TRAUMATIC BRAIN INJURY DEFENSE, VERSION 3 - CUTTING EDGE DEVELOPMENTS IN TBI LAW AND SCIENCE

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CLE Materials - Table of Contents

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Course Agenda - Traumatic Brain Injury Defense, Version 3

Co- Presenters: Jeffrey A. Brown, MD, JD, David Mahalick, PhD and William DeVito, Esq.

September 22, 2016 at 5:30 pm.

1. Introduction - DeVito – 10 minutes
   - Why talk about brain injury cases
   - Increasing financial stakes in brain injury claims (dollar values)
   - New Science
   - Resilience, Reliability and Recovery
   - How is plaintiff going to present the case and tests that come up
   - New cases on Diffusion Tensor Imaging
   - People Recover - the case of Bob Woodruff

2. Using the Latest Science and Understanding of Brain Injuries to Help You Work Constructively with Your Adversary to Settle Cases

   A. Dr. Brown - **30 minutes**
   - The difference between head and brain injuries
   - The Hidden Power of Negative DTI Studies
   - The Impact of the new DSM-5
   - Motivating Plaintiffs and their lawyers to want to get better
   - Using the neuroscience of Resilience
   - The Use of Vocational Rehabilitation and ADA Accommodations to accelerate recovery
   - The importance of giving TBI patients hope of recovery

   B. Dr. Mahalick - **30 minutes**
   - Types of Brain Injuries
   - What to look for in Medical Records
   - Identifying TBI
   - Clinical Testing by a neuro-psychologist
   - Framing the degree of alleged damage
   - Co-occurring psychiatric disorders
   - Treatment

10 minute break

3. Translating the neuroscience into winning legal strategies – DeVito - **25 minutes**
    - The Ideal Plaintiff case – what you may face
    - Using discovery to find out what baseline you started with – what brain were you dealing with before the accident
    - New Cases on the admissibility of diagnostic exams for TBI:
      White v Deere and Ali v Connick
    - Review of Past cases on Admissibility of DTI and other tests "the greatest hits"
    - Using sensitivity and specificity arguments to win the case

4. Finale - Q&A – panel discussion – **15 minutes** open discussion

VOLUME 2

PART II. BRAIN INJURY LITIGATION FROM A NEUROPSYCHIATRIST'S PERSPECTIVE

Chapter 11. Avoiding Predictable Case Blunders

§11:14.10 Catastrophic reliance on "mind/body dualism" [New]

The editors believe that there is not one single living physician (except some afflicted with cognitive damage of their own(!) truism, for many decades it has been indisputable that the brain is intertwined both with the "mind" or "psyche" as well as with the rest of the physical body.

Even in healthy, non-brain damaged brains, it is indisputable that brain impulses travel from pain receptors through nerves well within the spinal cord and ultimately to the thalamic region of the brain. It is equally well-known – with all the above concepts frankly subject in the editor's view to judicial notice given the universality of their being accepted in all fields of medicine – that the brain in turn sends down via the sympathetic and parasympathetic nervous systems nerves that influence everything from speed of digestion to heart speeding up or slowing down in rate and rhythm, with virtually every area of the

body sending nervous impulses to and in turn receiving nervous impulses from the brain in even healthy individuals.[1]

It is equally indisputable in modern times and again in the editors' view worth of judicial notice that even in healthy non-brain-damaged individuals there are hormonal influences that involve the pituitary gland, the hypothalamus, and end organ receptors that are responsible for, for example, ovulation and timing of same in women, body temperature regulation and metabolism, the "flight or fight" responses of every human being to potential danger, etc., etc..

It is further well known since the time of Aristotle in the 4th century BCE and in specific research going back to the 1950's, that even one's response to a systemic bodily disease like tuberculosis[2] has been known to demonstrate even the type of response one gets to an infectious disease like tuberculosis in turn is a dependent on the effects of stress and the suppression of the immune system by stress.

The March, 2016 issue of *The Journal of Clinical Psychiatry* explodes and discredits any vestiges of any distinction between "psychological" and

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[1] See, i.e., Truex and Carpenter, Human Neuroanatomy, Sixth Edition (Baltimore: Williams & Wilkins Company, 1969); see also Kasper, et al, Harrison's Principles of Internal Medicine, 19th Edition (New York: McGraw Hill, 2016) at 2708-2725.

[2] See, e.g., Lerner, Can stress cause disease? Revisiting the tuberculosis research of Thomas Holmes, 1949-1961, 124(7) Ann Intermed, 673-680 (1966).

"emotional" causes and injuries on one hand and the relationship to the rest of the body on the other.

Note specifically the following:

1. The article by Coccaro, et al describing a protozoan parasite, Toxoplasma gondii infection and its "relationship with aggression in psychiatric subjects."[3]

The results of the study were that infection with this parasite was in fact associated with both higher behavioral aggression and impulsivity especially with regard to aggression.

Their findings "were not accounted for by the presence of other syndromal/personality disorders or by states or traits related to depressed or anxious moods."

The authors concluded that the data were consistent with those of previous studies that indeed suggested a relationship between infection with this agent and even suicidal behavior and "further add to the biological complexity of impulsive aggression both from a categorical and dimensional perspective." [4]

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[3] Coccaro, et al, Toxoplasma gondii infection: relationship with aggression in psychiatric subjects, 77(3), The Journal of Clinical Psychiatry, 334-341 (2016).
[4] Coccaro, et al, Toxoplasma gondii infection: relationship with aggression in psychiatric subjects, 77(3), The Journal of Clinical Psychiatry, (2016) at 341.

2. An online article that had a title which is self-explanatory: "The clinical picture of Alzheimer's disease in the decade before a diagnosis: clinical and biomarker trajectories."[5]

3. Finally, an article clearly indicating that PTSD – often taken by many psychologists and plaintiff attorneys as indicating "pure" psychological problems or by a defense attorney as indicating a lack of organic problems again is a false distinction since the article documents that "PTSD symptoms lead to modifications in the memory of the trauma."[6]

It also is known that the central nervous system has a direct effect on the organs in which circulating lymphocytes[7] (including T-cells and cells now believed to be made by the body to combat cancer)[8] directly affected by CNS effects on the organs and producing these lymphocytes (the bone marrow, thymus, spleen and lymph nodes).

Furthermore, and again even in non-brain-damaged individuals and individuals not warranting a psychiatric diagnosis, it is indisputable that stress is

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[5]Ritchie, et al, The clinical picture of Alzheimer's disease in the decade before diagnosis: clinical and biomarker trajectories, 77(3), The Journal of Clinical Psychiatry online, 2016.

[6] Dekel, et al, PTSD symptoms lead to modifications in the memory of the trauma, 77(3), The Journal of clinical Psychiatry online, 2016.

[7] See also, Boomershine, et al, Stress and the pathogenesis of tuberculosis, 22, Clin Microbiol News 177-181 (2000) and Ishigami, The influence of psychic acts of the progress of pulmonary tuberculosis, 2 Ann Rev Tuberc, 470-485 (1919), and Lewis, et al, Early cell deprivation and non-human primates: long-term effects on survival and cell-mediated immunity, 47, Soc. of Biologic Psychiatry, 114-126 (2000), Selyi, Recent progress of stress research, with reference to tuberculosis, in: Sparer (Ed.), Personality, stress and tuberculosis (New York: Int. Univ. Press; 1956) at 45-64.

[8] See, e.g., Pollack, Setting body's 'serial killers' loose on cancer, The New York Times at A1, A3, A13 (8/2/16).

known to worsen the diverse range of neurological conditions – conditions which can be comorbid with traumatic brain injury in many cases.

These include Multiple Sclerosis, Tourette's syndrome, and others.

In cases of traumatic brain injury and "mind/body"/"psyche" dualism becomes even more absurd than it does in non-brain injured people because all of the known physical/"somatic" as well as "emotional"/"psychological" direct and indirect consequences of traumatic brain injury.

All have been documented for years and not just in specific texts of traumatic brain injury (e.g., by Silver, et al)[9] is the fact that traumatic brain injury is well-known in many individual to create a host of specific problems including imbalance, vestibular dysfunction in the head as well as chronic pain or fibromyalgia, and even mask cell (part of our cellular immune response throughout the body) dysregulation as well as psychiatric and family disturbances.

The Adams and Victors Principles of Neurology[10] text clearly and indisputably links traumatic brain injury to a host of "physical" and "emotional" psychological manifestations ranging from motor paralysis to tremor to gait disturbances to again pain in the back and neck, disturbances of vision, seizures, endocrine abnormalities, problems with respiration, psychiatric

[9] Silver, et al (Eds.), Textbook of Traumatic Brain Insjury, 2nd Edition (Washington, DC: American Psychiatic Publishing, Inc.) 2011.
[10] Ropper, et al (Eds.), Adams and Victors Principles of Neurology, 10th Edition (New York, NY: McGraw Hill, 2011).

disorders, and even (in cases of severe brain injury) an autonomic dysfunction "storm" syndrome.

The physical as well as "psychologic"/neurobehavioral consequences and direct results of traumatic brain injury also are discussed in Merritt's Neurology[11] which links traumatic brain injury to conditions including depression and anemia.

Then if one turns to a traditional classic medical text, e.g., Harrison's Principles of Internal Medicine[12], one sees multiple references to "psyche"/"mind/body" interfaces throughout the book, including there being specific references to stress in septic shock, stress ulcers, as well as in clear discussion of the hypothalamic-pituitary-adrenal axis[13], the hypothalamic-pituitary-gonadal axis[14] and the hypothalamic-pituitary-testes axis[15] as well as specific discussion of metabolic abnormalities including hypokalemia[16] (low potassium in the blood) and hyponatremia[17] (low sodium in the blood) from traumatic brain injury – with it being known of course that blood electrolyte

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[11] Louis, et al (Eds.), Merritt's Neurology, 13th Edition (Philadelphia: Wolters Kluwer) 2016.
[12] Kasper, et al (Eds.), Harrison's Principles of Internal Medicine, 19th Edition (New York: McGraw Hill) 2015.
[13] Kasper, et al (Eds.), Harrison's Principles of Internal Medicine, 19th Edition (New York: McGraw Hill) 2015 at 2310.
[14] Kasper, et al (Eds.), Harrison's Principles of Internal Medicine, 19th Edition (New York: McGraw Hill) 2015 at 335.
[15] Kasper, et al (Eds.), Harrison's Principles of Internal Medicine, 19th Edition (New York: McGraw Hill) 2015 at 2358.
[16] Kasper, et al (Eds.), Harrison's Principles of Internal Medicine, 19th Edition (New York: McGraw Hill) 2015 at 305.
[17] Kasper, et al (Eds.), Harrison's Principles of Internal Medicine, 19th Edition (New York: McGraw Hill) 2015 at 299.

abnormalities create neurobehavioral disturbances for the simple reason that blood goes everywhere in the body, including the brain....

Moreover, and "hot off the presses" was an important article describing and specifically "exploring the connection between diabetes and depression"[18] that blows a gigantic hole in any anachronistic vestigial claims that a "psyche"/"mind/body" split has any clinical or legal validity whatsoever.

The article clearly further discredits further any "mind/body" dichotomy with the statements that:

1. "*The relationship between diabetes and depression have been known for a long time. Each one is a risk factor for the other, and together and separately they are a risk factor for dementia*" (emphasis added).[19]
2. Although "certainly, many people's roots to depression and diabetes are just that, psychological reactions to having the other disease" but research shows that the *physiological relationship between the two* is much deeper."[20]
3. "A prospective study of 2,525 patient showed that those with depression and metabolic *risk factors* were more than six times

[18] Harris, Weight issues: exploring the connection between diabetes, depression, 40(7) Clinical Psychiatry News (July, 2016) at 5.
[19] Harris, Weight issues: exploring the connection between diabetes, depression, 40(7) Clinical Psychiatry News (July, 2016) at 5.
[20] Harris, Weight issues: exploring the connection between diabetes, depression, 40(7) Clinical Psychiatry News (July, 2016) at 5.

more likely to develop diabetes than patients who had depression alone, metabolic risk factors alone, are neither" (emphasis added).[21]

Indeed, the entire history of the field of "psychosomatic medicine" is one of course of exploring and researching "mind/body"/"psyche" relationships as well as demonstrating the absurdity of any false distinction between emotions and "mind" on one hand and physiological and neuroanatomical changes between the brain and the rest of the body on the other.

Consider an article on this very same July, 2016 issue of *Clinical Psychiatry News*: "Psychosomatic medicine – targeting vagal activity could improve breast cancer survival." [22]

Here there again was a clear "mind/body" clinically significant interchange, with the specific finding being that "*vagal [nerve] activity predicts survival in patients with metastatic or recurrent breast cancer"* (emphasis added).[23]

Amongst the most important recent explosions of the "mind/body" myth and corrolary emphasis of "mind/body" interactions and "comorbidities" was an

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[21] Harris, Weighty issues: exploring the connection between diabetes, depression, 40(7) Clinical Psychiatry News (July, 2016) at 5.
[22]Worcester, Psychosomatic medicine – targeting vagal activity could improve breast cancer survival, presented at the annual meeting of the American Psychiatric Association, 44(7) Clinical Psychiatry News (July, 2016) at 35.
[23] Worcester, Psychosomatic medicine – targeting vagal activity could improve breast cancer survival, presented at the annual meeting of the American Psychiatric Association, 44(7) Clinical Psychiatry News (July, 2016) at 35.

article appearing the summer, 2016 issue of *The Journal of Neuropsychiatric and Clinical Neurosciences.* The virtual entirety of this issue exploring – and exploding -- false "mind/body" relationships and false "emotional/brain" anachronistic and now clearly absurd dualisms.

Just the titles of some of these articles is revealing:

1. "Functional Neuroanatomy and Neurophysiology of Functional Neurological Disorders (Conversion Disorder)" which describes "bottom-up limbic influences interacting with and influencing basic motor function."[24]
2. "Apathy is Associated with Ventral Striatum Volume in Schizophrenia Spectrum Disorder."[25]
3. "Psychiatric Comorbidities in Restless Leg Syndrome." [26]

Even the perhaps traditional bastion of "pure" emotion and "psychological" versus "brain" and "body" has been discredited as well by, e.g., imaging studies that showed clear neurophysiologic and even neuroanatomic differences in individuals suffering from borderline personality disorder versus those who did not.

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[24] Voon, et al, Functional neuroanatomy and neurophysiology of functional neurological disorders (conversion disorder), 28(3) The Journal of Neuropsychiatric and Clinical Neurosciences 168-190 (2016).
[25] Roth, et al, Apathy is Associated with Ventral Striatum Volume in Schizophrenia Spectrum Disorder, 28(3) The Journal of Neuropsychiatric and Clinical Neurosciences 191-194 (2016).
[26] Kallweit, et al, Psychiatric comorbidities in restless legs syndrome, 28(3) The Journal of Neuropsychiatric and Clinical Neurosciences 239-242 (2016).

Regarding traumatic brain injury in particular, *The New England Journal of Medicine* 7/14/16 issue devoted an article to other things including collecting blood samples for genetic and protein analysis "of veterans who have had traumatic brain injury," as well as the falseness of distinguishing between traumatic brain injury "as a physical injury."

The authors instead consider "PTSD as a psychiatric condition" when "*the symptoms overlap, and the diagnoses coexist in many of these veterans" with "treating one often" alleviating symptoms "ascribed to the other*" (emphasis added).[27]

Furthermore, in the prior 7/7/16 issue of this journal, there had been specific reference to a "cutting edge" technology that goes way beyond issues related to traumatic brain injury but indeed provides perhaps the newest link weaving an inextricably intertwining web between the "brain," the "mind," emotions and the rest of the body.

Indeed here, biomarkers – the forefront of "unlocking precision medicine" throughout the body were linked to "genomic" – inherited – information that holds promise in fact "in selecting therapy that improves clinical care" despite the (*Daubert*-type) barriers to these being routinely used clinically, let alone in litigation because "potentially useful tests have not been adapted into clinical

[27] Okie, TBI's long-term follow-up, slow progress in science and recovery, 375(2) The New England Journal of Medicine 180-184 (2016).

practice rapidly, in part because we lack common evidentiary standings for regulatory, clinical, coverage, and reimbursement decisions.

Furthermore, clinical implication will require the consistent collection and sharing of data on biomarker tests, treatments, and patient outcomes.[28]

[28] Lyman, et al, Biomarker test for molecularly targeted therapies – the key to unlocking precision medicine, 375(1) The New England Journal of Medicine, 4-6 (2016).

§11:14.20 Critical "mind/psyche/body" issues regarding the accuracy, validity, and reliability of retroactive recollection of the traumatic event

One of the first things that one of the editors (JB) learned during his first day of medical school in 1963 was that it is essential to indicate in any medical record to what extent the patient giving "his or her" story – the "history" – is an accurate factual historian whose recollections, perceptions, and claims of past events should or should not be taken as gospel truth.

The ability of an interviewee to give an accurate history often is included by one or more of the following events, even in individuals warranting any "brain injury and/or formal psychiatric diagnosis but even in completely "normal" individuals. The history-contaminating factors are known by any sophisticated physician to range from simple pride to desires for attention and sympathy (part of "secondary gain") to the psychological reasons to misperceive a problem as being physical as opposed to "mental" ("primary" gain) to pressure from parents (particularly in child custody disputes) to conscious and/or unconscious desires to create a picture of "damages" for litigation purposes beyond that which is truly objectively justified.

This accuracy has two elements: validity (the history reflecting what actually occurred) on one hand and "reliability" (the ability of the same history to be given consistently over time).

One of the most striking experiences this examiner had during his first week of medical school and during the years thereafter when he has taught clinical interviewing in virtually all areas of medicine, not just psychiatry, was how a patient's history – recollection – relating of claimed events dramatically can and has changed, even from week to another – and even when there is no litigation involved.

It indeed is for that reason that this examiner still when he teaches second year medical students about clinical interviewing always tries to have the students re-interview patients if they are in the hospital for more than one week since the histories given so often and so dramatically change.

One of the more dramatic experiences one of the editors (JB) had was when he interviewed a four year old child after having been appointed by a Family Court judge to help resolve a child custody dispute. There was nothing physically, psychiatrically, or in any "brain damaged" diagnostic way anything wrong with this four year old girl.

Then, when this examiner took her history, she spontaneously exclaimed that, "My daddy is a menace to society!" She in fact was quite empathic on that point and spontaneously repeated that sentence at least three times during the interview.

Then when this examiner asked her what the word "menace" meant, she had shrugged her shoulders, smiled and had said, "I don't know."

Then when he asked her what the word "society" meant she actually laughed and said "I don't know!"

When this examiner finally asked her then why she said that sentence to him, this completely otherwise "normal" child smiled again and said, "Well, I don't know, my mommy told me to tell you that!"

The importance of investigating the reliability and validity of a patient's "story" even in otherwise "normal" individuals not only requires investigation of any "external" influences and motivations that could influence the historian's reports but is particularly important even in those not formally diagnosed with "brain" and/or "psychiatric" problems, when complaints made that are at issue in a lawsuit are largely subjective. These latter complaints, which clearly involved "mind/body" interactions rather than a "one or the other" approach range from subjective symptoms and complaints including headaches, somatic pain, complaints of sensory loss, and many others that require behavioral medicine investigation even when a plaintiff and/or attorney formally drops any and all "mental" and/or "psychiatric" claims of injury.

Again, the entire modern field of pain medicine, "psychosomatic" medicine, and what DSM-5 characterizes as "Somatic Symptom Disorders," "Illness Anxiety Disorder," "Functional Neurological Symptom Disorder," "Psychological Factors Affecting Other Medical Conditions," "Factitious Disorder," "Other Specific Somatic Symptoms and Related Disorders," and

"Unspecified Somatic Symptom and Related Disorder[29] all involve recognition that one simply cannot artificially separate "mental" and "psychiatric" perceptions, misperceptions, claims and true dysfunction in an artificial "mind/body" dichotomy.

The absurdity of having any artificial "mind/body" dichotomy and ignoring not only how motivation affects history accuracy in even "healthy" people making no "psychiatric" damages claims on one hand as well as the absurdity of ignoring the reality that the nervous system itself literally inextricably ties together the "brain" with the "rest of the body" is demonstrated through DSM-5, with one example being the conditions required for "Functional Neurological Symptom Disorder" of which there are eight different subtypes (with their International Classification of Diseases Codes):

1. "(F44.4) With weakness or paralysis."

2. "(F44.4) With abnormal movement (e.g., tremor, dystonic movement, myoclonus, gait disorder);
3. "(F44.4) With swallowing problems";
4. "(F44.4) With speech symptom (e.g., dysphonia, slurre4d speech)";
5. "(F44.5) With attacks or seizures";

[29] American Psychiatric Association, Desk Reference to the Diagnostic Criteria from DSM-5 (Washington, DC: American Psychiatric Publishing 161-167 (2015).

6. "(F44.6) With anesthesia or sensory loss";

7. "(F44.6) With special sensory symptoms (e.g., visual, olfactory or hearing disturbance); and

8. "(F44.7) With mixed symptoms."

Note then that further that physicians of all specialties – including those who are not dealing with presentations of "psychiatric" or "psychological" disturbance but instead of "physical" problems ranging from paralysis to weakness to tremors to seizures and sensory loss must be familiar with the psychiatric Diagnostic Manual and all of the "mind/body" relationships clearly set forth therein.

♦**PRACTICE NOTE**: Any attempt to "plead away" issues related to the factual accuracy of a historian and the corollary need to have behavioral medicine expertise in investigating this as well as any attempt by any attorney – or judge – to ignore the reality of the fact that the mind and the body are inextricably intertwined and that behavioral medicine expertise is required to determine the interrelationship between physical complaints and mental state/motivation even in individuals making no "pure" psychiatric claim is fraught with the risk of grave mischaracter of justice which should lead to appellate findings of reversible error.

Simply put, clinging to anachronistic notions that there is a "mind/body" choice in pleadings, claims, and testimony risk making the entire trial a travesty clinically and legally.

As a consequence of the ignoring of the critical importance of determining history accuracy and including "mind/body" interactions and specifically the "somatoform" disorder as part of admitted testimony, even when formal "psychiatric" claims are dropped, the result has been all too often chaos and confusion for attorneys and jurors alike.

Indeed, what happens all too often is that an attorney (on either side) simply has taken what a patient claims as recalled fact as gospel truth and simply passes that erroneous perception and/or erroneous history on to the next clinician or lawyer, with there being the same kind of potentially horrific clinical and legal consequences regarding this "telephone game" as there are underlying all of the exclusions and exceptions to the "hearsay" rule and the law.

♦**PRACTICE NOTE**: Simply put, repeated witness statements often actually contradict themselves, having not only no intrinsic reliability but often quite the opposite. One only has to look at the movie "Twelve Angry Men" with Henry Fonda, the movie "Rashomon" and even to the lyrics of the song, "Yes, I remember it well" with this point to be driven home.

Yet all too often physicians commit the grave error that the hearsay rule prevents attorneys from committing at least in court: blindly repeating and taken as fact history which has its own inherent unreliability in many cases.

The sources of this potential witness and plaintiff factual unreliability are worth repeating since they have been so often ignored by so many over the years. Again, these range from true memory disturbance resulting from traumatic brain injury on one hand to unconscious "hysterical" symptom exaggeration and history distorting behavior fueled not necessarily by litigation but by classic "secondary gain" needs for sympathy and assistance, to infections that affect memory (e.g., neurosyphilis, tuberculosis, and HIV, amongst others), to the cognitive effects of brain cancer.

Moreover, even conditions when thought to be essentially "psychological" in nature – including posttraumatic stress disorder and depression – not only have well-known cognitive and memory distorting effects but the fact that there are medications that are known to improve cognitive functioning (including classically Ritalin and Adderall in some instances and antidepressants in others) further make it imperative for attorneys on both sides to investigate carefully with the help of neuropsychiatrists as well as objective "collateral" sources of behavioral data (not just friends of the plaintiff but pre-accident medical and pharmacy records) to determine to what extent a

plaintiff patient's retroactive account of a trauma and its effects can be believed by a clinician and attorney, let alone a juror.

Ironically, one of the easiest ways for this to be done and in the editor's experience assiduously avoided by both plaintiff and defense attorneys is the taking of serial neuroradiological and electrophysiological studies (CAT scans, MRIs with diffusion tensor imaging, electroencephalograms, and x-rays of reportedly broken bones).

Instead, the usual approach appears to be that all studies that are taken are taken right after the accident – with there being an implied assumption that these injuries don't heal, when there is a tidal wave of material cited in this and other volumes related to the concept of neuroplasticity and resilience.

♦**PRACTICE NOTE:** As an example, if a person has had intracerebral bleeding at the time of an impact, and if that person is continuing to complain for years about memory and other cognitive problems, it would make sense for there to be an MRI with DTI and/or CAT scan and/or EEG (especially a 72 hour video EEG) done within the months before a trial.

That way it could be determined if there is a lesion in the brain or not and whether or not if there is a lesion its presence and size correlates with the results of what typically all too often again are neuropsychological tests that are never repeated.

> If both sides simply could agree that these studies should be done, the editors predict that many more cases would be settled for far less money and far less emotional angst for the plaintiff patients than otherwise would be the case....

In any case, clearly, the need to determine the accuracy, validity and reliability of a patient's history is an area that clearly mandates neuropsychiatric investigation, regardless on which "side of the aisle" the attorney sits.

Such an investigation has nothing whatsoever to do with whether are not there are specific "emotional" and/or "psychiatric" claims as part of the original or amended Bills of Particular and Complaints but strike at the core of whether or not the jury should be alerted to factors that would make the plaintiff's testimony one which should be given substantial or little signs of credibility.

Even though a determination of factors are within the province of the jury, nonetheless the whole basis of jurors hearing scientific testimony and expert testimony is to inform the jury to what extent the facts as presented by the attorneys (plaintiffs and defendants) are given great or little weight.

It is of course the ultimate decision of the juror to weigh the facts, but the editors see nothing in the legal literature or case law to date prohibiting a neurobehavioral expert from providing data that would help the jury more effectively reach its own conclusions about when and how much to believe the

history given to it either by a plaintiff or by what frankly is second hand repetition of patient history of patient plaintiff's themselves....

To put all of this in other terms, neuropsychiatric testimony and neuropsychological test should ***not*** be precluded by the mere fact that a plaintiff attorney decides to "drop" any "mental" claims and/or any "psychiatric" and/or "emotional" claims of damage or injury.

§11:14.30 Intended and unintended consequences of "mind/body" dualism on admissibility of psychotherapeutic records [New]

One of the most important challenges raised for judges and attorneys accepting the reality that even in the absence of "psychiatric" claims it is essential to seek and admit expert behavioral medicine testimony related to patient factual reliability, "mind/body" interconnections including the "somatoform" disorders, etc. even in the absence of/dropping of "psychiatric" or "mental" claims is what types of information are most likely to provide the most clinically and probatively important data to create the most accurate picture possible of even a non-brain damaged, non-"psychiatric" patient's clinical presentation and information related to "physical" complaints like pain or physical disability.

The answer is clear if one asks one's self the following question: What professional is most likely to be told all historical data in an open and trusting fashion that would help a behavioral medicine specialist give a fair and accurate and complete picture both of patient factual reliability in any clinically significant "mind/body" issues?

The answer? Their treating mental health professional.

Yet, there are many and frankly understandable barriers to simply permitting a behavioral medicine expert during litigation to have free access to records of a plaintiff patient's psychotherapist or other mental health professionals. The reason why is that the need for this often critically important clinical information contained in the mental health professional's records collides with an equally important need – that of respecting patient privacy.

Consider for example §90.503 of the Florida statutes (Eff. 7/10/14): That statute provides amongst other things sub-section (2) that "a patient has a privilege to refuse to disclose, and to present any other person from disclosing, confidential communications or records made for the *purpose of diagnosis or treatment of the patient's mental or emotional condition, including alcoholism and other drug addiction*, between the patient and the psychotherapist, or persons who are participating in the diagnosis or treatment under the direction of the psychotherapist. This privilege includes any diagnosis made, and advice given, by the psychotherapist in the course of that relationship" (italics added).

