Traumatic Brain Injury Claims
Assessing Claims, Negotiating Settlements, and Effectively Using Witnesses

FRIDAY, NOVEMBER 9, 2012

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

Today’s faculty features:

Dr. Glenn T. Goodwin, Consulting Neuropsychologist, Edmonds, Wash.
Paul Zukerberg, Founder, Zukerberg Law Center, Washington, D.C.
John Jerry Glas, Partner, Deutsch, Kerrigan & Stiles, New Orleans
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A Neuropsychological Approach for Evaluation of Persistent Postconcussive Syndrome

Postconcussive complaints are a common sequelae following mild traumatic brain injury (MTBI). Postconcussive symptomatology is understood to represent the interactive and synergistic effects of physical, cognitive and psychological sequelae. However, postconcussive syndrome (PCS) often lacks uniform specificity, and symptoms do not cohere with sufficient reliability to form a true syndrome. Postconcussive type symptoms are reported and may be observed in many other clinical populations and non-head injury litigating individuals (Fox, 1995, Lees-Haley & Brown, 1993). Subjective complaints alone are not considered to be a reliable basis for evaluating PCS. Research review of postconcussive studies, (Gasquoine, 1997) summarizes the five most commonly reported symptoms to be headache, dizziness, irritability, decreased concentration and memory problems. Headache, dizziness, nausea in the ER after MTBI is strongly associated with the severity of PCS at six months after injury. Although the reports of duration of postconcussive symptomatology vary from one study to the next, as a general rule, the pattern of symptoms tends to decrease substantially over the first twelve months following injury, with a majority of patients experiencing improvement within the first three to six months.

A smaller percentage of patients continue to have a more protracted recovery and develop persistent postconcussive syndrome (PPCS). While there is no clear consensus for defining PPCS, typically this population of patients displays persistence of symptoms beyond one year and longer. Comprehensive neuropsychological examination is essential in helping to verify and provide objective documentation of legitimate neurocognitive and neurobehavioral symptoms of MTBI that may be contributing to PPCS, as well as providing differential diagnosis of potential secondary psychological complications such as posttraumatic stress, pain disorder, somatoform disorder, adjustment disorder, anxiety and depression.

A variety of neuropsychological outcome studies (Alves, 1986, Dikmen, 1995, Lees-Haley & Brown, 1993) have investigated the etiology of persistent neurocognitive and psychological sequelae following MTBI, with general indications of multiple factors contributing to prolonged recovery as time goes on. Meta-analytic review (Binder, 1997) of eleven studies revealed minimal primary effect of history of mild traumatic brain injury on persistent neurocognitive residuals. Among the factors that can be contributing to the
maintenance of PPCS are on-going orthopedic problems and chronic pain, lower educational status, unstable occupational status, history of premorbid vulnerabilities, age, gender, posttraumatic stress, somatization, effects of litigation, and malingering.

Although most patients with an uncomplicated mild traumatic brain injury can expect a favorable prognosis, research has consistently reported that there are a small group of patients (typically 5-15%) who may have less favorable or incomplete recovery. This unfortunate minority may experience primary neurocognitive and neurobehavioral residuals related to the history of mild traumatic brain injury. All patients who are experiencing some persistence of postconcussive symptomatology should have the opportunity of neuropsychological consultation to provide a more comprehensive diagnostic assessment.

The Task for the Consulting Neuropsychologist

- The starting position should be one of "clinical neutrality"
- An opportunity to review and examine the injury issues within the context of all available background information
- To determine the probability of specific factors that may be contributing to the persistence of residual symptomatology
- To what extent is PCS related to the MTBI incident, chronic pain, PTSD, anxiety, depression, personality style or other injury and accident related factors?
- To what extent are non-injury factors contributing to the maintenance of PCS
- Differential Diagnosis

Why get a Neuropsychological Consultation?

Subjective complaints alone are not a reliable or valid basis for assessing postconcussive symptoms and sequelae of mild traumatic brain injury.

Neuropsychological examination is essential in helping to verify residual neuropsychological symptoms of mild traumatic brain injury and injury related sequelae from other factors.

Pre-existing conditions and vulnerabilities are almost always factors that should be identified and considered in explaining current functioning after accident or injury.
By its very nature and methodology, neuropsychological examination addresses the issues of:

- Postconcussive syndrome
- PTSD, anxiety/depression, psychological overlay
- Effects of pain, sleep disturbance, medications
- Frontal Lobe Injury
- Premorbid issues and vulnerability
- Primary and secondary gain
- Malingering

The contextual environment of standardized neuropsychological testing may not always be analogous to the complexities and rigor of everyday life, where executive functioning, multitasking, processing speed and levels of concentration may be more diverse and demanding. It is imperative to look beyond the quantitative assessment and test scores and probe into the functional capacity and behavioral dynamics of patients who have persistent symptomatology.

References


**Duration of Postconcussive Syndrome**

- There is a general trend reported in the research literature of diminishing symptomatology with time, but no definitive consensus.
- Studies report a few weeks to years following injury.
- Less postconcussive symptomatology in severe head injury than in mild head injury.

Persistence of symptoms typically involves multiple factors.
By its very nature and methodology, neuropsychological examination addresses the issues of:

- Postconcussive syndrome
- PTSD, anxiety/depression and psychological overlay
- Effects of pain, sleep disturbance, medications
- Frontal lobe Injury
- Premorbid issues and vulnerability
- Primary and secondary gain
- Malingering

**Posttraumatic Stress Disorder (PTSD) and Mild Traumatic Brain Injury (MTBI)**

- PTSD may be superimposed on a legitimate MTBI resulting in deficits above and beyond brain injury.
- PTSD may masquerade as MTBI, confusing the diagnosis.
- Trauma may reactivate unresolved emotional trauma from the past, again creating a symptom picture that mimics MTBI, but is not MTBI.

**Postconcussive Symptoms and PTSD after MTBI**

- Survivors of MVA who either sustained a MTBI (N = 46) or no MTBI (N = 59) were assessed 6 months post-trauma for PTSD and postconcussive symptoms.
- Postconcussive symptoms were more evident in MTBI patients with PTSD than those without PTSD.
- Postconcussive symptoms more evident in MTBI patients than non-MTBI patients.
- CONCLUSION: postconcussive symptoms may be mediated by an interaction of neurological and psychological factors after MTBI.

*Bryant RA, Harvey AG. (1999) J Nervous and Mental Disease, May, 187, (5)*
CRITICAL REVIEW

Neuropsychology and clinical neuroscience of persistent post-concussive syndrome

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Abstract

On the mild end of the acquired brain injury spectrum, the terms concussion and mild traumatic brain injury (mTBI) have been used interchangeably, where persistent post-concussive syndrome (PPCS) has been a label given when symptoms persist for more than three months post-concussion. Whereas a brief history of concussion research is overviewed, the focus of this review is on the current status of PPCS as a clinical entity from the perspective of recent advances in the biomechanical modeling of concussion in human and animal studies, particularly directed at a better understanding of the neuropathology associated with concussion. These studies implicate common regions of injury, including the upper brainstem, base of the frontal lobe, hypothalamic-pituitary axis, medial temporal lobe, fornix, and corpus callosum. Limitations of current neuropsychological techniques for the clinical assessment of memory and executive function are explored and recommendations for improved research designs offered, that may enhance the study of long-term neuropsychological sequelae of concussion. (JINS, 2008, 14, 1–22.)

Keywords: Concussion, Mild TBI, Biomechanics, Neuroimaging, Neuropathology, Neuropsychology

INTRODUCTION: BRIEF HISTORY OF CONCUSSION

That concussion occurs and is commonplace is not in dispute. The United States Government’s Center for Disease Control (CDC) estimates that there are more than one million concussions that occur annually in the United States, using their definitional statement of concussion being a condition “of temporarily altered mental status as a result of head trauma (www.cdc.gov, see Rutland-Brown et al., 2006).”

