgow Coma Scale.

The Gasgow Coma Scale score for a patient is based upon clinical assessment at the time of the injury. It is a 15 point assessment of eye-opening response, verbal response, and motor response. The accuracy of the results of the assessment depends upon when and by whom it was conducted. This classification system can be misleading as all traumatic injuries to the brain are serious and even those classified as "mild" under this system can result in catastrophic and life-long consequences. As such, this paper will concentrate on "mild" traumatic brain injuries (MTBI), their recognition, diagnosis, and sequelea.

2. ACRM Definition of Mild TBI (mTBI).

The first clear definition of mild traumatic brain injury (mTBI) was developed by the American Congress of Rehabilitation Medicine (ACRM).[15] The ACRM defines mTBI as follows:

> "A patient with mild traumatic brain injury is a person who has had traumatically induced 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;

[13] D. Frank Benson & Dietrich Blumer, *Personality Changes with Frontal and Temporal Lobe Lesions*, in PSYCHIATRIC ASPECTS OF NEUROLOGIC DISEASE 151-170 (1975).

[14] A. Earl Walker & Dietrich Blumer, *The Localization of Sex in the Brain* (1975), *reprinted in* Cerebral Localization (K.J. Zulch et al. eds., 2011).

[15] Thomas Kay, *Neuropsychological Treatment of Mild Traumatic Brain Injury*, J. HEAD TRAUMA REHABILATATION, Sept. 1993, at 75.

- After 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and
- Posttraumatic amnesia (PTA) not greater than 24 hours." (Figure 7)

This definition includes the head being struck, the head striking an object, and the brain undergoing an acceleration/deceleration movement (i.e. whiplash) without direct external trauma to the head. (Figure 8) This definition has gained widespread acceptance and is recognized by many neurologists, psychiatrists, physiatrists, and neuropsychologists.

3. Other Definitions of TBI.

Realizing a clear, concise definition of traumatic brain injury (TBI) was fundamental for reporting, comparison, and interpretation of studies on TBI. The Demographics and Clinical Assessment Working Group of the International and Interagency Initiative toward Common Data Elements for Research on Traumatic Brain Injury and Psychological Health formed an expert group who proposed the following definition:

> TBI is defined as "an alteration in brain function, or other evidence of brain pathology, caused by an external force".[16]
>
> a) *Alteration in brain function* is defined as one of the following clinical signs:
>
> 1) Any period of loss of or decreased level of consciousness;
> 2) Any loss of memory for events immediately before (retrograde amnesia) or after the injury (PTA);
> 3) Neurologic deficits (weakness, loss of balance, change in vision, dyspraxia paresis/plegia (paralysis), sensory loss, aphasia, etc); or
> 4) Any alteration in mental state at the time of the injury (confusion, disorientation, slowed thinking, etc).
>
> b) *or other evidence of brain pathology:* Such evidence may include visual, neuroradiologic or laboratory confirmation of damage to the brain.
> c) *Caused by an external force* may include any of the following events:
>
> 1) The head being struck by an object;
> 2) The head striking an object;
> 3) The brain undergoing an acceleration/deceleration movement without direct external trauma to the head;
> 4) A foreign body penetrating the brain;
> 5) Forces generated from events such as a blast or explosion; or
> 6) Other force yet to be defined.

Because nomenclature and definitions are more likely to be a problem in mild TBI, the criteria presented in this definition under (*A) Alteration in brain function* is compatible with the diagnostic criteria presented in the ACRM definition. This has increased the understanding that both milder insults and less typical presentations now fit under the TBI diagnostic umbrella.

III. SIGNS AND SYMPTOMS OF MILD TBI.

The personal injury practitioner should be knowledgeable of the symptoms of mild TBI and should investigate the facts to determine if they are manifested in the client.

A. When to Look for Signs and Symptoms.

In any case in which the client has sustained a potential trauma to the brain through external force, investigation should be made into the presence of symptoms. The external force can be through a direct physical impact to the skull or through acceleration/deceleration (whiplash) of the head.

B. What Signs and Symptoms to Look For. (Figure 9)

1. Loss of Consciousness.

After establishing that the client sustained an impact to the head from an external force or an acceleration/deceleration event, inquiry should be made into whether the client lost consciousness at the scene. The loss of consciousness need not be prolonged in order to meet the criteria for establishing that a mild TBI occurred. Even a brief loss of consciousness demonstrates that the impact was sufficient to have disrupted and damaged structures and systems in the brain.

[16] David K. Menon et al., *Position Statement: Definition of Traumatic Brain Injury*, 91 ACRHIVES PHYSICAL MED. REHABILITATION 1637 (2010).

2. Amnesia/Confusion.

It is now well recognized that loss of consciousness is not necessary to confirm the client suffered mild TBI. Any loss of memory of events surrounding the incident, any dazed feeling, disorientation, or confusion by the client is sufficient to establish that the external force to the brain resulted in damage to and disruption of functions of the brain.

3. Post-Trauma Effects.

Investigation should be made into the presence of the following symptoms of a TBI:

a) Headaches;
b) Reduced ability to speak clearly;
c) Reduced ability to understand clearly;
d) Reduced ability to focus thoughts or to concentrate;
e) Reduced ability to read;
f) Unusual fatigue;
g) Changes in sleep patterns;
h) Changes in sex life;
i) Unusual temper;
j) Vision changes;
k) Changes in sense of smell;
l) Changes in sense of taste;
m) Irritability, feelings of hopelessness or depression;
n) Reduced ability to perform at school or work;
o) Reduced memory;
p) Reduced ability to sequence or perform complex tasks;
q) Reduced ability to exercise good judgment;
r) Difficulty organizing daily tasks;
s) Problems with balance, dizziness, or vertigo;
t) Reduced ability to perform in a noisy environment;
u) Development of antisocial behavior.[17]

C. Where to look for Signs and Symptoms.

The investigation into the potential signs and symptoms of mild TBI should include interviews and review of the following:

1. Client Interviews.

The client and family members (and potentially close friends, co-workers, and accident scene witnesses) should be interviewed for evidence of any of the signs and symptoms of mild TBI described above.

2. Accident Report.

The accident report may have evidence of the force of impact, the mechanism of injury, loss of consciousness, confusion/amnesia of events, as well as identification of scene witnesses and emergency medical personnel.

3. Accident Witnesses.

Scene witnesses may have evidence of the force of impact, mechanism of injury, loss of consciousness, confusion/amnesia, and client complaints (headaches, dizziness, nausea, etc.).

4. Ambulance/EMT Records and Interviews.

The ambulance crew may have conducted a Glasgow Coma Scale assessment at the scene and/or documented loss of consciousness, amnesia or confusion, bruising, laceration, etc.

5. Hospital Records.

It is important to review not only the initial history, assessment, and diagnosis but also the nursing notes which may contain references to cognitive, emotional, and behavioral symptoms consistent with TBI.

6. Post-accident Medical/Rehab Records from All Practitioners. (Including Physiotherapists, Chiropractors, Massage Therapists, Naturopaths, and Counselors).

These records may contain references to complaints of headaches, dizziness, nausea, memory/concentration problems, and other symptoms consistent with TBI.

7. Prior Medical Records.

These records may contain evidence of a prior brain injury or other medical conditions which would increase the vulnerability to TBI to help explain why the client is one of the 10-20% of victims of TBI that never recover.

8. Scene Photographs.

These photographs depicting damage to vehicles, equipment, etc., may assist in demonstrating the force of impact and mechanism of injury consistent with TBI.

9. School Records.

These records, including standardized testing and performance scores both before and after injury, can demonstrate decreased scholastic aptitude and/or behavioral problems consistent with TBI. These records should be made available to the neuropsychologist for consideration.

[17] Bruce Stern & Dr. Jeffrey Brown, *Litigating Brain Injuries*, Vol. 1 §3.2 (2006).

10. Employment Records.

Job performance, attendance, pay raises, etc., can be contrasted pre- and post-accident to indicate changes consistent with TBI.

IV. MEDICAL EVALUATION OF TBI.

Once the investigation shows that the client probably sustained a TBI, the practitioner should assure that the client receives a thorough medical evaluation of the nature and extent of the client's injury. Often emergency medical treatment of the acute injury will not include such an evaluation. Typically, emergent care of a head trauma will be overseen by a trauma physician and be limited to x-ray, CT scan, and/or MRI which, in many cases, are not effective to detect and define the injury to the brain.

A. Neurological Examination.

The client should be thoroughly assessed by a qualified neurologist in order to evaluate and document the loss of consciousness, amnesia/confusion, and any and all symptoms of the TBI. If warranted, the neurologist can make the referral for neuropsychological and neuroradiological examination of the client to augment the diagnosis and quantify the resulting deficits in brain function.