Note that in sub-section (1) "A 'patient' is defined as *one seeking treatment for a mental or emotional disorder as opposed to one consulting a psychotherapist for business or other professional purposes"* (italics added) – with the latter presumably including consulting a psychotherapist in connection with a lawsuit.

That sub-section goes on to be explicit in stating that "*the rationale for extending confidentiality to the psychotherapist-patient relationship is to assist successful treatment, and there is no plausible reason to extend it to communications where a businessman seeks psychological advice on consumer purchasing motivations or a lawyer prepares materials for a law review article concerned psychiatry or psychology and its relation to law"* (emphasis added).

> ♦**PRACTICE NOTE:** Note then that even here, at least by implication, the privilege should not apply when the primary purpose for the patient seeking the treatment is related to ongoing litigation, and that assessment of the litigation claims, even seemingly purely "physical ones" like pain and physical limitations themselves are part of the litigation claims and should be assessed by experts capable of determining whether that complaint/claim of disability/functional limitation is potentially a manifestation of "somatoform disorder" and/or "mind/body" confusion and/or influence by others and/or motivational factors even in someone not making psychiatric claims but nonetheless having been troubled by some sort of stress or problem to the point that they had sought out psychotherapeutic treatment in the first place.

Regarding the Florida Courts' interpretation of the statute and the times when a psychotherapist/patient privilege can or cannot be waived are contained in two cases this examiner found, both of which are over twenty years old (with this writer's search for more recent cases unfortunately not being successful):

The 1993 case of Sykes v. St. Andrew's School,[30] and the 1995 case of Helmic v. McKinon. [31]

The first case was decided in the 4th district of the District Court of Appeals of Florida and the second in the 5th District of the District Court of Appeals of Florida.

In the first of these, the 4th District Court of Appeals held that patient's initially waiving the psychotherapist/patient privilege in fact had been revoked "when patient abandoned claim for emotional distress." This was true even the patient already had voluntarily submitted to a defense expert "mental examination."[32]

The statutory authority cited did indicate that there was no privilege if the patient relied upon the psychiatric condition "as an element of his claim or defense" with the Court in this case stating that "the burden rests

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[30] Sykes v. St. Andrew's School, 619So.2d467 (1993).
[31] Helmick v. McKinnon (5th District Court of Appeals; 1995) at 1279.
[32] Sykes v. St. Andrew's School, 619So.2d467 (1993) at 468.

on the party seeking to depose a psychotherapist to show that the patient has introduced his mental condition as an issue in the case.

Note, however, that if one accepts modern science – and indeed takes judicial notice of the clear inextricable interconnections between the "mental condition" a person might have and the physical condition, the reality of "psychosomatic medicine" and recognition by physicians who are not just psychiatrists of physical complaints of pain and neurologic dysfunction that are in reality "conversion" of "mental" conditions into the *misperception* of physical complaints, this case does not rule out but rather rules in as necessary exploration of psychotherapeutic records that at least arguably would contain important data that would document one or more of the following:

1. Whether or not a patient claiming only "physical" complaints had a documented factual history in the psychotherapy records of having been physically abused or battered at any time in that person's life;
2. Whether or not the patient as part of the psychotherapeutic process – as often happens – had psychometric tests given that have the ability to uncover "mind/body" confusion on the part of the patient, suppression of emotional problems and "translation" of them into misperceptions of exaggerated physical problems, and

indeed whether or not the dropping of "mental" claims is frankly done either accidentally or even deliberately by some attorneys to conceal from the Court and confuse a jury into believing that the person's physical complaints might not be objectively validated, might lead to unnecessary invasive surgery, and ultimately be harmful to the plaintiff litigant as well as confusing and misleading to the jury.

In such cases, even if there is an initial question in the mind of a judge about whether or not a plaintiff patient implicitly waived the psychotherapist-patient confidentiality privilege in order to obtain such data as a matter of general principle, at the least such privilege should be waived when testing that is done by an expert in behavioral medicine which had not been done before in fact reveals the presence of findings and/or diagnoses including:

a. "Hysteria";

b. "Hypochondriasis";

c. "Somatic delusions";

d. "Somatoform disorder" and/or "somatic symptom disorder";

e. Findings pointing to neurologic problems;

f. Findings pointing to "mental" complaints being inaccurately dressed in "physical" clothing and symptoms;

g. Discrepancies between subjective physical complaints and lack of objective substantiation of them on examinations, complaints and retroactively exaggerated history by litigants of "brain damage" when no objective signs of such are documented in the emergency room, when patients claim they have been subject to radiological studies showing "brain damage" when the radiological studies show no such thing, etc., etc..

The issues here again go well beyond those involved in cases with alleged "traumatic brain injury" and/or alleged "psychiatric disorders" to include issues of determining to what extent the patient is an accurate and valid factual historian whose claims of events can be relieved, whether or not the plaintiff/litigant has been pressured by others to give an inaccurate history and/or make palpably false factual statements (as noted has occurred in one of the editors' experience in child custody cases), etc., etc..

As the Court said, in Sykes "one purpose of the waiver or exclusionary portions of the rule and the statute it to prevent a party from losing the privilege as both as a sword and shield, that is, *seeking to recover for damage to the emotions on the one hand while hiding behind the privilege on the other***"** (emphasis added; last page of decision).

Here, the Court found that because after first putting her "mental condition at issue" to "recover damages for her own emotional stress" but that "she subsequently abandoned that attempt" does **not** and with all due respect to the Court in this case mean that "her mental condition is no longer an issue…"

Moreover, the Court claimed at the end of the decision that there was "no residual effect from petitioner's early position that causes prejudice to the defense" frankly this examiner believes at least in the 21st century this is not a correct and scientific valid position to taken since the reason why a plaintiff would in many cases and has in many cases withdrawn a "mental" claim was to further enable the patient suffering from a true state of mind/body confusion and mis-presenting and mis-perceiving physical complaints as not being "mental" gets lost – and lost to a point of potentially fatally compromising the defense's ability to introduce 21st century behavioral medicine and neuroscience to the jury with the Courts of course certainly having the right to subject any motions to have the psychotherapist-privilege waived subject to *Daubert* and *Frye* analyses related to scientific credibility, "general acceptance," etc..

♦**PRACTICE NOTE:** The editors expect that defense counsel more and more will get judges to take formal judicial notice of modern 21st century neuroscience and the universally

> recognized by physicians in all specialties of the absurdity of any artificial "mind/body" distinction which may have existed in the minds of physicians as well as jurors in the previous centuries but is clearly outmoded today.
>
> Furthermore, the editors predict that "psychosomatic" books, testimony, articles, and tests (e.g., biomarker tests, brain-sensory biofeedback studies, etc.) addressing the inextricable interconnections between the brain/"mind" and the "physical" body will have little trouble getting past any *Frye* and/or *Daubert* exclusionary motions.

The second case found, the 5th District Court of Appeal's Helmick v. McKinnon 1995 case, at the outset again must be pointed out as being twenty years old and rooted in 20th and not 21st century generally accepted scientific beliefs.[33]

In this particular case, the plaintiff's pre-accident psychological and psychiatric records were relevant in view of the fact that the plaintiff's alleged accident caused brain damage that caused personality disorders and that defendant's medical expert asserted that such records were necessary to

[33]Helmick v. McKinnon (5th District Court of Appeals; 1995) at 1280.

formulation of opinion as to whether accident-caused plaintiff's disorders or whether they pre-existed the accident.[34]

Here the Court did acknowledge that the plaintiff had placed "mental health in issue" with the "relevancy" of the inquiry at issue being the expert's need to review records "in order to formulate an opinion as to whether the accident caused the plaintiff's current personality disorder or if they existed [in part or otherwise] prior to the accident. "

"Without these materials, Helmick will be unable to properly formulate his defense, and there is no better or available source for this information other than the counselor's notes"[35](emphasis added).

Note that even in this case there was no issue made about the inquiry in order to determine specific facts related to the plaintiff's actual ability to accurately remember events even though she was alleging that she had brain damage in the accident.

Furthermore, ironically, her claim that brain damage in fact can create personality disorders is a proposition that has been getting more and more support from 21st century "biomarker" neuroradiological metabolic studies.

♦**PRACTICE NOTE:** In the future, the issue of to what extent, if any, an alleged traumatic brain injury substantially causes and/or

[34] Helmick v. McKinnon (5th District Court of Appeals; 1995) at 1279.
[35] Helmick v. McKinnon (5th District Court of Appeals; 1995) at 1280.

> exacerbates the chemical, radiological, including clinical manifestations of personality disorder likely will become a question of not "if" but "how much," "when," and "why."
>
> Thus, here, unlike the likelihood that defense counsel will prevail on motions to "pierce" the psychotherapist-patient privilege in cases of alleged physical pain and an exacerbation of "preexisting" conditions, here the editors predict that defense counsel likely will lose in any attempts to claim to jurors that because a "personality disorder" is present.

Personality disorders *by definition* are chronic, life-long, and of necessity would pre-date the accident.

The 21st century clinical reality is that traumatic brain injury not only is known to cause personality changes that would "test" an incidence like the MMPI-2 that relies on Axis II "personality disorder" formats as a life-long "personality disorder" when not only can traumatic brain injury exacerbate prior "personality disorder" symptomatology but create new and emerging personality disturbances as the direct result of the traumatic injury, objectively validated orthopedic injuries and pain, the side effects of new post-accident medications needed to treat the person's pain, cognitive problems, and emotions, etc., etc..

CHAPTER 15. STRATEGIC PLANNING FOR FUTURE LITIGATION

§15.27.1 Update On Brain Injury Biomarker Explosion [New][36]

Some attorneys still claim that "biomarkers" do not exist and/or reveal themselves with all due respect to be completely ignorant of what the term means and its time-honored history in medicine.

For that reason, Mr. Jared Squires, a Harvard pre-med graduate and primary author of this section (his biography appears at the front of this physician) undertook a review of the history of "biomarkers," their time-honored use not just in neurology and neuropsychiatry but in general medicine, and some of the most recent literature specifically addressing their limitations in traumatic brain injury diagnoses and litigation.

First, an issue of definition: "Biomarkers" means simply that – biologically-generated substances (and/or behaviors) that have the potential to "mark" or identify the presence of an underlying diagnostic entity. Their presence, typically the result of injury on a cellular level than the result of trauma and/or a host of other potential "causes" unrelated to trauma do not permit as of yet

[36] The principal author of this section was Mr. Jared Squires, whose credentials, biography, and contact information appears at the beginning of this volume.

biomarkers generally to be deemed *specific* "fingerprints" for a particular diagnostic problem although they often are highly *sensitive* to the presence of the problem.

Amongst the most time-honored "biomarkers" are enzymes, and one of the most known contexts of their use in fact has not been in traumatic brain injury but in cardiology.

Consider, for example, statements contained in the 2011 issue of the widely read Merck Manual Diagnosis and Therapy[37] addressing acute coronary syndromes.

Note that "markers" for acute coronary events are "cardiac enzymes (e.g., CK-MB) and cell contents (e.g., troponin I, troponin T, myoglobin) that are released into the bloodstream after myocardial cell necrosis." [38]

Moreover – and in fact unfortunately in the editors' experience has not been appreciated in the context of brain injury biomarkers – these cardiac "markers appear *at different times after injury and decrease at different rates*" (emphasis added).[39]

Furthermore, cardiology also has been one of the areas in which "cutting edge" imaging studies have already started to be used, at least in a research

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[37] Porter, et al, The Merck Manual of Diagnosis and Therapy (eds), Nineteenth Edition (New Jersey: Merck, Sharp & Dohme Corp.) 2011 at 1999-2105.
[38] Porter, et al, The Merck Manual of Diagnosis and Therapy (eds), Nineteenth Edition (New Jersey: Merck, Sharp & Dohme Corp.) 2011 at 2104.
[39] Porter, et al, The Merck Manual of Diagnosis and Therapy (eds), Nineteenth Edition (New Jersey: Merck, Sharp & Dohme Corp.) 2011 at 2104. See also the chat at 2105.

fashion, to investigate not just abnormal cardiac functions but changes in normal cardiac function; e.g., in normal physiological changes in part of the heart (the left ventricles specifically) during normal pregnancy.[40]

> ♦**PRACTICE NOTE**: The editors consistently have found that jurors invariably understand and "relate" to analogies and issues related to bones, and automobiles particularly well. The concept that damaged or "broken" car tissue will leak chemicals into the blood which can be detected really is the basic image behind valid biomarker studies.
>
> Similarly, the detection of biomarkers on x-rays from broken bones demonstrates tissue damage at a cellular level, and even a "marker" for car engine damage which consists of finding pools of oil on a garage floor when there is a "broken" or injured or damaged seal or gasket in the parts of a car that normally would contain engine oil to the engine….

So, one of the most striking features of cardiac enzyme "biomarkers" again is that they c*hange in nature and in presence over time.* The *combination* of the presence of varying biomarkers of heart injury that appear and disappear over time in a specifically known fashion the *overall pattern of*

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[40] Cong, et al (including Jared Squires), Structural and functional changes in maternal left ventricle during pregnancy: a three-dimensional speckle-tracking echocardiography study, 13 (6), Cardiovascular Ultrasound 1-10 (2015).

the cardiac biomarkers over time are not just sensitive indicators of cardiac injury but specific "fingerprints" for them as well that indeed permit when one looks at the entire pattern of biomarkers over time medically justifiable statements that this particular pattern of biomarkers of heart injury over time indeed does represent a specific as well as sensitive "fingerprint" for cardiac injury.

♦**PRACTICE NOTE:** Unfortunately, the writers of this section (primarily Mr. Squires as noted) could not find as of yet any body of literature in refereed journals that has even undertaken to taken the somatic attempt to examine and correlate the appearance and disappearance of reported "biomarkers" for traumatic brain injury over time to fashion a "fingerprint" of the type that already exists in cases of cardiac tissue damage.

1. When such emerges, the entire field of traumatic brain injury in the writers' views will be transformed. On one hand, plaintiff attorneys who have the studies done likely will be able to exclude at the outset the clients where all the known biomarkers for brain injury are negative, thereby saving time and expense for both them and their clients.

2. Defense counsel on the other hand undoubtedly in the future will use known "fingerprints" for brain injury via patterns of biomarkers appearing and disappearing over time as important to win arguments that the plaintiff litigants indeed did not suffer any true clinically significant traumatic brain injury at all.

One final caveat about cardiac illness "biomarkers" as well as other cardiovascular biomarkers: Despite all of the research already done and literature published about cardiac injury biomarkers, the needs of these markers are not the "be-all and end-all" of diagnosing heart attacks.

See, for example, articles published as early as 2010 noting that even the presence of "serum biomarkers" and even EKG changes did not reveal any "biomarkers *specific to myocardium"* (emphasis added).

Furthermore, although there have been "a high incidence of major and unequivocal myocardial infarctions" on the EKG especially related to ST elevation there were also "occasional more borderline syndromes," leading the articles to note that here (as, the writer's note, in traumatic brain injury) "*symptoms continue to be the key diagnosis and ECG technology has not changed....The focus of improving diagnosis has been the development of rapid assays of much more sensitive and specific biomarkers," but even a*

traditional biomarker, creatinine kinase itself has "now been swept away from troponin revolution" (emphasis added).[41]

Yet even with the continued emergence of troponin specifically as the "hot new" cardiac illness biomarker, caveats and conditions remain, particularly of importance to attorneys who ignore the fact that even here this biomarker has "emerging" significance.

Note specifically the article in Hoff, et al, "Troponin in Cardiovascular Disease Prevention: Updates and Future Direction,"[42] which again highlight how most recently "cardiac troponin" in the abstract "has been well described as the preferred biomarker for diagnosis of myocardial infarction due to the high sensitivity and specificity of myocardial injury" – yet even here the issue of specificity raised its head since "numerous other conditions apart from acute coronary syndrome can also lead to small elevations in troponin level" noting consequently "the development of newer generations of high-sensitivity cardiac troponin assays."[43]

The conclusion is that although "evidence continues to show that high-sensitivity troponin is emerging as one of the most powerful prognostic biomarkers for the assessment of cardiovascular risk in the general population" –

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[41] Mann, Diagnosing a Heart Attack – Are Biomarkers the Be-All and End-All? 64 New Zealand Journal of Medical Laboratory Science (2010) at 38.
[42] Hoff, Tropinin in Cardiovascular Disease Prevention: Updates and Future Direction 18 (12), Current Atheroscler. Rep. (2016).
[43] Hoff, Troponin in Cardiovascular Disease Prevention: Updates and Future Direction 18 (12), Current Atherosclerosis. Rep. (2016).

note the word "emerging" here as opposed to "established with reasonable medical probability" let alone "certainty." [44]

Furthermore, the attempt to expand the use of biomarkers to investigate the nature, causes, and correlations with the presence of biomarkers and transient ischemic attacks and/or strokes also demonstrates the historical limitations of biomarkers even here.

First of all, as noted in a 2014 article, "Premature Death After Transient Ischemic Attacks"[45] is "more often because of heart disease or cancer than stroke" – again destroying any concept of "causal connection" or specific "fingerprints" for the outcome of death after stroke or stroke itself....

Furthermore, previous studies found blood biomarkers *not "usefully predictive of non-fatal stroke"* and only "possibly" of "all-cause death."[46]

What is important also with stroke – as with cardiac disease – was the significance of using *more than one biomarker* with the authors concluding that when they use four different biomarkers "the independent contribution of the four biomarkers *taken together added prognostic information and improved*

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[44] Hoff, Troponin in Cardiovascular Disease Prevention: Updates and Future Direction 18 (12), Current Atherosclerosis. Rep. (2016).
[45] Greisenegger, et al, Biomarkers and Mortality After Transient Ischemic Attack and Minor Ischemic Stroke Population-Based Study, 46 Stroke (2015) at 659.
[46] Greisenegger, et al, Biomarkers and Mortality After Transient Ischemic Attack and Minor Ischemic Stroke Population-Based Study, 46 Stroke (2015) at 659.

model discrimination (integrated discrimination improvement) at the 0.0001 level."[47]

Yet even here the authors acknowledge the limitations of their studies – a limitation that attorneys planning to rely on these results or have their experts rely on this literature clearly state that "*our results require validation in future* studies" (emphasis added).[48]

> ♦**PRACTICE NOTE:** Keeping all of the above qualifications in mind, consider the potential positives of at least using the presence of "biomarkers" of brain tissue injury in a fashion that as part of a global picture of data *consistent with* the presence of traumatic brain injury – and even with statements related to their presence being *consistent with* the presence of the biomarkers being the direct result of such brain injury.

Regarding brain injury biomarkers, as noted in previous sections of this book (§20:19.20, §20:19.21 and §20:19.70),[49] the editors discussed the ongoing search for objective (sensitive and specific) traumatic brain injury markers that

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[47] Greisenegger, et al, Biomarkers and Mortality After Transient Ischemic Attack and Minor Ischemic Stroke Population-Based Study, 46 Stroke (2015) at 659.
[48] Greisenegger, et al, Biomarkers and Mortality After Transient Ischemic Attack and Minor Ischemic Stroke Population-Based Study, 46 Stroke (2015) at 659.
[49] Dotson and Brown, et al, Emotional Injuries (Thomson West: Eagan, MN) at 1339-1344.

appeared in research in both neuroendocrinology, behavioral medicine, brain injury, and neuroimaging.

Also addressed in §20:76.10 had been a discussion of "The Future: Neurochemical and Neuroanatomical Causal Connection Markers[50] which specifically highlighted "the current explosion of neuromolecular, neuroanatomical, and behavioral research related to essential courtroom applications – and limitations – of the use of molecular, radiological, and neurobehavioral "biomarkers" to "prove" the presence specific brain abnormalities and/or their causes.[51]

> ♦**PRACTICE NOTE:** The editors' warning that, as a practical matter, studies like all other recent "cutting edge" technologies have as their valid place in court *only their use not as "stand alone" independent "proof" of diagnosis and/or causation but rather as part of the package of information that should be deemed "consistent with" other studies* (emphasis added) has often nonetheless been ignored by over-zealous attorneys on both sides.

1. For example, at least one of the editors' (JB) has had all too much continuing experience with some plaintiff attorneys who still attempt

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[50] Dotson and Brow Dotson and Brown, et al, Emotional Injuries (Thomson West: Eagan, MN) at 1484-1487.n, et al, Emotional Injuries (Thomson West: Eagan, MN) at 1484-1487.
[51] Dotson and Brown, et al, Emotional Injuries (Thomson West: Eagan, MN) at 1486.

to "prove" diagnoses and even causation by relying too much on "biomarker" studies as "stand alone" evidence.

2. At the same time, he also has had the equally unfortunate experience of being pressed by defense counsel who have made over zealous attempts to completely exclude "biomarker" data on *Daubert* and/or *Frye* grounds.

After all, due to the subjective nature of the current protocols for diagnosing TBI as well as the inconsistent ability of imaging technology to quickly and efficiently confirm the presence of said injury, a more objective measure would be ideal. Biomarkers have a great amount of potential to accomplish this. The biological markers of TBI that are present in the bloodstream post-trauma could serve as a non-invasive and easy to obtain source of samples from a patient, lead to a quicker and more accurate initial diagnosis of TBI, and would have prognostic value throughout the course of treatment to help track the progress of recovery.[52]

As discussed by Frederick Korley, M.D., Ph.D. at the Johns Hopkins University of School of Medicine, the improved ability to generate an early diagnosis and prognosis for TBI represented by biomarkers could allow for

[52] Foerster, Serum Biomarkers to Diagnose Mild Traumatic Brain Injury in Adults, 122 Issues in Emerging Health Technologies (Ottawa: Canadian Agency for Drugs and Technologies in Health, 2014).

doctors to develop a more complete and informative course of action for their patients, including identifying the patients who will need the most extensive amount of aid as well as the best candidates for clinical trials of new drugs[53]. A rapid blood based test may save time that would have been spent on inconclusive imaging[54], or complement an imaging test to help avoid underestimating how severe a TBI is.[55,56]

Currently, blood based biomarkers as applied to TBI have been tested for clinical use in small studies conducted in Spain, Switzerland, Austria, the Netherlands, and New York City[57], with a collaboration between 22 European countries and Israel for a longitudinal study currently being conducted at the time of this writing.[58]

In 2008, the American College of Emergency Physicians acknowledged the potential of biomarkers to contribute to TBI research: they concluded that

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[53] Cashin-Garbutt, Diagnosing Traumatic Brain Injury Through a Blood Test: An Interview with Dr. Korley, News Medical; http://www.news-medical.net/news/20150901/Diagnosing-traumatic-brain-injury-through-a-blood-test-an-interview-with-Dr-Korley.aspx (September, 2015).

[54] Carpenter, System, Local and Imaging Biomarkers of Brain Injury: More Needed and Better Use of Those Already Established? 26(6) Frontiers in Neurology (February 2015) at 1-20.

[55] Jagoda, et al, Clinical Policy: Neuroimaging and Decision Making in Adult Mild Traumatic Brain Injury in the Acute Setting, 52 American College of Emergency Physicians; Centers for Disease Control and Prevention, Annals of Emergency Medicine (2008) at 714–748.

[56] Undén, et al, Scandinavian Guidelines for Initial Management of Minimal, Mild and Moderate Head Injuries in Adults: An Evidence and Consensus-Based Update, 11 BMC Medicine (2013) at 50.

[57] Foerster, Serum Biomarkers to Diagnose Mild Traumatic Brain Injury in Adults, 122 Issues in Emerging Health Technologies (Ottawa: Canadian Agency for Drugs and Technologies in Health, 2014).

[58] Maas, et al, Collaborative European NeuroTrauma Effectiveness Research in Traumatic Brain Injury (CENTER-TBI): A Prospective Longitudinal Observational Study, 76(1) Neurosurgery (2015) at 67-80.

S100B had potential as an aid in pre-CT screening, and in tandem with NSE or Tau it may improve detection of intracranial abnormalities.[59]

Furthermore, the economic costs that were unknown and only speculated on at the time[60] have since been analyzed.[61]

As of 2007 the Scandinavian Neurotrauma Committee officially approved the usage of the biomarker S100B as an option to evaluate TBI patients with a risk of intracranial complications,[62] which was continued in updated guidelines 6 years later in 2013.[63]

In 2014, the Canadian Agency for Drugs and Technologies in Health also ran a review of TBI biomarker studies,[64] showing that there is worldwide interest in the potential.

As samples for these biomarkers would be taken with blood samples from a patient, the acquisition and testing process itself does not pose an additional

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[59] Jagoda, et al, Clinical Policy: Neuroimaging and Decision Making in Adult Mild Traumatic Brain Injury in the Acute Setting, 52 American College of Emergency Physicians; Centers for Disease Control and Prevention, Annals of Emergency Medicine (2008) at 714–748.

[60] Jagoda, et al, Clinical Policy: Neuroimaging and Decision Making in Adult Mild Traumatic Brain Injury in the Acute Setting, 52 American College of Emergency Physicians; Centers for Disease Control and Prevention, Annals of Emergency Medicine (2008) at 714–748.

[61] Ruan, et al, The Economic Impact of S-100B as a Pre-Head CT Screening Test on Emergency Department Management of Adult Patients with Mild Traumatic Brain Injury, 26(10) Journal of Neurotrauma (2009) at 1655-1664.

[62] Calcagnile, et al, Clinical Validation of S100B Use in Management of Mild Head Injury, 12(10) BMC Emergency Medicine (2012) at 13.

[63] Undén, et al, Scandinavian Guidelines for Initial Management of Minimal, Mild and Moderate Head Injuries in Adults: An Evidence and Consensus-Based Update, 11 BMC Medicine (2013) at 50.

[64] Foerster, Serum Biomarkers to Diagnose Mild Traumatic Brain Injury in Adults, 122 Issues in Emerging Health Technologies (Ottawa: Canadian Agency for Drugs and Technologies in Health, 2014).

risk for the patient. It is extremely important for biomarkers avoid a false negative test as that result could delay diagnosis and treatment.[65] This means that it is ideal for a biomarker based test to have a high degree of sensitivity and/or specificity.

When a medical test is determined to be highly sensitive, then that means in a population positive for a disease, it will correctly confirm that a large number are in fact positive: for example, if a test is 95% sensitive for skin cancer, then 95% of a group believed to have skin cancer will have the diagnosis confirmed.[66]

A test with a high degree of sensitivity can be an excellent tool for eliminating potential diagnoses: if the test had a sensitivity of 98% and is negative, then it is extremely unlikely (a 2% chance) that the result was actually a false negative.[67]

Specificity of a test is the opposite in that it is determined by the proportion of positive results that occur in a population known to be negative for

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[65]Undén, et al, Scandinavian Guidelines for Initial Management of Minimal, Mild and Moderate Head Injuries in Adults: An Evidence and Consensus-Based Update, 11 BMC Medicine (2013) at 50.

[66] Remington, et al, Statistics with Applications to the Biological and Health Sciences (New Jersey: Prentice Hall, 2000) at 58-59.