What is controversial is whether one fully recovers without symptoms from having sustained a concussion. Given the commonness of concussions along with the adaptive nature of brain function combined with neural plasticity (Duffau, 2006; Giza & Prins, 2006; Moucha & Kilgard, 2006; Priestley, 2007), it might be assumed that any transient impairment as a result of concussion would not result in any neurological sequelae. Indeed, historically the original Latin term “commotio cerebri” was used to describe concussion, thought to occur because of “traceless disturbances” that produced momentary functional impairment without any damage to brain tissue (see reviews by McCrory & Berkovic, 2001; Vos et al., 2002). Hence, for decades, one of the venerable definitions in standard neurology textbooks, exemplified by the following quote from Grinker’s Neurology was as follows: “the usual patient loses consciousness briefly, soon recovers and thereafter is without symptoms” (Vick, 1976; p. 651). In that concussion was thought to be mostly benign, the non-biological and psychodynamic theories that dominated the beginnings of clinical psychology and psychiatry minimized the effects head injury could have on behavior. This is captured by the 1947 quote by Page (1947) in an abnormal psychology textbook that “Head injuries and gunshot wounds involving damage to the brain occasionally produce mental disturbances, but such injuries are not an important cause of mental disease (p. 330)”. Persistent maladaptive symptoms in this time frame were believed to be more an expression of a “neurosis” than anything possibly “organic.” So,
“persistent” problems following concussion were interpreted within a psychogenic framework. In fact, one of the most cited publications in the clinical literature on concussion is that of Miller (1961) whose series of articles centered on the theme of concussion being nothing more than “Accident Neurosis”, which others have labeled as “Compensation Neurosis” (Levy, 1992) because of the prevalence of lawsuits involving mild head injury (Hall & Chapman, 2005; Mooney et al., 2005). No doubt “psychological” factors play an important role in the residuals from concussion (Meares et al., 2006; Whittaker et al., 2007; Wood, 2007), but they and other “functional” factors are also the source of intense debate and controversy over the existence of post-concussive symptomatology (Cantu, 2007; Evans, 1994; King, 2003). These controversies will be discussed in greater detail later in this review.

Part of this controversy has to do with nomenclature and definition. Years ago, Vick (1976) also stated that terms like concussion are “. . . of little value” because of “such wide and indefinite connotations” (p. 650). Much has been written about the definition of concussion (Blostein & Jones, 2003; Boake et al., 2005; Ruff & Jurica, 1999), including the definitional statements by major organizations and consensus panels as presented in Table 1, wherein the terms concussion and mild traumatic brain injury (mTBI) are used interchangeably. For this review, in referring to studies, if the authors used the term concussion then that term will be used in referring to the study and likewise, if the term mTBI is used by the authors, that term will be used; otherwise, mTBI and concussion will be used interchangeably in the current review. However, this review focuses on the persistence of symptoms following concussion or what has been referred to as post-concussive syndrome (PCS), but this term then brings up additional controversies. In the majority of those concussed, symptoms abate within minutes to hours to days post-injury. Thus, some refer to PCS if the symptoms persist for more than a few days and in particular, if the symptoms persist for more than a week (Anderson et al., 2006; Sheedy et al., 2006). If the symptoms last more than 3 months then the term persistent post-concussive syndrome or PCS has been used (Begaz et al., 2006; Chameilian et al., 2004; Iverson, 2006; Rees, 2003; Satz et al., 1999; Stalnacke et al., 2005; Willer & Leddy, 2006). Whereas there is a relationship between severity of concussion and who develops PCS (Hessen et al., 2006), concussion severity by itself is a poor predictor of who develops PCS (Guskiewicz et al., 2004).

DSM-IV lists PCS as a disorder under its “research” classification and some have referred to it as a syndrome (King, 2003; Rees, 2003; Ryan & Warden, 2003) and there are differences in symptom criteria between DSM-IV and the International Classification of Disorders (ICD-10) that further cloud this taxonomy issue (Kashluba et al., 2006; McCauley et al., 2005). Whether PCS is a disorder or syndrome is another ongoing debate (Hall & Chapman, 2005; Smith, 2006). Neither DSM-IV or ICD use the PCS label. Nosological issues are not the focus of this review and there are several excellent recent reviews on this topic ((Hall & Chapman, 2005; Silver et al., 2005; Smith, 2006; Zasler et al., 2007). Thus, for the current review PPCS is operationally defined as symptoms that persist beyond three months following a concussion (having met at least one of the definitions as listed in Table 1), implicating chronic sequelae.

As demonstrated by Table 1, there are many definitional statements about what constitutes a concussion or mTBI. Neuropsychological research on this topic would be aided to have a universally accepted definition as the standard (see Tagliaferri et al., 2006). Nonetheless, all definitions in Table 1 have general agreement that “mTBI is defined as the consequence of blunt (non-penetrating) impact with sudden acceleration, deceleration or rotation of the head with a Glasgow Coma Score (GCS) of 13–15 . . . (Vos et al., 2002, p. 207).” Thus, concussion occurs because of impact physical forces affecting the brain and if, physical forces are insufficient to injure the brain, no injury has occurred. Regardless of the etiology, recovery from concussion is typically rapid and ostensibly complete in most individuals. Clearly, the best-controlled studies examining outcome following concussion, demonstrate good to complete recovery in the majority of individuals (Iverson et al., 2007). Additionally, at least with sports concussion, major consensus statements of the past five years have resulted in statements like “concussion typically results from the rapid onset of short lived impairment of neurological function that resolves spontaneously” and that “concussion may result in neuro-pathological changes but the clinical symptoms largely reflect a functional disturbance rather than structural injury” as reviewed by (Cantu, 2007, p. 963). So this review focuses on the minority of subjects who sustain a concussion, who remain symptomatic after three months. Large-scale studies demonstrate approximately 70% of all head injury cases seen in the emergency room (ER) are in the mTBI category (Udekwu et al., 2004). However, as pointed out by the CDC and other studies (Delaney et al., 2005), a substantial number of concussions is never evaluated in the ER, making it difficult to obtain precise numbers as to the true annual incidence rate. Bazzarin et al. (2005) estimate that the annual mTBI incidence rate is 503.1/100,000, of which PPCS rates have been conservatively estimated at 10% (Ruff et al., 1996; Wood, 2004). Thus, despite the overall good to complete recovery rates from concussion, this remains a major public health concern (Langlois et al., 2005) and the field of neuropsychology should better understand the disorder (Kelly, 1999; Langlois et al., 2005).

From a neuropsychological standpoint, symptoms of impaired attention, memory, and executive function along with changes in emotional regulation dominate the clinical picture of PPCS (Lundin et al., 2006). An objective of this review is to understand these features in terms of a common pathological basis. To accomplish this, how evolutionary factors may have shaped recovery from concussion, followed by an up-to-date review of important new studies on the biomechanics of concussion and a thorough discussion
**Table 1. The Multiple Definitions and Grading Systems of Concussion**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Cantu</th>
<th>Colorado</th>
<th>Roberts</th>
<th>American Academy of Neurology</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No LOC; PTA &lt;30 min</td>
<td>No LOC; confusion without amnesia</td>
<td>“Bell ringer”; no LOC; no PTA</td>
<td>No LOC; transient confusion; concussion symptoms or mental status abnormality resolve in &lt;15 min</td>
</tr>
<tr>
<td>1</td>
<td>LOC &lt;5 min; PTA &gt;30 min and &lt;24h</td>
<td>No LOC; confusion with amnesia</td>
<td>LOC &lt;5 min; PTA &gt;30 min and &lt;24h</td>
<td>No LOC; transient confusion; concussion symptoms or mental status abnormality last &gt;15 min</td>
</tr>
<tr>
<td>2</td>
<td>LOC &gt;5 min or PTA &gt;24h</td>
<td>LOC</td>
<td>LOC &gt;5 min or PTA &gt;24h</td>
<td>Any LOC, either brief or prolonged</td>
</tr>
</tbody>
</table>

LOC = loss of consciousness; PTA = post-traumatic amnesia (from Leclerc et al., 2001)

**2nd International Conference on Concussion in Sport, Prague 2004**

"Sports concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathological, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include the following:

(1) “Concussion may be caused by a direct blow to the head, face, neck or elsewhere on the body with an ‘impulsive’ force transmitted to the head. (2) Concussion typically results in the rapid onset of short lived impairment of neurological function that resolves spontaneously. (3) Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury. (4) Concussion results in a grade set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course. (5) Concussion is typically associated with grossly normal structural neuroimaging studies. Two Classifications of Concussion: (1) Simple Concussion—In simple concussion, an athlete suffers an injury that progressively resolves without complication over 7–10 days.” (2) “Complex Concussion—Complex concussion encompasses cases where athletes suffer persistent symptoms (including persistent symptom recurrence with exertion), specific sequelae (such as concussive convulsions), prolonged loss of consciousness (more than one minute), or prolonged cognitive impairment after the injury” (p. 196–197) (McCrory et al., 2005)

**European Federation of Neurological Societies—2002 Task Force**

"mTBI is defined as the consequence of blunt (non-penetrating) impact with sudden acceleration, deceleration or rotation of the head with a GCS scores of 13–15 on admission to hospital (p. 209).