B. Neuropsychological Examination.

The client should be examined by a qualified neuropsychologist to evaluate the nature and extent of any neurological dysfunction. This should include taking of a history, review of medical records including neuroimaging studies, and the administration and interpretation of a battery of standardized tests. Cognitive deficits and abnormal behavior should be correlated to areas of the brain that control related functions and any clinical abnormalities noted there. These standardized tests measure memory, complex or sequenced tasks, I.Q., reasoning, emotional response, vision, and other brain functions. Where possible, post-morbid levels of function should be compared to pre-morbid levels in order to evaluate the reduction in brain function attributable to the TBI. (Figure 10)

C. Neuroradiological Evaluation.

Various neuroimaging techniques play an imperative role in TBI diagnosis. Certain of these techniques are capable of outlining neuroanatomical abnormalities, as well as cellular and metabolic dysfunction on the microscopic level. Therefore, it is possible to correlate neurophysiological damage caused by TBI with neuropsychological deficits incurred by the client.[18]

1. CT Scan.

Computed Tomography is capable of detecting skull fracture and subarachnoid hemorrhage, and can differentiate acute hemorrhage of the parenchyma from edema or swelling. However, it is not reliable to show specific deficits related to regional damage to the brain. (Figure 11)

2. MRI.

Magnetic Resonance Imaging is the preferred imaging technique for detecting sub-acute and chronic TBI; however, both traditional MRI and CT are not reliable to detect mild TBI microscopic shear injury or metabolic dysfunction on the microscopic level. (Figure 12)

3. FLAIR.

Fluid Attenuated Inversion Recovery uses a pulse to selectively reduce signal from cerebrospinal fluid (CSF). (Figure 13) FLAIR imaging increases the detection of contusions, white matter injuries, and subarachnoid hemorrhages. It also improves the detection of diffuse axonal injuries.[19]

4. DTI.

Diffusion Tensor Imaging measures the random motion of water molecules in brain tissue. (Figures 14 and 15) The white matter tracts are clearly shown by DTI. It also shows disruption in those tracts and is an excellent technique for showing diffuse axonal injury. DTI can reveal pathology where a conventional MRI is negative or normal in appearance.[20]

5. SPECT.

Single Photon Emission Tomography (Figure 16) measures cerebral blood flow in brain tissue.[21] Measuring blood flow is an indirect measurement of brain metabolism. SPECT is highly sensitive for detecting regional blood flow disturbances in patients with TBI. It is particularly more effective than CT or conventional MRI in cases of mild TBI.[22]

6. PET.

Positron Emission Tomography (Figure 17) can evaluate glucose metabolism in various regions of the

[18] Benjamin J. Hayempour et al., *The Role of Neuroimaging in Assessing Neuropsychological Deficits Following Traumatic Brain Injury*, 39 J. PSYCHIATRY LAW 537 (2011).

[19] Tuong H. Le & Alisa D. Gean, *Neuroimaging of Traumatic Brain Injury*, 76 MOUNT SINIAI J. MED. 145 (2009).

[20] Thierry A.G.M. Huisman et al., *Diffusion Tensor Imaging as Potential Biomarker of White Matter Injury in Diffuse Axonal Injury*, 25 AM. J. NEURORADIOLOGY 370 (2004).

[21] Bruce G. Gray et al., *Technetium-99m-HMPAO SPECT in the Evaluation of Patients with a Remote History of Traumatic Brain Injury: A Comparison with X-Ray Computed Tomography*, 33 J. NUCLEAR MED. 52 (1992).

[22] Daniel K. Kido et al., *Traumatic Brain Injuries: Predictive Usefulness of CT*, 182 RADIOLOGY 777 (1992).

brain. Slowed glucose metabolism indicates neuronal dysfunction in that region of the brain. Thus, PET has a significant advantage for illustrating regional brain dysfunction.

7. NeuroQuant®

(Figure 18) is an FDA-approved method of analyzing MRI data in measuring brain volume of a patient and comparing it to normal controls. Brain atrophy or shrinkage is associated with damage to the brain. NeuroQuant® can measure atrophy to various areas of the patient's brain which can then be correlated to the patient's TBI symptoms.[23]

V. EXPERT WITNESSES IN TRAUMATIC INJURY CLAIMS.

The litigation of traumatic brain injury claims can be expert-intensive. The fact of injury, its cause, the nature and extent of the injury, and the past and future damages from the injury can each require expert testimony in order to effectively persuade the trier of fact. As such, expert witness selection, qualification, and presentation are often the battleground in these cases. The following is to assist the practitioner in the selection, preparation, and presentation of these important witnesses.

A. Neurologist.

In any case in which a traumatic brain injury is suspected, it is important to assure that the plaintiff is evaluated by a qualified neurologist. A neurologist is a medical doctor or osteopath who is trained in the diagnosis and treatment of nervous system disorders, including injuries to and diseases of the brain, spinal cord, nerves, and muscles. Neurologists perform neurological examinations of the nerves of the head and neck, movement, balance, sensation, memory, speech, language, and other cognitive abilities. A neurologist qualified to examine and treat a TBI victim should also be familiar with advanced neuroimaging techniques such as CT, MRI, DTI, SPECT, FLAIR, PET, and NeuroQuant®. She should be capable of interpreting the results of such imaging methods and correlation of positive findings with any neurological deficits reported by history and/or neuropsychological testing. The neurologist should be familiar with documented long-term sequelea of traumatic brain injury. That is, the neurologist should understand that TBI is a "disease process" and not a one-time injury. The neurologist should also be capable of diagnosing the TBI and formulating a treatment plan including medications, therapy, and other recommendations for future medical and custodial care. The neurologist should opine as to the causation of the injury, the nature and extent of the injury, its probable duration, and the resultant limitations. The neurologist should interface with the neuroradiologist, neuropsychologist, vocational expert, rehabilitation expert, life care planner, and biomechanical expert.

B. Neuroradiologist.

A neuroradiologist is a medical doctor or osteopath that practices a sub-specialty of radiology focusing on the diagnosis and characterization of abnormalities of the central and peripheral nervous systems, spine, and brain by the use of neuroimaging techniques. The neuroradiologist should be familiar with not only traditional imaging modalities, CT and MRI, but also advanced modalities for the diagnosis of TBI such as DTI, FLAIR, SPECT, PET, and NeuroQuant®. In addition to coordination with the neurologist, the neuroradiologist should be able and willing to coordinate with medical illustrators and video production professionals to assist in the formulation and authentication of exhibits to illustrate the positive neuroradiological findings.

C. Neuropsychologist.

A neuropsychologist is educated and trained to understand the relationship between the brain and emotions, behavior, memory, perception, motivation, and motor skills. The neuropsychologist should be trained in the interpretation of neuroimaging studies and the administration and interpretation of the results of standardized testing of the patient so as to correlate structural abnormalities with resultant brain dysfunctions. The clinician should be able to relate the brain trauma to the abnormality and resulting impairment. The neuropsychologist should also assist in the development of treatment modalities and estimations of the probable effect of brain dysfunction on the patient's future family, social, and work performance. The neuropsychologist should interface with the neurologist, neuroradiologist, biomechanical engineer, and vocational experts.

D. Biomechanical Engineer.

The forensic biomechanical engineer is educated and trained in the analysis of mechanical forces generated from a given impact and the probable disruption caused to anatomical regions of the human body. In cases where it is disputed that a given event would cause a diagnosed traumatic brain injury, a biomechanical engineer should be employed to conduct such an analysis. Typically, in a motor vehicle collision, this analysis will involve a three-step approach. In the first step, the type and severity of the collision is determined, including the change in velocity of the vehicle (delta-v), and the associated

[23] David E. Ross et al., *NeuroQuant® Revealed Hippocampal Atrophy in a Patient with Traumatic Brain Injury*, 24 J. NEUROPSYCHIATRY & CLINICAL NEUROSCIENCES E33 (2012).

acceleration time-history of the occupant compartment. Step two is the occupant dynamics analysis which models the kinematics of the occupant when subjected to the forces determined in step one. Step three, the injury biomechanics analysis, consists of comparing the occupant loading states determined in step two with experimentally determined injury tolerance thresholds of the human body.[24] From this analysis, the biomechanical engineer can opine as to whether or not the collision probably caused the traumatic brain injury that has been diagnosed.

E. Life Care Planner.

In cases where TBI is likely to result in substantial future care needs, a life care planner should be considered for the plaintiff. "The Life Care Plan is a dynamic document based upon published standards of practice, comprehensive assessment, data analysis and research, which provides an organized, concise plan for current and future needs with associated costs for individuals who have experienced catastrophic injury or have chronic health needs."[25] The life care planner should be experienced in planning life care for the traumatic brain injured and have an understanding that TBI is a disease process and not a finite injury with finite sequalea. The life care plan may address the following areas:

- Projected patient evaluations
- Projected therapeutic modalities
- Medication
- Diagnostic testing and educational assessments
- Supply needs
- Wheelchair needs (including accessories and maintenance)
- Home care or facility-based care needs
- Transportation needs
- Projected surgical or aggressive medical needs
- Home furnishings and accessories
- Architectural renovations
- Aides for independent function

The life care planner may be from diverse fields of practice, including rehabilitative medicine, rehabilitative nursing, rehabilitative counseling, psychiatry, case management, and other areas. The planner may also be board certified in life care planning (CLP), which is granted by the Commission on Health Care Certification. The life care planner should interface with the neurologist, neuropsychologist, vocational expert, and other related experts and medical providers for the preparation of the life care plan. The plan, its projected care needs, and associated costs should also be approved and agreed to by the testifying physician. The methodology for preparation of the life care plan may include the following:

- Review of medical records and supporting documents
- Clinical interview with the patient and/or family members for comparison of pre-morbid and post-morbid conditions
- Consultation with medical treatment team
- Research to develop clinical practice guidelines and costs of care, supplies, equipment, etc.
- The life care services and supply costs, as reflected in the Life Care Plan, should then be totaled and reduced to their present value by a qualified economic expert.