[67] Remington, et al, Statistics with Applications to the Biological and Health Sciences (New Jersey: Prentice Hall, 2000) at 58-59.

the condition: if 3% of the negative samples test positive, then the test has a specificity of 97% and correctly rules out a condition that percent of the time.[68]

A high degree of specificity can be quite useful in confirming that a patient has a disease: if a highly specific tests rules a condition as positive, it is almost certain that the patient does indeed have the disease: using the above example, there would only be a 3% chance that it was a false positive.[69]

A key element to keep in mind is that a solid estimate of the prevalence disease, in this case TBI, in a population needs to be known to help quantify the effectiveness of a test, and that if that there is a small population being analyzed, then the positive results are more vulnerable to random chance such as false positive occurring.[70]

However, if these tests were used in addition to the current methods of diagnosing TBI, then the risks are mitigated and would only enhance the efficacy of the diagnostic process and each test of examination used can help cover for the deficits of the others. In terms of costs for testing, one biomarker in

[68]Remington, et al, Statistics with Applications to the Biological and Health Sciences (New Jersey: Prentice Hall, 2000) at 58-59.
[69]Remington, et al, Statistics with Applications to the Biological and Health Sciences (New Jersey: Prentice Hall, 2000) at 58-59.
[70]Remington, et al, Statistics with Applications to the Biological and Health Sciences (New Jersey: Prentice Hall, 2000) at 58-59.

particular, the S-100B protein, should be approximately $20.00 USD when accounting for Medicare's coverage of similar immunoassays.[71]

The last element to account for any additional time the patient has to hold a spot waiting for those results, which is holding a bed that could be used for another patient;[72] in the case of S-100B, an automated test can be completed in 18 minutes.[73]

[71]Ruan, et al, The Economic Impact of S-100B as a Pre-Head CT Screening Test on Emergency Department Management of Adult Patients with Mild Traumatic Brain Injury, 26(10) Journal of Neurotrauma (2009) at 1655-1664.
[72]Ruan, et al, The Economic Impact of S-100B as a Pre-Head CT Screening Test on Emergency Department Management of Adult Patients with Mild Traumatic Brain Injury, 26(10) Journal of Neurotrauma (2009) at 1655-1664.
[73]Ruan, et al, The Economic Impact of S-100B as a Pre-Head CT Screening Test on Emergency Department Management of Adult Patients with Mild Traumatic Brain Injury, 26(10) Journal of Neurotrauma (2009) at 1655-1664.

Specific Biomarkers

1. S100B

One of the biomarkers most extensively researched today for TBI research is the protein S100B. It is found primarily in glial cells, where it is expressed by two types: most prominently in astrocytes that line the blood vessels, and by oligodendrocytes expressing NG2. [74]

Due to the former, it is considered a biofluid biomarker of astrocyte injury.[75] Its primary purpose in the body is to assist in the regulation of intracellular calcium levels[76], and S100B levels in serum elevate after cells expressing the protein in the body suffer damage.[77]

Its main flaw as a biomarker for TBI is that orthopedic injuries such as bone fractures can also inflict the type of trauma necessary to allow its release,[78] limiting its potential sensitivity in cases where a patient has suffered multiple traumas. It also has a short half-life and is cleared rapidly by the body, limiting

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[74] Wang, et al, The Astrocyte Odyssey, 86(4) Progressive Neurobiology (2009) at 342-367.
[75] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[76] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[77] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[78] Nimer, et al, Comparative Assessment of the Prognostic Value of Biomarkers in Traumatic Brain Injury Reveals an Independent Role of Serum Levels of Neurofilament Light, PLOS ONE (July 2015).

the window of opportunity that it can be utilized on certain patients as a marker.[79]

However, it is still a biomarker candidate that is excellent negative predictive value: normal levels of S100B serve as extremely strong evidence that a given patient has not sustained a TBI because it reportedly demonstrates serum elevations arguably only in response to trauma.[80]

Furthermore, while it is not the best biomarker to use in order to independently diagnose a TBI, it has a great amount of potential to predict favorable vs unfavorable outcomes in those who have been confirmed to have a TBI.[81]

> ♦**PRACTICE NOTE:** Note that like diffusion tensor imaging, because this biomarker because reportedly is highly sensitive to the presence of traumatic brain injury, this biomarker usefully could be used in conjunction with diffusion tensor imaging by plaintiff experts to help advise plaintiffs whether or not a particular case is worth pursuing, simply because the combination of a negative DTI with a normal level of S100B pointing to the lack of traumatic brain injury.

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[79] Undén, et al, Scandinavian Guidelines for Initial Management of Minimal, Mild and Moderate Head Injuries in Adults: An Evidence and Consensus-Based Update, 11 BMC Medicine (2013) at 50.

[80] See Calcagnile, et al, Clinical Validation of S100B Use in Management of Mild Head Injury, 12(10) BMC Emergency Medicine (2012) at 13.

[81] DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology at 100.

Due to these features, S100B has been evaluated in several European countries, and has been implemented as part of the TBI protocol in Scandinavia, with further investigation being done in North America. In Scandinavia, S100B is used to evaluate the need for CT scans and to hold a patient in a certain subset of patients. [82]

It was recommended that adults with GCS scores of 14 with no other risk factors or a score of 15 with a loss of consciousness and/or repeated vomiting with no other risk factors, who have had head trauma occur less than 6 hours prior to admission be tested for S100B: if they have levels of the protein less than 0.10 µg/l and their other injuries have been treated, it is strongly recommended that they are discharged without a CT scan.[83]

It was found that patients that meet these criteria are extremely unlikely to experience intracranial complications or need for neurosurgical interventions,[84] therefore saving the time that would be spent on a CT scan and helping to give these patients peace of mind. These are similar findings and policies to those published by the American College of Emergency Physicians for mild TBI in

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[82] Undén, et al, Scandinavian Guidelines for Initial Management of Minimal, Mild and Moderate Head Injuries in Adults: An Evidence and Consensus-Based Update, 11 BMC Medicine (2013) at 50.

[83] Undén, et al, Scandinavian Guidelines for Initial Management of Minimal, Mild and Moderate Head Injuries in Adults: An Evidence and Consensus-Based Update, 11 BMC Medicine (2013) at 50.

[84] Undén, et al, Scandinavian Guidelines for Initial Management of Minimal, Mild and Moderate Head Injuries in Adults: An Evidence and Consensus-Based Update, 11 BMC Medicine (2013) at 50.

2008.[85] This can also be economically feasible for a hospital perform in lieu of a CT scan when there are a high proportion of mild TBI patients (≥78%) or the CT scans require longer than an hour and a half more than the blood test for results.[86]

Other studies implicate the value of S100B in predicting patient outcome after TBI.

For example, a 2015 study measured S100B levels among others from a group of 85 adult TBI patients (60 severe, 25 moderate), taking samples at admission, 6, 12 and 24 hours post injury, and then evaluating patient outcome at discharge, 28 days after injury and 6 months after injury. [87]

The patients who experienced unfavorable outcomes had plasma levels of S100B approximately four times higher than those who had favorable outcomes[88]. Despite S100B's short half-life, the level of S100B stayed elevated at roughly double the serum concentration of those with favorable outcome at the three measured time points over 24 hours after injury.[89]

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[85] Jagoda, et al, Clinical Policy: Neuroimaging and Decision Making in Adult Mild Traumatic Brain Injury in the Acute Setting, 52 American College of Emergency Physicians; Centers for Disease Control and Prevention, Annals of Emergency Medicine (2008) at 714–748.
[86] Ruan, et al. The Economic Impact of S-100B as a Pre-Head CT Screening Test on Emergency Department Management of Adult Patients with Mild Traumatic Brain Injury, 26(10) Journal of Neurotrauma (2009) at 1655-1664.
[87]DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology (2015) at 100.
[88]DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology (2015) at 100.
[89]DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology (2015) at 100.

In patients who died, S100B levels were close to fivefold compared to those who lived, with levels of S100B dropping significantly after 6 hours only to escalate again dramatically compared to patients who lived at 24 hours[90]. It was theorized that the heightened S100B levels could have produced an immune response in patients who died, with the ensuing inflammation leading to eventually fatal complications. [91]

At a minimum, it appears that repeated hits to the head that do not lead to concussions can elevate levels of S100B, and with it cause the production of anti-S100B autoantibodies[92]: the immune system produces antibodies to attack a protein that it itself produces. These autoantibodies are associated with lowered performances on cognitive tests and react very strongly against glial and neuronal cells[93], posing a risk long term for the health of the nervous system.

Overall, S100B has exhibited much potential as a biomarker, with its abilities to rule out cases of TBI, serve as an economically and time efficient method of determining the need for CT scans in some patients, and its prognostic value in predicting a patient's outcome from TBI.

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[90]DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology (2015) at 100.
[91]DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology (2015) at 100.
[92] Hazeldine, et al, Traumatic Brain Injury and Peripheral Immune Suppression: Primer and Prospectus, 6(11) Frontiers in Neurology (2015) at 1-17.
[93]Hazeldine, et al, Traumatic Brain Injury and Peripheral Immune Suppression: Primer and Prospectus, 6(11) Frontiers in Neurology (2015) at 1-17.

2. Brain-Derived Neurotrophic Factor (BDNF)

Brain-Derived Neurotrophic Factor (BDNF) is a neurotrophic essential to the growth, development, and survival of neurons. [94] In particular, it helps neurons to better interact and communicate with each other. [95] Due to its very specific use in the body, it is found within the nervous system and has a unique diagnostic and prognostic value compared to some other biomarkers of TBI.

Research conducted by Dr. Frederick Korley and his team at the Johns Hopkins School of Medicine explored the potential of BDNF as a biomarker. Their study initial recruited 151 patients with TBI and 150 control patients, finding that patients who has sustained a TBI had a lower serum level of BDNF (17.5 ng/mL and 13.8ng/mL at the two test sites) compared to controls (60.3 ng/mL).

In their pilot study of 159 patients, they found that the lower an individual's levels of serum BDNF, the more severe their TBI: mild TBI patients averaged 8.3 ng/mL of BDNF in their serum, compared to the 4.3 ng/mL average found in moderate patients and the 4.0 ng/mL average in severe patients. [96]

In addition, the patients with the lowest serum levels of BDNF had the lower odds of making a complete recovery 6 months after their injury compared

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[94] Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.
[95] Doidge, The Brain That Changes Itself (New York: Penguin Books, 2007) at 80.
[96] Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.

to those with higher values[97]. These results were believed to be because BDNF has the ability to promote neuronal recovery as well as protection from injury, and that lower levels of BDNF may prevent new neuronal connections from being forged when then the brain is in such a damaged state[98].

[97] Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.
[98]Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.

3. Glial Fibrillary Acid Protein (GFAP)

Glial Fibrillary Acid Protein (GFAP) is another widely studied biomarker. Unlike S100B, GFAP is a key monomeric intermediate protein found primarily in the cytoskeletons of astrocytes within the brain's gray and white matter that assists with the maintenance of the blood-brain barrier and intercellular communication[99]. It is normally not found in circulation at all and only passes through the blood-brain barrier to be released into serum when astrocytes in the brain sustain structural damage or die.[100,101]

Within one hour of an individual sustaining a concussion, it reaches detectable levels in serum, and has been shown as able to differentiate between a TBI and an orthopedic injury.[102]

Furthermore, the levels of GFAP as well as its breakdown products (GFAP-BDP) can be used in testing to distinguish between different levels of TBI, as well as having the potential to predict which subjects who have intracranial

[99] Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.
[100] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[101] Lei, et al, Glial Fibrillary Acidic Protein as a Biomarker in Severe Traumatic Brain Injury Patients: a Prospective Cohort Study, 19(10) Critical Care (2015) at 1-12.
[102]Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).

injuries[103] thanks to its extremely high specificity for TBI induced brain hemorrhages and lesions.[104]

A 2015 study with 215 patients (83% with mild TBI, 4% moderate and 12% severe, 51% of patients with intracranial lesions) found that GFAP-BDP could reduce the scans given to patients between 12% and 30%[105]: similarly to S100B, it may help reduce the number of unnecessary CT scans performed. The body also produces autoantibodies against GFAP and GFAP-BDP within 4 days after injury which are capable of causing damage to glial cells *in vitro,*[106] which provides more options related to GFAP that do not have as strict a time limit for testing as S100B.

Furthermore, GFAP has some prognostic value: elevated serum levels of GFAP at hospital admission and the following days was linked to negative outcomes, including death, 6 months afterwards. [107,108,109]

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[103] McMahon, et al, Measurement of the Glial Fibrillary Acidic Protein and Its Breakdown Products GFAP-BDP Biomarkers for the Detection of Traumatic Brain Injury Compared to Computed Tomography and Magnetic Resonance Imaging, 32(4) Journal of Neurotrauma (2015) at 527-55.
[104] Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.
[105] McMahon, et al, Measurement of the Glial Fibrillary Acidic Protein and Its Breakdown Products GFAP-BDP Biomarkers for the Detection of Traumatic Brain Injury Compared to Computed Tomography and Magnetic Resonance Imaging, 32(4) Journal of Neurotrauma (2015) at 527-558.
[106] Hazeldine, et al, Traumatic Brain Injury and Peripheral Immune Suppression: Primer and Prospectus, 6(11) Frontiers in Neurology (2015) at 1-17.
[107] Lei, et al, Glial Fibrillary Acidic Protein as a Biomarker in Severe Traumatic Brain Injury Patients: a Prospective Cohort Study, 19(10) Critical Care (2015) at 1-12.
[108]Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.
[109]DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology (2015) at 100.

4. Tau

Tau is an intracellular protein closely involved in the functioning of microtubules: it helps to create microtubule packages and then supporting anterograde axonal transport, explaining why it is found in very high concentrations within the axons of neurons.[110]

Due to this, when it or its cleaved form c-tau are found in serum, it strongly implies that neurons in the body have sustained axonal damage.[111] Tau is frequently studied as a measure of TBI suffered due to blasts[112], such as battlefield explosions, although it is also elevated in professional boxers, concussed athletes and in cases of severe TBI.[113]

Another 2015 study conducted on a total of 98 participants (70 military personnel with self-reported TBI and 28 controls) demonstrated that tau was elevated to levels approaching double that of the controls in those who

[110]Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[111]Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[112] Olivera, et al, Peripheral Total Tau in Military Personnel Who Sustain Traumatic Brain Injuries During Employment, 72(10) JAMA Neurology (August 2015) at 1109-1116.
[113] Olivera, et al, Peripheral Total Tau in Military Personnel Who Sustain Traumatic Brain Injuries During Employment, 72(10) JAMA Neurology (August 2015) at 1109-1116.

believed they had suffered a TBI compared to controls (1.13 pg/mL on average compared to 0.63 pg/mL).[114]

Furthermore, those who reported that they had suffered 3 or more TBIs had an even greater disparity in tau serum concentrations compared to those who reported less than 3 (1.52 pg/mL compared to 0.82 pg/mL)[115], indicating the importance of repeated damage on levels of the biomarker and prognostic outlook. It should be noted that the serum levels of tau were still extremely low, requiring very sensitive assays to attempt to detect the protein.[116]

Tau can potentially stay elevated in serum for 18 months or longer in a patient, although this appears to be a dose-dependent effect[117].

Notably, tau is implicated in the degenerative brain disease Chronic Traumatic Encephalopathy (CTE) becoming notorious for its appearance in ex-NFL players post-mortem (in September 2015, 87 out of 91 deceased NFL players studied were determined to be positive for the disease[118]): after repeated blows

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[114]Olivera, et al, Peripheral Total Tau in Military Personnel Who Sustain Traumatic Brain Injuries During Employment, 72(10) JAMA Neurology (August 2015) at 1109-1116.
[115]Olivera, et al, Peripheral Total Tau in Military Personnel Who Sustain Traumatic Brain Injuries During Employment, 72(10) JAMA Neurology (August 2015) at 1109-1116.
[116] Peskind, et al, Biofluid Biomarkers of Mild Traumatic Brain Injury, 72(10) JAMA Neurology (October 2015) at 1103-1105.
[117] Peskind, et al, Biofluid Biomarkers of Mild Traumatic Brain Injury, 72(10) JAMA Neurology (October 2015) at 1103-1105.
[118] Breslow, PBS Frontline, News: 87 Deceased NFL Players Test Positive for Brain Disease, accessed from http://www.pbs.org/wgbh/frontline/article/new-87-deceased-nfl-players-test-positive-for-brain-disease (September 2015).

to the head, the damaged tau protein leaves axons and clogs the blood vessels in the brain, slowly starving nerve cells and atrophying the brain itself.[119]

One of the major weaknesses with tau and c-tau as a prognostic biomarker is that there is no correlation between serum levels of the two and clinical outcome despite its elevation in severe TBI[120], and that even in something directly related to it with such a dramatic effect on the brain such as CTE can only be diagnosed with absolute certainty after death.[121]

[119] Wexler, PBS Frontline, How CTE Affects the Brain, accessed from http://www.pbs.org/wgbh/frontline/article/the-four-stages-of-cte (October 2013).
[120]Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[121] Breslow, J. M. PBS Frontline. New: 87 Deceased NFL Players Test Positive for Brain Disease. Accessed from http://www.pbs.org/wgbh/frontline/article/new-87-deceased-nfl-players-test-positive-for-brain-disease/ Published September 2015

5. Enzyme Biomarkers: Neuron-Specific Enolase (NSE)

Neuron-Specific Enolase (NSE) is an isozyme, a naturally occurring variant of the enzyme Enolase that helps in the breakdown of glucose, and is found primarily in neurons, erythrocytes and neuroendocrine cells.[122] With a half-life of 24 hours,[123] there is a larger theoretical window to test for NSE than S100B.

Extensive study has been performed on this enzyme, with it being shown as an efficient biomarker for diagnosing and creating a prognosis for small cell lung cancer[124,125] alongside its potential applications for TBI.[126,127] Its use in cancer diagnosis is similar to its use as biomarker in TBI: to help diagnose the type of cancer in such a way that a patient minimizes their exposure to radiation based screening.[128]

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[122]Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[123] Scolletta, et al. Biomarkers as Predictors of Outcome After Cardiac Arrest, 5(6) Expert Rev Clin Pharmacol (2012) at 687-699.
[124] Ahn, et al, Current Serum Lung Cancer Biomarkers, J Mol Biomark Diagn S4:001 (February 2013).
[125] Li, et al, Biomarkers in the Lung Cancer Diagnosis: A Clinical Perspective, 59(5) Neoplasma at 500-507.
[126] Papa, et al, Exploring Serum Biomarkers for Mild Traumatic Brain Injury, Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Florida: CRC Press/Taylor & Francis) 2015.
[127]DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology (2015) at 100.
[128] Ahn, et al, Current Serum Lung Cancer Biomarkers, J Mol Biomark Diagn S4:001 (February 2013).

The Tianjin Medical University Cancer Institute and Hospital in China has the fee for using NSE as a marker in cancer testing at ¥100,[129] which is approximately $15.33 USD. Like with the S100B protein, this is typically done via an immunoassay; in this case, a biopsy sample is stained using antibodies or immunoglobulins that react to the presence of NSE. Due to its essential role in glycolysis within neurons, it is not typically found in serum or cerebral spinal fluid, and reaches its highest levels of extracellular concentration when neurons are damaged[130] or within tumors of neuroendocrine nature.[131]

Careful handling has to be done when testing for NSE levels in serum because the lysis of erythrocytes in a sample can unintentionally increase the concentration of the enzyme, leading to potential false-positives.[132] However, it is also detectable in cerebral spinal fluid (CSF) after injury,[133] meaning there are multiple options available to minimize the risk. NSE has shown prognostic value in severe TBI, with serum concentrations being higher in patients who went on to have negative outcomes, including death, 6 months after their hospital

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[129] Wang, et al, Clinical Evaluation and Cost-Effectiveness Analysis of Serum Tumor Markers in Lung Cancer, BioMed Research International, Article ID 195692 (August 2013).
[130] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[131] Li, et al, Biomarkers in the Lung Cancer Diagnosis: A Clinical Perspective, 59(5) Neoplasma (June 2012) at 500-507.
[132] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[133] Zetterberg, et al, Biomarkers of Mild Traumatic Brain Injury in Cerebrospinal Fluid and Blood, 99(2) Nat. Rev. Neurol. (2013) at 201-210.

admission and samples being taken compared to those with positive outcomes.[134]

[134]DiBattista, et al, Blood Biomarkers in Moderate-to-Severe Traumatic Brain Injury: Potential Utility of a Multi-Marker Approach in Characterizing Outcome, 6(5) Frontiers in Neurology (2015) at 100.

6. Ubiquitin C-Terminal Hydrolase (UCH-L1)

Ubiquitin C-Terminal Hydrolase (UCH-L1) is an enzyme that assists in preparing proteins for metabolism by either adding or removing ubiquitin from its substrates[135]. It is nearly exclusively found in neurons, proven to be elevated for at least one week after injury, and was capable of distinguish between patients who sustained orthopedic injuries and those who suffered TBI.[136]

It potentially can be found in both serum[137] as well as CSF within an hour after a patient has sustained a TBI,[138] leading to multiple sources for the biomarker from a given patient for testing, as well as a relatively large window of time to perform a test. One study with 262 subjects (95 severe TBI patients and 167 normal controls) found that CSF levels of UCH-L1 were more highly elevated than that in serum 6 hours of injury, though both sources were highly

[135] Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.
[136] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).
[137] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015); Welch, et al, Ability of Serum Glial Fibrillary Acidic Protein, Ubiquitin C-Terminal Hydrolase-L1 and S100B to Differentiate Normal and Abnormal Head Computed Tomography Findings in Patients with Suspected Mild or Moderate Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 203-214.
[138] Papa, et al, Chapter 22, Exploring Serum Biomarkers for Mild Traumatic Brain Injury. In Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects (Gainsville, Florida: CRC Press/Taylor & Francis, 2015).

detectable[139]. Furthermore, the levels in CSF stayed elevated compared to that in serum slowly declining.[140]

Finally, the levels of UCH-L1 could distinguish between survivors and non-survivors when measured 6 hours after injury, as well as showed a correlation with severity of TBI.[141] Studies have also shown that it has can differentiate between positive and negative CT scans[142], and that it can distinguish between those who have made a complete recovery from TBI and those who still have a lingering injury.[143]

One of, if not the most recent "cutting edge" article – and intended publicity connected with it – is in the writer's views, unlike some of the biomarkers here, there was a specific explanation both of the "time course and diagnostic accuracy" of biomarkers, glial and neuronal blood, glial fibrillary acidic protein (GFAP) and ubiquitin terminal hydrolase L1 (UCH-L1).[144]

Note that this article is widely read and published in the 3/28/16 edition of the Washington Post as well as cited on the on-line cite of CBS News.

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[139] Mondelo, et al, Clinical Utility of Serum Levels of Ubiquitin C-Terminal Hydrolase as a Biomarker For Severe Traumatic Brain Injury, 70(3) Neurosurgery (March 2012) at 666-675.
[140] Mondelo, et al, Clinical Utility of Serum Levels of Ubiquitin C-Terminal Hydrolase as a Biomarker For Severe Traumatic Brain Injury, 70(3) Neurosurgery (March 2012) at 666-675.
[141] Mondelo, et al, Clinical Utility of Serum Levels of Ubiquitin C-Terminal Hydrolase as a Biomarker For Severe Traumatic Brain Injury, 70(3) Neurosurgery (March 2012) at 666-675.
[142] Welch, et al, Ability of Serum Glial Fibrillary Acidic Protein, Ubiquitin C-Terminal Hydrolase-L1 and S100B to Differentiate Normal and Abnormal Head Computed Tomography Findings in Patients with Suspected Mild or Moderate Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 203-214.
[143]Korley, et al, Circulating Brain-Derived Neurotrophic Factor Has Diagnostic and Prognostic Value in Traumatic Brain Injury, 33(1) Journal of Neurotrauma (2016) at 215-225.
[144] Online version of JAMA Neurology, 3/28/16 at E1, E10.

As noted in the CBS statement, it was claimed that the researchers at Orlando Regional Medical Center had a "simple blood test" that can detect whether or not someone has suffered a concussion as long as a week after the initial injury. This test may even help regarding treatment in individuals.

Furthermore, this study was cited as having "showed" that the other biomarkers studied, GFAP even "exceeded that of UCH-LI, helping distinguish patients with mild traumatic brain injury from the study's control group of patients within seven days after injury," as well as that "patients who exhibited traumatic brain injury symptoms on CT scans had higher levels of the two biomarkers too, especially GFAP.

"Once more, the authors said GFAP appears to 'predict neurosurgical intervention consistently after seven days of injury, whereas the ability of UCH-L1 seem to be more limited to the earliest time points after injury."[145]

The authors also stated that "a biomarker blood test could be very useful in people who don't seek medical treatment immediately but show up in the emergency room or the doctor's office days later with possible concussion symptoms, including dizziness, headache, memory problems, fatigue, and

[145] Papa, Time Course and Diagnostic Accuracy of Glial and Neuronal Blood Biomarkers GFAP and UCH-L1in a Large Cohort of Trauma Patients With and Without Mild Traumatic Brain Injury, online publication of JAMA Neurology (March 28, 2016) at E1-E10.

feeling dazed" since "about 20% of people who have concussions don't come in right away" according to Dr. Papa.[146]

Since summaries in the lay press often translate into at least subliminal juror knowledge, the authors note some of the data appearing in the article itself as opposed to the lay translation and dissemination of what the article reportedly revealed.

Note specifically the following:

1. As seen in other articles already cited, there does – finally! – appear to be emerging attempts to look at multiple biomarkers, as well as their appearance and disappearance over time that in the writer's view will indeed *ultimately* lead to a composite fingerprint for brain injury that will be much closer in accuracy, including specificity and sensitivity, to the ways that cardiac biomarkers are now being used.
2. Particularly noteworthy was the objective – but unfortunately not the clear unambiguous – finding in the article of "understanding" the "diagnostic accuracy" of these biomarkers but instead that will establish with reasonable medical probability at least issues related to other possible diagnoses being excluded and causation statements being clinically valid as well as legally relevant.

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[146] Papa, Time Course and Diagnostic Accuracy of Glial and Neuronal Blood Biomarkers GFAP and UCH-L1in a Large Cohort of Trauma Patients With and Without Mild Traumatic Brain Injury, online publication of JAMA Neurology (March 28, 2016) at E1-E10.

3. One of the strengths of this study was that it was prospective but – again the article clearly states that even in the abstract that these studies (only) show "promise" for "clinical usefulness"[147] proof with reasonable medical probability of anything....

4. Note also that when using GFAP, clearly lauded in the article and in the press as in many ways a "better" biomarker than the previously discussed (in this section) UCH-L1, sensitivity of the test in detecting mild to moderate traumatic brain injury was not 100% but ranged from 0.73 to 0.94, whereas UCH-L1 demonstrated diagnostic range as little as 0.30 and as high as 0.67.

These biomarkers were more accurate in detecting actual intracranial lesions, with GFAP having a diagnostic range from 0.80 to 0.97 and UCH-L1 a range as low as 0.31 to a high of 0.77.

The "CONCLUSIONS AND RELEVANCE" section of the article indicated that although "GFAP performed consistently in detecting MMTBI" (apparently mild and moderate brain injury were lumped together under this heading – further limiting the use of this test in the classic litigation cases of mild traumatic brain injury, the writer's note) did "perform consistently" in detecting this as well as "CT

[147]Papa, Time Course and Diagnostic Accuracy of Glial and Neuronal Blood Biomarkers GFAP and UCH-L1in a Large Cohort of Trauma Patients With and Without Mild Traumatic Brain Injury, online publication of JAMA Neurology (March 28, 2016) at E1-E10.

lesions and neurosurgical intervention across seven days," there again was no claim of uniqueness either in diagnosis of "causal" connections. UCH-L1 was deemed to perform "best in the early post injury period" only. [148]

5. Finally, the authors stated that "*at the present time and most importantly there is insufficient knowledge as to when these biomarkers should be used for the clinical evaluation of the trauma patient with suspected mild TBI*" (emphasis added)[149], let alone, the writer's note, any truly believed defensible justification for these biomarkers to be used in the *legal* "evaluation of the trauma patient with suspected mild TBI"....[150]

Note once again that the publicity of these study results appeared to over-exaggerate *current* ability to identify true sensitive and specific biomarkers for mild traumatic brain injury, let alone justify their being acknowledged as anything other than research techniques that hold great clinical promise and which can at most be used as part of a total package of complete clinical data

[148]Papa, Time Course and Diagnostic Accuracy of Glial and Neuronal Blood Biomarkers GFAP and UCH-L1in a Large Cohort of Trauma Patients With and Without Mild Traumatic Brain Injury, online publication of JAMA Neurology (March 28, 2016) at E1-E10.
[149] Papa, Time Course and Diagnostic Accuracy of Glial and Neuronal Blood Biomarkers GFAP and UCH-L1in a Large Cohort of Trauma Patients With and Without Mild Traumatic Brain Injury, online publication of JAMA Neurology (March 28, 2016) at E1-E10.
[150] Papa, Time Course and Diagnostic Accuracy of Glial and Neuronal Blood Biomarkers GFAP and UCH-L1in a Large Cohort of Trauma Patients With and Without Mild Traumatic Brain Injury, online publication of JAMA Neurology (March 28, 2016) at E1-E10.

and history as data *consistent with* other clinical findings and laboratory data by any expert (either plaintiff or defense) or attorney (either plaintiff or defense) attempting to use these biomarkers at the current "state of the art" to argue for these tests justifying as "stand alone" measures either the presence or absence of mild traumatic brain injury let alone with reasonable medical probability, let alone certainty, the "cause" of the findings....