<table>
<thead>
<tr>
<th>Mild</th>
<th>Category</th>
<th>GCS</th>
<th>Clinical Description</th>
<th>*Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>15</td>
<td>No LOC, no PTA, = head injury, no TBI, No risk factors*</td>
<td>Unclear or ambiguous accident history, continued post-traumatic amnesia, retrograde amnesia longer than 30 min, skull fracture, severe headache, vomiting, focal neurological deficit, seizure, age &lt; 2 years, age &gt; 60, coagulation disorders, high energy accident, intoxication with alcohol/drugs</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>15</td>
<td>LOC &lt; 30 min, PTA &lt; 1 hr No risk factors*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>GCS = 15 + Risk factors present*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>13–14</td>
<td>LOC &lt; 30 minutes, PTA &lt; 1 hr. With or without risk factors present*</td>
<td>(Vos et al., 2002)</td>
<td></td>
</tr>
</tbody>
</table>

American Congress of Rehab Medicine Definition

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:

(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)
(4) Focal neurological deficit(s), that may or may not be transient but where the severity of the injury does not exceed the following:
   (A) loss of consciousness of approximately 30 minutes or less;
   (B) after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13–15; and
   (C) post-traumatic amnesia (PTA) not greater than 24 hours. (American Congress of Rehabilitation Medicine Head Injury Interdisciplinary Special Interest Group, 1993)

The WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury (mTBI)

"mTBI is an acute brain injury resulting from mechanical energy to the head from external physical forces. Operational criteria for clinical identification include: (i) 1 or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, and/or other transient neurological abnormalities such as focal signs, seizure, intracranial lesion not requiring surgery; (ii) Glasgow Coma Scale score of 13–15 after 30 minutes post-injury or later upon presentation for healthcare, These manifestations of mTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries or intubation), caused by other problems (e.g., psychological trauma, language barrier or coexisting medical conditions) or caused by penetrating craniocerebral injury, p. 115). (Carroll et al., 2004a)
of what is now currently known about the neuropathological and pathophysiological basis of concussion will be offered. The last part of the review will focus on more traditional neuropsychological concepts as they relate to concussion and conclude with suggestions on improved research tactics on this topic. There may be nothing more controversial in contemporary clinical neuropsychology, than the issues to be discussed in this review. At the outset, these controversies are acknowledged and the approach of this review is to first overview the contemporary neuroscience of concussion and deal with the most controversial issues at the end of the review.

EVOLUTIONARY ASPECTS OF INJURY

Undoubtedly concussions have been part of mammalian life since the beginning. The universality of concussions is that the stunned, motorically wobbly appearance commonly observed in an athlete, particularly a boxer who has been concussed, is replicated with animal models (Shaw, 2002). Survivability across mammalian species following concussion is testament to the fact that most concussions are but transient disruptions in normal brain function allowing the animal (including humans) to recover quickly and fully return to pre-injury abilities and activities. Because of the commonness of concussions, it is likely that genes that promoted certain brain morphologies and/or positive recovery characteristics have been passed down. However, concussions prior to the modern era would have occurred only from falls, falling or thrown objects, fisticuffs, combat, and the like. All of these remain major sources of concussions but with the modern era, concussions also occur from high-speed impacts that simply were never the source of injury in earlier times. So, whatever evolutionary advantages occurred they did so prior to the modern era. Likewise, genes selective for their ability to promote survivability of a brain injury were most likely associated with simple concussion and not more severe brain injury, because prior to modern medical treatment the majority of moderate-to-severe injuries would not have been survivable or lead to disability that could not be sustained.

From a structural standpoint, the position of the irregular skull base to the dural surface of the frontal and temporal lobes, housed with in the anterior and middle cranial fossa provides a means for holding the brain in position, in response to movement and/or mild trauma to the head (Bigler, 2007). Likewise, the position of the ventricles dissipates some of the strain effects with movement, including that of concussion (Ivarsson et al., 2002). Both of these have significant evolutionary advantage. It is also very likely that a selective bias occurred that favored rapid brain reparative mechanisms once a concussion occurred (Diaz-Arrastia & Baxter, 2006). In fact the most common of injuries for a particular organ system are the very ones most likely shaped by evolution (Martin & Leibovich, 2005). So key to recovery from concussion is a fast acting reparative system and this would emphasize a transient cellular response that immediately re-establishes neural homeostasis. In addition to reparative metabolic and cellular responses, redundancy and back-up neural circuitry activated once a primary system were injured would be critical to recovery (Bach-y-Rita, 2004; Desmurget et al., 2007; Duff, 2001; Guigon et al., 2007; Kercel et al., 2005). These redundant systems can either share in or take over function for injured neurons and networks. A neural systems reserve capacity probably directly relates to how rapid recovery from brain injury occurs, including concussion (Berker, 1996; Stern, 2007) and the role of genes in this recovery process is being examined (Alexander et al., 2007; McAllister et al., 2006).

One final evolutionary speculation will be made and that is based on the appearance and “design” of the fornix (see Fig. 1), the major white matter output from the hippocampus. At least half of the fornix is suspended beneath the corpus callosum and loosely connected with the septum pellucidum as it dives toward its connection with the mamillary body and septum (Andersen et al., 2006). One look at this delicate anatomical structure and it is obvious that it was not selected for its ability to withstand brain trauma. Evolutionarily, in lower mammals the fornix is clearly imbedded in brain parenchyma, but moving up the evolutionary tree with the expansion of the cerebral hemispheres, and the ventricular system, the fornix becomes more progressively suspended (see Crosby & Schnitzlein, 1982). The importance of the hippocampus and fornix in understanding mTBI is a major part of this review.

Physics of TBI

Given that concussions are so commonplace it must be easy to at least transiently impair the brain through mechanical deformation and there must be common neurological structures affected [see Figs. 1 and 2; (Ropper & Gorson, 2007)]. In a most innovative experiment by Bayley et al. (2005) human volunteers were studied using MRI to determine momentary brain parenchymal deformation when the head falls just 2 cm. MRIs of the brain were obtained before and immediately after the drop, comparing the degree of brain deformation or warping by measuring changes in fixed points between the two scans. These movements were far below the threshold for concussion and the authors liken this to the type of head (and brain) acceleration when jumping vertically a few inches and landing flat-footed. The authors estimated that it was 10% to 15% of the acceleration of “heading” a soccer ball. However, even with this mild impact the brain deforms. Bayley et al.’s conclusions were as follow: “When the skull decelerates, the brains center of mass continues to move, but the motion of the base of the brain appears constrained near the sellar and supra-sellar space. Tethering loads may be borne by the vascular; neural; and dural elements, which bind the brain to the base of the skull. Such anatomic structures might include the distal internal carotid arteries, the optic nerves, the olfactory tracts, the oculomotor nerves, and the pituitary stalk. All these structures pass through fixed bony or dural rings, which
restrict their movement. These features attach to or penetrate the more mobile brain parenchyma. As a result, the brain begins to rotate about this region, while material anterior is compressed and material posterior is stretched by initial effects. As the brain rotates backward and upward relative to the skull, the superior-frontal surface of the brain appears to compress against the top of the cranial vault. Normal forces, tangential forces, and possibly tension in the bridging veins on the superior surface of the brain eventually arrest the rotation of the brain in front of the superior contact region is compressed and pushed forward. Behind the superior points of contact, the brain is elongated as the brains inertia pulls it backward and clockwise. Finally, behind the basal tethering region, material in the brainstem experiences shortening and shear as the posterior and inferior parts of the brain continue rotating downward and forward (p. 852).” Bayly et al. (2006) have also performed this type of modeling on the rat pup brain with similar findings of significant transient mechanical deformation of the brain.