F. Vocational/Economic Experts.

Persons that suffer even "mild" traumatic brain injuries can suffer deficits affecting behavior and performance that last for varying periods of time and often last for life. The deficits often result in difficulty comprehending what is read, problems with attention to tasks, and not hearing or understanding what is said by others. These patients cannot process multiple commands or new information. They have memory problems, especially when there are two people talking or there are interruptions. They also complain of dizziness, irritability, anti-social behavior, anxiety, and depression. All of these common complaints can be devastating to affect employment even though the patients, at first, appear normal to the casual observer. These patients often can gain or return to employment following injury, but cannot sustain over time.[26] [27] On average, the brain injured person works less over a life time than the non-injured person and, when they do work, the brain injured person earns less.[28]

A vocational economic analysis compares the injured person's pre-accident earning capacity (based on education, work experience, and earning levels) to

[24] Wilson C. Hayes et al., *Forensic Injury Biomechanics*, 9 ANN. REV. BIOMEDICAL ENGINEERING 55 (2007).

[25] International Association of Rehabilitation Professionals, *Standards of Practice* §1, http://www.rehabpro.org/sections/ialcp/focus/standards/section-i-introduction (last visited May 6, 2014).

[26] Laurence M. Binder, *Persisting Symptoms After Mild Head Injury; a Review of Past Concussion Syndrome*, 8 J. CLINICAL & EXPERIMENTAL NEUROPSYCHOLOGY 323 (1986).

[27] Deborah Abrams et al., *The Economies of Return to Work for Survivors of Traumatic Brain Injuries: Vocational Services are Worth the Investment*, J. HEAD TRAUMA REHABILITATION, Dec. 1993, at 59.

[28] U.S. Bureau of the Census, Series P-23, *Labor Force Status and Other Characteristics of Persons with a Work Disability: 1981-1988*, No. 160, Table 9 (1989).

their post-injury earning capacity (based on medical, neuropsychological, and vocational information) and makes a two-step determination:

1) can the injured person return to competitive employment and, if so, what are the types of employment to which they can realistically return and retain; and
2) if partially or completely disabled, what is the projected loss of earning capacity over their lifetime in terms of "present value".

In determining the foregoing, five key elements are addressed:

- Pre-Injury Earning Capacity: The vocational expert will consider the person's education, training, experience, and earning history to estimate his "capacity" to earn before the brain injury.
- Pre-Injury Work Life Expectancy: The expert will estimate pre-injury with life expectancy based upon the person's education, health status, profession, population averages, and expression of intentions by the person.
- Post-Injury Earning Capacity: The expert will consider the person's post-injury disability and his residual capacity to earn. Statistically, disabled persons earn about 35% less than non-disabled persons.
- Post-Injury Work Life Expectancy: Work life expectancy varies greatly based upon disability status. The expert should consider that brain injuries are progressing in nature and are likely to shorten the person's work life. Studies show that a 45 year old nondisabled worker with 12 years of education has a work life expectancy of 16.5 years while a disabled person with similar age and education has a work life expectancy of 4.2 years.[29]
- Present Value Calculation: The economic expert, whether the same or separate from the vocational expert, should then calculate the "present value" of the pre-injury earning capacity per year multiplied by the pre-injury work life expectancy minus the post-injury earning capacity multiplied by the post-injury work life expectancy.

The vocational expert should be qualified by his education, training, and experience to testify as to the five key elements. The expert typically will have at least a bachelor's degree but preferably a master's or doctoral level degree in economics, accounting, vocational rehabilitation, counseling, or psychology. Board certifications are available through the American Board of Vocational Experts, International Association of Rehabilitation, and the American Rehabilitation Economics Association.

G. Economist.

In cases in which the Plaintiff has suffered a TBI resulting in lost future earning capacity, lost profits from business activities, and/or substantial medical and/or custodial expenses, a qualified economist should be retained. An economist typically has a degree in economics, finance, or accounting and has sufficient education, training, and experience to offer opinions concerning the "present value" of future lost income earning capacity, lost profits, and medical expenses. The economist should review the life care plan and its projected future costs so as to reduce those costs to their present value.

VI. TRAUMATIC BRAIN INJURY AS A DISEASE PROCESS.

Too often, traumatic injury to the brain is seen as "an event". It is often mistakenly treated as a broken bone, the final outcome to an injury of an isolated body system. That is, once it is "fixed" and given some therapy, no further treatments would be needed in the future and, certainly, there will be no affect on other organs of the body. However, brain injury is not an event. It is a disease. It never, ever, ever goes away.[30]

A. TBI Effects on Mortality.

Those suffering a TBI are more likely to die or have a reduced life span.

1) Those with moderate to severe TBI have a 7 year reduction in life expectancy. Those with mild TBI have a small but statistically significant reduction in long term survival.[31]
2) Of 767 subjects with mild-moderate-severe TBI followed for 13 years, those surviving over a year, death rate was 2.5 times higher than controls and 2 times higher than controls for mTBIs.[32]
3) As to the causes of death of TBI victims, they are 37 times more likely to die from

[29] Anthony M. Gamboa Jr., *The New Work Life Expectancy Table*, Tables 1 & 4 (1990).

[30] Brent Masel, *Brain Injury as a Disease*, Address at North American Brain Injury Society (NABIS) (Mar. 19-21, 2014).

[31] Cindy Harrison-Felix et al., *Mortality Following Rehabilitation in the Traumatic Brain Injury Model Systems of Care*, 19 NEUROREHABILITATION 45 (2004).

[32] Thomas McMillan et al., *Death After Head Injury: The 13 Year Outcome of a Case Control Study*, 82 J. NEUROLOGY NEUROSURGERY & PSYCHIATRY 931 (2011).

seizure, 12 times more likely to die from septicemia, 4 times more likely to die from pneumonia, and 3 times more likely to die from other respiratory conditions as were the non-TBI controls.[33]

B. TBI Effects on Morbidity.

Those suffering TBIs are more likely to develop other diseases of the body.

1) TBI Victims have an increased risk of stroke, even higher risk than those with hypertension.[34]
2) TBI is the leading cause of epilepsy in young adults. TBI victims are 1.5 to 17 times more likely to develop seizures than the general population.[35]
3) 70% of chronic TBI out-patients have subjective complaints of sleep disturbances.[36]
4) Patients with TBI are 5 times more likely to develop malignant brain tumors.[37]
5) There is an increased incidence of neuroendocrine dysfunction in the TBI victims. 30% of severe TBIs have hypopituitarism after 1 year.[38]
6) 20% of moderate to severe TBI victims after 1 year have Growth Hormone Dysfunction with increased osteoporosis, high cholesterol, increased abdominal fat, cardiovascular disease, and decreased cognitive functioning.[39]
7) Sexual Dysfunction is reported by 40-50% of individuals post-TBI.[40]
8) There is a higher incidence of psychiatric disease in patients with chronic TBI; 20% report psychosis, 18-61% report depression, 1-22% have mania, 3-59% PTSD, and 20-40% have aggression. TBI is associated with
9) higher rates of suicidal ideation, suicide attempts, and completed suicides.[41]
10) Moderate to severe TBIs in adults result in 2 times the risk of developing Alzheimer's and other forms of dementia later in life.[42]

VII. RESOURCES FOR FURTHER TBI RESEARCH.

This paper is merely a "primer" on the subject of litigation of traumatic brain injuries. Organizations for plaintiff and defense counsel exist that provide in-depth programs and materials on the subject as well as excellent publications for the interested practitioner.

A. Organizations for Plaintiff Counsel.

1) The American Association for Justice (AAJ) has a Traumatic Brain Injury Group that sponsors conferences at least two times a year with excellent topics and speakers. www.justice.org.
2) The North American Brain Injury Society (NABIS) is a society of professionals involved in the issues surrounding brain injury. It provides education programs, scientific updates, and a platform for communication and professional exchange. www.nabis.org.

B. Organizations for Defense Counsel.

The Defense Research Institute (DRI) is the leading organization of defense attorneys and in-house counsel. It sponsors seminars that regularly feature speakers and papers that treat the issues surrounding traumatic brain injury litigation from the defense perspective. www.dri.org.

[33] Cindy Harrison-Felix et al., C*auses of Death Following 1 Year Post Injury Among Individuals with Traumatic Brain Injury*, 21 J. HEAD TRAUMA & REHABILITATION 22 (2006).