Understanding Traumatic Brain Injury

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Neuropsychology:

- **Typically defined as being the study of brain-behavior relationships.**
- **Utilizes objective testing to quantify performance on cog.-neuro. measures.**
  - **Performance is evaluated relative to applicable data in normative samples**

Objective NP Tests

A Comprehensive Neuropsychological will typically take approximately 5-8hrs.

- **Behavioral Measures**
- **Malingering/Motivation**
- **Sensorium**
- **Attention/Concentration**
- **Motor functions**
- **Language functions**
- **Memory**
  - **STM, LTM**
  - **Verbal, Visual**
- **Visuospatial processing**
- **Intellectual functions**

Diagnostic Criteria for mTBI:

- **Positive Loss of Consciousness (LOC)**
- **If no LOC- the patient must have an alteration of Mental Status (MS).**
- **Will later discuss:**
  - **Retrograde Amnesia**
  - **Anterograde Amnesia**
  - **Post-traumatic Amnesia (PTA)**
  - **HI secondary to whiplash with neg. LOC & neg. altered MS- QUESTIONABLE**

Features of concussion frequently observed

- **Vacant stare** (befuddled facial expression).
- **Delayed verbal and motor responses** (slow to answer questions or follow instructions).
- **Confusion and inability to focus attention** (easily distracted and unable to follow through with normal activities).
- **Disorientation** (walking in the wrong direction, unaware of time, date. and place).
- **Slurred or incoherent speech** (making disjointed or incomprehensible statements).
- **Gross observable incoordination** (stumbling, inability to walk tandem/straight line).
- **Emotions out of proportion to circumstances** (distraught, crying for no apparent reason).
- **Memory deficits** (exhibited by the patient repeatedly asking the same question that has already been answered, etc.)
- **Any period of loss of consciousness** (paralytic coma, unresponsiveness to arousal)

American Academy of Neurology Standards

The Quality Standards Subcommittee of the American Academy of Neurology, in June 1996, adopted practice parameters for the management of concussions (Published in Neurology 1997; 48:581-585).

Grades of Cerebral Concussion

- Grade 1
  - Transient confusion
  - No LOC
  - Concussion symptoms or mental status abnormalities on examination resolve in less than 15 minutes.

- Grade 2
  - Transient confusion
  - No LOC
  - Concussion symptoms or mental status abnormalities on examination last *more* than 15 minutes
- Grade 3
  - Any LOC, either brief (seconds) or prolonged (minutes).

Retrograde Amnesia (RA)

- **The inability to recall events immediately preceding the injury.**
- **Usually measured in seconds, however, in more severe cases may be hours, months and sometimes years.**
- **RA is predictable and is not selective.**
- **Very important with Children.**

Anterograde Amnesia

- **The period of time wherein there is no recall for events subsequent to the injury.**
- **May last seconds, hours, months, etc.**
- **When patchy recall evolves PTA comes into effect.**

Post-traumatic Amnesia (PTA)

- **The patient's inability to appreciate his/her moment to moment psychological. environment in a consistent and continuous fashion.**
- **Duration of PTA is the gold standard for evaluating the severity of neurotrauma and its post-acute neurobehavioral sequealae.**
- **State of being groggy/dazed/confused**

Classification of Head Injury

- **Mild**
  - **(90% of all TBI's, i.e., Cerebral Concussions).**
  - **PTA less than 24 hours.**
- **Moderate**
  - **PTA 24 hours to 1 week**
- **Severe**
  - **PTA greater than 1 week**

Course of Neurobehavioral Recovery

- **Most recovery will take place within the first 12 months**
- **Significant recover continues between 12-24 months**
- **Spontaneous recovery terminates at about 3 years.**
- **Deficits should be most severe at a time most proximal to the injury.**
- **Serial (f/u) examination in real HI cases will demonstrate improvement vs. deterioration (n.b., atypical).**
- **Deterioration may result from some secondary underlying condition such as Chronic SDH, seizures, etc.**

Important Sources of Information for TBI Cases

- **Medical records relating to the HI.**
- **Records relating to past and present treatment.**

- **Previous neuropsychologicals.**
- **Premorbid records.**
- **Clinical Interview material.**
- **All objective NP test evidence.**

Records Proximal to the Head Injury

- **Police report.**
- **EMT/Paramedic report (? LOC or disorientation).**
- **Emergency Room Record.**
- **Nursing notes.**
- **GCS.**
- **Progress notes.**
- **Consultant reports (neurology, NP, speech).**
- **Social Work notes.**
- **Discharge directives.**

Premorbid Records

- **Academic Transcripts.**
- **CST Evaluations.**
- **Achievement Testing (SAT's, CAT's)**
- **Job performance.**
- **Family Practice Records.**
- **Pediatric/well-baby records.**
- **Testings from any previous injuries.**
- **Prior P.I., W.C., or Divorce proceedings.**

Clinical Interview Material

- **Ptx's account of the accident in detail.**

- **Acute complaints**
- **PMHx.**
- **Social/family Hx**
- **Educational Hx.**
- **Employment Hx.**
- **Military Hx.**
- **Hx of arrests.**
- **Current complaints.**

2016 WL 462960
Only the Westlaw citation is currently available.
United States District Court,
D. Colorado.

Miriam White, Plaintiff,
v.
Deere & Company, John Deere Limited, and John Does 1-5, Defendants.

Civil Action No. 13-cv-02173-PAB-NYW
|
Signed February 8, 2016

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ORDER

PHILIP A. BRIMMER, United States District Judge

***1** This matter is before the Court on defendants' Motion to Exclude Randall Benson's Opinions Derived from Neuroimaging [Docket No. 103].

I. BACKGROUND

This is a products liability action that arises out of an accident that occurred on August 17, 2011 while plaintiff Miriam White was operating her Deere Model 4600 compact utility tractor and Model 460 loader. Ms. White claims that she suffered facial injuries and traumatic brain injury ("TBI") as a result of a hay bale falling onto her head while she was operating the tractor. Docket No. 103 at 1. Ms. White alleges that her tractor had design defects that created an unreasonable risk of injury from falling hay bales and that her injuries resulted from these defects. Docket No. 150 at 2-3.

Ms. White has designated Randall Benson, a board-certified neurologist, as a medical expert. Docket No. 103 at 1. Dr. Benson opines that Ms. White suffered a traumatic brain injury as a result of the August 17, 2011 incident. Docket No. 116-3 at 18. He bases his opinion, in part, on results derived from a Magnetic Resonance Imaging ("MRI") sequence called diffusion tensor imaging ("DTI"). *Id* at 20-21. Defendants move to exclude Dr. Benson's DTI findings on two grounds. First, defendants argue that Dr. Benson's DTI findings are unreliable. Docket No. 103 at 3. Second, defendants argue that Dr. Benson's DTI findings will not assist the trier of fact to determine whether Ms. White's alleged brain injuries were caused by the August 17, 2011 accident. *Id.* at 4.

II. FEDERAL RULE OF EVIDENCE 702

Rule 702 of the Federal Rules of Evidence provides that:

> A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if: (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue; (b) the testimony is based on sufficient facts or data; (c) the testimony is the product of reliable principles and methods; and (d) the expert has reliably applied the principles and methods to the facts of the case.

Fed. R. Evid. 702. As the rule makes clear, while required, it is not sufficient that an expert be qualified based upon knowledge, skill, experience, training, or education to give opinions in a particular subject area. Rather, the Court must "perform[ ] a two-step analysis." *103 Investors I, L.P. v. Square D Co.*, 470 F.3d 985, 990 (10th Cir. 2006). After determining whether the expert is qualified, the specific proffered opinions must be assessed for reliability. *See id.*; Fed. R. Evid. 702 (requiring that the testimony be "based on sufficient facts or data," be the "product of reliable principles and methods," and reflect a reliable application of "the principles and methods to the facts of the case").

Rule 702 imposes on the district court a "gatekeeper function to 'ensure that any and all scientific testimony or evidence admitted is not only relevant, but reliable.' " *United States v. Gabaldon*, 389 F.3d 1090, 1098 (10th Cir. 2004) (quoting *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 589 (1993)). To perform that function, the Court must "assess the reasoning and methodology underlying the expert's opinion, and determine whether it is both scientifically valid and applicable to a particular set of facts." *Dodge v. Cotter Corp.*, 328 F.3d 1212, 1221 (10th Cir. 2003) (citing *Daubert*, 509 U.S. at 592-93). Where an expert relies on experience, the expert " 'must explain how that experience leads to the conclusion reached, why that experience is a sufficient basis for the opinion, and how that experience is reliably applied to the facts.' " *United States v. Medina-Copete*, 757 F.3d 1092, 1104 (10th Cir. 2014) (quoting Fed. R. Evid. 702, advisory committee notes).

***2** Although it is not always a straightforward exercise to disaggregate an expert's method and conclusion, when the conclusion simply does not follow from the data, a district court is free to determine that an impermissible analytical gap exists between premises and conclusion. *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997). In examining an expert's method, however, the inquiry should not be aimed at the "exhaustive search for cosmic understanding but for the particularized resolution of legal disputes." *Daubert*, 509 U.S. at 597. It is the specific relationship between an expert's method, the proffered conclusions, and the particular factual circumstances of the dispute that renders testimony both reliable and relevant.

In addition to the expert having appropriate qualifications and methods, the proponent of the expert's opinions must demonstrate that the process by which the expert derived his or her opinions is reliable. *United States v. Crabbe*, 556 F. Supp. 2d 1217, 1220 (D. Colo. 2008). When assessing reliability, "the court may consider several nondispositive factors: (1) whether the proffered theory can and has been tested; (2) whether the theory has been subject to peer review; (3) the known or potential rate of error; and (4) the general acceptance of a methodology in the relevant scientific community." *103 Investors I*, 470 F.3d at 990 (citing *Daubert*, 509 U.S. at 593-94). These considerations are not exhaustive. Rather, "the trial judge must have considerable leeway in deciding in a particular case how to go about determining whether particular expert testimony is reliable." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 152 (1999). Ultimately, the test requires that the expert "employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Id.*

While plaintiff, as the proponent of the challenged testimony, has the burden of establishing admissibility, the proffer is tested against the standard of reliability, not correctness; she need only prove that "the witness has sufficient expertise to choose and apply a methodology, that the methodology applied was reliable, that sufficient facts and data as required by the methodology were used and that the methodology was otherwise reliably applied." *Crabbe*, 556 F. Supp. 2d at 1221.

Once the standard of reliability "is met, the court will still consider other non-exclusive factors to determine whether the testimony will assist the trier of fact: (1) whether the testimony is relevant; (2) whether it is within the juror's common knowledge and experience; and (3) whether it will usurp the juror's role of evaluating a witness'[ ] credibility." *United States v. Rodriguez-Felix*, 450 F.3d 1117, 1123 (10th Cir. 2006).

In sum, assuming an objection is properly made, expert testimony must be excluded if the expert is unqualified to render an opinion of the type proffered, if the opinion is unreliable, if the opinion will not assist the trier of fact, or if the opinion is irrelevant to a material issue in the case.

III. ANALYSIS

Defendants do not challenge Dr. Benson's qualifications, the application of MRI techniques other than DTI,[1] or the four sources of data other than DTI on which Dr. Benson bases his conclusions. defendants' challenge focuses squarely on Dr. Benson's use of DTI and his opinions based on DTI. The Court's Practice Standards regarding Rule 702 objections require that the party seeking to exclude an opinion of an opposing expert identify the opinion sought to be excluded. *See* Practice Standards (Civil Cases), Judge Philip A. Brimmer, § III.G. The only specific opinion that defendants identify in their motion is Dr. Benson's fifth piece of evidence regarding brain imaging, including DTI. Docket No. 103 at 2. The Court therefore assumes that the opinion defendants seek to exclude is that finding in Dr. Benson's report that states as follows: "DTI voxel-wise analysis revealed a large number of white matter tracts with abnormally reduced FA." Docket No. 116-3 at 20. Dr. Benson also refers to these findings later in his report in support of his conclusion that the DTI "reveals axonal injury predominantly in bilateral frontal lobes." *Id.* at 21-22.

A. Reliability of DTI for Identifying a TBI

***3** Defendants argue Dr. Benson should be precluded

from presenting his opinions based on DTI because DTI is unreliable as a means for diagnosing individual patient injuries. Docket No. 103 at 3. Defendants cite a November 2014 research paper by Wintermark et al. that finds DTI to be suitable only for research and concludes that there is insufficient evidence to support its routine clinical use at the individual patient level. Docket No. 103 at 3-4; Docket No. 103-1 at 76.

Plaintiff responds that the non-exclusive *Daubert* reliability factors establish that Dr. Benson's opinions based on DTI are admissible. Docket No. 116 at 11-14. While the Wintermark article may undermine the weight of Dr. Benson's DTI findings, plaintiff cites articles that support DTI's reliability. *See, e.g.*, Docket No. 116-1 at 7, ¶ 10; Docket No. 116-6. The articles cited by plaintiff appear to support the conclusion that DTI is a generally accepted diagnostic measure for TBI. One peer-reviewed article cited by plaintiff reviews the last decade of research conducted on DTI and finds that "[a] unifying theme can be deduced from this large body of research: DTI is an extremely useful and robust tool for the detection of TBI-related brain abnormalities. The overwhelming consensus of these studies is that low white matter FA [fractional anisotrophy] is characteristic of TBI." M.B. Hulkower et al., *A Decade of DTI in Traumatic Brain Injury: 10 Years and 100 Articles Later*, 34 AM J NEURORADIOL 2064, 2071 (2013). This article also found "an overwhelming consensus that imaging abnormalities detected with DTI are associated with important clinical outcomes. This further validates DTI as a meaningful measure of clinically important brain injury." *Id*. Another peer-reviewed article cited by plaintiff states that the "overwhelming consensus of a substantial body of scientific inquiry supports DTI for detecting pathology in [mild TBI ("mTBI")] patients," Docket No. 116-6 at 4, and directly challenges the criticisms of DTI proffered by defendants' expert, Dr. Hal Wortzel. *Id*. at 2 ("The misleading and often entirely unsubstantiated opinions and positions of Wortzel, Tsiouris, and Filippi (2014), in opposition to diffusion tensor imaging (DTI) as a useful measure in mTBI, are at odds with the clear consensus of the scientific literature regarding [mTBI], its clinical assessment, and its natural history."). The Court notes that the November 2014 research paper cited by defendants acknowledges that "there is evidence from group analyses that DTI can identify TBI-associated changes in the brain across a range of injury severity, from mild to severe TBI. Evidence also suggests that DTI has the sensitivity necessary to detect acute and chronic TBI-associated changes in the brain, some of which correlate with injury outcomes." Docket No. 103-1 at 78. Thus, the Court finds that defendants have not shown that the November 2014 research paper, or other evidence, establishes that DTI is an unreliable technology to detect mild TBI-associated changes in the brain.

In his affidavit, Dr. Benson discusses some of the testing that he has conducted "to demonstrate the clinical validity and reliability of DTI in TBI" as part of his work with the U.S. Army Telemedicine and Advanced Technology Research Command at a "Diffusion MRI TBI Roadmap Development Workshop." Docket No. 116-1 at 11-12, ¶ 18. As part of his research for his presentation at that workshop, Dr. Benson found "excellent correlation between DTI and injury severity" and "repeatability of DTI for a single mTBI case scanned in two different cities." *Id*. Dr. Benson also notes that "[o]ther speakers presented data showing the correlations of DTI with neurocognitive outcome and experience using DTI on Iraq war veterans." *Id*. Dr. Benson states the known rate of error for DTI analysis is .4%, Docket No. 116-1 at 14, ¶ 28; however, he provides no support for this rate.

***4** Application of the four non-dispositive *103 Investors* factors supports plaintiff's argument that DTI is a reliable methodology. *See 103 Investors I*, 470 F.3d at 990 (citing *Daubert*, 509 U.S. at 593-94). Regarding whether DTI can be and has been tested, Dr. Benson's affidavit discusses the testing he has conducted to confirm DTI results. Docket No. 116-1 at 11-12, ¶ 18. The publications and workshops cited by Dr. Benson support the conclusion that DTI has been subjected to peer review and is generally accepted in the medical community as a tool for detecting TBI. *Id.* at 10-12, ¶¶ 16, 18. While plaintiff has not supported her argument that DTI has a known error rate, no single *103 Investors* factor is dispositive. *See 103 Investors I*, 470 F.3d at 990 (citing *Daubert*, 509 U.S. at 593-94). The Court notes that DTI findings have been admitted by multiple courts. *Andrew v. Patterson Motor Freight, Inc.*, 2014 WL 5449732, at *8 (W.D. La. Oct. 23, 2014) ("In sum, the evidence submitted shows DTI has been tested and has a low error rate; DTI has been subject to peer review and publication; and DTI is a generally accepted method for detecting TBI.") (citation omitted); *Ruppel v. Kucanin*, 2011 WL 2470621, at *6 (N.D. Ind. June 20, 2011) (finding DTI to be a reliable method); *Booth v. KIT, Inc.*, 2009 WL 4544743, at *3 (D.N.M. Mar. 23, 2009) (denying motion to exclude expert testimony regarding findings from DTI). Accordingly, the Court finds that plaintiff has carried its burden of showing that DTI is a reliable technology and that Dr. Benson applied a reliable methodology in arriving at his challenged opinion.

B. "Fit" of Dr. Benson's DTI Findings

Defendants argue that Dr. Benson's opinions derived from DTI do not "fit" this case. Docket No. 103 at 4; *see Bitler v. A.O. Smith Corp.*, 400 F.3d 1227, 1234 (10th Cir. 2004) ("A trial court must look at the logical relationship between the evidence proffered and the material issue that the evidence is supposed to support to determine if it advances the purpose of aiding the trier of fact. Even if an expert's proffered evidence is scientifically valid and follows appropriately reliable methodologies, it might not have sufficient bearing on the issue at hand to warrant a determination that it has relevant 'fit.' ") (citing *Daubert*, 509 U.S. at 591). Defendants assert that Dr. Benson's DTI findings show that plaintiff has only one or two white matter lesions and that Dr. Benson has not adequately addressed other possible causes for such findings in light of Ms. White's medical history, specifically, her injuries after being kneed in the head by a horse. Docket No. 103 at 5-6. On June 10, 2012, Ms. White was hit on the left side of her face by a horse's knee. Docket No. 81-3 at 6. After emergency medical services arrived and evaluated Ms. White, they determined that she should be transferred to the Medical Center of the Rockies. *Id*. There, Chris Cribari, M.D., noted that Ms. White was admitted with a diagnosis of a concussion and that the EMTs said she was repeating herself, had retrograde amnesia, and was slow to respond. *Id*. Defendants claim that these are signs of brain trauma that Dr. Benson ignores. Docket No. 103 at 5. Defendants also argue that Dr. Benson does not "adequately consider or explain why the white matter lesions are so definitively attributable to the 2011 incident and not to [p]laintiff's psychiatric issues." *Id.* at 6. The Court notes that both the June 10, 2012 incident and plaintiff's psychiatric history are mentioned in Dr. Benson's report. *See* Docket No. 81-3 at 6, 8. Defendants also argue that "a fact-finder needs to determine ...whether [p]laintiff's alleged brain injury was caused by the 2011 incident at issue in this case" and claim that Dr. Benson's DTI findings are not relevant to the issue of causation. Docket No. 103 at 5.

In support of his conclusion that "[i]t is probable that [Ms. White's] permanent cognitive, emotional, and physical symptoms...are the direct result of the 8/17/11 injury and not the subsequent injury of 6/10/12," Dr. Benson relied on five sources of data: (1) the available biomechanical information regarding the August 17, 2011 injury event; (2) Ms. White's symptoms following the August 17, 2011 injury event; (3) findings from a neurobehavioral examination; (4) findings from a neuropsychological assessment; and (5) Ms. White's neuroimaging. Docket No. 81-3 at 18-20. Thus, DTI is not the only source of information Dr. Benson uses to diagnose TBI. The neuroimaging he relies upon consists of FLAIR, SWI, and Gradient Echo imaging in addition to DTI. *Id*. at 20. Dr. Benson pairs the neuroimaging results with the neuropsychological assessment, which notes impaired processing speed and working memory and delayed verbal memory, coding, and symbol search, to determine the presence of brain damage. *Id.* at 21. The reasons Dr. Benson articulates for identifying the August 17, 2011 incident as the source of plaintiff's traumatic brain injury are not based on DTI, and Dr. Benson readily admits that "[n]o standalone imaging technique allows for unequivocal determination of etiology absent clinical information." Docket No. 116-1 at 6. Dr. Benson compares the imaging findings to the other data sources and states that the "imaging findings match the biomechanics, chronic symptoms, neurobehavioral and neuropsychological findings." Docket No. 116-1 at 9. Applying the differential diagnosis procedure, Dr. Benson asserts that Ms. White's "injury/accident of 8/17/11 was the much more significant injury and rendered her vulnerable to the more mild[ ] concussion of 6/10/12." Docket No. 116-4 at 6. He also states that the "injury of 6/10/12, while inducing a mild concussion, does not explain her clinical deficits that began when her head was crushed under the weight of a heavy hay bale on 8/7/11." *Id*.

***5** The Court finds that defendants present no basis to exclude Dr. Benson's causation opinions on the grounds of the alleged unreliability or irrelevance of DTI for identifying a TBI suffered by Ms. White.

IV. CONCLUSION

For the foregoing reasons it is

ORDERED that defendants' Motion to Exclude Randall Benson's Opinions Derived from Neuroimaging [Docket No. 103] is **DENIED**.

All Citations

Slip Copy, 2016 WL 462960

Footnotes

1 In their reply, defendants appear to broaden their argument to include Dr. Benson's conclusions drawn from Susceptibility Weighted Imaging (SWI) and Fluid Attenuated Inversion Recovery (FLAIR) imaging. Docket No. 130 at 3.

Defendants admit that SWI and FLAIR are "methodologically sound." *Id*. A party generally may not raise an issue for the first time in a reply brief. *See* *Ulibarri v. City & Cty. of Denver*, No. 07-cv-01814-WDM-MJW, 2011 WL 1336388, at *2 (D. Colo. April 6, 2011) (citing *Hill v. Kemp*, 478 F.3d 1236, 1250 (10th Cir. 2007)); *LNV Corporation v. Hook,* No. 14-cv-00955-RM-CBS, 2015 WL 5679723, at *3 (D. Colo. Sept. 25, 2015) (citing *Conroy v. Vilsack*, 707 F.3d 1163, 1179 n.6 (10th Cir. 2013)). Accordingly, the Court will not consider defendants' arguments related to SWI and FLAIR imaging.

2016 WL 3002403
United States District Court,
E.D. New York.

Imran Ali, Plaintiff,
v.
Police Officer William Connick and Sergeant Donald Kipp Defendants.

11-CV-5297 (NGG) (VMS)
|
Signed 05/23/2016

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MEMORANDUM & ORDER

NICHOLAS G. GARAUFIS, United States District Judge

***1** Plaintiff Imran Ali brings this action against two employees of the New York City Police Department ("NYPD"): Police Officer William Connick ("Connick") and Sergeant Donald Kipp ("Kipp"). (Am. Compl. (Dkt. 7).) At this stage of the litigation, the only claims remaining are: (1) use of excessive force in violation of 42 U.S.C. § 1983, against Kipp; (2) conspiracy in violation of 42 U.S.C. §§ 1983 and 1985, against Connick and Kipp; and (3) violation of equal protection in violation of 42 U.S.C. § 1983, against Kipp. (Joint Pre-Trial Order (Dkt. 55) at 2; see also Summ. J. Mem. & Order ("Summ. J. Decision") (Dkt. 50); Stip. of Partial Voluntary Dismissal & Discontinuance (Dkt. 34).) The parties filed a joint pre-trial order on December 22, 2015. (Joint Pre-Trial Order.) An amended joint pre-trial order was filed on May 19, 2016 (Am. Joint Pre-Trial Order ("JPTO") (Dkt. 72)), and entered by the court on May 20, 2016 (May 20, 2016, Order). Trial is scheduled to begin on May 31, 2016.

Before the court are the parties' respective pre-trial motions in limine. (See Pls.' Mot. in Lim. ("Pl.'s Mot.") (Dkt. 62); Defs.' Mot. in Lim. ("Defs.' Mot.") (Dkt. 63).) For the reasons stated below, the court holds that Plaintiff's motion is GRANTED in part and DENIED in part, and Defendants' motion is GRANTED in part, DENIED in part, and RESERVED in part.

I. BACKGROUND

The court assumes familiarity with the underlying facts and procedural history, but will briefly describe the aspects relevant to these motions. The court refers to its memorandum and order deciding Defendants' motion for partial summary judgment for additional factual background for further procedural detail. (See Summ. J. Decision at 1-4.)

Plaintiff was involved in a car accident on the morning of July 17, 2009. Plaintiff's car struck a parked vehicle at the intersection of 142nd Street and Lakewood Avenue in Queens, New York. (Id. at 1.) Defendant Connick arrived at the scene of the accident and placed Plaintiff under arrest. (Id. at 2.) There appears to be a factual dispute as to whether Plaintiff was intoxicated and whether he was driving his car when the accident occurred. (Compare Defs.' Mem. of Law in Opp'n to Pl.'s Mots, in Lim. ("Defs.' Opp'n") (Dkt. 64) at 2-4 (citing Plaintiff's deposition testimony), with Pl.'s Mot. at 4 (noting that "Ali was intoxicated [is] a fact conceded by Ali").)

Two non-party police officers subsequently took Plaintiff to the 103rd Precinct, while Connick remained at the scene. (Summ. J. Decision at 2.) At the police station, Plaintiff was brought to the desk of Defendant Kipp, and was asked certain pedigree information. (Id.) According to Plaintiff, Kipp "became belligerent ... after we exchanged words." (Id.) He was then "brought, dragged, pushed" into a holding cell, where Kipp drove Plaintiff's head into the wall three or four times. (Id.) Plaintiff also states that Kipp directed slurs at him during this time, including comments about Plaintiff's "race and stuff" and "about Muslim[s]." (Id. at 2-3.) Kipp allegedly slammed Plaintiff's head into the cell gate and wall until he passed out from his injuries. (Id. at 2.) According to Kipp, he was at his desk when he heard bangs coming from the cell where Plaintiff was held, and from the monitor at his desk that displayed the cell, he saw Plaintiff sitting on the ground. (See Mar. 7, 2013, Dep. of Donald Kipp (Decl. of James F. Desmond, Jr. (Dkt. 39), Ex. H (Dkt. 39-8)) at 85:9-25.) Emergency services were called and Plaintiff was transported to Queens Hospital Center. (Summ. J.

Decision at 3.)

II. LEGAL STANDARD

A. Motion in Limine

***2** "The purpose of a motion in limine is to allow the trial court to rule in advance of trial on the admissibility and relevance of certain forecasted evidence." Gorbea v. Verizon N.Y., Inc., No. 11-CV-3758 (KAM), 2014 WL 2916964, at *1 (E.D.N.Y. June 25, 2014) (citing Luce v. United States, 469 U.S. 38, 40 n.2 (1984); Palmieri v. Defaria, 88 F.3d 136, 141 (2d Cir. 1996); Nat'l Union Fire Ins. Co. of Pittsburgh v. L.E. Myers Co., 937 F. Supp. 276, 283 (S.D.N.Y. 1996)). "Evidence should be excluded on a motion in limine only when the evidence is clearly inadmissible on all potential grounds." United States v. Paredes, 176 F. Supp. 2d 179, 181 (S.D.N.Y. 2001). "[C]ourts considering a motion in limine may reserve decision until trial, so that the motion is placed in the appropriate factual context." Jean-Laurent v. Hennessy, 840 F. Supp. 2d 529, 536 (E.D.N.Y. 2011) (citing Nat'l Union Fire Ins. Co., 937 F. Supp. at 287). Further, a district court's ruling on a motion in limine is preliminary and "subject to change when the case unfolds." Luce, 469 U.S. at 41.