Viano et al. (2005b) used a different approach by simulating movement within the cranium by a “finite element analysis using a detailed anatomic model of the brain and head accelerations from laboratory reconstructions of game impacts (p. 891)” based on National Football League (NFL) players who experienced on the field concussions that were videotaped. The exceptional innovativeness of this study was the ability to model the brain, including white and gray matter, the ventricular system, meninges, and in particular the falx cerebri and tentorium cerebelli along with the skull (most of these anatomical regions are shown in Figs. 1 and 2). In a number of those concussed, the initial strain occurred in the temporal lobe adjacent to the impact and then migrated though the temporal lobe to other brain regions. This is depicted in Figure 3. In all subjects concussed the largest strains that occurred in the migration of the brain deformation occurred in the fornix, midbrain and corpus callosum. Dizziness correlated with early strain in the orbital-frontal cortex and temporal lobe.

The Viano et al. (2005b) modeling study found, in general, excursions of the concussed brain to be between 3–6 mm at 24–26 ms post-impact. Of particular importance to neuropsychology is that this modeling shows 4–5 mm displacements of the hippocampus, caudate, amygdala, anterior commissure, and midbrain (again refer to Fig. 2). In addition to these brain regions showing significant displacement, they also related to various cognitive and physical symptoms from concussion in this group of NFL players. Also, increased strain at the level of the hypothalamus was associated with at least transient cranial nerve symptoms.
The models described earlier occur in well-controlled experimental conditions. Obviously, high speed impact head injuries are not a controlled experiment and likely involve more significant pressure and shear-strain forces than what are seen in sports concussion (Bradshaw et al., 2001; Zhang et al., 2006a). Regardless of these factors, the same brain regions as described earlier and as shown in Figures 1 and 2 are likely involved in all concussions, just a matter of degree. Similarly, much of the cognitive and neurobehavioral symptoms of concussion can be explained by the involvement of the brain regions highlighted in Figures 1 and 2. In these Figures, note the proximity of the medial temporal lobe to the midbrain, the fact that the free-edge of the tentorium makes contact with the medial temporal lobe and midbrain as well as the nearness of the basal forebrain to these regions along with the hypothalamus, hypophysis and pituitary stalk, and the arterial vasculature. So, within a few centimeters are critical brain structures that, if affected, could represent the structural basis to many symptoms associated with concussion.

Pathophysiology of Concussion

Iverson (2005) and Hovda (2004) provide an excellent and detailed reviews of the pathophysiology of concussion, which need not be re-elaborated here. Whereas initiated by immediate biomechanical forces, as describe above, much of the pathology of acute concussion is believed to be transient biochemical induced neurotransmitter disruptions initiated within 25–50 msec of impact. Tensil forces also disrupt the cytoskeletal status of the axon and its ability to function, including disrupted axonal permeability and transport (Povlishock & Katz, 2005). Disrupted cytoskeletal architecture, renders cells less functional and may have widespread effects on the injured brain (Hall et al., 2005), albeit transient in concussion.

Since the Iverson (2005) and Hovda (2004) publications there are several important studies that add to our understanding of the potential microscopic pathology that can occur from concussion. Zetterberg et al. (2006) examined cerebrospinal fluid (CSF) taken by from lumbar puncture in 14 amateur boxers 7 to 10 days and 3 months after a bout compared to matched controls without any contact. They used several markers of neuronal and astroglial injury that can be readily detected in the CSF, finding significant indicators of neuronal injury byproducts in CSF that were positively related to the actual number of hits during a bout, most apparent in the initial samples taken after the amateur boxing contest. None of the boxers received a knock-out punch and likely did not meet any of the behavioral criteria.
for concussion. This study confirms the presence of acute pathological changes in the brain that can occur from boxing, in blows to the head that are below the threshold for producing what behaviorally would be classified as a concussion in these conditioned athletes.

Using a different approach, Zhang et al. (2006b) examined conventional MRI along with diffusion tensor imaging (DTI) in a group of professional boxers. While the majority had normal clinical imaging, 7 of the 42 examined had abnormal white matter findings, which should not be evident in an otherwise young, healthy subjects (Hopkins et al., 2006). More importantly, even those without clinical abnormalities as a group exhibited DTI differences from their matched controls, suggesting subtle white matter abnormalities, particularly at the level of the corpus callosum. Recall that Viano et al. (2005b) showed that the corpus callosum was one of the brain regions receiving the biggest strain effect in concussion. Similarly, Chappell et al. (2006) using DTI methods demonstrated similar white matter pathology in a group of 81 professional boxers. These studies focused on professional boxers without known neurological impairment, otherwise they would not be boxing, and show that sensitive MRI methods do detect with a higher frequency abnormalities of white matter. Along these lines Cohen et al. (2007) have shown MR spectroscopic and subtle brain volume loss in mild TBI. Such imaging findings demonstrate that pathological changes in brain parenchyma can be detected in mild TBI using contemporary neuroimaging methods.

Bigler (2004) demonstrated hemosiderin and residual inflammatory reaction in the post-mortem brain of an individual with PPCS, where the autopsy was performed seven months post-injury. Similar findings were observed in a post-mortem of a professional football player who had developed cognitive decline later in life (Omalu et al., 2005). Combining the imaging and neuronal injury biomarker studies discussed earlier, with the Bigler (2004) and Omalu et al. (2005) post-mortem studies provide indisputable evidence that structural pathology can be present in mTBI. Additionally, these type of hemorrhagic lesions can be observed with specialized high-field MRI studies (see Ashwal et al., 2006; Scheid et al., 2006) as shown in Figure 4. As such, some aspects of the so-called “traceless injury” of concussion are being revealed with newer techniques.

Fig. 3. From Viano et al. (2005b) published with permission from Lippincott Williams & Wilkins. The model on the left represents a coronal (top) and axial (bottom) view of the tagged brain model with the ventricle in pink and the skull encasing in yellow. The left hand column represents the baseline, where no movement occurred; notice the midline is vertical in the coronal plane and straight in the axial plane. Time in msec is shown on the x-axis. By 25 msec the model indicates that this player’s brain had a maximal shift, where it is evident that there is particular distortion in the medial temporal and hypothalamic region. This was from a player concussed on a kick return who had brief LOC, and PCS symptoms of headache, fatigue, dizziness, and photophobia and sleep disorder as physical symptoms. Note that the modeling of this subject's brain would involve all of the structures identified in Figure 1, and indeed, the high strain findings modeled in this subject supported such a locus of injury (see Appendix 1B of the Viano et al., 2005b paper that detail individual characteristics of the subjects).
What is the significance of these pathological residua in those concussed, even when ostensibly reparative and restorative mechanisms return function to apparent baseline? Are there still potential sequelae that can be elicited and are these expressed overtime? Do these lesions relate to neuropsychological function? What Gronwall and Wrightson (1975) demonstrated years ago suggested that concussion may not be as benign as Miller (1961) had implied, but may be very dependent on the cognitive demands placed on a patient. Routine cognitive tasks may be unaffected, whereas more complex functions affected. This has been revisited more recently by Chen et al. (2003) using functional brain imaging in a small group of subjects ($N = 5$) who had sustained concussion, only two of whom had brief LOC (less than 2 minutes). In this study the concussed patients, none of whom were in litigation, all had neurobehavioral symptoms of PPCS, but their resting PET metabolism did not differ from controls. However, when given a spatial working memory task to perform, differences in regional cerebral blood flow were detected in prefrontal cortex in PPCS subjects. In other words, unless a significant cognitive demand was placed on the subject that required more than typical cognitive effort, no differences could be determined. Similarly, Bernstein (2002) demonstrated that by increasing the complexity of a dual task involving auditory and visual discrimination and measuring evoked responses that those with a history of concussion but ostensibly no residual complaints could be differentiated from controls (Dockree et al., 2006a).