[34] James F. Burke, et al., *Traumatic Brain Injury May be an Independent Risk Factor for Stroke*, 81 NEUROLOGY 33 (2013).

[35] John F. Annegars et al., *A Population Based Study of Seizures After Traumatic Brain Injuries*, 338 N. ENG. J. MED. 20 (1998).

[36] Brent Masel et al., *Excessive Day Time Sleepiness in Adults with Brain Injuries*, 821 ARCHIVES PHYSICAL MED. & REHABILITATION 1526 (2001).

[37] Yi-Hua Chen et al., Association *Between Traumatic Brain Injury and the Risk of Brain Cancer*, 29 J. NEUROTRAUMA 1328 (2012).

[38] Harald Jorn Schneider et al., *Hypothalamopituitary Dysfuction Following Traumatic Brain Injury and Aneurysmal Subarachnoid Hemmorage: A Systematic Review*, 298 J. AM. MED. ASS'N 1429 (2007).

[39] Gianluca Aimaretti, *Traumatic Brain Injury and Hypopituitarism*, 5 SCI. WORLD J. 777 (2005).

[40] Nathan Zasler et al., *Brain Injury Medicine* (2006).

[41] Edward Kim et al., *Neuropsychiatric Complications of Traumatic Brain Injury: A Critical Review of the Literature (A report by the ANPA Committee on Research)*, 19 J. NEUROPSYCHIATRY & CLINICAL NEUROSCIENCES 106 (2007).

[42] Scott Gottlieb, *Head Injury Doubles the Risk of Alzheimer's Disease*, BRIT. MED. J., Nov. 4, 2000, at 1100.

C. Publications.

1) Litigating Brain Injuries, Vols. 1 and 2, Stern, Bruce. Thomson-West Publishing. 2006.
2) Neuropsychological Assessment, Lezak, et al., 5th Edition, Oxford University Press. 2012.
3) The Handbook of Clinical Neuropsychology, Gurd, et al. Oxford University Press. 2012.
4) The Handbook of Functional Neuroimaging of Cognition. Cabeza, et al, Second Edition.

VIII. CONCLUSION.

Recent events have brought public awareness to traumatic brain injuries and their devastating effects on the lives of victims, including war heroes and athletic legends. Scientific advances in neuroimaging have enabled objective verification of many of the injuries. Even so called "mild" traumatic brain injuries (which may go undetected during emergent care of the acute injury) can have life-long effects. The prudent practitioner must be vigilant in identifying these injuries and thorough in the assessment and presentation of them. It is imperative that personal injury lawyers understand the symptoms and science related to TBI and the proper modalities for verifying and explaining such injuries.

APPENDIX

Table of Figures

Figure 1:

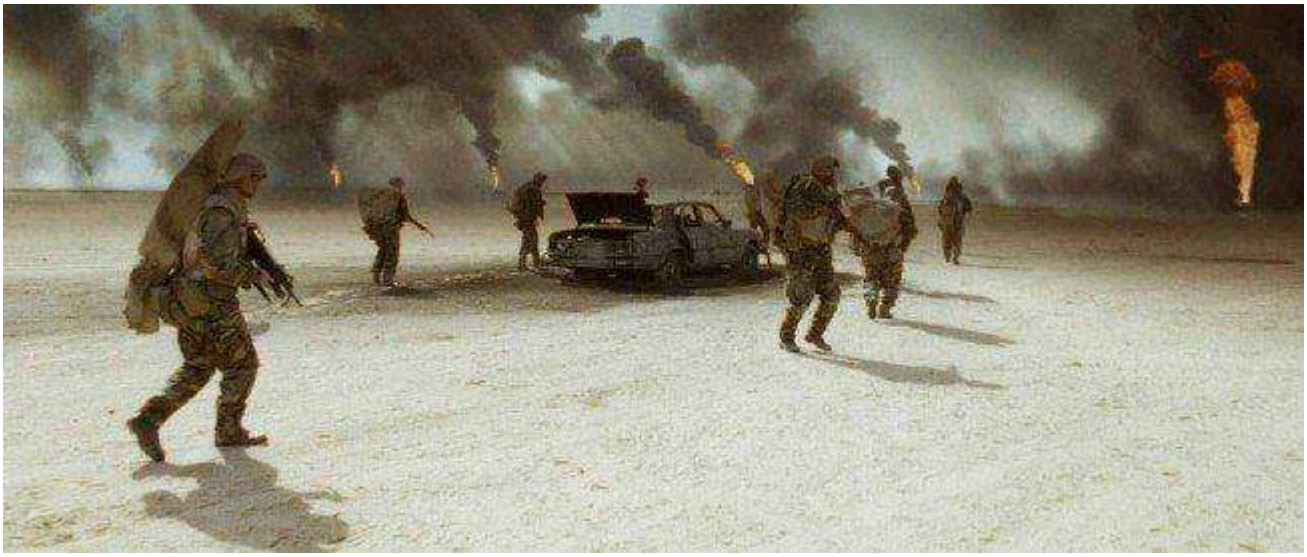


Figure 2:

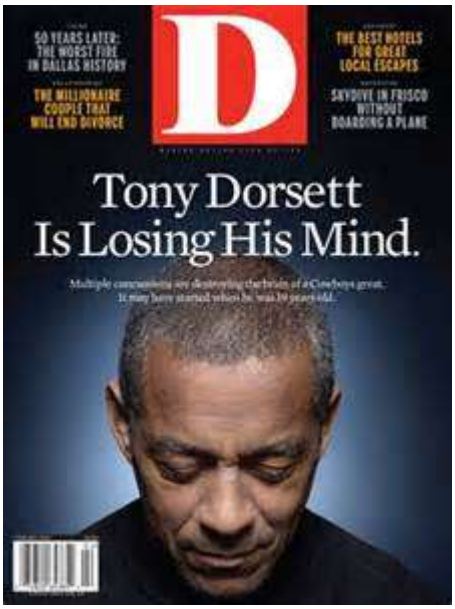

50 YEARS LATER: THE WORST FIRE IN DALLAS HISTORY
THE MILLIONAIRE COUPLE THAT WILL END DIVORCE
D
THE BEST HOTELS FOR GREAT LOCAL ESCAPES
SKYDIVE IN FRISCO WITHOUT BOARDING A PLANE
Tony Dorsett Is Losing His Mind.

Figure 3:

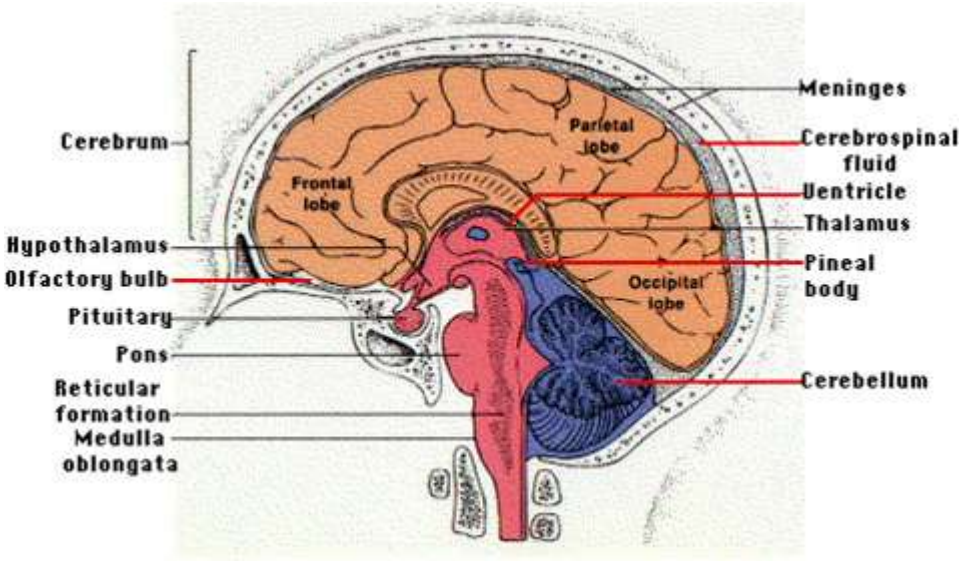

Meninges
Cerebrum
Parietal lobe
Cerebrospinal fluid
Frontal lobe
Ventricle
Thalamus
Hypothalamus
Pineal body
Olfactory bulb
Occipital lobe
Pituitary
Pons
Cerebellum
Reticular formation
Medulla oblongata

Figure 4:

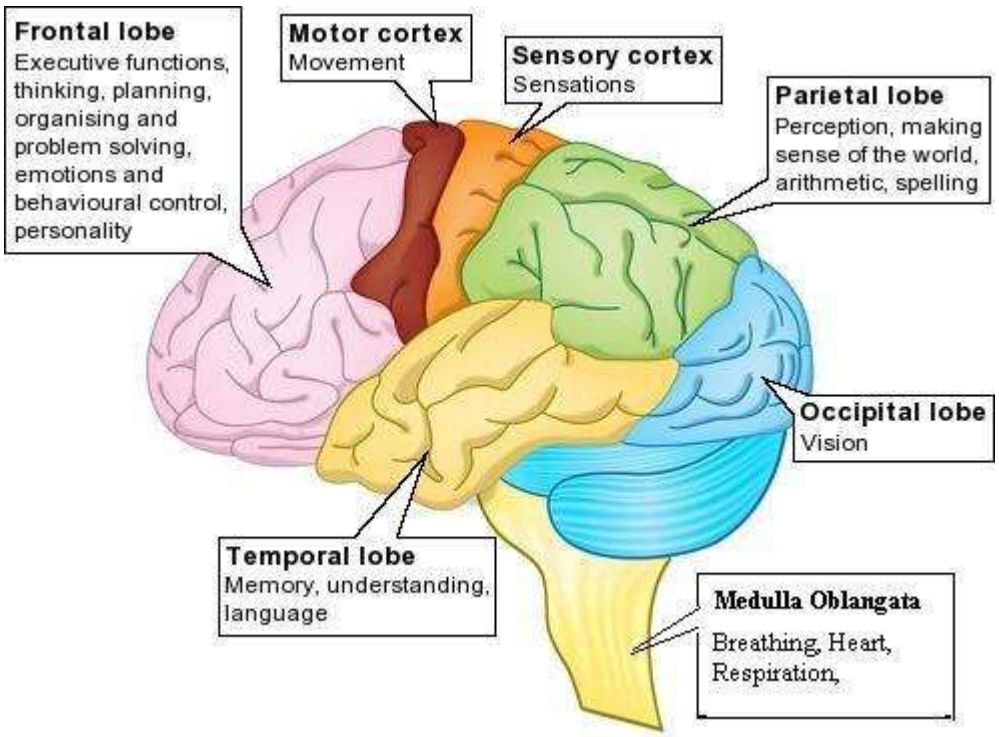

Frontal lobe
Executive functions, thinking, planning, organising and problem solving, emotions and behavioural control, personality
Motor cortex
Movement
Sensory cortex
Sensations
Parietal lobe
Perception, making sense of the world, arithmetic, spelling
Occipital lobe
Vision
Temporal lobe
Memory, understanding, language
Medulla Oblangata
Breathing, Heart, Respiration,

Figure 5:

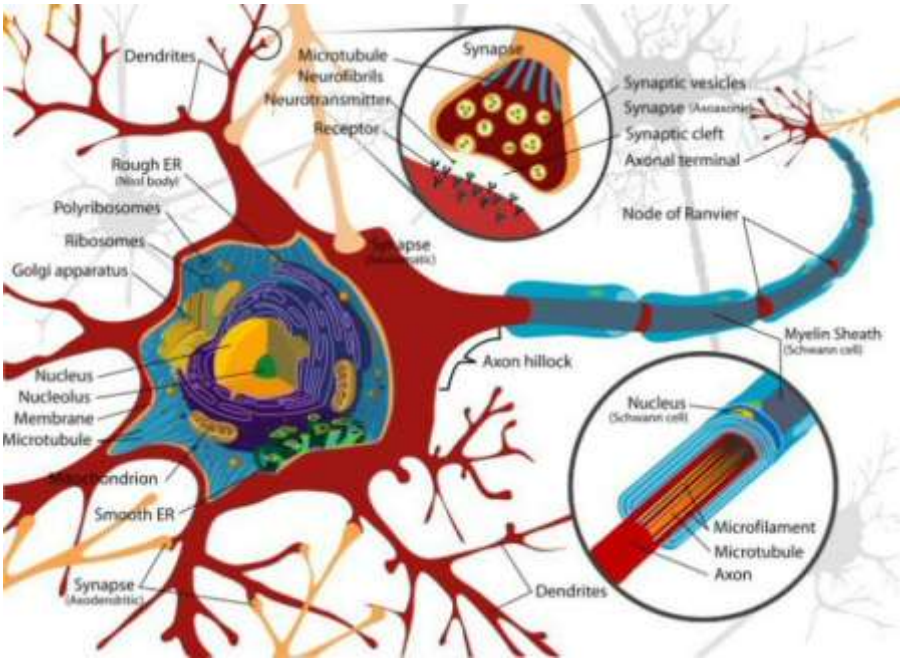

Dendrites
Microtubule
Neurofibrils
Neurotransmitter
Receptor
Synapse
Synaptic vesicles
Synapse
Synaptic cleft
Axonal terminal
Rough ER
(Nissl body)
Polyribosomes
Ribosomes
Golgi apparatus
Node of Ranvier
Myelin Sheath
(Schwann cell)
Axon hillock
Nucleus
Nucleolus
Membrane
Microtubule
Mitochondrion
Smooth ER
Synapse
(Axodendritic)
Nucleus
(Schwann cell)
Microfilament
Microtubule
Axon
Dendrites

Figure 6:

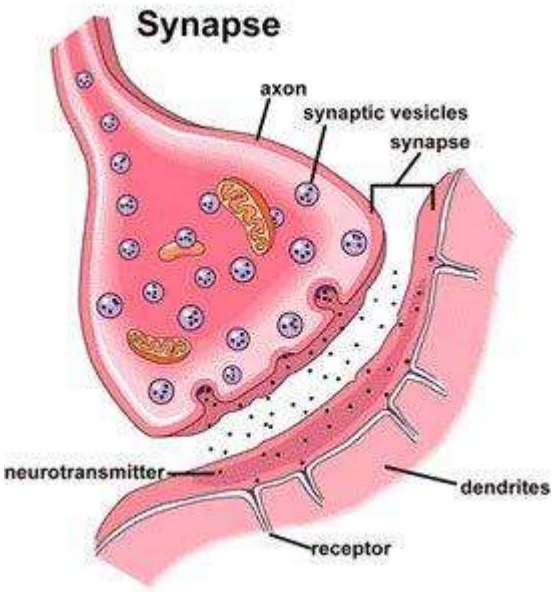

Synapse
axon
synaptic vesicles
synapse
neurotransmitter
dendrites
receptor

Figure 7:

ACRM* Definition of Mild TBI

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

1. Any period of loss of consciousness;

2. Any loss of memory for events immediately before or after the accident;

3. Any alteration in mental state at the time of the accident (e.g. feeling dazed, disoriented or confused); and

4. 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) of 13-15; and
- Post-traumatic amnesia (PTA) not greater than 24 hours."

Figure 8:

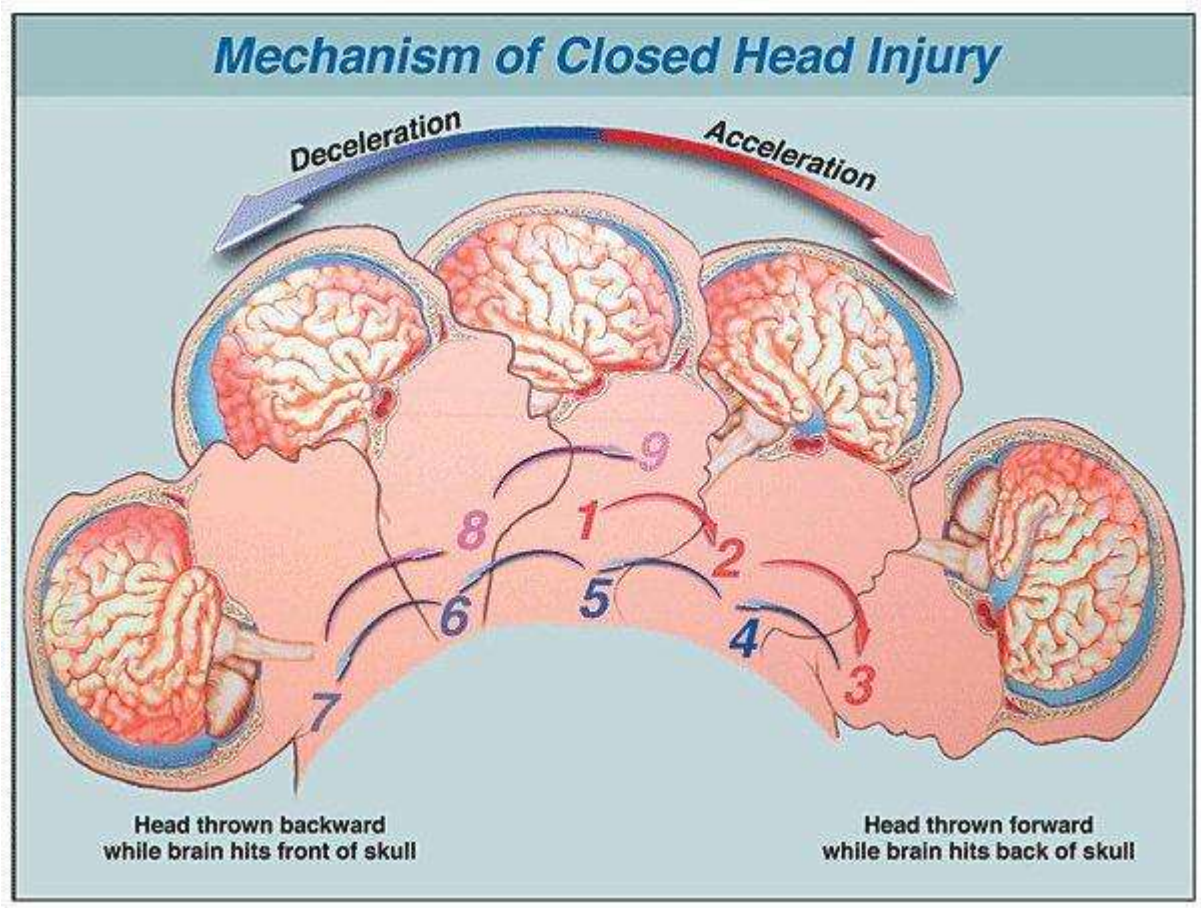

Mechanism of Closed Head Injury
Deceleration
Acceleration
9
8
1
2
6
5
4
7
3
Head thrown backward
while brain hits front of skull
Head thrown forward
while brain hits back of skull