B. General Rules of Admissibility

Federal Rule of Evidence 402 provides that "[r]elevant evidence is admissible unless any of the following provides otherwise: the United States Constitution; a federal statute; these rules; or other rules prescribed by the Supreme Court. Irrelevant evidence is not admissible." Fed. R. Evid. 402. Thus, "unless an exception applies, all '[r]elevant evidence is admissible.' +" United States v. White, 692 F.3d 235, 246 (2d Cir. 2012) (quoting Fed. R. Evid. 402). Federal Rule of Evidence 401 provides that "[e]vidence is relevant if: (a) it has any tendency to make a fact more or less probable than it would be without the evidence; and (b) the fact is of consequence in determining the action." Fed. R. Evid. 401. The Second Circuit has characterized this relevance standard as "very low." See White, 692 F.3d at 246 (quoting United States v. Al-Moayad, 545 F.3d 139, 176 (2d Cir. 2008)). Indeed, "[t]o be relevant, evidence need not be sufficient by itself to prove a fact in issue, much less to prove it beyond a reasonable doubt." United States v. Abu-Jihaad, 630 F.3d 102, 132 (2d Cir. 2010).

Under Federal Rule of Evidence 403, "[t]he court may exclude relevant evidence if its probative value is substantially outweighed by a danger of one or more of the following: unfair prejudice, confusing the issues, misleading the jury, undue delay, wasting time, or needlessly presenting cumulative evidence." Fed. R. Evid. 403. "[W]hat counts as the Rule 403 'probative value' of an item of evidence, as distinct from its Rule 401 'relevance,' may be calculated by comparing evidentiary alternatives." Old Chief v. United States, 519 U.S. 172, 184 (1997). In short, Rule 403 requires "the district court [to] make a conscientious assessment of whether unfair prejudice substantially outweighs probative value" with regard to each piece of proffered evidence. Al-Moayad, 545 F.3d at 160 (quoting United States v. Salameh, 152 F.3d 88, 110 (2d Cir. 1998) (per curium)).

III. DISCUSSION

The court addresses in turn each of the parties' requests for admissions and exclusions.

A. Plaintiff's Motion

Plaintiff seeks to preclude Defendants from introducing nineteen exhibits that "document Ali's intoxication at the time of the motor vehicle accident and that Ali was arrested for and ultimately pled guilty to driving while intoxicated under New York's [Vehicle Traffic Law] 1192.1." (Pl.'s Mot. at 2.) The objected-to documents are:

- NYPD Highway District – IDTU Technician Tech Report ("JPTO Ex. A");

***3** • Arresting Officer's Report ("JPTO Ex. B");

- Intoxicated Driver Examination ("JPTO Ex. D");

- NYPD Intoxicated Driver Examination Instruction Sheet ("JPTO Ex. E");

- NY State DMV Report of Refusal to Submit to Chemical Test ("JPTO Ex. F");

- Chemical Test Analysis ("JPTO Ex. G");

- Property Clerk's Invoice ("JPTO Ex. H");

- NYPD Arrest Report ("JPTO Ex. I");

- NYPD Complaint Report ("JPTO Ex. J");

- Criminal Court Complaint: Queens Criminal Court ("JPTO Ex. K");

- Intoxylizer – Alcohol Analyzer Report ("JPTO Ex. L");

- Certificate of Disposition ("JPTO Ex. M");
- Decision and Order from Suppression Hearing ("JPTO Ex. N");
- DMV Order of Suspension of Revocation ("JPTO Ex. O");
- DMV Order of Suspension Pending Prosecution ("JPTO Ex. P");
- Pre-Sentence Conditions (Criminal Court City of New York) ("JPTO Ex. Q");
- Accident Report ("JPTO Ex. S");
- IAB DVD – Surveillance Video from "Underground Nightclub" ("JPTO Ex. U"); and
- Transcript of Plaintiff's Guilty Plea ("JPTO Ex. V").

(Id.) Plaintiff argues that documents relating to his intoxication would cause undue prejudice (id. at 3-5), and documents relating to the violation arising out of the underlying accident are irrelevant and unduly prejudicial (id. at 5). Plaintiff further asserts that certain of the documents are inadmissible hearsay. (Id.)

1. Intoxication

Plaintiff moves to preclude from trial documents evidencing his intoxication. (Id. at 4.) Plaintiff argues that because he concedes that he was intoxicated, submitting ten documents to prove that fact "would be cumulative and tend to confuse the jury" and would only serve to "disparage Ali in the eyes of the jury." (Id. at 4-5.) Defendants counter that Plaintiff had denied that he was intoxicated in numerous prior statements, including in his deposition testimony. (Defs.' Opp'n at 2-3.) Defendants contend that his intoxication—and the extent of his intoxication—are "highly probative of Plaintiff's memory and perception of the incident, as well as his credibility." (Id. at 2.)

To the extent that Plaintiff argues that his intoxication is irrelevant to this case (see Pl.'s Mot. at 5), the court disagrees. As Plaintiff himself notes in his motion in limine, Defendants may argue at trial that "Ali's in juries were sustained as a result of his intoxication." (Id. at 4.) Whether Plaintiff had been drinking on the night of the accident and the extent to which he had been drinking thus are plainly relevant. See Kokoska v. Carroll, No. 12-CV-1111 (WIG), 2015 WL 1004303, at *3 (D. Conn. Mar. 6, 2015) (finding Plaintiff's alleged intoxication probative in an excessive force case because "it may have influenced [the plaintiff's] actions and behavior").

Defendants are also correct that the level of Plaintiff's supposed drunkenness is probative of his memory and perception of the events on the night in question. Courts have long held that "[i]t is, of course, within the proper scope of cross-examination to determine whether a witness was under the influence of drugs or narcotics or alcohol at the time of observation of events in dispute ...." United States v. DiPaolo, 804 F.2d 225, 229 (2d Cir. 1986). Thus, evidence of intoxication may be used to impeach the witness's ability to observe and recall critical events. Nibbs v. Goulart, 822 F. Supp. 2d 339, 346 (S.D.N.Y. 2011); see also Kokoska, 2015 WL 1004303, at *3. Defendants are therefore permitted to raise the issue of Plaintiff's drinking on cross-examination to test Plaintiff's credibility.

***4** Having concluded that evidence of Plaintiff's drinking is relevant and probative, the court next addresses arguments that documents concerning Plaintiff's intoxication should nonetheless be excluded. As an initial matter, it is unclear whether Plaintiff's intoxication is in dispute. In his motion in limine, Plaintiff asserts that he concedes that he was intoxicated on the night of the accident. (Pl.'s Mot, at 1, 5.) However, Defendants correctly point out that Plaintiff has testified to the contrary on multiple occasions. (Defs.' Opp'n at 2-3.) The court also notes that as recently as during briefing on Defendants' partial motion for summary judgment, Plaintiff maintained that "he was not drunk." (Pl.'s Rule 56.1 Statement (Dkt. 41) at 2.) The court is not aware of any stipulation or concession since the summary judgment phase of this case that altered Plaintiff's position. In any event, even if the parties had stipulated to the fact of Plaintiff's intoxication, the stipulation would not be grounds to preclude the Defense from offering evidence demonstrating the same fact. Defendants are entitled to submit to the jury evidence that shows Plaintiff was intoxicated and place this fact in the appropriate context as part of a narrative to prove their case. As the Supreme Court noted, "[a] syllogism is not a story, and a naked proposition in a courtroom may be no match for the robust evidence that would be used to prove it." Old Chief, 519 U.S. at 189.

This is not to say, however, that Defendants have unfettered leave to present the jury with all the evidence they desire on the issue of intoxication. Defendants may not introduce evidence of Plaintiff s drunkenness if "unfair prejudice substantially outweighs probative

value." Salameh, 152 F.3d at 110. This is the case for JPTO Exs. F, G, L, and N: documents indicating that Plaintiff refused a blood alcohol test. Especially if intoxication is contested at trial, there is a danger that a jury would view the refusal as an admission or that Plaintiff had something to hide. Defendants have not argued that the refusal to submit to a blood alcohol test, in and of itself, is probative or relevant, and the information in these exhibits tending to show Plaintiff was intoxicated are found elsewhere. (Compare, e.g., JPTO Ex. F ("strong odor of alcohol, bloodshot eyes & [Ali] stated he was drunk"), with JPTO Ex. D ("unsteady on feet, smell of alcohol, bloodshot eyes & [Ali] stated he was drunk").) The probative value of these blood alcohol test documents—as "calculated by comparing evidentiary alternatives"— is thus low, if not nonexistent. Old Chief, 519 U.S. at 184. JPTO Exs. F, G, L, and N, and any other document or testimony reflecting Plaintiff's refusal to take a blood alcohol test are inadmissible.

The court will not exclude any other proposed exhibits at this stage. "Where possible, a party should be allowed 'to prove its case by evidence of its own choice.' +" Int'l Bus. Machines Corp. v. BGC Partners, Inc., No. 10-CV-128 (PAC), 2013 WL 1775437, at *8 (S.D.N.Y. Apr. 25, 2013) (quoting Old Chief, 519 U.S. at 186). Defendants propose at least seven exhibits (not including the blood alcohol test documents) on the issue of intoxication, and Plaintiff argues that they would be "cumulative and tend to confuse the jury." (Pl.'s Mot. at 5.) Plaintiff may well be right that if Defendants introduced all these exhibits at trial, in addition to eliciting testimony on intoxication from other witnesses such as Defendant Connick, the author of many of the objected-to exhibits, the evidence would become cumulative sooner rather than later. See Fed. R. Evid. 403; United States v. Jamil, 707 F.2d 638, 643 (2d Cir. 1983) ("Evidence is cumulative when it replicates other admitted evidence."). However, the court cannot predict when Defendants' evidence will become cumulative because the court does not know what evidence Defendants will present. Should Defendants cross the line at trial, Plaintiff should renew his challenge then.

Accordingly, the court finds that evidence of Plaintiff's intoxication is admissible, with the exception of JPTO Exs. F, G, L, and N, and any other documents or testimony reflecting Plaintiff's refusal to submit to a blood alcohol test.

2. Traffic Violation

Plaintiff also moves to exclude documents relating to his violation of New York's Vehicle Traffic Law 1192.1, for driving while intoxicated. (Pl.'s Mot. at 5-6.) This traffic violation arose out of the events underlying this action. (See id.) Plaintiff maintains that these documents are either irrelevant or more prejudicial than probative. (Id.) He additionally argues that evidence of his guilty plea to the violation is inadmissible under FRE 609(a). (Id.) Defendants respond that they do not seek to admit evidence of his conviction pursuant to FRE 609(a). (Defs.' Opp'n at 4.) Instead, Defendants argue that they are admissible to test Plaintiff's credibility because Plaintiff testified in the past that he was not the driver of the car when the accident occurred, but by pleading guilty to a charge of driving while intoxicated, he necessarily admitted he was the driver. (Id.)

***5** The court agrees that FRE 609 is inapplicable. Rule 609(a)(1) states that evidence of a conviction for a crime punishable for more than one year must be admitted, subject to FRE 403. Fed. R. Evid. 609(a)(1). Rule 609(a)(2) states that evidence of a conviction for which a dishonest act or false statement is an element of the crime must also be admitted. Fed. R. Evid. 609(a)(2). Because the traffic violation to which Plaintiff pleaded guilty was not punishable by more than one-year imprisonment, and because neither a dishonest act nor a false statement is an element of the violation, the Rule 609 is inapposite.

The court additionally finds that Plaintiff's guilty plea to a violation arising out of the accident at issue in this civil action is irrelevant to whether Defendants violated Plaintiff's constitutional rights after he was taken into custody. Defendants appear to agree, and instead argue that evidence of the violation is admissible to test Plaintiff's credibility. (Defs.' Opp'n at 4.) The court agrees that Plaintiff's seemingly contradictory positions on whether he was the driver of the car on the night in question is probative of his credibility in the eyes of the jury. However, to be admissible, evidence of the violation must still be more probative than prejudicial under Rule 403. Nibbs, 822 F. Supp. 2d at 344. The court recognizes that references to Plaintiff's guilty plea for driving while intoxicated may cast him in a negative light. "The plain fact of the matter is jurors don't tend to like people who drive drunk, and their view of [the guilty plea] might affect their ability to view fairly the evidence in this case." United States v. Landry, 631 F.3d 597, 604 (1st Cir. 2011) (citation and internal quotation marks omitted). But unlike Stephen v. Hanley, No. 03-CV-6226 (KAM) (LB), 2009 WL 1471180 (E.D.N.Y. May 21, 2009), a case upon which Plaintiff relies, Defendants do not seek to impeach Plaintiff's credibility using the simple fact of the guilty plea. Rather, Defendants seek to use documents

relating to the guilty plea to contradict statements Plaintiff made during the course of these proceedings. There was no such question of inconsistent statements in Stephen. The court therefore concludes that the resultant prejudice is outweighed by the probative value provided as to his credibility.

Accordingly, the court finds that evidence of Plaintiff's underlying violation is admissible for impeachment purposes.

3. Hearsay

Plaintiff also argues in a cursory manner that certain of the documents he objects to are inadmissible hearsay. (Pl.'s Mot. at 5.) In their opposition, Defendants assert that the seventeen documents Plaintiff seek to exclude as hearsay fall within the exceptions to the rule against hearsay because "they are NYPD business records made and kept in the ordinary course of NYPD business." (Defs.' Opp'n at 4-5 (citing Fed. R. Evid. 803(6)).) Defendants also argue that even if certain documents are hearsay, they are admissible for impeachment purposes. (Id. at 5.)

The one case Plaintiff cites in support of his argument is inapposite. (Id.) The plaintiff in Wilson v. Roberson, No. 92-CV-2709 (KMW), 1996 WL 63053 (S.D.N.Y. Feb. 14, 1996), moved in limine to exclude certain memo book entries and complaint reports on hearsay grounds. Id. at *5. The defendants opposed the exclusion only if the plaintiff testified at trial to a specific fact. Id. at *5. The court then granted the plaintiff's motion but noted that it would reconsider its ruling if the plaintiff did so testify. Id. The court, for all intents and purposes, granted the motion as unopposed. See id.

***6** Plaintiff has not addressed the documents individually to explain why each is objectionable and why exceptions to the hearsay rule do not apply. It is also unknown at this stage which documents Defendants intend to introduce and for what purpose—i.e., whether to prove a fact or for impeachment. The burden to establish that evidence is inadmissible for any purpose, and thus excludable on a motion in limine, is on the movant. United States v. Pugh, ___ F. Supp. 3d ____, No. 15-CR-116 (NGG), 2016 WL 627347, at *2 (E.D.N.Y. Feb. 12, 2016). Plaintiff has failed to carry this burden. Accordingly, the court denies Plaintiff's motion to exclude documents as inadmissible hearsay. Plaintiff may renew his application at trial.

B. Defendants' Motion

Defendants move in limine to preclude Plaintiff from offering:

- Testimony from any of Plaintiff's treating physicians;
- Evidence relating to a Diffusor Tensor Imaging exam Plaintiff undertook;
- Testimony from two named witness (Alex Rodriguez and Kavita Samaroo), and certain unnamed witnesses from Jamaica Medical Center and the NYPD Internal Affairs Bureau ("IAB");
- Evidence relating to Defendants' disciplinary history, lawsuits, and personnel files;
- Defendants' memo book entries, command logs, medical treatment of prisoner forms, IAB reports, and certain photographs;
- Evidence relating to the NYPD Patrol Guide;
- Any reference of counsel for Defendants as "City Attorneys" or suggesting that the City of New York may indemnify Defendants; and
- Any request for a specific amount of damages.

(See generally Defs.' Mot.) Plaintiff opposes only a portion of these requests. (Pl.'s Mem. of Law in Opp'n to Defs.' Mot. in Lim. ("Pl.'s Opp'n") (Dkt. 67) at 2-3.)

1. Uncontested Issues

The court first disposes of the portions of Defendants' motion in limine that are not in dispute. In his opposition, Plaintiff states that he does not oppose the exclusion of evidence of Defendants' disciplinary histories, lawsuits, and personnel files, testimony of unidentified healthcare providers from Jamaica Medical Center or members of the NYPD IAB, the NYPD Patrol Guide, references to Corporation Counsel as "City Attorneys," suggestions that the City may indemnify Defendants, or requests for a specific dollar amount of damages from the jury. (Id.) Accordingly, Defendants' motion as to these requests is granted.

2. Plaintiff's Treating Physicians

Defendants seek to preclude from trial the testimony of Plaintiff's five treating physicians: Dr. David L. Cohen, Dr. Igor Cohen, Dr. Gregory Lawler, Dr. Thugmann, and Dr. Ahid Elfiky. (Defs.' Mot. at 2-4.) Defendants argue that Plaintiff failed to provide the disclosures required by Federal Rule of Civil Procedure 26 for expert witnesses, and thus should be prevented from offering these individuals at trial. (Id.) Specific to Dr. Igor Cohen, Defendants also dispute whether he qualifies as Plaintiff's treating physician. (Id. at 3-4.) Plaintiff responds that "treating physicians are not expert witnesses," and therefore they are exempt from the disclosure requirements of Rule 26. (Pl.'s Opp'n at 3-4.) According to Plaintiff, the treating physicians will be testifying as fact witnesses in their capacities as Plaintiff's treating physicians. (Id.) The parties agree that Plaintiff has neither named any of the treating physicians as experts under Rule 26 nor satisfied the corresponding disclosure requirements. The issue before the court then, is whether the five treating physicians can nonetheless testify—and the scope of their testimony if admissible at all—as fact witnesses.

i. Treating Physicians as Fact Witnesses

***7** Courts in this circuit have held that treating physicians may testify as fact, rather than expert, witnesses. See, e.g., Puglisi v. Town of Hempstead Sanitary Dist. No. 2, No. 11-CV-0445 (PKC), 2013 WL 4046263, at *6 (E.D.N.Y. Aug. 8, 2013); Zanowic v. Ashcroft, No, 97-CV-5292 (JGK) (HBP), 2002 WL 373229, at *2 (S.D.N.Y. Mar. 8, 2002). Even without being declared an expert under Rule 26, treating physicians "may testify as to facts acquired and opinions formed during [their] personal consultation ...." Puglisi, 2013 WL 4046263, at *6 (emphasis in original). Opinions formed during consultation "are considered an explanation of treatment." Turner v. Delta Air Lines, Inc., No. 06-CV-1010, 2008 WL 222559 (NG) (CLP), at *1 (E.D.N.Y. Jan. 25, 2008). Specifically, a treating physician who testifies as a fact witness can opine on "causation, severity, disability, permanency and future impairments" as part of the doctor's explanation of treatment. Puglisi, 2013 WL 4046263, at *6 (quoting Williams v. Regus Mgmt. Grp., No. 10-CV-8987 (JMF), 2012 WL 1711378, at *3 (S.D.N.Y. May 11, 2012)).

However, the testimony of a treating physician who has not been declared an expert under Rule 26 or complied with its disclosure requirements is not without bounds. It is confined to "information he/she has acquired through observation of the Plaintiff in his/her role as a treating physician [and] limited to the facts in Plaintiff's course of treatment." Spencer v. Int'l Shoppes, Inc., No. 06-CV-2637 (AKT), 2011 WL 4383046, at *3-4 (E.D.N.Y. Sept. 20, 2011). It must not include testimony "with regard to another physician's records, opinion or recommendations ... because this information cannot be characterized as being within the personal knowledge of [the treating physician]." Motta v. First Unum Life Ins. Co., No. 09-CV-3674 (JS) (AKT), 2011 WL 4374544, at *4 (E.D.N.Y. Sept. 19, 2011); see also Spencer, 2011 WL 4383046, at *4 ("The treating physician may not introduce information provided by other physicians to whom the Plaintiff may have been referred nor may the doctor present any medical reports received from other physicians regarding the Plaintiff or opine on any information provided by another doctor."). Equally off limits is "information acquired during preparations for [+] testimony at trial." Puglisi, 2013 WL 4046263, at*6. "[T]he key to what a treating physician can testify to without being declared an expert is based on his[/her] personal knowledge from consultation, examination and treatment of the Plaintiff, 'not from information acquired from outside sources.' +" Motta, 2011 WL 4374544, at *3 (quoting Mangla v. Univ. of Rochester, 168 F.R.D. 137, 139 (W.D.N.Y. 1996)) (emphasis in original).

Defendants nonetheless argue that treating physicians must be declared as experts and satisfy the disclosure requirements of either Rule 26(a)(2)(B) or (C) in order to testify at trial. (Defs. Mot. at 2-4.) As an initial matter, it is clear that treating physicians are not subject to the expert report requirement of Rule 26(a)(2)(B). Rule 26(a)(2)(B) provides that the disclosure of an expert "must be accompanied by a written report—prepared and signed by the witness—if the witness is one retained or specially employed to provide expert testimony in the case or whose duties as the party's employee regularly involve giving expert testimony." Fed. R. Civ. P. 26(a)(2)(B). The Advisory Committee's Notes make explicit, however, that "[a] treating physician ... can be deposed or called to testify at trial without any requirement for a written report." Fed. R. Civ. P. 26(a)(2) advisory committee's note to 1993 amendment; see also Spencer, 2011 WL 4383046, at *2; Zanowic, 2002 WL 373229, at *2 ("It is well settled that a treating physician is not subject to the disclosure obligations set forth in Fed. R. Civ. P. 26(a)(2)(B)."). It is therefore unnecessary for treating physicians to provide Rule 26(a)(2)(B) expert reports in order to testify, even as expert witnesses.

***8** A closer question is whether the 2010 addition of the new disclosure requirement of Rule 26(a)(2)(C) affected how and when treating physicians may be called at trial. Rule 26(a)(2)(C) states that, "if the [expert] witness is not required to provide a written report, [the Rule 26

disclosure] must state (i) the subject matter on which the witness is expected to present evidence under Federal Rule of Evidence 702, 703, or 705; and (ii) a summary of the facts and opinions to which the witness is expected to testify." Fed. R. Civ. P. 26(a)(2)(C). Prior to the 2010 amendment, no written disclosures were required for treating physicians. The Advisory Committee's Notes explain that, "[t]his amendment resolves a tension that has sometimes prompted courts to require reports under Rule 26(a)(2)(B) even from witnesses exempted from the report requirement." Fed. R. Civ. P. 26(a)(2)(C) advisory committee's note to 2010 amendment. It goes on to list physicians as a common example of witnesses that do not need to provide an expert report under subdivision (B), but may still testify as a fact witness or provide expert testimony. Id.

Some courts appear to conclude that Rule 26 now requires all treating physicians to provide a Rule 26(a)(2)(C) summary disclosure before they can testify. See, e.g., Ziegenfus v. John Veriha Trucking, No. 10-CV-5946 (RJS), 2012 WL 1075841, at *7 (S.D.N.Y. Mar. 28, 2012) (finding a treating physician must "at a minimum, provide Defendants with a [Rule 26(a)(2)(C)] summary of his opinions about Plaintiff's medical condition"); Barack v. Am. Honda Motor Co., 293 F.R.D. 106, 108 (D. Conn. 2013) (same). Others maintain that "[n]o expert report [pursuant to Rule 26(a)(2)(B)] or summary of testimony [pursuant to Rule 26(a)(2)(C)] is required for [a treating physician] to testify as a fact witness." Puglisi, 2013 WL 4046263, at *6 (emphasis in original); see also Spencer, 2011 WL 4383046, at*2-3; Motta, 2011 WL 4374544, at *4.

As noted previously, testimony of treating physicians as to facts acquired and opinions formed during consultation are considered factual and not expert testimony, and thus fall without the reach of Rule 26. Nothing in the amended Rule or the accompany notes of the Advisory Committee suggests an enlargement of the scope Rule 26 to now encompass facts acquired and opinions formed during treatment. Instead, it was amended to "resolve[+] a tension that has sometimes prompted courts to require reports under Rule 26(a)(2)(B) even from witnesses exempted from the report requirement." Fed. R. Civ. P. 26(a)(2)(C) advisory committee's note to 2010 amendment. While the note is silent as to the precise tension, it is likely a reference to the fact that prior to the 2010 amendment, courts either limited a treating physician's testimony to facts acquired or opinions formed during treatment, see Geary v. Fancy, No. 12-CV-796W(F), 2016 WL 1252768, at *2 (W.D.N.Y. Mar. 31, 2016), or required a treating physician to provide a full Rule 26(a)(2)(B) expert report if the testimony included any information gained outside of consultation, such as another doctor's records or recommendations, see Lewis v. Triborough Bridge, No. 97-CV-607 (PKL), 2001 WL 21256, at * 1 (S.D.N.Y. Jan. 9, 2001). In other words, courts either limited a treating physician's testimony to facts based on personal knowledge acquired during treatment, or required a full expert report if testimony exceeded that limited scope, even if the treating physician was not "retained or specially employed to provide expert testimony in the case" within the meaning of Rule 26(a)(2)(B). It is understandable why, in the latter situation, courts have required a full report "even from witnesses exempted from the report requirement." Fed. R. Civ. P. 26(a)(2)(C) advisory committee's note to 2010 amendment. Courts operated with a limited toolbox. It was either a full expert report, or none at all. Where a treating physician's testimony includes information acquired from sources other than personal knowledge, the opposing party would have no notice of the potential testimony absent a written report, "thus creating the possibility of unfair surprise and delay." Geary, 2016 WL 1252768, at *2. Such dangers are obviated where testimony is limited to facts acquired and opinions formed during consultation, because, typically, the opposing party would have received as part of discovery the medical records pertaining to the doctor's treatment, thus providing sufficient notice. Id. The introduction of Rule 26(a)(2)(C) summary of opinions represent a middle ground to the all-or-nothing approach of Rule 26 disclosures previously facing parties and courts.

***9** The court thus concludes that treating physician testimony can be of three different types: (1) testimony limited to facts acquired and opinions formed during consultation; (2) testimony that also includes reliance on outside sources, such as another doctor's records or opinions or facts acquired as part of litigation; and (3) testimony where circumstances suggest the doctor was "retained or specially employed to provide expert testimony." Fed. R. Civ. P. 26(a)(2)(B). The first category is outside the purview of Rule 26 and is considered factual testimony. The second falls under the domain of Rule 26(a)(2)(C). The third is governed by Rule 26(a)(2)(B). Here, the parties agree that the five treating physicians are not experts, so these potential witnesses must only provide testimony falling within the first category.

ii. Dr. Igor Cohen

As to one of the treating physicians, Dr. Igor Cohen ("Dr. Cohen"), Defendants argue that he was not Plaintiff's

treating physician at all. (Defs. Mot. at 3-4.) If Dr. Cohen was not Plaintiff's treating physician, then his testimony must be excluded as a result of Plaintiff's failure to properly disclose him under Rule 26. While the law on who qualifies as a "treating physician" is not well defined, the critical inquiry centers around why the physician was retained: whether to treat the Plaintiff or to provide expert testimony at trial. See Evans v. United States, 978 F. Supp. 2d 148, 153 (E.D.N.Y. 2013) (citing Zanowic, 2002 WL 373229, at *2). Courts consider factors such as why the plaintiff saw the doctor, the reason for the referral to the doctor if referred, how often the doctor was consulted, whether medication was prescribed, the time spent treating the patient compared to providing testimony, and whether there is a continuing relationship between doctor and patient. Zanowic, 2002 WL 373229, at *2-3. Here, the only reasons Defendants put forth in support of their argument that Dr. Cohen was not Plaintiff's treating physician are that Dr. Cohen treated Plaintiff twice in the seven years since the incident, and that the first consultation occurred four years after the incident. (Defs.' Mot. at 3.) It is unclear from the parties' submissions the substance of Dr. Cohen's consultation, whether Dr. Cohen was referred by another of Plaintiff's physician, if medication was prescribed, and why the consultations started years after the incident and ended after two sessions. These uncertainties make it impossible for the court to determine on the papers whether Dr. Cohen was Plaintiff's treating physician. The court therefore reserves ruling until trial.