Moreover, confirmation of the likely residual pathology from concussion is clearly demonstrated in the second-injury circumstance, where a prior concussion increases the likelihood of a second concussion and greater morbidity of the second concussion in both human and animal studies (Huh et al., 2007; Longhi et al., 2005; Manville et al., 2007; Moser et al., 2005; Omalu et al., 2005; Pellman et al., 2004; Wall et al., 2006). The most straightforward explanation of the pathology of the second injury concussion is that the first concussion is not simply not benign, but that the brain adapts quickly to the injury in most cases. It should be noted that there is some controversy over the second injury hypothesis (Iverson et al., 2006b; Schnadower et al., 2007) and much more animal and human research is needed to fully understand this phenomena (Laurer et al., 2001). From a clinical management standpoint, repeated concussions are the basis for recommendations to retire from sports (Cantu, 2003) and reported to be related to the presence of neuropsychiatric symptoms in professional North American Football players (Guskiewicz et al., 2007).

While petechial hemorrhage associated with concussion has been well documented neuropathologically for decades (Ashwal et al., 2006), the shearing phenomena may only be part of the pathological story of vascular injury in concussion. A most intriguing animal study by Ueda et al. (2006), inducing what would be at least a moderate brain injury, has shown that the perivascular nerve network is injured in TBI as well. It is often overlooked that there is a neural regulation of blood vessels and blood vessels can contract and expand under neurogenic control. In fact, it is the dispersion of blood in response to autoregulation and localized activation that is at the basis of functional neuroimaging. If a blood vessel has a subtle abnormality in its ability to regulate regional flow, this may contribute to the neuropsychological sequelae expressed in a concussed individual. This remains to be investigated and represents speculation at this time. Thus, in TBI the same mechanisms that stretch the neuron can stretch the blood vessel and this may impair the neurogenic response of the blood vessel. Thus, the functional neuroimaging findings in concussion may not just be a consequence of brain parenchymal injury, but vascular and blood-brain barrier disruptions (Korn et al., 2005).
Along these same lines, is how the peri-vascular spaces that house cerebral vasculature are affected by injury, because much of the surrounding tissue is white matter. Numerous studies have shown the vulnerability of white matter damage in TBI (de la Plata et al., 2007; Inglese et al., 2005a; MacKenzie et al., 2002) have all shown that in mTBI increased frequency of dilated perivascular spaces, changes in white matter volumetry and chemical composition occur and relate to persistence of symptoms. Significant inflammatory reactions and hemosiderin deposits occur in the perivascular space in response to injury and their presence is considered a marker of white matter injury (Beschorner et al., 2002; Konsman et al., 2007). What is potentially so important about these observations is that inflammatory reactions that may originally injure white matter parenchyma, at least experimentally, have been shown to disrupt dopaminergic function (Roy et al., 2007), which heuristically, could be the basis for some of the neuropsychiatric symptoms associated with damage to white matter.

Another neuropathological complexity that is only beginning to be understood is the individual differences and heterogeneity of injury to individual cells (Buki & Povlishock, 2006; Reeves et al., 2005; Singleton & Povlishock, 2004). This too may be under genetic control where individual differences to injury susceptibility relates to outcome. It just may be that certain neurons are more susceptible to injury and certain injury forces or dynamics than others (Park et al., 2006).

There are other biomarkers of injury that have also been examined in human mTBI. For example, Stalnacke et al. (2005), using a blood biomarker of brain injury, serum concentrations of S-100B and neuron-specific enolase, found that S-100B levels during the acute phase of mTBI related to long-terms sequelae. S-100B findings have not been universal in mTBI (see also Bazarian et al., 2006a; Bazarian et al., 2006b; De Kruijk et al., 2002; Savola & Hillbom, 2003) and these observations are but some of the first. The level of initial CSF tau, a microtubular binding protein, believed to be a marker of axonal injury, correlates with outcome in severe TBI (Ost et al., 2006), but it has not been systematically studied in mTBI.

### Functional Neuroanatomy of Concussion and PPCS

In concussion, regardless of the definitional criteria used as outlined in Table 1 and the variability in clinical presentation, it is clear that 4 features dominate concussive symptoms—(1) brief alteration in consciousness or neurological function with at least acute changes in mentation and speed of processing; (2) physical symptoms of headache, dizziness and/or vertigo along with increased fatigability; (3) impairments in short-term memory, attention and concentration (particularly for multi-tasking); and (4) increased likelihood for changes in mood and emotional function. Where and how can these symptoms be integrated in understanding the functional neuroanatomy of concussion? The assumption is that there must be a common origin to these symptoms.

Figure 1 is a sagittal MRI view of the brain. The average adult brain weighs somewhere between 1150 and 1450 grams (2.5–3 pounds), with most of that weight located in the cerebrum, above the cerebellum in the figure. The anterior aspect of midbrain region of the upper brainstem is comprised of the cerebral peduncles which house all of the major ascending and descending white matter pathways connecting the cerebrum with the periphery of the body and the connections between the cerebrum and the cerebellum. In an earlier review, these anatomic regions, pathway and structures have been outlined in detail (Bigler, 2007). As can be clearly visualized in the Figure 1, the midbrain at the level of the cerebral peduncle is small, opposed to frontal-occipital linear dimension, and in the vertical position the tegmental aspect of the upper midbrain “rests” on or is adjacent to the dorsum sellae and the anterior clinoid, partially shown in Figures 1 and 2. Just in front of the tegmentum is the hypophyseal fossa that house the infundibulum (or pituitary stalk), the neural connection between the ventral hypothalamus and the pituitary, situated in the sella turcica. Immediately lateral to the cerebral peduncle is the carotid groove of the sphenoid bone wherein the internal carotid artery ascends into the brain to form the anterior and medial cerebral arteries. Next, moving laterally just past the carotid groove is the inner edge of the greater wing of the sphenoid and the beginning medial surface of the temporal lobe (see Fig. 2). The entrance of internal carotid into the cranium through the carotid canal occurs just adjacent to the midbrain. What is particularly interesting about this region of the brain is that the tentorium cerebelli extends from a covering of the cerebellum to attach at the junction of the clinoid and lesser wing of the sphenoid. As the tentorium projects to its clinoid-lesser wing of the sphenoid connection, the lateral surface of the upper brainstem touches the “free edge” of the tentorium cerebelli, and just on the other side of the tentorium at this level is the medial surface of the temporal lobe, where the perihinal and entorhinal surfaces also touch the “free edge” of the tentorium (Bigler, 2007; Van Hoesen et al., 1999). What is also of particular interest with regards to consciousness is that arterial branches of the posterior circulation of the brain actually cross the free edge of the tentorium and these arterial branches supply blood to the brainstem (Blinkov et al., 1992).

Biomechanics of concussion inform us that concussions are more likely to occur if there is some rotational force present (Fijalkowski et al., 2006; Viano et al., 2005a; Vorst et al., 2007). Returning to this midbrain region of the brain, if there is any stretching and/or rotational force at this level, note what occurs: the upper brainstem stretches across the clinoid and lesser wing of the sphenoid, with its lateral margins potentially striking the free-edge of the tentorium, the pituitary stalk stretches disrupting hypothalamic-pituitary connections, the internal carotid stretches against the carotid canal and posterior circulation to the brainstem is also dis-
ruptured, the medial temporal lobe strikes the lateral surface of the free-edge of the tentorium as well as the medial wall of the sphenoid. Just in from this medial surface of the temporal lobe are the amygdala and hippocampus, with the hippocampus giving rise to the fornix that not only connects with the anterior thalamus via the mammillary body but also to the septum and pituitary (McDonald et al., 2006). Just anterior to the hypophysial-hypothalamic region is the basal forebrain; just posterior are the mammillary bodies. So at one level, if there is slight mechanical deformation either in terms of compression or uplift and particularly if rotation occurs, there are putative functional neuroanatomical connections disrupted for consciousness (upper brainstem, reticular activating system), memory (mechanical compression of perihinal and entorhinal cortices disrupting input to the hippocampus or hippocampal output via the fornix and its connection with the anterior thalamus and cingulate), emotional regulation (medial temporal lobe and basal forebrain), post-traumatic migraine (stretching the internal carotid and all vasculature that forms the circle of Willis as well as stretching/irritation of the dura and other vessels) and fatigue as well as hormonal changes secondary to hypothalamic-pituitary disruption. 