Figure 9:

THINKING/ REMEMBERING	PHYSICAL	EMOTIONAL/ MOOD	SLEEP DISTURBANCE
• Difficulty thinking clearly • Feeling slowed down • Difficulty concentrating • Difficulty remembering new information	• Headache • Nausea or vomiting (early on) • Balance problems • Dizziness • Fuzzy or blurry vision • Feeling tired, having no energy • Sensitivity to noise or light	• Irritability • Sadness • More emotional • Nervousness or anxiety	• Sleeping more than usual • Sleeping less than usual • Trouble falling asleep

Figure 10:

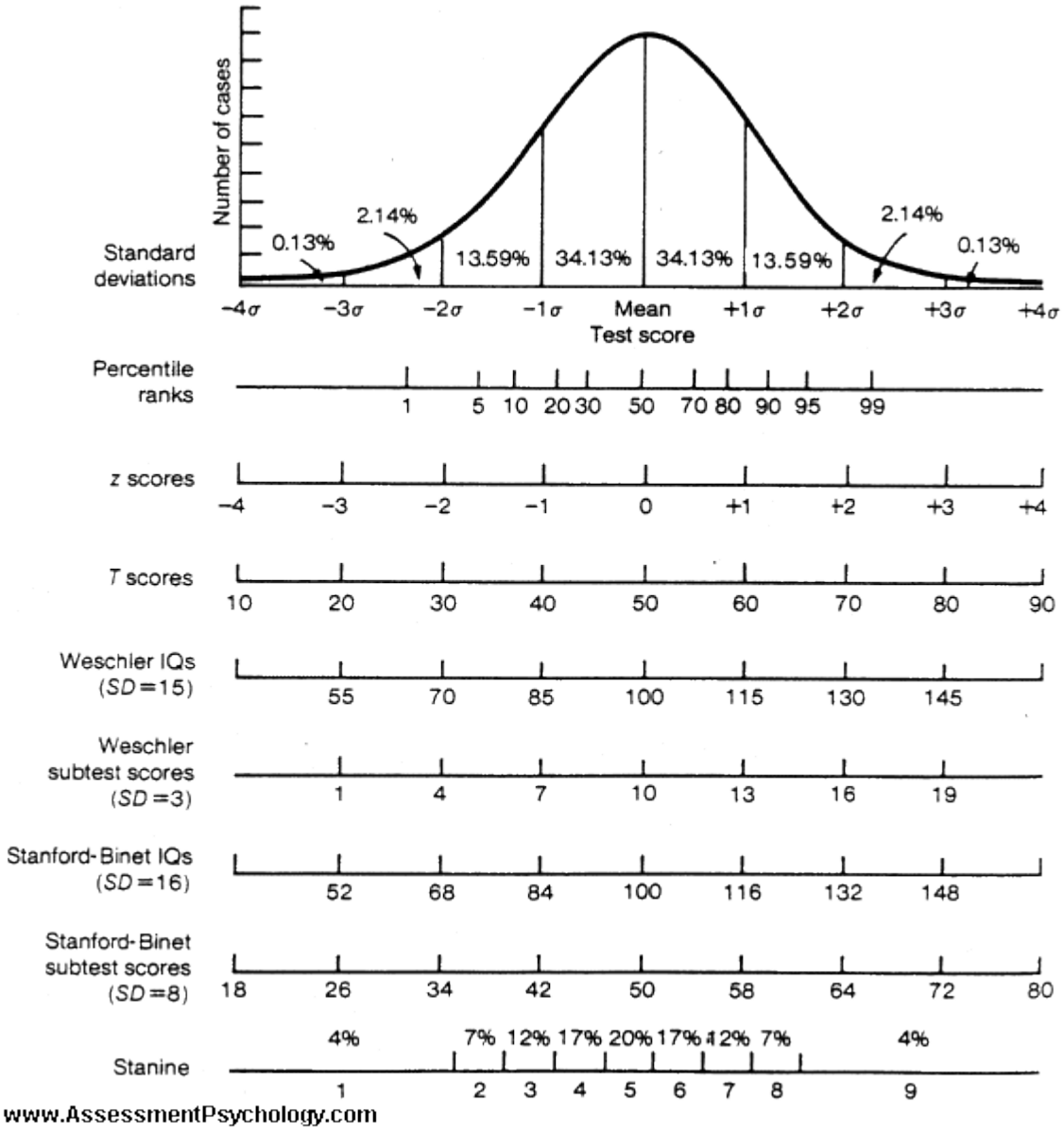

Number of cases
0.13%
2.14%
13.59%
34.13%
34.13%
13.59%
2.14%
0.13%
Standard deviations
−4σ −3σ −2σ −1σ Mean +1σ +2σ +3σ +4σ
Test score
Percentile ranks
1 5 10 20 30 50 70 80 90 95 99
z scores
−4 −3 −2 −1 0 +1 +2 +3 +4
T scores
10 20 30 40 50 60 70 80 90
Weschler IQs (SD = 15)
55 70 85 100 115 130 145
Weschler subtest scores (SD = 3)
1 4 7 10 13 16 19
Stanford-Binet IQs (SD = 16)
52 68 84 100 116 132 148
Stanford-Binet subtest scores (SD = 8)
18 26 34 42 50 58 64 72 80
Stanine
4% 7% 12% 17% 20% 17% 12% 7% 4%
1 2 3 4 5 6 7 8 9
www.AssessmentPsychology.com

Figure 11:

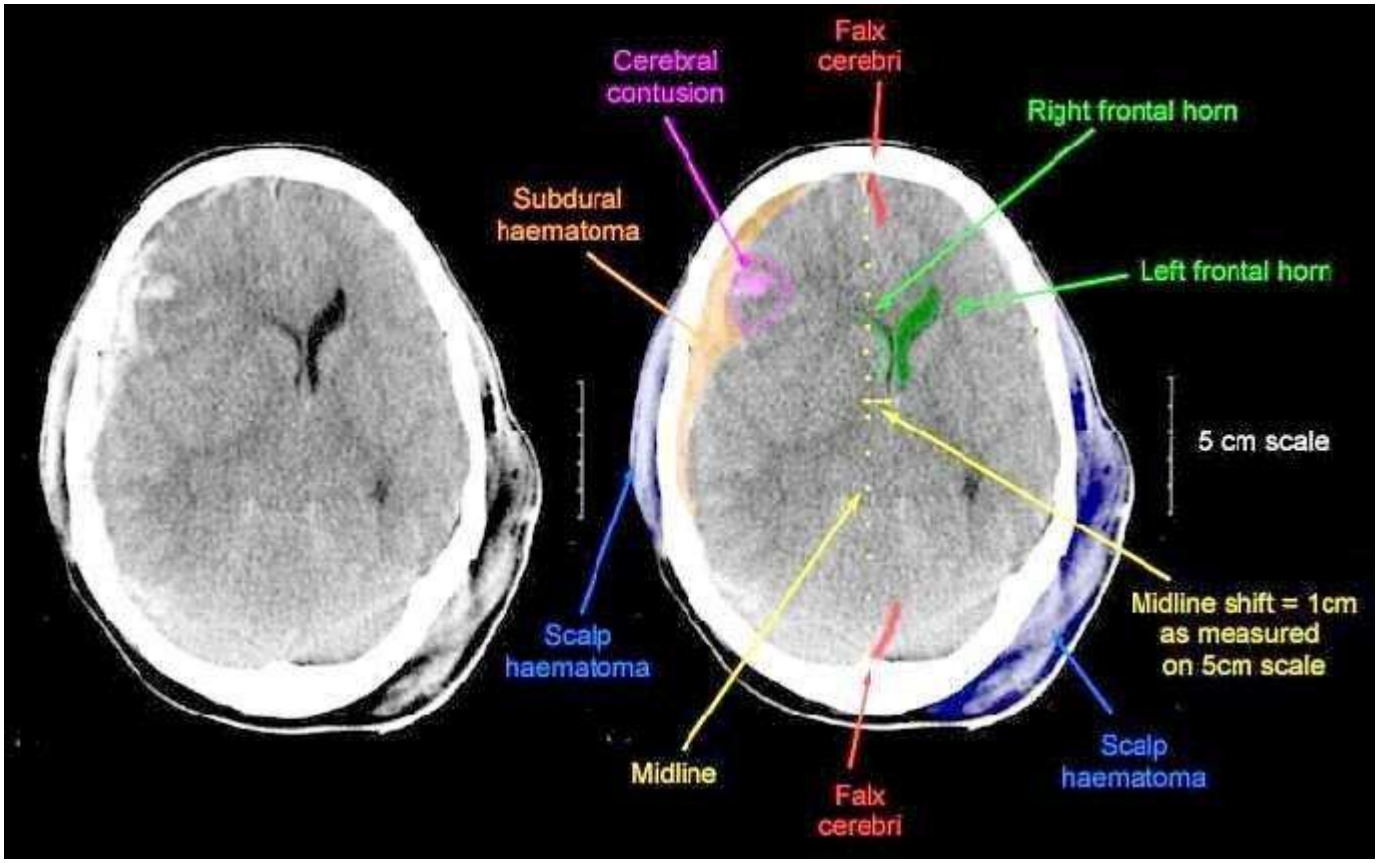
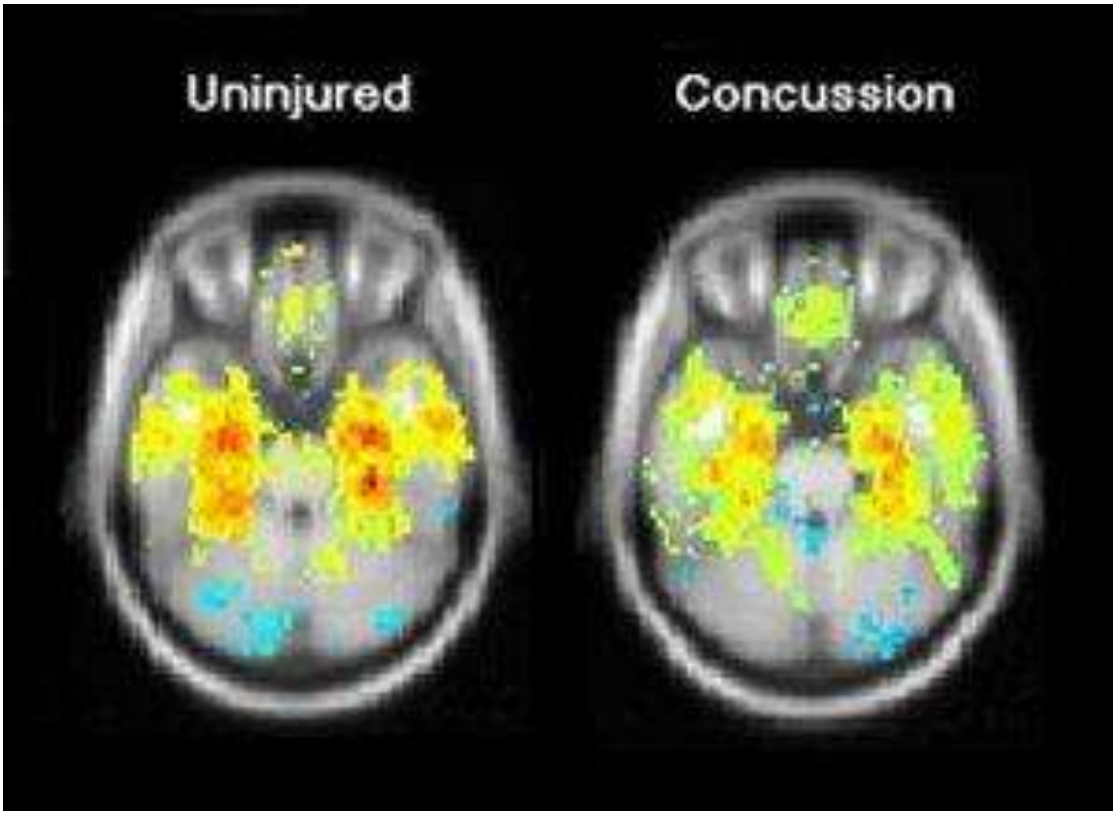

Cerebral contusion
Falx cerebri
Right frontal horn
Subdural haematoma
Left frontal horn
5 cm scale
Midline shift = 1cm as measured on 5cm scale
Scalp haematoma
Midline
Falx cerebri
Scalp haematoma

Figure 12:



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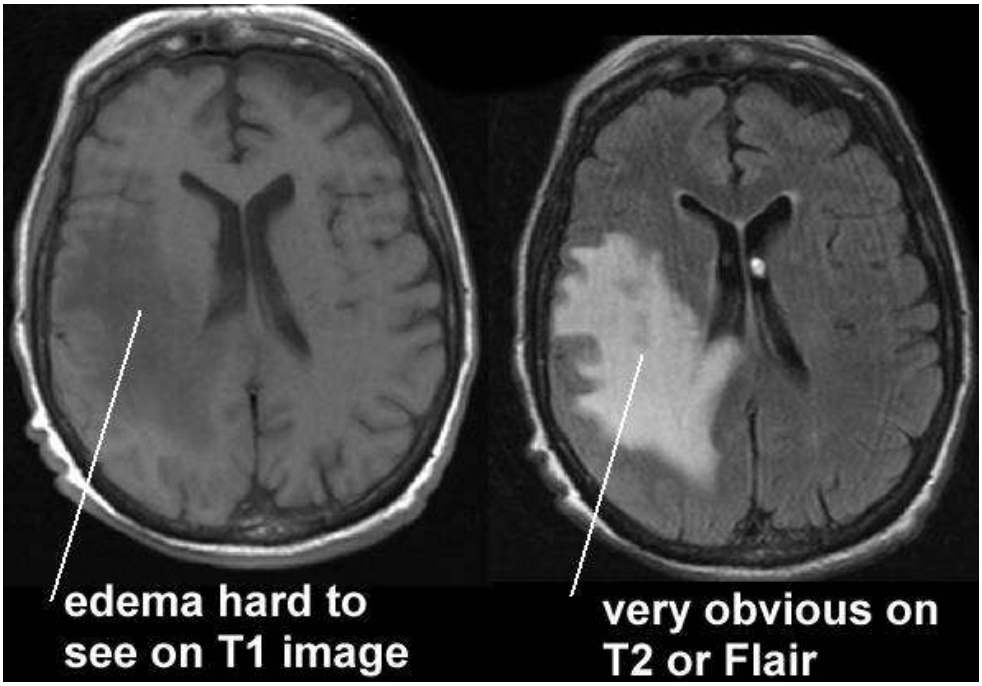
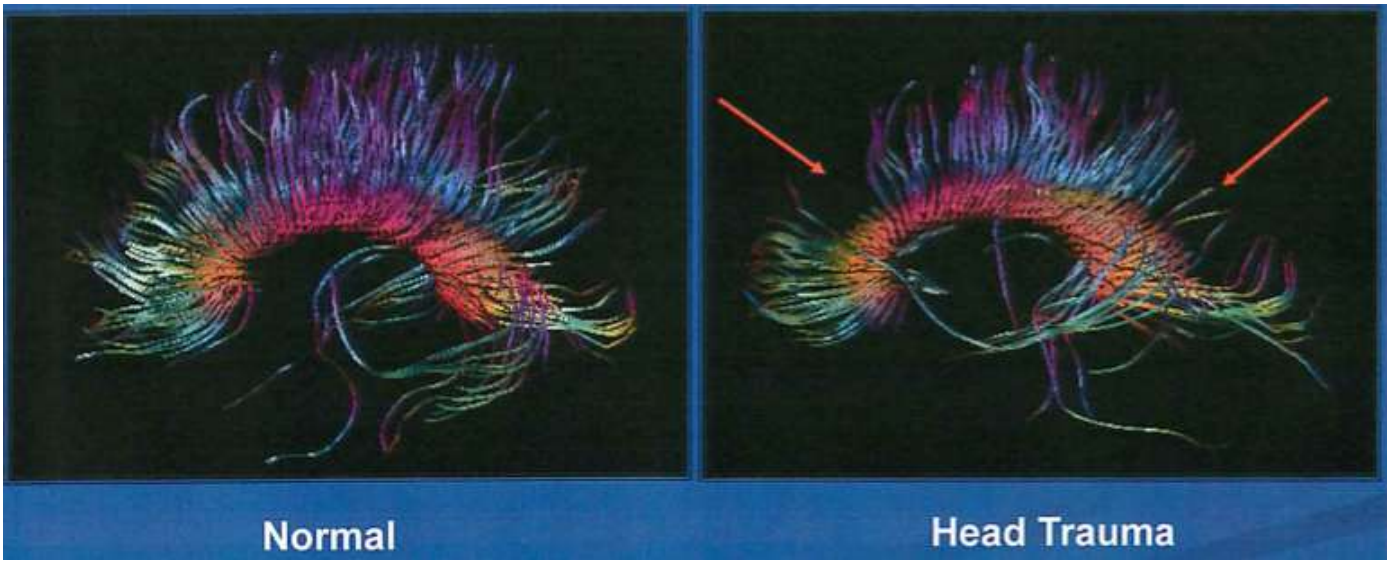