3. Diffusor Tensor Imaging Exam

Defendants next seek to preclude Plaintiff from offering any reports or opinions resulting from a Diffusor Tensor Imaging ("DTI") exam undertaken by Plaintiff in 2013. (Defs.' Mot. at 4-5.) Defendants argue that without a properly disclosed expert witness, no one can testify to "knowledge of DTI testing and its relationship to traumatic brain injury." (Id.) Alternatively, Defendants argue that Plaintiff has not offered any evidence tending to show the reliability of DTI under Daubert v. Merrell Dow Pharms., 509 U.S. 579 (1993). (Id. at 5.) Plaintiff contends that he had previously disclosed that one of his treating physicians, Dr. Gregory Lawler ("Dr. Lawler"), would testify to the results of the DTI exam that he performed. (Pl.'s Opp'n at 6-7.) Plaintiff also argues that "DTI testing has been recognized as authoritative and reliable in many District Courts around the country." (Id. at 7-8.)

Under the scope of the non-expert treating physician testimony previously delineated, treating physicians may testify "as to facts acquired and opinions formed during [+] personal consultation ...." Puglisi, 2013 WL 4046263, at *6 (emphasis in original). It follows that, theoretically, Dr. Lawler would be permitted to testify as to the fact that he conducted a DTI test on Plaintiff, and the conclusions that he drew and opinions that he formed based on the DTI exam results at the time of consultation. However, as a non-expert witness, his testimony cannot rely upon "scientific, technical, or other specialized knowledge" gained outside of his personal consultation with Plaintiff. In re World Trade Ctr. Lower Manhattan Disaster Site Litig., No. 21-MC-102, 2014 WL 5757713, at *3-4 (S.D.N.Y. Nov. 5, 2014). Dr. Lawler thus cannot testify as to the general theory of the science and medicine behind DTI exams and its relationship with traumatic brain injury. To allow a non-expert to provide such expert testimony would subvert the disclosure requirements of Rule 26.

***10** The question then, is whether allowing Dr. Lawler to testify to the DTI exam results without any expert explanation would confuse the jury or cause undue prejudice to Defendants under FRE 403. Cf. United States v. Mejia, 545 F.3d 179, 189 (2d Cir. 2008) ("[E]xpert testimony is called for when the 'untrained layman' would be unable intelligently to determine 'the particular issue' in the absence of guidance from an expert." (quoting Fed. R. Evid, 702 advisory committee's note to 1972 proposed rule)). DTI testing has only gained medical acceptance in recent years. See Ruppel v. Kucanin, No. 3:08-CV-591, 2011 WL 2470621, at*7 (N.D. Ind. June 20, 2011) ("DTI is a relatively new technology [that] is gaining general acceptance as a method for detecting [traumatic brain injury]."). Its reliability is challenged by Defendants (Defs.' Opp'n at 5), and Plaintiff does not have any expert witness who can establish its reliability. Furthermore, as a medical doctor, the jury may already view any testimony from Dr. Lawler with an aura of authority. See In re Agent Orange Prod. Liab. Litig., 611 F. Supp. 1267, 1283 (E.D.N.Y. 1985). If Dr. Lawler is permitted to testify to the results of the DTI exam, the jury may be confused as to the connection between the DTI results and potential traumatic brain injury or the weight they should afford such testimony, or they may give the testimony undue weight because of Dr. Lawler's status as a physician. On balance, the court finds that the dangers of juror confusion and undue prejudice outweigh any probative value of testimony regarding the DTI exam.

In his opposition, Plaintiff argues that courts generally permit treating physicians to testily as to radiology reports, even those of other physicians. (Pl.'s Opp'n at 7.)

The cases he cites are inapposite, however, because the treating physicians in those cases were all properly designated as expert witnesses. See, e.g., Shamanskaya v. Ma, No. 07-CV-1974 (RRM), 2009 WL 2230709, at *6 (E.D.N.Y. July 24, 2009); Williams v. Elzy, No. 00-CV-5382 (HBP), 2003 WL 22208349, at *5 (S.D.N.Y. Sept. 23, 2003). Dr. Lawler is not an expert. Plaintiff made the strategic decision not to designate any expert in this case. This issue with the DTI exam could easily have been resolved if Plaintiff had designated Dr. Lawler as an expert treating physician under Rule 26. Dr. Lawler would only have been subject to the "considerably less extensive" summary of opinions disclosure requirement of Rule 26(a)(2)(C), rather than a full expert report under Rule 26(a)(2)(B). Fed. R. Civ. P. 26(a)(2)(C) advisory committee's note to 2010 amendment. Plaintiff did not do so, and must now live with the consequences.

To be clear, Dr. Lawler may still testify to the fact that he examined Plaintiff, performed tests, including but not limited to an MRI exam, which Defendants have not contested, and that based on his examination, he diagnosed Plaintiff (presumably) with traumatic brain injury. He may not, however, testify to the results of the DTI exam. Accordingly, Defendants' motion to exclude any evidence of the DTI exam is granted.

4. Alex Rodriguez

Defendants also move to preclude Plaintiff from calling witness Alex Rodriguez ("Rodriguez") at trial. (Defs.' Mot. at 5.) Defendants argue that because Plaintiff failed to provide any contact information for Rodriguez, he has failed to comply with Rule 26(a) and the testimony should be excluded. (Id. at 5-6.) Defendants also assert that they would be substantially prejudiced if Rodriguez were found at the eleventh hour on the eve of trial, a witness Defendants have never had a chance to depose. (Id.) Plaintiff maintains that he does not intend to call Rodriguez, and his failure to provide the requisite contact information is the result of Plaintiff's own inability to locate Rodriguez. (Pl.'s Opp'n at 9.) At this time, Plaintiff only seeks to reserve the right to call Rodriguez in the event that he can be located. (Id.)

Rule 26(a)(1) and (3) require that parties disclose the identity of, and contact information for, potential witnesses. Failure to comply with its dictates can result in the exclusion of the testimony as a sanction under Rule 37(c). See Virgin Enters. Ltd. v. Am. Longevity, No. 99-CV-9584 (CSH), 2001 WL 34314729, at*2 (S.D.N.Y. Mar. 1, 2001). In determining whether to exclude trial testimony, courts consider "(1) the surprise or prejudice suffered by the moving party; (2) the ability of that party to cure the prejudice; (3) whether waiver of the rule against calling unlisted witnesses is appropriate; (4) bad faith or willfulness in failing to comply ...." Id. However, "courts recognize that preclusion is a drastic remedy appropriate only in rare cases where a party's conduct represents flagrant bad faith and callous disregard of the Federal Rules of Civil Procedure." Jenkins v. City of New York, No. 96-CV-4421 (LMM), 2004 WL 2624872, at*2 (S.D.N.Y. Nov. 17, 2004) (citations and internal quotation marks omitted). Defendants likely are correct that they would be prejudiced if Rodriguez suddenly appeared to testify on Plaintiff's behalf. Rodriguez is alleged to have been in the car with Plaintiff when Plaintiff's car struck the parked vehicle, the accident that precipitated the events giving rise to this case. If he unexpectedly turned up at the last minute, the court may need to postpone trial to give Defendants an opportunity to depose Rodriguez and to seek rebuttal evidence. See, e.g., Baptiste v. Rohn, No. 13-CV-104, 2016 WL 1060237, at*5 (D.V.I. Mar. 15, 2016) (excluding two witnesses whose contact information were not disclosed until seventeen days before trial).

***11** Nevertheless, preclusion is a drastic measure only appropriate where "a party's conduct represents flagrant bad faith and callous disregard of the Federal Rules." Jenkins, 2004 WL 2624872, at*2. Plaintiff notes that he does not know where Rodriguez is, and does not intend to call on him to testify. The court cannot assess whether there is any bad faith until Plaintiff actually comes forth with new information about Rodriguez—and how that information came about—and states his intention to call him to testify. The court therefore concludes that the most prudent path forward is to reserve decision on Defendant's motion to preclude Rodriguez until it becomes an issue and the court is in possession of all the necessary information. Cf. Morgutia-Johnson v. Hustedde, No. 14-CV-127 (LJO), 2015 WL 3507130, at *6 (E.D. Cal. June 3, 2015) (denying as moot motion to preclude plaintiff's witnesses for which contact information has not been properly disclosed because plaintiff has not been able to find them).

5. Kavita Samaroo

Next, Defendants request that the court preclude witness Kavita Samaroo ("Samaroo") from testifying. (Defs.' Mot, at 6.) Defendants reason that Plaintiff's parents "will testify to Plaintiff's damages," and "[t]o allow Ms. Samaroo to testify on the same topic would be

cumulative, a waste of time, and would not make any fact at issue more or less likely." (Id.) Plaintiff argues that it would be premature to make any judgments on whether the testimony of potential witnesses would be cumulative or a waste of time. (Pl.'s Opp'n at 8-9.) The court agrees.

"It would ordinarily be inappropriate for any party to call two witnesses to offer the same testimony because such testimony would be cumulative. However, at this time it is impossible to know whether [one witness's] testimony will be cumulative of [another witness's] because neither has testified." Giladi v. Strauch, No. 94-CV-3976 (RMB) (HBP), 2007 WL 415365, at *10 (S.D.N.Y. Feb. 6, 2007); see also Jamil, 707 F.2d at 643 ("[W]hen the trial has not yet commenced and no evidence has yet been put before a jury, it is premature to conclude that this evidence is cumulative."). The issue of "damages" is expansive, and Defendants have not yet indicated that Plaintiff's parents and Samaroo would testify to the same facts or opinions, either by pointing to deposition testimony or other evidence. Defendants then, are asking the court to blindly cap the number of witnesses that Plaintiff should be permitted to call to testify on the topics of damages and arbitrarily select Plaintiff's parents as more appropriate witnesses than Samaroo. This, the court will not do. If it turns out at trial that the testimony of Plaintiff's parents and Samaroo is cumulative, Defendants may raise their objection then. At this time, however, Defendants' motion to preclude Samaroo from testifying is denied.

6. Memo Book Entries, Command Logs, Medical Treatment of Prisoner Form, Internal Affairs Bureau Documents, and Photographs

Lastly, Defendants seek to exclude wholesale Defendants' memo book entries, command logs, a medical treatment prisoner form, Internal Affairs Bureau documents, and certain photographs taken of the police precinct where the alleged excessive force took place. (Defs.' Mot. at 13-15.) Defendants claim in a conclusory manner that the memo books contain little relevant information and that any relevant information is contained in other unidentified proposed exhibits for trial. (Id. at 13.) Defendants also argue that Plaintiff's sole purpose for introducing the memo books would be "to cause undue embarrassment and harassment to defendants for their entries or alleged omissions." (Id. at 13-14.) Defendants failed, however, to include any of the disputed memo book entries with their moving papers, or even summarize for the court their objectionable content. The court has no idea what relevant information may be contained in the memo books, much less what other potential trial exhibits could serve as alternatives. The court also does not know how introduction of the memo books could cause Defendants "undue embarrassment and harassment" or why any "alleged omissions" would not be relevant in this case.

***12** As to the other documents, Defendants claim that the only relevant information contained in the command logs, IAB documents, and medical treatment of prisoner form are "duplicative of information that plaintiff cannot dispute—namely, that on July 17, 2009, Plaintiff was transported in an ambulance [+] from the vicinity of the 103rd Precinct in Queens, New York to Jamaica Hospital, and later diagnosed with and treated for a head injury." (Id. at 14.) Defendants also claim that there are some "handwritten statements" contained in the medical treatment of prisoner form that is hearsay. (Id. at 15.) As to the photographs, Defendants argue that they were taken after the precinct had been renovated, and so do not accurately depict the precinct at the time of the incident. (Id.) Defendants also broadly charge that all of the documents are inadmissible hearsay under Rule 802 and lack authentication under Rule 901. Again, without the benefit of actual documents, the court is unable to assess their admissibility. Plaintiff also points out—and the court is unable to reconcile at this juncture—the fact that Defendants themselves listed the command logs and the medical treatment of prisoner form as potential Defense exhibits. (See Pl.'s Opp'n at 10; JPTO at 16, 18.)

It is Defendants' burden to establish evidence is inadmissible for any purpose, and they have failed to meet their burden. See Pugh, 2016 WL 627347, at *2. Accordingly, Defendants' motion as to these documents and photographs is denied. See Viada v. Osaka Health Spa, Inc., No. 04-CV-2744 (VM) (KNF), 2005 WL 3435111, at *1 (S.D.N.Y. Dec. 12, 2005) (denying "vague" motions in limine). Defendants are invited to make more particularized objections at trial.

IV. CONCLUSION

For the reasons stated above, the court finds that:

- Plaintiff's motion to preclude evidence of his intoxication is GRANTED in part and DENIED in part. (See infra Part III.A. 1 & Part III.A.3.) Specifically, evidence of Plaintiff's intoxication is admissible, but JPTO Exs. F, G, L, and N, and any other documents or testimony reflecting Plaintiff's refusal to submit to a blood alcohol test are inadmissible.

- Plaintiff's motion to preclude evidence of his guilty plea to his traffic violation is DENIED. (See infra

Part III.A.2 & Part III.A.3.)

• Defendants' motion to preclude evidence of Defendants' disciplinary histories, lawsuits, and personnel files, testimony of unidentified healthcare providers from Jamaica Medical Center or members of the NYPD Internal Affairs Bureau, the NYPD Patrol Guide, references to Corporation Counsel as "City Attorneys," suggestions that City of New York may indemnify Defendants, or requests for a specific dollar amount of damages from the jury is GRANTED. (See infra Part III.B.1.)

• Defendants' motion to preclude Plaintiff's treating physicians from testifying is GRANTED in part, DENIED in part, and RESERVED in part. (See infra Part III.B.2.) Specifically, Plaintiff's treating physicians may not testify as expert witnesses, but they are permitted to testify as fact witnesses. The court reserves judgment on whether Dr. Igor Cohen qualifies as Plaintiff's treating physician.

• Defendants' motion to preclude evidence of Plaintiff's Diffusor Tensor Imaging Exam is GRANTED. (See infra Part III.B.3.)

• Defendants' motion to preclude Alex Rodriguez from testifying is RESERVED. (See infra Part III.B.4.)

• Defendants' motion to preclude Kavita Samaroo from testifying is DENIED. (See infra Part III.B.5.)

• Defendants' motion to preclude Defendants' memo book entries, command logs, a medical treatment prisoner form, Internal Affairs Bureau documents, and certain photographs is DENIED. (See infra Part III.B.6.)

SO ORDERED.

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United States District Court,
W.D. Louisiana,
Lafayette Division.

Robert Craig ANDREW, et al.
v.
PATTERSON MOTOR FREIGHT, INC., et al.

Civil Action No. 6:13CV814.
|
Signed Oct. 23, 2014.

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MEMORANDUM RULING

REBECCA F. DOHERTY, District Judge.

***1** Currently pending before the Court are the following motions: (1) plaintiff's "Motion in Limine to Strike and/or Limit Certain Testimony of Lay Witness, George 'Tracy' Latiolais" [Doc. 47]; (2) "Defendants' Motion in Limine/ *Daubert* Challenge to Exclude or Limit the Trial Testimony and Evidence of Dr. Eduardo Gonzalez–Toledo and Request for Hearing" [Doc. 51]; and (3) "Defendants' Motion in Limine/ *Daubert* Challenge to Exclude the Trial Testimony and Evidence of Dr. Mark S. Warner, Ph.D" [Doc. 52] .[1]

Considering the law, the facts in the record, and the arguments of the parties, the Court GRANTS plaintiffs' motion to limit the testimony of George "Tracy" Latiolais [Doc. 47]; the Court DENIES IN PART and DEFERS IN PART defendants' motion in limine/ *Daubert* challenge to Dr. Eduardo Gonzalez–Toledo [Doc. 51]; and the Court DENIES IN PART and DEFERS IN PART defendants' motion in limine/ *Daubert* challenge to Dr. Mark S. Warner [Doc. 52].

I. Factual Background

This matter involves a motor vehicle accident occurring on June 29, 2012, in the town of Broussard, Louisiana. [Doc. 1, ¶¶ 6, 7] According to the complaint, plaintiff Robert Andrew was injured when he was struck by a tractor-trailer operated by defendant Cecil A. French. [*Id.* at ¶ 7] Plaintiff alleges Mr. French was in the course and scope of his employment with defendant Patterson Motor Freight, Inc. at the time of the collision. [Doc. 5, ¶ 3] Plaintiff alleges as a result of the accident, he "sustained a Traumatic Brain Injury to the frontal lobe resulting in residual deficits in the areas of emotion, impulsivity, personality, and short term memory." [Doc. 48, p. 3] Plaintiff additionally alleges he sustained a fracture of a thoracic vertebrae (for which he underwent a T8 Kyphoplasty), and damages to the facets at the L4–5 region of the spine (with a recommendation of an L3–4 and L4–5 fusion with rods). [*Id.*] Plaintiff asserts he "has suffered and continues to suffer with severe back pain and general body pain, cognitive difficulties, headaches, sleep deprivation and disturbances, mood uncertainties, and confusion."[2] [*Id.*] Trial of this matter is scheduled for December 8, 2014. [Doc. 26]

II. Standards of Review

A. Lay Testimony

Rule 602 of the Federal Rules of Evidence states in pertinent part: "A witness may testify to a matter only if evidence is introduced sufficient to support a finding that the witness has personal knowledge of the matter. Evidence to prove personal knowledge may consist of the witness's own testimony." Fed.R.Evid. 602. If it is determined the witness does have personal knowledge of the matters to which he intends to testify, the nature of the witness' testimony is further limited by Rule 701, which provides:

> If a witness is not testifying as an expert, testimony in the form of an opinion is limited to one that is:
>
> (a) rationally based on the witness's perception;
>
> ***2** (b) helpful to clearly understanding the witness's testimony or to determining a fact in issue; and
>
> (c) not based on scientific, technical, or other specialized knowledge within the scope of Rule 702.

Fed.R.Evid. 701; *see also U.S. v. Ebron*, 683 F.3d 105, 137 (5th Cir.2012)("A lay opinion must be based on

personal perception, must be one that a normal person would form from those perceptions, and must be helpful to the jury.")

B. Expert Testimony

To be admissible at trial, expert testimony must satisfy the conditions of Federal Rule of Evidence 702, which provides:

> A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if:
>
> (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;
>
> (b) the testimony is based on sufficient facts or data;
>
> (c) the testimony is the product of reliable principles and methods; and
>
> (d) the expert has reliably applied the principles and methods to the facts of the case.

Fed.R.Evid. 702. A district court has considerable discretion in deciding whether to admit or exclude expert testimony. *See Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 152, 119 S.Ct. 1167, 143 L.Ed.2d 238 (1999) ("[W]e conclude that the trial judge must have considerable leeway in deciding in a particular case how to go about determining whether particular expert testimony is reliable."); *General Elec. Co. v. Joiner*, 522 U.S. 136, 139–40, 118 S.Ct. 512, 139 L.Ed.2d 508 (1997)(abuse of discretion is the standard of review).

"Rule 702 requires trial courts to ensure that proffered expert testimony is 'not only relevant, but reliable.' " *Brown v. Illinois Cent. R. Co.*, 705 F.3d 531, 535 (5th Cir.2013)(quoting *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 589, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993)). "To determine whether proffered testimony is reliable, the trial court must make 'a preliminary assessment of whether the reasoning or methodology underlying the testimony is ... valid and of whether that reasoning or methodology properly can be applied to the facts in issue.' " *Id.* (quoting *Daubert* at 592–93). Courts should consider scientific expert testimony in light of factors that help determine the reliability of that testimony. *Daubert* at 589, 592–94. In this reliability analysis, courts may rely on factors such as those suggested by the *Daubert* court: "whether the theory or technique the expert employs is generally accepted; whether the theory has been subjected to peer review and publication; whether the theory can and has been tested; whether the known or potential rate of error is acceptable; and whether there are standards controlling the technique's operation." *Broussard v. State Farm Fire and Cas. Co.*, 523 F.3d 618, 630 (5th Cir.2008). "*Daubert* makes clear that the factors it mentions do *not* constitute a 'definitive checklist or test.' " *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 150, 119 S.Ct. 1167, 143 L.Ed.2d 238 (1999) (emphasis in original). "The district court's responsibility is 'to make certain that an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field.' " *Pipitone v. Biomatrix, Inc.*, 288 F.3d 239, 247 (5th Cir.2002)(quoting *Kumho*, 526 U.S. at 152)). The focus of reliability "must be solely on principles and methodology, not on the conclusions they generate." *Daubert*, 509 U.S. at 595.

***3** "[A]s a general rule, questions relating to the bases and sources of an expert's opinion affect the weight to be assigned that opinion rather than its admissibility...." *United States v. 14.38 Acres of Land*, 80 F.3d 1074, 1077 (5th Cir.1996)(internal quotations and citations omitted). "It is the role of the adversarial system, not the court, to highlight weak evidence...." *Primrose Operating Co. v. Nat'l American Ins. Co.*, 382 F.3d 546, 562 (5th Cir.2004). "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence." *Daubert* at 596 (citation omitted).

III. Mr. George "Tracy" Latiolais

In 2005, plaintiff and Mr. Tracy Latiolais formed A & L Repair Service, LLC, an oilfield service company specializing in the repair of oilfield equipment, such as drill pipe spinners, kelly spinners, and power tongs.[3] [Doc. 48, pp. 6–7] Plaintiff and Mr. Latiolais each owned fifty percent of the company. [*Id.* at 6; Doc. 64, p. 2] In August 2013, Mr. Latiolais unilaterally made the decision to close down A & L Repair. [Doc. 48, pp. 7–8; Doc. 64, pp. 3–4] According to both plaintiff and Mr. Latiolais, Mr. Latiolais made the decision to close down A & L Repair because he was concerned the medications plaintiff was prescribed to address injuries sustained in the motor vehicle accident impaired plaintiff and might cause an accident, thereby exposing the business (and Mr. Latiolais) to liability. [*See e.g.* Doc. 64–1, pp. 15, 18–19; Doc. 47–6, pp. 3–4] According to plaintiff, he tried to explain to Mr. Latiolais the behaviors about which Mr. Latiolais was concerned were due to effects of the brain

injury he incurred, rather than his prescribed medications. [Doc. 64–1, pp. 18–19] However, Mr. Latiolais was adamant that unless plaintiff discontinued his medications, the business would be closed. [*Id.*] As noted, Mr. Latiolais closed A & L Repair in August 2013.

By this motion, plaintiff seeks an order prohibiting Mr. Latiolais from testifying certain behaviors of plaintiff were caused by plaintiff's use of prescribed medications. [Doc. 48, pp. 16, 17] Plaintiff agrees Mr. Latiolais may testify as to: "his perceptions that after the crash Mr. Andrew's *behavior* changed," the behavior change affected plaintiff's work performance, and the behavior change led to Mr. Latiolais' decision to shut down the business. [*Id.* at 17 (emphasis in original)] However, plaintiff argues Mr. Latiolais should not be allowed to testify the *cause* of plaintiff's behavior change was due to medication. [*Id.* at 16–17] Counsel for plaintiff notes Mr. Latiolais testified in his deposition he did not know what medications plaintiff was taking, the dosage of those medications, or the side effects caused by the medications.

Defendants argue such testimony is properly admissible based upon Mr. Latiolais' observation of plaintiff, and because Mr. Latiolais had been told by plaintiff he was taking medications due to the injuries sustained in the accident. [Doc. 64, p. 6] Defendants additionally argue this testimony is relevant to the issue of damages for loss of wages, because Mr. Latiolais testified the reason they closed the business "was because of Andrew's medication usage and the resulting impairment."[4] [*Id.*] Finally, defendants argue, "[a]ny concerns Plaintiffs may have can be fully addressed in cross-examination."

***4** The Court finds Mr. Latiolais lacks the qualifications necessary to provide his opinion as to the *cause* of plaintiff's behavior, and thus, his opinion plaintiff's behavior was caused by prescribed medications lacks foundation. Fed.R.Evid. 701 (where witness is not testifying as an expert, opinion testimony is limited to opinions based on perception, if helpful, and if not based on scientific, technical, or other specialized knowledge). Again, Mr. Latiolais testified he does not know what medications plaintiff was taking or their dosage; other than "a broken back," he does not know what injuries plaintiff sustained; and he has no experience dealing with someone with abrain injury. [Doc. 47–6, pp. 16–18, 20, 22]

The Court additionally finds the foregoing testimony should be excluded pursuant to Federal Rule of Evidence 403, which provides: "The court may exclude relevant evidence if its probative value is substantially outweighed by a danger of one or more of the following: unfair prejudice, confusing the issues, misleading the jury, undue delay, wasting time, or needlessly presenting cumulative evidence." Here, the Court finds any probative value of the testimony at issue would be substantially outweighed by the danger of unfair prejudice, confusion of the issues, and/or misleading the jury, in that it would present plaintiff to the jury as a potential drug abuser, where no evidence has been presented regarding same, and there are alternative explanations for the behavioral changes (*i.e.* the effects of abrain injury).

For all of these reasons, the Court finds while Mr. Latiolais may properly testify about his observations of plaintiff's behavior, he lacks sufficient personal or scientific knowledge to testify as to *the cause* of such behavior changes. *See e.g. Graves ex rel. W .A.G. v. Toyota Motor Corp.*, 2011 WL 4590772, *8 (S.D.Miss.)("An opinion based upon the assumption of the existence of an important fact cannot meet the Rule 701 test.") Accordingly, the Court grants plaintiff's motion, and Mr. Latiolais will be prohibited from testifying plaintiff's behavior changed *due to his use of prescribed medications.*

IV. Dr. Eduardo Gonzalez–Toledo

By this motion, defendants assert plaintiff's neuroradiology expert, Dr. Eduardo Gonzalez–Toledo, should be prohibited from testifying at trial, and "all evidence associated with him" should be excluded. [Doc. 51, p. 1] Alternatively, defendants move for an Order limiting his testimony, "to exclude the images created with the Brain Suite program." [*Id.; see also* Doc. 56, p. 3] Defendants request a "pre-trial '*Daubert* Hearing' on this motion...." [*Id.* at 2] In support of their motion, defendants argue: (1) Dr. Gonzalez–Toledo is not qualified in the field of neuroradiology; (2) "the methodology that he utilized for his analysis is not widely accepted for the diagnosis of traumatic brain injury (TBI)"; and (3) "his testimony will be cumulative with that of Plaintiff's treating physicians and other expert and will not be helpful to the court." [Doc. 51–2, p. 1]

A. Qualifications

***5** Defendants argue Dr. Gonzalez–Toledo "does not meet the criteria of having sufficient specialized knowledge to assist the trier of fact," because "he does not possess the necessary board certification to be recognized as a neuroradiologist or a neurosurgeon in the United States." [*Id.* at 4] According to defendants, Dr. Gonzalez–Toledo's "designation as 'neuroradiologist' is

self-selected." [*Id.*] Defendants note Dr. Gonzalez–Toledo "has prior certifications in neurosurgery and radiology from Argentina, but he is only licensed to practice radiology in Louisiana." [*Id.*]

According to Dr. Gonzalez–Toledo's affidavit: he is "a medical doctor specializing in neuroradiology," licensed by the Louisiana State Board of Medical Examiners; he is the Director of Neuroradiology at LSU Health Sciences Center in Shreveport and the Director of Research for the Department of Radiology at University Health (formerly known as LSU Health Sciences Center in Shreveport); he is a tenured professor of Radiology, Neurology and Anesthesiology at University Health; for over forty-five years, he has been teaching, researching, practicing, and publishing articles about neurology, radiology, neurosurgery, CT technology, MR technology and neuroimaging; he has published nearly 200 publications, including books, chapters in books, and articles in journals in the fields of radiology, neurology, and neuroradiology; he is a member of many professional societies, including the American College of Radiology and the American Society of Neuroimaging; he became board certified in neurosurgery by the Argentine College of Neurosurgeons in 1971, and was certified in radiology by the Ministry of Public Health in Argentina in 1977; he was board certified in both diagnostic imaging and neurosurgery by the National Academy of Medicine's Council for Certifications of Medical Professionals in Argentina shortly after it was created in 1994; in 2010, the United States' Accreditation Council for Graduate Medical Education ruled the foregoing credentials "were equivalent to board certification by the American Board of Radiology." [Doc. 59–5, ¶¶ 1, 3–4, 44, 46–47, 53–54]

The Court finds the foregoing credentials qualify Dr. Gonzalez–Toledo to testify as an expert in the field of neuroradiology and notes, however, that defendants will have full opportunity to traverse Dr. Gonzalez–Toledo as to his qualifications at trial, if defendants so desire.