Indisputably, clearly demonstrated immediately after concussion (Barrow et al., 2006a; Barrow et al., 2006b), even in those who go on to fully recover, is slow speed of processing (Crawford et al., 2007; De Monte et al., 2005). Speed of processing is dependent on the integrity of white matter pathways maintaining their optimal inter-connectiveness. Returning to the biomechanical deformation effects reviewed above, long-coursing axons are going to be more vulnerable, particularly interhemispheric connections, especially the corpus callosum and anterior commissure (Cecil et al., 1998; Holshouser et al., 2006; Inglese et al., 2005b; Mathias et al., 2004; Wilde et al., 2006a; Wilde et al., 2006c). Thus, neuropsychological tasks that require interhemispheric integration and/or multiple intracortical connections often show differences in the form of slowed responding, even in those with mTBI (Mathias et al., 2004). 

So, the hypothesis put forth in this section is that the biomechanics of brain injury simultaneously disrupt neuro logical function in the upper brainstem, pituitary-hypothalamic axis, medial temporal lobe, and basal forebrain concomitant with irritative injury to the vasculature and meninges, which gives rise to the symptoms observed in the post-concussive state and the neuropsychological sequela associated with such an injury. How rapidly these neural, dural, and vascular areas return to homeostasis or recovery from some adaptive mechanism or do not recover, provides the biological basis for the symptoms expressed.

**Animal Models of Concussion**

The advantage of animal models is the controlled environment where the reproducibility of an adverse effect can be tested, in this case a concussive brain injury. There have been numerous animal models of brain injury over the years (Leker et al., 2002), but most focused on what would be moderate-to-severe brain injury with focal cortical impact, producing not only focal brain injury but diffuse injury readily demonstrated by histological analysis. It has been challenging to develop an animal model of concussion that mimics human concussion, because of a host of differences associated with brain morphology, skull-brain interface, and species differences (Leker et al., 2002).

Nonetheless, several excellent animal models of concussive injury have recently been established (Gurkoff et al., 2006; Henninger et al., 2005; Milman et al., 2005; Tang et al., 1997; Tashlykov et al., 2007; Ucar et al., 2006; Yoshiyama et al., 2005; Zohar et al., 2003, 2006). For example, Henninger et al. (2005) modifying methods of Tang et al. (1997) have used a weight drop device to the exposed skull that replicates human concussion. As stated by these researchers, “immediately after impact, all TBI animals lost their muscle tone and righting reflex response (p. 450)” but it shortly returned. This is analogous to what is observed acutely in sports concussion (McCrory & Berkovic, 2000). After reflex recovery the concussed rats behaved “normally” in comparison to sham controls. In this study, memory was assessed using the Morris Water Maze (MWM), where the concussed animals also showed no differences from controls in ability to swim and other species typical behaviors. Thus, in terms of ordinary rat behavior, function returned without discernable abnormality following concussion. However, given time to heal from the minor surgery to expose the skull, the concussed animals exhibited memory deficits on the MWM when assessed nine days post-injury. This study also included high-field MRI which was negative. However, histology demonstrated several pathological changes including a reduction in the number of cortical neurons as well as in the hippocampus. A limitation of this study is that it only examined memory nine days post-injury but Milman et al. (2005) and Zohar et al. (2003, 2006) using somewhat similar methods, but in mice, have demonstrated these type of persistent cognitive differences in concussed animals for longer periods of time post-injury. Gurkoff et al. (2006) have demonstrated this in rats with a fluid percussion injury model and Tashlykov et al. (2007) have shown apoptotic changes in cerebral cortex and hippocampus using this weight drop technique as well.

So animal models of concussion do support the notion that persistent cognitive deficits can occur, although not all studies have found lasting effects (Gaetz, 2004; Leker et al., 2002). The difference between those studies that find persisting symptoms and those that do not is probably the severity of the concussion. For example, in the Tashlykov et al. (2007) study pellets of incremental weight from five to 30 g were dropped on the head of mice under light ether anesthesia. Of particular interest in this study is that none of the weight amounts produced any discernable change in the species typical behavior of the mice once recovered from the ether anesthesia, yet related to the weight amount of the pellet pathological changes were proportional to the impact. The 5 g weight drop was insufficient to produce any detect-
able pathological changes. A minimum of 10 g was necessary for showing pathological neuronal changes, but 15 g was necessary to initiate apoptotic changes. Thus, the threshold to produce injury varies depending on what pathological changes are under investigation and whether a certain injury threshold has been reached.

IS BRAIN INJURY ON A CONTINUUM: CONCUSSION → SEVERE TBI?

In examining the post-mortem brain of several human subjects who had sustained a “mild concussion,” but died for reasons other than the head injury, Blumbergs et al. (1994) demonstrated presence of axonal injury, particularly in the fornix. Blumbergs et al. (1995) in a follow-up study demonstrated that the microscopic pathology was on a continuum from mild (GCS of 13–15) to severe (GCS of 3–8), again demonstrating the susceptibility of the fornix. As shown by Viano et al. (2005b), the fornix is distinctly vulnerable to the stress/strain effects of concussion and is a common area of damage in moderate-to-severe TBI, as visualized using MRI (Gale et al., 1995; Tate & Bigler, 2000; Tomaiuolo et al., 2004), where the degree of atrophy is related to severity of injury (Bigler et al., 2006; Tate & Bigler, 2000; Tomaiuolo et al., 2004; Wilde et al., 2006b). Because the fornix is a white matter structure containing projecting axons from the hippocampus, disruption in fornix integrity likely relates to the concussive effects of disrupted short-term memory, at least transiently.

At the histopathological level, severity can be graded by the degree of cell loss, cytoskeletal changes, presence of inflammatory cellular reaction, biochemical markers of cell damage or death, etc. and all seem to relate to severity on some continuum (Anderson et al., 2003; Vorst et al., 2007). Taken together, in well controlled animal models there is a continuum associated with severity of impact injury supporting the contention that injury is on a continuum (Gurkoff et al., 2006; Igarashi et al., 2007; Kharatishvili et al., 2006; Maegle et al., 2005; Ucar et al., 2006). Understanding this continuum means that at the mildest level of brain perturbation there may, in fact, be no lasting effect. However, once a threshold is reached, lasting sequelae begin to occur (Zhu et al., 2006).

Human neuroimaging studies also support the concept of continuum of injury. For example, a linear relationship with cerebral atrophy relates to injury severity measures such as GCS, PTA, and duration of LOC (Bigler et al., 2006; Wilde et al., 2006b). Likewise, complicated mTBI is more likely to have positive neuroimaging findings (Levine et al., 2006; McAllister et al., 2001; Vorst et al., 2007) and significant residuals (Kennedy et al., 2006). If boxing is considered a model for detecting “pre-clinical” or asymptomatic brain injury, recent diffusion tensor imaging studies have demonstrated abnormalities in boxers (Chappell et al., 2006; Zhang et al., 2006b). Thus, animal and human studies support the contention of injury on a continuum, implicating that understanding the variables that relate to severity of injury are likely very important in understanding neuropsychological sequelae (see Wilde et al., in press; Lewine et al., 2007).