Figure 14:

Figure 15:



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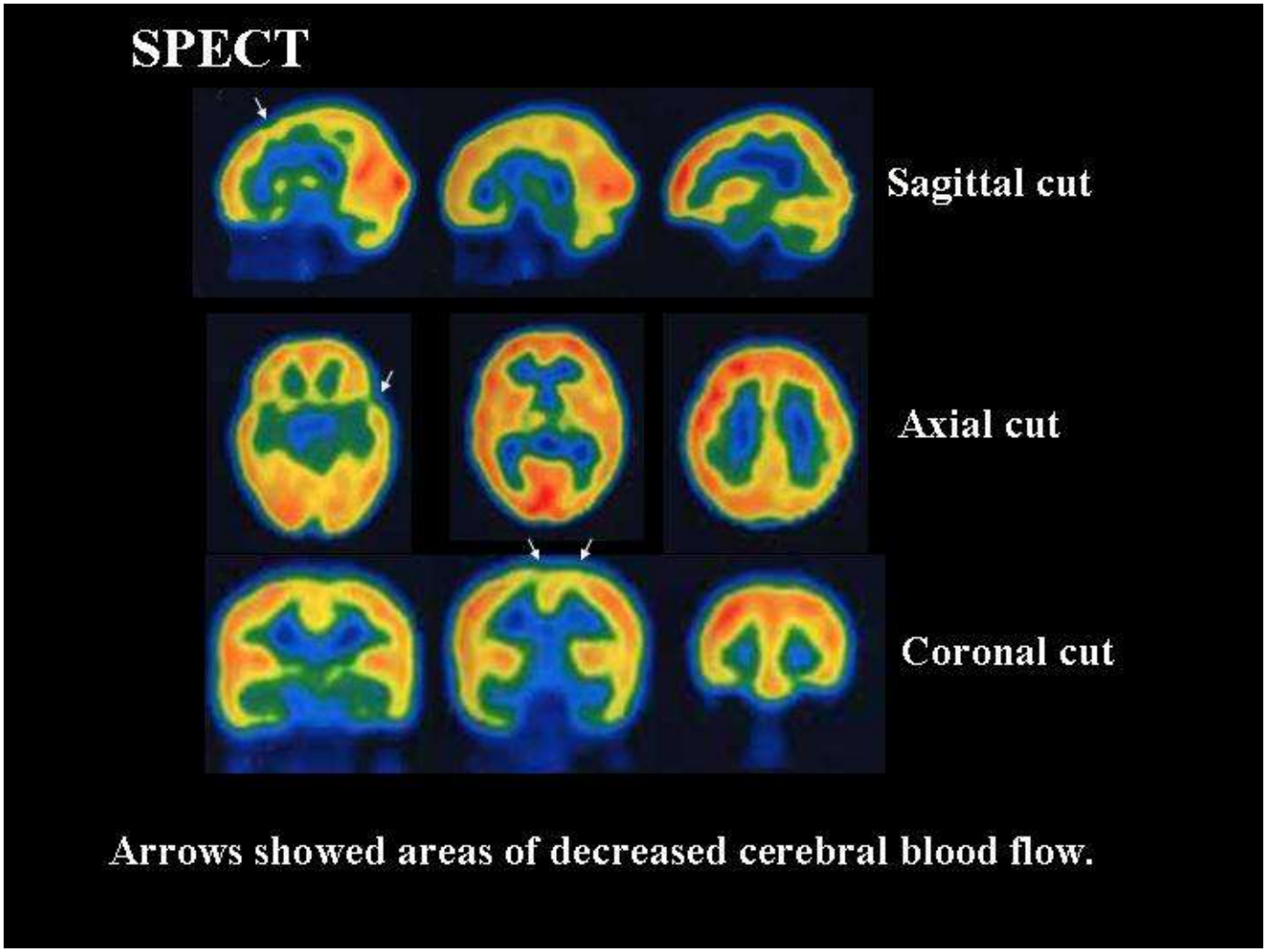
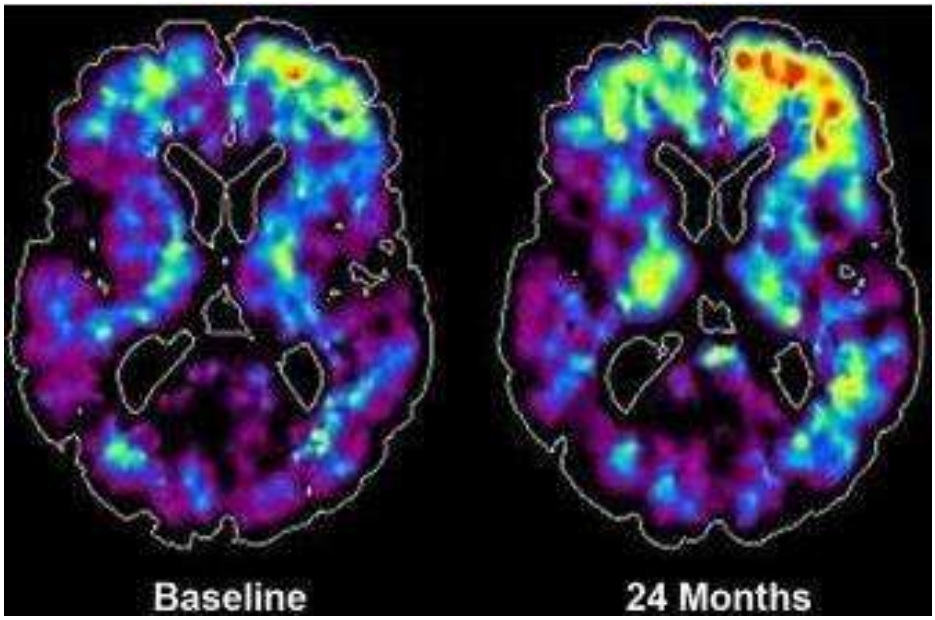

SPECT
Sagittal cut
Axial cut
Coronal cut
Arrows showed areas of decreased cerebral blood flow.

Figure 17:



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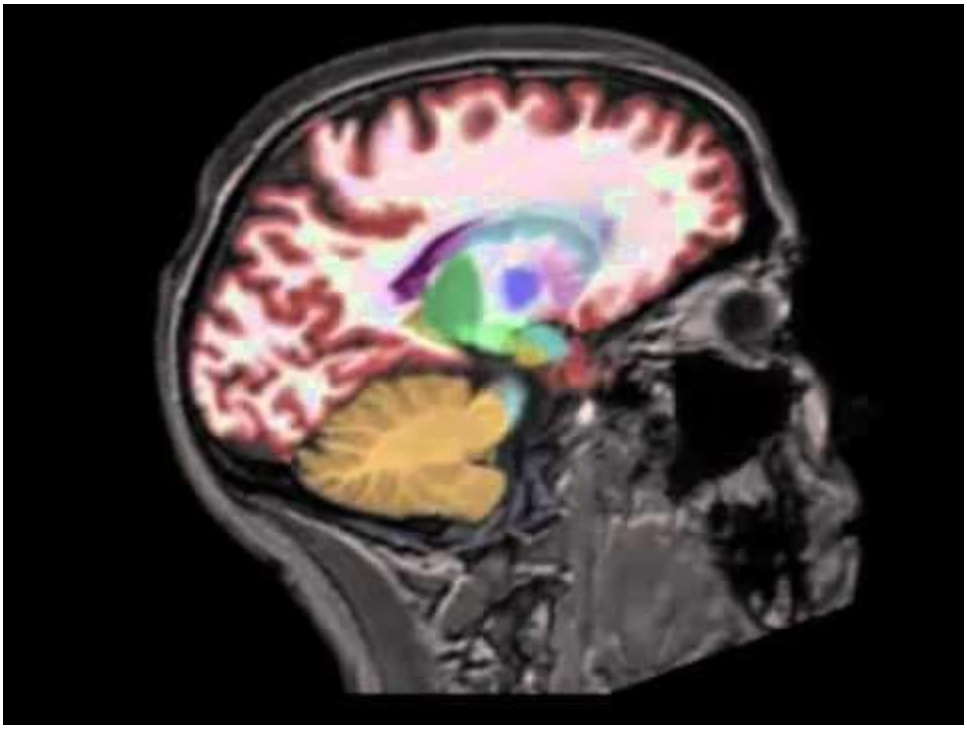

Figure 19:


LOST
CONFUSED
UNSURE
UNCLEAR
PERPLEXED
DISORIENTED
BEWILDERED