B. Methodology

1. Cortical Reconstruction/Cortical Thickness Measurement

According to Dr. Gonzalez–Toledo, Cortical Reconstruction or Cortical Thickness Measurement ("CTM") is a type of neuroimaging that detects changes in the cortical surface—*i.e.*, "the area where the gray matter covers the cerebral hemispheres, where the higher nervous system centers are located." [Doc. 51–4, p. 1; Doc. 59–5, ¶ 6] To conduct CTM, Dr. Gonzalez–Toledo performs an MRI, the data from the MRI is processed through BrainSuite software, resulting in 3D reconstructed images of the cortical surface. [Doc. 59–5, ¶¶ 32–33, 35; Doc. 59, p. 4; Doc. 51–4, p. 2] According to Dr. Gonzalez–Toledo, CTM "demonstrate[s] evidence of traumatic brain injury pathology and can reveal abnormalities that are not visible on standard MRIs." [Doc. 59–5, ¶ 21; Doc. 51–4, p. 3] As noted by defendants, according to the BrainSuite website:

> ***6** BrainSuite is a collection of software tools that enable largely automated processing of magnetic resonance images (MRI) of the human brain. The major functionality of these tools is to extract and parameterize the inner and outer surfaces of the cerebral cortex and to segment and label gray and white matter structures. BrainSuite also provides several tools for visualizing and interacting with the data.

[Doc. 51–2, p. 6 (citing *http://brainsuite.org/* (August 19, 2014))]

Defendants argue Dr. Gonzalez–Toledo's testimony should be excluded because "it is not based on sufficient data and facts, and the methodology that he utilized for his analysis, i.e., reconstructing images from MRI data through the use of Brain Suite software, is not widely accepted for the diagnosis of traumatic brain injury (TBI)." [Doc. 51–2, pp. 4–5] Alternatively, defendants move for an order "limiting the testimony and evidence ... to exclude the images created with the Brain Suite program." [Doc. 51, p. 1] Defendants note they "do not object to the underlying data [i.e. the MR images], but to the prejudicial and misleading reworking of the data and presentation of it by the created images produced by postprocessing software." [Doc. 80, pp. 1–2]

With regard to methodology, defendants argue "cortical mapping ... is currently a research tool and is not used in clinical diagnostics and decision-making," citing the affidavit of their expert neuroradiologist, Dr. Partington.[5] According to defendants, the images of plaintiff's brain attached to Dr. Gonzalez–Toledo's report are "excerpted from the MRI," and then "processed to show the surface of the brain with color of an arbitrary value superimposed on these images." [Doc. 52–2, p. 6] Defendants continue, "In his report, Dr. Gonzalez–Toledo stated that the areas that are color-coded in blue on these maps show evidence of traumatic brain injury."[6] [*Id.*] According to Dr. Partington, when the areas in blue on the CTM images are

compared to the same areas of the brain on the MRI images, no abnormality is observable. [*Id.; see also* Docs. 59–24, p. 12; 54–3, p. 3; 51–7, p. 2] In other words, defendants argue "[t]he data itself is normal and shows no evidence of traumatic injury."[7] [*Id.* at 7] In light of the foregoing, defendants conclude:

> Dr. Gonzalez–Toledo's use of the Brain Suite software for diagnostic purposes has not been sufficiently tested and subjected to peer review and publication in the field of traumatic brain injury to be reliable. The potential rate of error is unknown, Dr. Gonzlez–Toledo offered no standards controlling its operation; and it is not generally accepted within the neuroradiology field as a reliable clinical diagnostic tool. *Daubert, supra.* [*Id.* at 8][8]

In support of their argument that Dr. Gonzalez–Toledo's testimony is based on insufficient facts and data, defendants argue Dr. Gonzalez–Toledo "never met Plaintiff or observed his behavior" and, based solely upon the MRI he conducted and his "reconstruction of the data from that MRI in Brain Suite, ... he claims that Mr. Andrew suffered a traumatic brain injury during the motor vehicle accident." [Doc. 51–2, p. 5 (citing Dr. Gonzalez–Toledo's expert report)] However, according to defendants, in his deposition, Dr. Gonzalez–Toledo "admitted that he cannot say that this accident caused the alleged damage to the brain." [*Id.*] The Court will not exclude Dr. Gonzalez–Toledo's testimony on the basis of the argument now presented by defendants. Rather, after testimony and opportunity for objection, should CTM testimony be admitted at trial, this issue can be fully addressed on cross-examination. *See e.g. Daubert,* 509 U.S. at 592 ("Unlike an ordinary witness ..., an expert is permitted wide latitude to offer opinions, including those that are not based on firsthand knowledge or observation"); *Bryan v. John Bean Division of FMC Corp.,* 566 F.2d 541, 546 (5th Cir.1978)("experts particularly doctors customarily rely upon third party reports from other experts such as pathologists and radiologists in whom the testifying expert places his trust"); Fed.R.Evid. 703 ("An expert may base an opinion on facts or data in the case that the expert has been made aware of or personally observed").

***7** As their final argument, defendants assert "the probative value of Dr. Gonzalez–Toledo's reconstructed images and analysis is substantially outweighed by the likelihood that the jury will be confused or mislead by the compelling visuals of the images produced by the Brain Suite imaging technology." [Doc. 51–2, p. 9] According to defendants, "The images produced by the software, while not accurately reflecting the status of Plaintiff's brain, are colorful, arresting, and likely to impress the average juror who may not understand the nature and origin of the images and what they actually portray." [*Id.*]

With regard to CTM, itself, the Court finds, at this juncture, it has insufficient information to determine whether the testimony and evidence is reliable. While Dr. Gonzalez–Toledo has provided a number of *conclusory* statements and open opinions regarding the reliability of CTM, he has not provided an underlying bases for those opinions. "To establish reliability under *Daubert,* an expert bears the burden of furnishing 'some objective, independent validation of [his] methodology.' " *Brown v. Illinois Cent.R. Co.,* 705 F.3d at 536 (quoting *Moore v. Ashland Chemical Inc.,* 151 F.3d 269, 276 (5th Cir.1998)). Accordingly, the Court will grant defendant's motion for a pre-trial *Daubert* hearing to address the reliability of CTM and Dr. Gonzalez–Toledo's reliance thereon. At the hearing, plaintiff should focus his argument and evidence on factors such as: whether the theory or technique the expert employs is generally accepted; whether the theory has been subjected to peer review and publication; whether the theory can be and has been tested; whether the known or potential rate of error is known or if known, acceptable; and whether there are standards controlling the technique's operation. *Broussard,* 523 F.3d at 630. The hearing will be set by separate minute entry.

2. Diffusion Tensor Imaging ("DTI")

According to Dr. Gonzalez–Toledo, diffusion tensor imaging ("DTI") is "an MRI method that examines the microstructure of the white matter of the brain, allowing for the detection of microscopic pathology or abnormality of the white matter." [Doc. 59–5, ¶ 7] More specifically:

> DTI measures the direction of movement or flow (known as diffusion) of water molecules through tissue. Water moves through damaged tissue at different rates and in different directions than it does [in] healthy tissue. DTI is based upon the basic physics of the flow of water. With no barriers to flow, water will move in isotropic distribution, which means it Will move equally in all directions. If there are barriers to flow, it will move anisotropically or unequally in all directions like a perforated sprinkler-hose. As the water molecules flow through brain tissue, the water molecules follow

> the nerve fibers, and so by reconstructing these trajectories, DTI can image the nerve fibers.

[Doc. 59–5, p. 5] "The majority of people who have sustained mild traumatic brain injury (mTBI) have normal MRI and CT findings, even when significant neurological impairments exist as a result of the traumatic brain injury." [*Id.*] "DTI is a more sensitive technology that can reveal damage that is not visible on standard MRIs." [*Id.* at ¶ 9] To perform DTI, Dr. Gonzalez–Toledo performs an MRI, and then inputs the data obtained from the MRI into software called "3D Slicer," resulting in 3D reconstruction of the fiber tracts. [*Id.* at ¶¶ 32–35; Doc. 51–4, p. 2]

***8** At this juncture, the Court must note defendants make no attack against the use of DTI until their reply brief. While they ask this Court to exclude both DTI and CTM evidence in their original and supplemental motion in limine, all arguments contained in those documents are addressed toward the use of the BrainSuite software (and thus, CTM). The majority of defendants' argument against Dr. Gonzalez–Toledo's methodology (*i.e.* DTI is not widely accepted for the diagnosis of TBI) is based upon a single article entitled *Guidelines for the Ethical Use ofNeuroimages in Medical Testimony*. According to defendants, this article supports their position that "[t]he postprocessed images are vibrant and visually arresting, and likely to impress the average juror who will likely not understand how the images are created, what they actually show, and whether they are reliable." [Doc. 80, p. 3] Defendants additionally note the article "cites concerns about bias, such as the hindsight bias, by which radiologists are more likely to detect an abnormality on imaging when they are told in advance to expect one," as well as concerns that " 'in cases that use functional neuroimaging methods typically performed in the research setting, the expert may be influenced by a professional investment in promoting his or her research area or specific research findings.' " [*Id.*]

Defendants then state the same concerns "may very well be at play here...." [*Id.*] The Court finds these are all matters for cross-examination and not a basis for blanket exclusion of Dr. Gonzalez–Toledo's testimony.

Defendants note the article states DTI "results may vary by scanner field strength, scanner type, pulse sequence, and postprocessing." [*Id.* at 3–4; Doc. 74–3, p. 3] However, Dr. Gonzalez–Toledo has provided all the relevant information necessary for defendants to explore this topic on cross-examination. [*See* Doc. 59–5, ¶¶ 31–33, 35–38] Defendants additionally assert Dr. Gonzalez–Toledo was "required" to include a disclaimer in his report, but failed to do so. [Doc. 80, pp. 4–5] First, the Court notes the disclaimer is "suggested"—not required. Second, the Court notes the disclaimer is addressed toward physicians and not jurors. [*See* Doc. 74–3, p. 4; 59–21, p. 5] Regardless, this issue can be fully addressed on cross-examination. The remainder of defendants argument against admission of DTI evidence is based upon defendants' expert's assertion of the ways in which he alleges Dr. Gonzalez–Toledo did not follow the "proposed" guidelines set forth in the referenced article. Again, all of these issues are matters for cross-examination, and not the basis for blanket exclusion of evidence.

Unlike CTM, the Court finds plaintiff has submitted sufficient evidence to show the reliability of DTI. In sum, the evidence submitted shows DTI has been tested and has a low error rate [Doc. 59–5, ¶¶ 12, 20–21, 30; Doc. 59–9]; DTI has been subject to peer review and publication [Doc. 59–5, ¶ 30; Doc. 59–9]; and DTI is a generally accepted method for detecting TBI [Doc. 59–5 at ¶ 7–12, 14, 18–19, 21, 30–31]. *Daubert v. Merrell Dow Pharmaceuticals, Inc.* ., 509 U.S. 579, 593–94, 113 S.Ct. 2786, 125 L.Ed.2d 469. The Court additionally notes DTI testimony has been admitted by several courts. *See e.g. Ruppel v. Kucanin*, 2011 WL 2470621 (N.D.Ind.); *Hammar v. Sentinel Ins. Co., Ltd.*, No. 08–019984 (Fla.Cir.Ct.2010) [Doc. 59–11]; *Booth v. Kit*, 2009 WL 4544743 (D.N.M.). Accordingly, the Court denies defendants' motion to the extent it seeks to exclude evidence and testimony regarding DTI.

V. Dr. Mark S. Warner

***9** By this motion, defendants argue the evidence and testimony offered by plaintiff's neuropsychology expert, Dr. Mark S. Warner, should be excluded, or alternatively, limited. [Doc. 52, p. 1] In support of this position, defendants argue Dr. Warner's methodology is "flawed and unreliable," as well as cumulative. [Doc. 52–2, p. 1] Defendants argue Dr. Warner's methodology is flawed because: (1) he never met or examined plaintiff; (2) "[h]is opinion is based solely upon the reported findings of other treating professionals and his general knowledge of the science surrounding traumatic brain injury"; and (3) because one of the expert opinions upon which Dr. Warner relies is that of Dr. Gonzalez–Toledo, who is the subject of a defense *Daubert* motion. [*Id.* at 4–5] Defendants argue Dr. Warner's testimony is cumulative, because defendants anticipate plaintiff will present testimony from his treating physicians (*i.e* . his treating neurosurgeon, neuropsychologist, and psychiatrist). [*Id.* at 2, 6]

As to defendants' argument Dr. Warner's methodology is flawed because he never examined plaintiff, and his opinion is based "solely upon the reported findings of other treating professionals and his general knowledge of the science surrounding traumatic brain injury," the Court notes defendants have provided no legal authority in support of this argument. Rather, "experts [,] particularly doctors[,] customarily rely upon third party reports from other experts such as pathologists and radiologists in whom the testifying expert places his trust." *Bryan v. John Bean Division of FMC Corp.*, 566 F.2d 541, 546 (5th Cir.1978); *see also Daubert*, 509 U.S. at 592 ("Unlike an ordinary witness ..., an expert is permitted wide latitude to offer opinions, including those that are not based on firsthand knowledge or observation"). Federal Rule of Evidence 703 provides, "An expert may base an opinion on facts or data in the case that the expert has been made aware of or personally observed". As the notes to Fed.R.Evid. 703 make clear, the rule contemplates opinions based upon data provided to the expert "outside of court and other than by his own perception." Fed.R.Evid. 703 (1972 Notes). Furthermore, " '[a]s a general rule, questions relating to the bases and sources of an expert's opinion affect the weight to be assigned that opinion rather than its admissibility and should be left for the jury's consideration.' " *U.S. v. 14.38 Acres of Land, More or Less Sit. in Leflore County, Miss.*, 80 F.3d 1074, 1077 (5th Cir.1996)(quoting *Viterbo v. Dow Chemical Co.*, 826 F.2d 420, 422 (5th Cir.1987)). Accordingly, defendants' motion will be denied on the basis of this argument.

As to defendants' argument Dr. Warner's testimony should be excluded because it relies upon the opinion of Dr. Gonzalez–Toledo, the Court defers ruling until after the *Daubert* hearing regarding CTI testimony and Dr. Gonzalez–Toledo's reliance thereon. Should it be found evidence of CTI is inadmissible, then the Court will exclude any opinions of Dr. Warner based *solely* upon his reliance of Dr. Gonzalez–Toledo's CTM studies.

***10** The Court additionally defers addressing whether Dr. Warner's testimony is cumulative until the evidence is heard at trial, but cautions plaintiffs, cumulative testimony will not be allowed. Defendants (as well as plaintiff) may object to cumulative testimony from any witness if and when such an event occurs at trial.

VI. Conclusion

In light of the foregoing reasons, the Court GRANTS plaintiffs' motion to limit the testimony of George "Tracy" Latiolais [Doc. 47]; the Court DENIES IN PART and DEFERS IN PART defendants' motion in limine/ *Daubert* challenge to Dr. Eduardo Gonzalez–Toledo [Doc. 51]; and the Court DENIES IN PART and DEFERS IN PART defendants' motion in limine/ *Daubert* challenge to Dr. Mark S. Warner [Doc. 52].

All Citations

Not Reported in F.Supp.3d, 2014 WL 5449732

Footnotes

1 Additionally pending are: "Defendants' Motion in Limine/ *Daubert* Challenge to Exclude the Trial Testimony and Evidence of John W. Theriot and Request for Hearing" [Doc. 53], and plaintiffs' "Motion to Exclude Expert Witness, Frank Stagno, CPA/ABV and/or Motion in Limine as to Defendants' Proffered Expert Testimony and Report Regarding Mitigation of Damages and Reasonable Alternatives" [Doc. 67]. Those motions will be addressed by separate ruling.

2 Plaintiff's wife, Susan M. Andrew, asserts a claim for loss of consortium. [Doc. 1, ¶ 12] References herein to "plaintiff" are to Robert Andrew.

3 In 2006, plaintiff and Mr. Latiolais additionally formed A & L Construction, LLC, a real estate holding company that owned the A & L Repair office building/shop, and received rental payments from A & L Repair for the use of this space. [Doc. 60–2, pp. 4, 6]

4 Defendants argue Mr. Latiolais' reason for closing the business (*i.e.* his concern A & L would face liability in the event plaintiff's impairment from medications caused an accident) is relevant, because plaintiff is seeking "damages associated with the closure of the businesses...." [Doc. 64, pp. 2, 3, 6] However, as noted by plaintiff, "A & L Repair Services, LLC is not a party to this litigation and Mr. Andrew is not by pleading financial damages stemming from the closure of this entity on behalf of the LLC." [Doc. 67–3, p. 18; *see also* Doc. 48, p. 18] Rather, plaintiff is seeking damages for lost wages and lost earning capacity he *personally* incurred as a result of this accident. [*See e.g.* Doc. 1, ¶ 11; Doc. 48, p.18; Doc. 67–3, pp. 18–19]

5 According to Dr. Gonzalez–Toledo's affidavit, CTM is "used clinically at University Health as a diagnostic tool," and it is "used clinically in other parts of the country and is reimbursable by some health insurance companies." [Doc. 59–5, p. 4]

6 The Court notes Dr. Gonzalez–Toledo's states the "compromised portions of the cortex" are shown in "blue and yellow colors." [Doc. 51–4, p. 2]

7 Again, according to Dr. Gonzalez–Toledo, the reason one conducts CTM is precisely because it "demonstrate[s] evidence of traumatic brain injury pathology and can reveal abnormalities that are not visible on standard MRIs." [Doc. 59–5, ¶ 21] Additionally, the Court notes, when pressed by plaintiff's counsel on the issue of the purported inconsistencies between plaintiff's CTM and MRI images, Dr. Partington testified: "And I will admit that I am not well-versed enough in cortical mapping to know whether a normal person, are they absolutely homogenous red, absolutely homogenous blue.... And I just don't have enough experience with it and knowledge of it to know what the normal variations are." [Docs. 56–1, p. 6; 59–24, p. 13] He further admits it is speculation on his part as to whether the areas in blue shown on the CTM images must match the MRI images. [Doc. 59–24, p. 13]

8 To the extent defendants argue the cortical mapping images are unreliable because "it is impossible to discern what parameters Dr. Gonzalez–Toledo set to get the results he presented in his report," the Court disagrees. [Doc. 54–2, p. 3] This argument is based on testimony of Dr. Partington, wherein he was asked if he could explain why the MRI images show a normal brain, whereas the CTM images show abnormality. Dr. Partington could not explain, but stated, "[m]y guess would be, and its strictly speculation on my part," that one could change the parameters on the software to show increased abnormality where none existed. [Doc. 56–1, p. 8] However, Dr. Gonzalez–Toledo states in his affidavit "[t]he software has preset conditions and settings that are recommended by physicists at ... UCLA," and he "does not modify the settings, change the parameters or make any changes to the software." [Doc. 59–5, p. 10] Accordingly, the Court will not exclude Dr. Gonzalez–Toledo's testimony on the basis "it is impossible to discern what parameters Dr. Gonzalez–Toledo set to get the results he presented in his report."

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Not Reported in F.Supp.2d, 2011 WL 2470621 (N.D.Ind.), 85 Fed. R. Evid. Serv. 859
(Cite as: 2011 WL 2470621 (N.D.Ind.))

United States District Court,
N.D. Indiana,
South Bend Division.
Dale RUPPEL, Shelley Ruppel, Plaintiffs,
v.
Dragan KUCANIN, Fedex Ground Package System, Inc., Defendants.

No. 3:08 CV 591.
June 20, 2011.

Robert J. Ehrenberg, Barry R. Conybeare, Conybeare Law Office PC, Saint Joseph, MI, for Plaintiffs.

Christopher J. Spataro, Carl A. Greci, Baker & Daniels, South Bend, IN, for Defendants.

OPINION AND ORDER

JAMES T. MOODY, District Judge.

***1** Defendant Dragan Kucanin ("Kucanin") a driver for defendant FedEx Ground Package System, Inc. ("FedEx") drove his semi-tractor trailer rig into a semi-tractor trailer rig driven by plaintiff Dale Ruppel ("Ruppel") when Ruppel was stopped in a construction zone. The accident between Ruppel and Kucanin occurred on Interstate 80/94 East in Calumet Township, Lake County, Indiana, on January 8, 2008. Both vehicles were damaged in the collision. (Pls.' Exh. 2, DE # 57–2.) Ruppel and his wife Shelley Ruppel (collectively "the Ruppels") sued FedEx and Kucanin for damages that he allegedly sustained as a result of the accident. (DE # 1.) Defendants have admitted that Kucanin was negligent in operating his semi-tractor trailer rig causing the crash with Ruppel's semi-tractor trailer rig. (Responses to Plaintiffs' Requests to Admit to Dragan Kucanin and FedEx Ground Package system, Inc., Pls.' Exh. 1, DE # 57–1 at 1.) They also admit that Ruppel has no comparative negligence. (*Id.*) Defendants have moved to exclude Ruppel's evidence related to an alleged diffuse axonal brain injury under FEDERAL RULE OF EVIDENCE 702 and for summary judgment on Ruppel's claim for a diffuse axonal injury. (DE54–56.) As explained below, both motions will be denied.

Defendants argue that two pieces of Ruppel's proposed evidence should be excluded under FEDERAL RULE OF EVIDENCE 702. First, they argue that Dr. Christine Pareigis ("Dr.Pareigis") is unqualified to diagnose a diffuse axonal injury because she is not qualified to diagnose an injury. (DE # 56 at 13.) Second, they argue that Dr. Randall Benson's ("Dr.Benson") opinion as to Ruppel's condition of a diffuse axonal injury and its causation is unreliable under RULE 702 because it is based on two controversial methods: diffusion tensor imaging ("DTI") and fractional anisotrophy ("FA") quantification from that imaging and because the wording of his opinion is not sufficiently certain. (*Id.* at 15.) Defendants argue that once this evidence is excluded, Ruppel will have no evidence as to his diagnosis of diffuse axonal injury or to its causation, and therefore, summary judgment should be granted against Ruppel on his claim related to diffuse axonal injury. The court will begin with an analysis of whether the contested evidence should be excluded under *Daubert.*

I. MOTION TO EXCLUDE EVIDENCE

To be admissible, expert testimony must satisfy the conditions of FEDERAL RULE OF EVIDENCE 702 and *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993). *United States v. Parra,* 402 F.3d 752, 758 (2005). RULE 702 provides:

If scientific, technical, or other specialized

> knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

***2** Under *Daubert*, the court must be satisfied, first, that the expert can testify based on *valid* scientific, technical or specialized knowledge, *i.e.*, whether the expert's testimony is reliable, and second, whether that testimony will be of assistance to the trier of fact. 509 U.S. at 592; *United States v. Welch*, 368 F.3d 970, 973 (7th Cir.2004); *Ammons v. Aramark Uniform Services, Inc.*, 368 F.3d 809, 816 (7th Cir.2004). The reliability issue requires the court to determine whether the expert is qualified in the relevant field and used a reliable methodology to arrive at his or her conclusions. *Zelinski v. Columbia 300, Inc.*, 335 F.3d 633, 640 (7th Cir.2003); *Smith v. Ford Motor Co.*, 215 F.3d 713, 718 (7th Cir.2000).

A. Dr. Pareigis's qualifications

FEDERAL RULE OF EVIDENCE 702 provides that a witness qualified as an expert "by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise." Defendants are correct that under RULE 702, a witness may only offer an expert opinion on an area within his or her field of specialized knowledge. (DE # 56 at 15 (citing *Jones v. Elec. Co.*, 188 F.3d 709, 723 (7th Cir.1999)).) To determine if a witness is an expert, the court must compare the area in which the witness has superior skill, knowledge, education, or expertise to the area of her proposed testimony. *Jones*, 188 F.3d at 723.

The parties contest whether Dr. Pareigis can testify as to Ruppel's diagnosis of diffuse axonal injury. Defendants argue that Dr. Pareigis cannot testify as to Ruppel's diagnosis because she is an expert in rehabilitation, not diagnosis. (DE # 56 at 16.) Defendants also submit proposed testimony from their witness, neurologist Dr. John Talbott, that physiatrists normally do not make a diagnosis of diffuse axonal injury in a "neurology field." (John Talbott Dep. 37, Defs.' Exh. R, DE # 56–18.) In response, the Ruppels assert that Dr. Pareigis is "board certified in physical medicine and rehabilitation and is qualified by knowledge, skill, experience, training and education to testify in the form of opinion as to a diagnosis of closed head injury with diffuse axonal damage and the probable cause thereof." (DE # 57 at 4.)

Dr. Pareigis is board certified in physical medicine and rehabilitation, a practice speciality which she stated "includes the evaluation, diagnosis, and treatment of brain injury." (Dr. Christine Pareigis Aff., Pls.' Exh. 4, DE # 57–4 ¶ 5.) She is now the Medical Director of Rehabilitation at the Lakefront Medical Center in St. Joseph, Michigan. (*Id.* ¶ 2.) In that position, which she has held for 21 years, she regularly diagnoses, evaluates, and treats brain injury. (*Id.*) She also maintains a private practice in St. Joseph, Michigan where she regularly evaluates, diagnoses, and treats brain injury. (*Id.* ¶ 4.) Dr. Pareigis stated that she sees an average of ten new cases a year involving injuries like Ruppel's for a total of about two hundred cases over the course of her career. (Dr. Christine Pareigis Dep. 48, Defs.' Exh. D., DE # 56–4.)

***3** She previously served as the Medical Director of Rehabilitation at New Medico / Visitors Hospital in Buchanan, Michigan. (Pareigis Aff. ¶ 3.) This institution is a head injury clinic, affiliated with a national program, that evaluates, diagnoses, and treats head injury patients. (*Id.*) As the Medical Director, 90% to 100% of Dr. Pareigis's practice involved the evaluation, diagnosis, and treatment of closed head injury. (*Id.*)

First, defendants appear to argue that Dr. Pareigis

cannot testify as to Ruppel's diagnosis of diffuse axonal injury because her diagnosis was based in part on the results of DTI and she received help from a radiologist in deciding to run that scan. (Christine Pareigis Dep. 23.) They also take issue with that fact that she used the abbreviations SWY/DTI explaining that she needed to do so because they were radiology terms. (*Id.*) Dr. Pareigis testified that she ordered the magnetic resonance imaging ("MRI") with SWY/DTI because she felt that it would give her "more evidence regarding axonal diffuse injuries." (Pareigis Dep. 23.) At the time of the deposition, she had not received the results of the DTI scan and she did not expect it to change the course of treatment, but she thought it might help her to understand Ruppel's injury a little better. (*Id.*)

Dr. Pareigis's testimony that she consulted with a radiologist in deciding to order the MRI does not disqualify her as an expert because she can base her conclusion on the opinions of others as long as they are the type of materials reasonably relied upon by experts in her field. *United States v. Gardner*, 211 F.3d 1049, 1054 (7th Cir.2000). RULE 703, the corollary to RULE 702, is instructive on this matter. RULE 703 states that an expert can rely on facts and data not admissible into evidence as long as the facts and data are "of a type reasonably relied upon by experts in the particular field in forming opinions or inferences upon the subject." The Advisory Committee notes to the 1972 amendments to RULE 703 state that "a physician in his own practice bases his diagnosis on information from numerous sources and of considerable variety including statements by patients and relatives, reports and opinions from nurses, technicians and other doctors, hospital records and X-rays." Accordingly, the FEDERAL RULES OF EVIDENCE account for the reality that doctors, like Dr. Pareigis, rely on the opinions of other doctors in reaching their diagnoses.