VULNERABILITY OF THE MEDIAL TEMPORAL LOBE AND IN PARTICULAR, THE HIPPOCAMPUS

Elsewhere, I have reviewed research demonstrating that the brain-skull interface in the anterior and middle cranial fossa is a major factor for the vulnerability of these regions in TBI (Bigler, 2007). Potentially the most critical structure injured for neuropsychological sequelae in TBI is the hippocampus and its afferent and efferent connections (Wilde et al., 2007). The Viano et al. (2005b) study demonstrated that the typical deformation of the hippocampus to be 4–6 mm in concussion associated with professional football. Numerous human and animal studies have demonstrated the vulnerability of the hippocampus (and fornix) to injury in TBI (Bigler et al., 2006; Geddes et al., 2003; Royo et al., 2006; Tashlykov et al., 2007; Tasker et al., 2005; Wilde et al., 2006b) and functional neuroimaging studies using SPECT also demonstrate medial temporal lobe hypoperfusion in mTBI (Gowda et al., 2006). Thus, given the location of the hippocampus in the medial temporal lobe and its connection and location to the fornix, these brain regions are key to understanding PPCS neuropsychology, and should be the focus of intense neuropsychological investigation.

LIMITATIONS OF NEUROPSYCHOLOGICAL RESEARCH TO ADVANCE THE FIELD

The Litigation Conundrum: Forensic Implications for Clinical Neuropsychology

From the anatomical and pathophysiological discussions earlier, it is plainly evident that the brain is at least momentarily and transiently injured in concussion but for the majority of those injured persistent sequelae do not occur. Because animal models have demonstrated that lasting negative effects can occur with concussion (see Tashlykov et al., 2007), it is reasonable to assume that PPCS will exist in some individuals. It is in these individuals that neuropsychological research needs to direct its best and most unbiased research efforts. Unfortunately, as pointed out by the World Health Organization’s task force on mTBI, poor research designs and the cross-sectional nature of many of the studies on this topic, restrict generalizations of the findings (Carroll et al., 2004a; Carroll et al., 2004b). What can be done to correct short-comings of research in this area?

More than 40 years after Miller (1961) wrote about concussion and “compensation neurosis”, Kertesz and Gold (2003), reviewing outcome from concussion make the following statement: “the involvement of insurance claims, litigation, and the expense of rehabilitation makes this area very contentious (p. 629).” Belanger et al. (2005) per-
formed a meta-analysis of 39 studies involving 1463 cases of mTBI assessing clinical neuropsychological test findings. Their findings were similar to what has also been described by Binder et al. (1997), Frencham et al. (2005), and Schretlen & Shapiro (2003), implicating short-term, but not necessarily long-term neuropsychological effects, except for those cases who were in litigation, where either “stable or worsening of cognitive functioning over time (p. 215) was observed.” Mooney et al. (2005), in a university based rehabilitation service, examined those with “disappointing recoveries” and observed that “in cases of poor recovery after mTBI where compensation or litigation may be a factor, most of the variance in recovery seems to be explained by depression, pain, and symptom invalidity (p. 975).” With regards to symptom invalidity, Loring et al. (2007) reported 20% of subjects including those with history of head injury who were evaluated in a University-based clinical assessment laboratory but who were also in litigation did not pass symptom validity testing (SVT). Plainly, presence of litigation is a major confound in research in mTBI and its presence in research studies has likely obscured the true effects of concussion, including PPCS. Also, whenever analyzing group data, if all subjects with concussion are examined at a particular time period, the effects on individual subjects who may be symptomatic get washed out by the total group effects (Iverson et al., 2006a; Kent, 2007; McHugh et al., 2006; Sterr et al., 2006). This is a very important point, because few studies compare symptomatic versus non-symptomatic subjects who have been concussed and those who do, find those who are symptomatic to have greater neuropsychological impairment (Collie et al., 2006; Iverson et al., 2004; Sterr et al., 2006).

The fact that the litigation process is adversarial and that neuropsychological testimony occurs on both sides of the legal argument, raises the specter of potential bias in what has been written about PPCS depending on the type of forensic work an author may participate in. If one is exclusively retained in legal settings for one side or the other in a legal matter, that could have a bearing on what is studied and reported (Racette et al., 2006). The legal side that retains a clinician or researcher may influence directly or indirectly what is published by that individual (Bigler, 2006). For example, it would be difficult for the individual in private practice whose sole income is derived from their forensic work and consistently retained by the defense to publish on the subtle sequelae of PPCS, including its lasting and enduring adverse effects. Oppositely, but just as likely, the clinician who is exclusively retained by the plaintiff’s side is unlikely to publish on the “myth” of PPCS.

Neuropsychological research from countries that do not have the kind of litigation and medical care system that the United States has may provide important information about PPCS, if the proper large scale studies are done. There are cultural differences in the expression of whiplash associated disorders (WAD) (Obelieniene et al., 1999), and the same may be expected in PPCS. Incomplete effort is another major factor contaminating any study looking at long-term neuropsychological sequelae of concussion (Ross et al., 2006a; Ross et al., 2006b), which represents a topic of its own for review (Iverson & Binder, 2000).

Ecological Validity of the Clinical Neuropsychological Approach

Ecological validity of neuropsychological assessment remains an ever present concern (Chaytor et al., 2006; Moritz et al., 2004; Odhuba et al., 2005; Wood & Liossi, 2006). As an example, the antemortem clinical neuropsychological testing in the concussed patient previously described who met PPCS criteria and who at autopsy had verified pathology of brain injury, was all normal yet this individual had “real-world” difficulty running his business, problems not evident before his injury (Bigler, 2004). Standardized paper-and-pencil tests typically conducted in the sterile laboratory may simply not tap the cognitive symptom being experienced by the individual with PPCS. This very point has been made by Collie et al. (2006) in determining which kinds of measurements are most sensitive in detecting problems in those who remain symptomatic after concussion. Obviously, cognitive skills, in particular working memory and executive function, can place much higher demands on neural integrity in the real world than what can be assessed by any current clinical neuropsychological technique in the laboratory.

Assessment in sports concussion has recognized the need to move beyond traditional neuropsychological assessment with the development of more tailored assessment tools in the athlete with concussion (Broglio et al., 2007; Parker et al., 2007). Such assessments are also taking advantage of computerized and virtual assessment techniques as well as the ability to automate the assessment (Cernich et al., 2007; Iverson et al., 2005; Schatz & Putz, 2006; Slobounov et al., 2006). Likewise, various cognitive neuroscience measures either by themselves or combined with functional neuro-imaging methods hold great promise for more accurate assessment of the effects of TBI on behavior and cognition (Bergemalm & Lyxell, 2005; Casson et al., 2006; Chan, 2001; Chen et al., 2007; Cicerone et al., 2006; Dockree et al., 2006b; Jantzen et al., 2004; Mendez et al., 2005; O’Keeffe et al., 2007a; O’Keeffe et al., 2007b; Scheibel et al., 2007; Suh et al., 2006). These types of studies applied to PPCS will likely advance the field rather than another round of testing with traditional “clinical” neuropsychological measures (Heitger et al., 2004, 2005, 2006).

Confounding Factors That Must be Considered in the Design of PPCS Studies and the Accurate Determination of Neuropsychological Sequelae

The fact that the eight symptoms of PCS [i.e., (1) becoming fatigued easily, (2) disordered sleep, (3) headache, (4) vertigo or dizziness, (5) irritability, (6) anxiety, depression or
affective lability, (7) changes in personality, and (8) apathy or lack of spontaneity] as outlined by DSM-IV (pp. 704–705) all overlap such that all coexist with a myriad of other medical and psychiatric diagnoses, underscores how complicated the design of the ideal study has to be to truly assess PPCS. For example, Iverson (2006) points out the commonness of misdiagnosing PPCS when the symptoms are really driven by depression and how depression can be misattributed to concussion (Chamelian & Feinstein, 2006; Meares et al., in press). In fact every PPCS symptom can occur independent of a head injury (Iverson et al., 2007). Also, a threshold issue exists where symptoms have to rise beyond a baseline before PPCS can be diagnosed (Chan, 2005). Post-traumatic pain injuries correlate with presence of PPCS (Sheedy et al., 2006); and pain has its own set of correlates, by itself, potentially affecting cognitive performance and emotional status (Alfano, 2006; Karp et al., 2006). None of this even addresses the complexity of WAD, as already mentioned, and WAD pain-related problems (Holm et al., 2006; Johansson, 2006; Zumsteg et al., 2006), nor post-traumatic headaches (Lew et al., 2006; Weiss et al., 1991) which are commonplace in concussion, especially those caused by MVAs.

Not only is the brain concussed, but also other organs such as the eye, inner ear, and soft internal organs (Frater & Haindl, 2003; Keane & Baloh, 1992; Nolle et al., 2004); and injury to these organs can be a source of symptoms. With regards to organs of the head, vertigo, dizziness, tinnitus, and ocular disturbance are commonplace; and they relate to cognitive sequela associated with mTBI (Suh et al., 2006). Presence of any of these symptoms may confound the neuropsychological presentation and sequelae of the mTBI patient but are rarely controlled. What is particularly important about pain, regardless of its source, is that pain changes the functioning of the brain, demonstrated by both structural as well as functional imaging (Schweinhardt et al., 2006). Also, the nature and extent of early medication treatment in those who sustain mTBI, may also relate to who develops PPCS (Meares et al., 2006).

Fatigue is a common and persistent problem in PCSS (Stulemeijer et al., 2006; Ziino & Ponsford, 2006), and it too has its own set of neurochemical, neuroimaging, and neuropsychological differences (de Lange et al., 2004; Kozora et al., 2006). The same can be said about the co-occurrence of PTSD in those involved in accidental injury or assault as the source of their concussion (Bryant, 2001; Creamer et al., 2005; McCauley et al., 2001) and the role of stress hormones in the behavioral response to injury (Sojka et al., 2006). PTSD alone has its own unique effect on neuropsychological performance (Vasterling & Bremner, 2006; Vasterling et al., 2006; Veltmeyer et al., 2005). Even for those who do not develop PTSD, being in an accident (Mayou & Bryant, 2002) or an assault (Johansen et al., 2006) or just sustaining a brain injury (Prigatano et al., 2005) is stress producing.

Mooney and colleagues have documented that many with persistent symptoms following concussion meet criteria for conversion disorder (CD (Mooney & Speed, 2001). However, the neurobiology of CD, including neuroticism, is starting to emerge and it may not be as “functional” as believed (Allet & Allet, 2006; Atmaca et al., 2006; Ghaffar et al., 2006; Schonfeldt-Lecuona et al., 2006; Stonnington et al., 2006; Ward et al., 2003; Wright et al., 2006). Theories and functional neuroimaging studies of CD imply the involvement of limbic regions, inferior frontal and medial temporal lobe structures, the very regions most likely injured in TBI. Is there an increased prevalence of conversion disorder in individuals concussed because these areas are injured? Neuropsychology should be exploring the potential neurobiology of this observation, not merely writing this off as a mere functional manifestation of concussion (Ashman et al., 2006). Recently, Wood (2005) has put forth a diathesis-stress model as a beginning attempt to describe these relationships.

It has long been known that pre-morbid factors predispose those with history of neuropsychiatric disorder to be more likely to experience PPCS once concussed (Karzmark et al., 1995; Ponsford, 2005). As such, any study that examines PPCS that does not take into consideration pre-morbid factors likely overlooks important and relevant information that may contribute to the disorder.

It is interesting that only recently has research begun to examine the role of pituitary injury in TBI to functional outcome, even in concussion (Acerini et al., 2006; Kellstimur, 2005; Kelly et al., 2006; Tanriverdi et al., 2007). As shown by the Bayly et al. (2005) and Viano et al. (2005b) studies, the hypothalamic-pituitary axis is particularly vulnerable to physical strain in concussion. Pituitary dysfunction can be associated with many of the same symptoms as seen by PPCS (Casauieva et al., 2006; Powner et al., 2006), yet this has not been systematically investigated in PPCS. This is particularly important because of some pituitary associated physical and neuropsychiatric symptoms are treatable.

Similarly, the basal forebrain resides just anterior to the hypothalamus housing important nuclei and pathways for cholinergic innervation of the brain. The basal forebrain is another region that sustains significant strain effects in biomechanical modeling of concussion and in moderate or greater injury, is consistently damaged (Bigler, 2005; Conner et al., 2005). However, this region has never been systematically examined in PPCS.

The elegant reconstruction of concussion by Viano et al. (2005b) clearly demonstrates that each concussion places unique stress and strain on the brain. Just as clear from this research is that no two concussions are identical in terms of how the brain is impacted. So if one does not take into consideration the impact and physical dynamics of the injury and subject characteristics (including genetics), neuropsychological sequelae could vary widely with regards to the brain regions most likely injured even though all subjects had sustained a “concussion”. Unfortunately, for most research on PPCS such information has never been obtained and this has never been systematically investigated other
CONCLUSION

From a neuropathological standpoint, this review demonstrates that concussion can lead to structural damage. From the biomechanics of concussion, the vulnerability of the upper brainstem, hypothalamic-pituitary axis, medial temporal lobe and basal forebrain and long-coursing white matter fibers, particularly involving the corpus callosum and fornix are the brain regions most likely to give rise to post-concussive symptoms.

Confusion in the literature on this topic comes from differences in terminology and definitional standards as well as poor research designs where small sample sizes, samples of convenience, selected clinical sub-samples and research that may have an agenda behind it has created serious interpretative problems with regards to the neuropsychology of concussion and its sequelae. Prospective studies of concussion where large trauma centers assess, follow and track patients with concussion and follow such a cohort prospectively using uniform and more ecologically valid cognitive assessment protocols simply have not been done. In such a group it would be reasonable that additional data could be obtained that would provide more information about the biomechanics of injury and a host of other medical and demographic factors, including attempts to be establish pre-injury level of function. In one of the largest reviews of mTBI, the WHO task force that reviewed mTBI literature up to 2004 concluded that mTBI research is “of varying quality and causal inferences are often mistakenly drawn from cross-sectional studies (p. 84,” (Carroll et al., 2004a), see also (Ragnarsson, 2006) The only correction for this gaffe in the neuropsychology of concussion, and potential long-term sequelae of PPCS, will be large, unbiased prospective studies that address the issues raised in this review. The importance of understanding this more accurately and completely is the fact that concussion is reportedly the most common of all neurological injuries and this is also true of the Iraq and Afghanistan war (Das & Moorthi, 2005; Okie, 2005; Warden, 2006; Warden & French, 2005), where unofficial estimates place the numbers in the tens of thousands (Bob Woodruff Reports. February 27, 2007: www.abc.com), potentially as high as, “1 out of every 10 returning service men and women” [p. 16, American Academy of Neurology News, 20(6), 2007]. Neuropsychology needs to better understand PPCS and this review offers a number of very testable hypotheses for future research.

ACKNOWLEDGMENTS

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REFERENCES

Persistent post-concussive syndrome

... and proton magnetic resonance spectroscopy in assessment of outcome after pediatric traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 87, 50–58.


Persistent post-concussive syndrome


Mayou, R. & Bryant, B. (2002). Outcome 3 years after a road traffic accident. Psychology Medicine, 32, 671–676.


Persistent post-concussive syndrome


St concussion disorder. American Journal of Psychiatry, 163, 1510–1517.


Crushin A Concussion: 
Attacking Claims Of Impairment Following Mild TBI

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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

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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.").
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. 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. 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.”

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.

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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 potion 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) cingulated; (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.

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. 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 < 1 hr
- **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.

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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”:

<table>
<thead>
<tr>
<th>Group</th>
<th>TFC</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TFC &lt; 1hr</td>
</tr>
<tr>
<td>2</td>
<td>TFC = 1-23 hr</td>
</tr>
<tr>
<td>3</td>
<td>TFC = 1-6 days</td>
</tr>
<tr>
<td>4</td>
<td>TFC = 7-13 days</td>
</tr>
<tr>
<td>5</td>
<td>TFC = 14-28 days</td>
</tr>
<tr>
<td>6</td>
<td>TFC &gt; 28 days</td>
</tr>
</tbody>
</table>

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.

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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 pupilary reaction (equal constriction) to light?
The oculomotor nerve is the 3rd cranial nerve. An examination of pupilary function includes inspecting the pupils for equal size (1mm or less of difference may be normal), regular shape, and reactivity to light. To test pupilary 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 (Trigemial 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.

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 activates 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.