Further, Dr. Pareigis did not rely on the DTI scan alone in making her diagnosis. In fact, she stated that she thought the DTI scan would help her learn more about the injury but that it probably would not change her course of treatment. So her testimony is not unreliable because she consulted with another doctor in deciding the course of treatment for her patient. Instead, evidence that Dr. Pareigis consulted a radiologist to order the MRI would go to the weight that the jury may give her testimony.

***4** Apart from her reliance on the DTI scan, defendants argue that Dr. Pareigis is not qualified to testify at all as to Ruppel's diffuse of axonal brain injury diagnosis because making a diagnosis is outside of her expertise. In making this argument defendants cite to two cases, *Jones* and *Cunningham v. Masterwear, Inc.* In both, the court determined that qualified experts cannot testify on subjects that are outside of their field of expertise. In *Jones*, the United States Court of Appeals for the Seventh Circuit found that the witness, a doctor in metallurgy, the study of metals, was not qualified to testify as to how manganese affects the human body and is processed by the lungs. 188 F.3d at 723. In his testimony, the witness admitted that toxicology and how the body absorbs certain substances was outside of his expertise. *Id.* Similarly in *Cunningham*, the court held that witness medical doctors could not testify as to whether a hazardous chemical caused the plaintiffs' illnesses because the witnesses did not have any training in epidemiology or toxicology. No. 1:04–cv–1616, 2007 WL 1164832, at *10 (S.D.Ind. Apr.15, 2007).

In this case, Dr. Pareigis stated that the diagnosis of brain injuries is firmly within her area of expertise. The Seventh Circuit has noted that while "extensive academic and practical expertise" may be sufficient to qualify a witness as an expert, RULE 702 "specifically contemplates the admission of testimony by experts whose knowledge is based on experience." *Smith*, 215 F.3d at 718 (internal quotations and citations omitted). As described above, in her affidavit [FN1] Dr. Pareigis stated that she has over thirty years of experience in diagnosing brain injuries. This is the type of "exten-

sive hands-on experience over a meaningful period of time" that qualifies someone as an expert under RULE 702. *Jones*, 188 F.3d at 724. Thus the evidence before the court shows that Dr. Pareigis is qualified to testify as to Ruppel's diagnosis of a diffuse axonal brain injury.[FN2]

FN1. Defendants argue that Dr. Pareigis's affidavit cannot be used to show her qualifications when her qualifications were not established through her deposition. It is true that an "affidavit cannot be used to create a genuine issue of material fact where the affidavit differs from the prior deposition testimony to the point that it is ble." *Patterson v. Chicago Ass'n for Retarded Citizens*, 150 F.3d 719, 720 (7th Cir.1998). However, when "deposition testimony is ambiguous or incomplete ... the witness may legitimately clarify or expand upon that testimony by way of an affidavit." *Shepherd v. Slater Steels Corp.*, 168 F.3d 998, 1007 (7th Cir.1999). Dr. Pareigis's affidavit does not contradict her deposition testimony. Rather, the deposition testimony did not cover her qualifications and experience related to brain injury diagnosis.

FN2. Defendants do not argue that Dr. Pareigis was not qualified to testify as to causation. Accordingly, plaintiffs have not produced much evidence that she is qualified to testify as to causation. However, medical doctors do testify as to the issue of specific causation. *See e.g.*, *Cunningham*, 2007 WL 1164832, at *10–11 (citing Mary Sue Henifin, Howard M. Kipen & Susan R. Poulter, *Reference Guide on Medical Testimony* 444–45, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2nd ed.2000)). Further, in her deposition, Dr. Pareigis testified that she had seen "a great number of people" who suffered brain injury after motor vehicle accidents. (Christine Pareigis Dep. 47.) Thus her deposition testimony indicated that she does have experience in determining the specific causes of brain injury for her patients. Accordingly, at this time, the court will not exclude Dr. Pareigis's testimony as to the cause of diffuse axonal injury.

B. Dr. Benson's testimony

1. Dr. Benson's reliance on DTI

Defendants assert that Dr. Benson's expert testimony on diffuse axonal injury is unreliable under *Daubert* and RULE 702 because he relies on DTI which defendants argue is an unreliable technology that has not gained acceptance and because his reliance on FA quantification based on DTI comparisons is not the most accurate way to diagnose diffuse axonal brain injuries.

To begin, the court will give a brief overview of diffuse axonal brain injury, closed head injury, DTI, and how Dr. Benson used DTI to diagnose diffuse axonal injury in Ruppel. According to Dr. Benson, brain injury is classified as either focal or diffuse. (Dr. Randall Benson Aff., Pls.' Exh. 7, DE # 58–1 at ¶ 5.) A focal injury is a localized injury, such as that caused by a stroke, a direct blow to the head, or a aneurysm, and is typically a contusion on the surface of the brain, visible by conventional scanning. (*Id* .) On the other hand, a diffuse axonal injury involves scattered damage to the brain substance, particularly the white matter that is comprised of axon fibers. (*Id.*) A closed head (non-penetrating) brain injury, the most common type of traumatic brain injury, can include focal injury, diffuse injury, or both. (*Id.*) A brain injury can include only evidence of diffuse axonal injury. when it is a result of "relatively little direct impact to the skull such as during a motor vehicular collision with a restrained passenger and little or no impact to the head."

(*Id* .)

***5** According to Dr. Benson:

> Diffuse axonal injury is the hallmark pathology in closed head injury and is not visible on conventional MRI imaging in milder cases. Diffuse axonal injury results from acceleration or deceleration of the head (skull) which causes deformations (stretch and strain) of the brain substance leading to shear injury of white matter fibers.

(*Id.*) A traditional MRI shows the structure of the brain and the majority of people with mild brain injury will have a normal MRI even if they have significant impairment. (*Id.* ¶ 6.) DTI is a more sensitive, three-dimensional type of MRI that examines the microstructure of the white matter in the brain. (*Id.* ¶¶ 7–8.) DTI can show reduction in fractional anisotrophy ("FA") meaning that the white matter in the brain has been damaged. (*Id.* ¶ 12.) Because the reduction in FA caused by a milder traumatic brain injury ("TBI") cannot be seen by looking at a single scan standing alone, a TBI patient's imaging is evaluated for damage by comparing it to images of non-TBI control group's brains. (*Id.* ¶ 13.)

First, defendants cannot exclude Dr. Benson's opinion simply because DTI is not the most reliable way to diagnose a brain injury. They argue, and Dr. Benson testified, that the only definite way to identify a diffuse axonal brain injury is by autopsy. Barring that, they argue, as their expert Dr. Valerie Drnovsek ("Dr.Drnovsek") explains, that reduced FA may be detected through analysis with fiber-tracking algorithms. (DE # 56 at 10.) As defendants acknowledge, it is not reasonable to expect that Ruppel would have to submit to an autopsy in order to provide proof of his injuries. Contrary to defendants' contentions, expert opinions may be admitted even if they are not stated with absolute certainty. Indeed, in *Daubert* the Court stated, "[o]f course, it would be unreasonable to conclude that the subject of scientific testimony must be 'known' to a certainty; arguably, there are no certainties in science." *Daubert*, 509 U.S. at 590.

It is also unnecessary for Dr. Benson to have used fiber-tracking algorithms. The court's focus is on whether Dr. Benson's opinion is based on a reliable method, not on a method that defendants deem to be most reliable. *See e.g.*, *Cunningham*, 2007 WL 1164832, at *3 (stating "as long as [plaintiffs' proposed witness] used a reliable method to come up with his conclusions, it is not a problem that he did not use the method that Defendants claim is 'useful' "); *cf. Cooper v. Carl A. Nelson & Co.*, 211 F.3d 1008, 1020 (7th Cir.2000) (stating "[o]ur case law has recognized that experts in various fields may rely properly on a wide variety of sources and may employ a similarly wide choice of methodologies in developing an expert opinion.").

Further, Dr. Drnovsek identified fiber tracking algorithms analysis as a way to address certain deficiencies with FA quantitative analysis. (Dr. Drnovsek Report 4, Defs.' Exh. H, DE # 56–8.) In his affidavit, Dr. Benson stated that is not necessary. But Dr. Benson contends that this is not necessary because the problems addressed by this method are presented by scans that look at gray matter, not those that look only at white matter such as the ones he employs. (Dr. Benson Aff. ¶ 34.) The difference in opinion between the two experts is something that can be addressed at trial and does not make Dr. Benson's method so unreliable that his opinion need be excluded.

***6** As will be discussed, DTI and FA quantification based on comparative scans appear to be reliable methods for Dr. Benson to arrive at his expert opinion of both Ruppel's diagnosis of diffuse axonal injury and the cause of that injury. A district court has great latitude in determining not only how to measure the reliability of the proposed expert testimony but also whether the testimony is, in fact, reliable. *United States v. Pansier*, 576 F.3d 726, 737 (7th Cir.2009).

The Seventh Circuit has advised that "[t]o determine reliability, the court should consider the proposed expert's full range of experience and training, as well as the methodology used to arrive [at] a particular conclusion." *Id.* Defendants do not take issue with Dr. Benson's qualifications; they focus instead on the reliability of the methods he employed.

The Supreme Court, in *Daubert*, laid out four general criteria for determining the validity of an expert's methodology: (1) whether the theory has been or can be tested or falsified; (2) whether the theory or technique has been subject to peer review and publication; (3) whether there are known or potential rates of error with regard to specific techniques; and (4) whether the theory or approach has general acceptance. *Daubert*, 509 U.S. at 593–94. As "these factors do not establish a definitive checklist" for determining the reliability of expert testimony, the Seventh Circuit has described the *Daubert* test as a "non-exhaustive list of guideposts." *Trustees of Chi. Painters and Decorators Pension v. Royal Int'l Drywall & Decorating Inc.*, 493 F.3d 782, 787 (7th Cir.2007); *Am. Honda Motor Co., Inc. v. Allen*, 600 F.3d 813, 817 (7th Cir.2010). Further, the Seventh Circuit has employed other benchmarks which appear in the 2000 Advisory Committee's Notes to RULE 702 to gauge expert reliability, including whether the testimony relates to "matters growing naturally and directly out of research they have conducted independent of the litigation, or whether they have developed their opinions expressly for purposes of testifying"; "[w]hether the expert has adequately accounted for obvious alternative explanations"; and "[w]hether the expert is being as careful as he would be in his regular professional work outside his paid litigation consulting." *Id.* (alterations in *Allen*).

In this case, defendants argue that the DTI and FA quantification used by Dr. Benson are unreliable because 1) DTI is not generally accepted; 2) DTI cannot be tested 3) Dr. Benson has not considered alternative explanations for the comparatively decreased FA quantification found in the images; 4) Dr. Benson did not use proper methods and controls in his use of this imaging, especially considering that FA decreases with age; 5) Dr. Benson did not use the same level of intellectual rigor that is used by a regular expert in his field. (DE # 56 at 14.)

In response, the Ruppels argue that DTI is generally accepted in the relevant scientific community; DTI has been subjected to peer review and publication; DTI and FA quantification have low error rates; DTI and FA quantification was not developed for litigation; and DTI has been admitted by other courts. (DE # 57 at 20–23.) They also argue that defendants' experts lack the knowledge and qualifications to challenge the scientific reliability of DTI testing. (*Id.* at 25.) The court will now discuss the relevant factors in turn.

a. General acceptance of DTI

***7** The evidence shows that while DTI is a relatively new technology it is gaining general acceptance as a method for detecting TBI. First, as explained in further detail below, there have been numerous validation studies, published in peer reviewed journals, on the use of DTI to detect diffuse axonal injuries. (Dr. Benson Aff. ¶ 14.) Second, DTI is regularly used as a diagnostic tool at the Detroit Medical Center and at other locations throughout the country. (*Id.* ¶ 15.) Third, Dr. Benson, Dr. Pareigis, and Dr. Bradley Sewick, a neuropsychologist, all determined that DTI would be helpful in diagnosing Ruppel. (Dr. Bradley Sewick Aff. ¶ 10.) Fourth, the United States Army Telemedicine and Advanced Technology Research Command ("TATRC") sponsored a "Diffusion MRI TBI Roadmap Development Workshop" at which it was acknowledged: "DTI has detected abnormalities associated with brain trauma at several single centers." (Benson Aff. ¶ 4.) It was also stated that "the workshop seeks to identify and remove barriers to rapid translation of advanced diffusion MRI technology for TBI ... in order to expedite getting the benefits of diffusion MRI to reach those who need it most, espe-

cially injured soldiers and veterans." (*Id.*)

Fifth, in 2001, the Food and Drug Administration ("FDA") approved the product "Diffusion Tensor Imaging Option for MRI" for marketing as a Class II Special Control device. (Pl.'s Exh. 8, DE # 57–8.) Ruppel, citing to 21 U.S.C. § 360c(a)(3)(A), states that the FDA tested the software for safety and effectiveness before granting marketing permission. (DE # 57 at 21.) The letter from the FDA does not say this specifically. However, 21 U.S.C. § 360c(a)(3)(A) provides that approved Special Control devices are determined to be effective:

> on the basis of well-controlled investigations, including 1 or more clinical investigations where appropriate, by experts qualified by training and experience to evaluate the effectiveness of the device, from which investigations it can fairly and responsibly be concluded by qualified experts that the device will have the effect it purports or is represented to have under the conditions of use prescribed, recommended, or suggested in the labeling of the device.

So although the FDA letter itself does not address the effectiveness of DTI, but its approval for marketing by the FDA indicates that its effectiveness was determined pursuant to 21 U.S.C. § 360c(a)(3)(A). In fact, other courts that have found DTI to be a reliable method have noted that it is "FDA approved, peer reviewed and approved, and a commercially marketed modality which has been in clinical use for the evaluation of suspected head traumas including mild traumatic brain injury." *Hammar v. Sentinel Ins. Co., Ltd.*, No. 08–019984 at *2 (Fla.Cir.Ct.2010).

Sixth, Ruppel has pointed to several decisions in which trial court judges admitted DTI into evidence. *See e.g., Hammar*, No. 08–019984 at *2 (allowing DTI evidence to be admitted under the *Frye* standard); *Whilden v. Cline*, No. 08–cv–4210 (Col.Ct.Dist. May 10, 2010) (allowing an expert witness to rely on DTI evidence when testifying as to the diagnosis of mild TBI and its possible causation from an automobile accident as long as the expert's opinion was not based solely on DTI).

***8** On the other side, defendants' argument that DTI is not generally accepted is based primarily upon testimony that Dr. Benson provided in his deposition. (DE # 56 at 13 (citing Dr. Randall Benson Dep. 13, Defs.' Exh. F, DE # 56–6).) Defendants point to this portion of Dr. Benson's deposition:

> Q: I think at the beginning of your question you said some insurance companies would cover [DTI] and some wouldn't. Take your average hundred mild TBI patients, all things being equal, approximately how many of them after one or two regular MRIs showing no abnormalities would be able to get this more advanced MRI?
>
> A: I think very few, and the reason is that this technique that we're hoping will become a standard operating technique, it is clearly not something that is far enough along. I mean in terms of the commercialization of it, that insurance companies routinely will cover.
>
> Now having said that, we add these sequences onto standard sequences, and insurance companies do pay for it. But if a patient has already had one or two negative MRIs, I think its going to be, it is going to be very very difficult, you know, to convince the insurance company, which is why we're doing this work obviously.

(Dr. Benson Dep. 13–14.) This testimony focuses mostly on insurance companies' acceptance of DTI. Surely insurance companies' willingness to pay for a test is not dispositive of its reliability. Further, Dr. Benson also testified that some insurance companies would pay for DTI after an MRI showing no abnor-

mality and some would not because "that is just kind of a state of where we're at with insurance these days." (*Id.* at 12.) He did not say that insurance companies do not find DTI helpful, but only that they are reluctant to pay for it after a regular MRI shows no problems.

As shown above, DTI has been accepted within the medical community. It is regularly used at some hospitals even though it is not the regular standard of care at the average hospital. (*Id.* at 24.) Importantly, as discussed below, there are many articles published in peer-reviewed publications that cover the effectiveness of DTI in detecting mild TBI. All of the factors shown above weigh towards a finding that while DTI is a relatively new and developing technology, it is well on its way to gaining general acceptance in the scientific community as a tool for identifying mild TBI. Thus, the evidence shows that DTI and analysis of white matter in DTI images are generally accepted methods for determining mild TBI.

b. Peer review and publication

As of early 2010, there were 3,472 papers on DTI published in peer review journals. (Dr. Benson Aff. ¶ 17.) Eighty-three of these articles involved DTI in relation to TBI. (*Id.*) Of these 83 papers, a control group was used for the statistical analysis of 35 of them. (*Id.*) In the case that defendants rely upon to show the DTI has not been accepted by the courts, the trial judge determined that DTI could not be admitted to show mild traumatic brain injury in large part because the party moving to admit DTI evidence had not pointed to any articles showing that DTI was used for that purpose. *Bowles v. Pennington,* No. 06–cv–11030, at *3–4 (Col.Ct.Dist. Aug. 14, 2009). As just explained, that problem does not exist here because the Ruppels have pointed to many articles that discuss how DTI is effective in detecting mild brain injury. In fact, Dr. Benson's affidavit includes quotes from fourteen peer-reviewed articles that discuss how DTI can help detect TBI. (Dr. Benson Aff. ¶ 18.) Eleven of these excerpts specifically address the effectiveness of DTI in detecting mild TBI ("mTBI"). (*Id.*) Here is an example:

> ***9** Detection of ultrastructural damage by using DT imaging is a major advance in diagnostic imaging. Several studies have supported the capability of FA to help identify white matter abnormalities in patients with traumatic brain injury including mTBI. As confirmed by our findings, abnormal FA is detected even in the absence of other imaging abnormalities.

Michael Lipton, *Diffusion–Tensor Imaging Implicates Prefrontal Axonal Injury in Executive Function Impairment Following Very Mild Traumatic brain Injury,* RADIOLOGY, Sept. 2009, Vol. 252: No. 3. (Dr. Benson Aff. ¶ 18.f.) Another article stated, "Our study shows that DTI can be used to detect differences between patients with cognitive impairment after mild TBI and controls." Calvin Lo, *Diffusion Tensor Imaging Abnormalities in Patients with Mild Traumatic Brain Injury and Neurocognitive Impairment,* COMPUT ASSIST TOMOGR, March/April 2009, Vol. 33, No. 2. (Dr. Benson Aff. ¶ 18.i.) Thus, there are peer-reviewed articles on the effectiveness of DTI and FA quantification based on comparative DTI scans for detecting diffuse axonal brain injury. Accordingly, the concern that drove the judge's decision in *Bowles* does not exist here.

c. Ability of DTI and FA quantification to be tested and their error rate

As to the ability to test DTI and the FA quantification based on it and their reliability, defendants' main arguments are that decreased FA in DTI scans cannot be challenged in an objective sense and cannot be replicated.[FN3] (DE # 56 at 13.) However, the Ruppels have presented evidence that the DTI scan and resulting FA quantification analysis can be tested and replicated and that the error rate is not higher than other methods commonly relied upon such as MRIs. (Dr. Benson Aff. ¶¶ 34–36.) According to Dr. Benson, DTI has "good test retest reliability." (Dr. Benson Dep. 15.) He stated that DTI scans have shown high

reproducibility. (Dr. Benson Aff. ¶ 34.) Dr. Benson explained the numerous steps he took to minimize the error rates in his DTI analysis and he stated: "Statistically speaking, the clusters of abnormal voxels found in areas of Dale Ruppel's brain were there by chance is next to impossible." (Dr. Benson Aff. ¶ ¶ 29–32.) He also stated that the quantitative analysis of FA is reproducible. (*Id.* ¶ 34.)

> FN3. Dr. Drnovesk also concludes that Dr. Benson's study of Ruppel is flawed because the DTI scan was performed 27 months after the accident at issue and that decrease in FA caused by mild TBI is not detectable after three months from the date of the cause of an injury. (Dr. Drnovesk Report 5.) Defendants do not appear to address this conclusion in their motion or reply. Still, the court notes that Dr. Drnovesk's conclusion does not operate to block Dr. Benson's testimony on DTI and FA quantification from coming in all together. Rather it is an argument that defendants can raise at trial as to the weight that the fact-finder should afford to Dr. Benson's opinion.

As explained above, Ruppel has produced evidence that Dr. Benson's methods can be tested and that the error rate is not higher than that of other commonly used methods. While defendants' expert Dr. Drnovsek disagrees with Dr. Benson (Dr. Drnovsek Report 3), she does not have as much experience in this area as Dr. Benson. Dr. Benson is a behavioral neurologist who has been involved in research using advanced MRI methods for eighteen years. (Dr. Benson Aff. ¶ 4.) He has focused his research on TBI imaging for the past five years and has published a paper on how DTI scans of FA correlate with TBI severity. (*Id.*) On the other hand, Dr. Drnovsek, a neuroradiologist, does not do diffusion tensor imaging and before becoming involved in this case her only experience with DTI was a basic familiarity with the literature about DTI and attendance at conferences that "elaborate[d] on [DTI] application in different pathologies, including traumatic brain injury." (Dr. Valerie Drnovsek Dep. 16–17, Pl.'s Exh. 15, DE # 57–15.) She has not done any personal research into DTI. (*Id.* at 17.) Her criticism of Dr. Benson's methods was based on her reading of two articles on the subject. (*Id.* at 42.)

***10** In *Wagoner v. Schlumberger Tech. Corp.*, a proposed expert witness, a neuroradiologist, had never reviewed a DTI scan before analyzing one for the trial and had only read one article on DTI. No. 07–CV–244, 2008 U.S. Dist. LEXIS 118764, at *2, 2008 WL 5120750 (D. Wyo. June 20, 2008). The trial judge found that the witness did not have any special expertise on DTI and excluded any testimony from the expert about his opinion on the DTI scans. *Id.* Here, the Ruppels have not moved to exclude Dr. Drnovsek's testimony. However, Dr. Drnovsek, like the expert in *Wagoner*, has not been shown to have special expertise in DTI and Dr. Benson has been shown to have this expertise. Therefore, the court will not exclude Dr. Benson's testimony based on conflicting testimony from Dr. Drnovsek as to DTI's error rate, testability, and replicablity. This disagreement can be explored at trial.

d. Alternative explanations for the decreased white matter in the DTI images

Defendants argue that Dr. Benson should not be able to testify as to his determination that the DTI image indicated that Ruppel had diffuse axonal brain injury because it showed that Ruppel's white matter had decreased in comparison to scans done of control patients because Dr. Benson did not consider alternative explanations, primarily aging, for the decreased white matter. However, this argument is not supported by the evidence. Dr. Benson testified that while Ruppel was 46 at the time of his DTI scan and the mean age of the control group was the 32, the analysis was corrected to account for age. (Dr. Benson Dep. 65.) He also stated that the age effect on FA is well-known and easily accounted for. (Dr. Benson Aff. ¶ 28.) He stated that he normalized the results to

account for the effect of age. (Dr. Benson Dep. 36.) The Ruppels have also submitted a chart that shows the amount of FA in Ruppel's scan as compared to a group of 50 controls many of whom are his age or older. (DE # 58–1 at 18.) The effect of aging is certainly an issue that can be probed at trial, but it is not a basis for excluding Dr. Benson's opinion.

Defendants, pointing to Dr. Drnovsek's report, also argue that Dr. Benson did not account for alternative explanations such as the variations in FA in structures abutting the basal ganglia and thalamic nuclei. (Dr. Drnovsek Report 4.) However, Dr. Benson contends that these problems are presented by scans that look at gray matter, not those that look only at white matter such as the ones he employs. The difference in opinion between the two experts is something that can be addressed at trial and does not make Dr. Benson's method unreliable.

Further, defendants point to Dr. Benson's testimony that other diseases can affect FA quantification. (Dr. Benson Dep. 67–69.) However, Dr. Benson explains that many of these diseases are rare, and that some of the more common ones, such as stroke and MS, would also come up on a regular MRI scan if they would come up on a DTI scan. (*Id.* at 69.) Defendants also raise the issue that Ruppel's DTI scan could have been affected by the medications he was on. (Dr. Drnovsek Report 3.) This is an issue they can address during cross-examination.

***11** Defendants also point to Dr. Benson's testimony that "So obviously you're going to have variance, okay, with any type of measurement, there is error, there's a number of different sources, some physiologic, some machine, right, and in this case, age is a factor as well." (Dr. Benson Dep. 35.) Defendants present their argument that Dr. Benson attributed this error just to FA quantification, but it appears that he thinks these errors can accompany any type of measurement. He stated: "I am going to always let's say err[ ] on the side of respecting the lack of absolute certainty that we have in our field. I mean it is the nature of medicine, not just science." Dr. Benson also corrected his results for motion during the scan. (*Id.* at 68.) In any case, Dr. Benson's deposition and affidavit testimony show that he was aware of possible alternative explanations of Ruppel's decreased white matter and that both the method and Dr. Benson's application of the method accounted for these possibilities. His conclusion took into account alternative explanations for his results and that the only way to diagnose diffuse axonal injury with complete certainty is autopsy. (*Id.* at 66.) Therefore, the possibility of alternative explanations does not bar Dr. Benson's testimony; rather it goes toward the weight to be given to his opinion. *See e.g.*, *Cooper v. Carl A. Nelson & Co.*, 211 F.3d 1008, 1021 (7th Cir.2000).

b. Nature of Dr. Benson's opinion and how careful he was in reaching it

In this case, it appears that Dr. Benson's opinion grew naturally and directly out of the research that he has conducted independently of the litigation and he has been as careful as he would be in his regular professional work outside his paid litigation consulting. First, the evidence shows that DTI and FA quantification is a regular focus of Dr. Benson's work and research. He has focused on TBI imaging for five years at the MR Research Center at Detroit Medical Center. (Dr. Benson Aff. ¶ 4.) He is also an investigator on a fifteen-year project entitled "Utility of MRI Techniques in Prediction of TBI Outcome" funded through a grant by the National Institute on Disability and Rehabilitation Research. (*Id.* ¶ 2.) In 2007, he published an article entitled *Global White Matter Analysis of Diffusion Tensor Images of Injury Severity in Traumatic Brain Injury* in the JOURNAL OF NEUROTRAUMA . (*Id.* ¶ 3.) In 2010, he testified before the United States House Judiciary about how DTI and other advanced imaging methods would improve the diagnosis and management of concussions in sports. (*Id.* ¶ 2.) Thus, the evidence shows that Dr. Benson regularly researches about and uses DTI and FA quantification to detect TBI. This is not a

method or area of research that he has adopted just for litigation. It appears that as the Ruppels' retained expert, he only applied his methods to Ruppel and reached his opinion because of his involvement in this litigation. However, because the methods he employed grew out of and is consistent with his regular work, Dr. Benson's opinion as to Ruppel appears reliable.

***12** Second, without pointing to any evidence, defendants accuse Dr. Benson of not using "the same level of intellectual vigor that characterizes the practice of an expert in the regular field." However, Dr. Benson's expert report, deposition, and affidavit do not show that he was not careful in reaching his conclusion or that he lacked intellectual vigor. Thus, there is no evidence to show that his opinion should not be admitted on this basis. Defendants can use cross-examination and their own witnesses's testimony to raise at trial the issue of the level of intellectual vigor that Dr. Benson employed.

Overall it is important to note that DTI is just one component of Dr. Benson's diagnosis of diffuse axonal injury for Ruppel. In *Whilden*, a Colorado state trial court found that an expert could base his opinion on DTI as long as he also considered the patient's history. No. 08–cv–4210 at 4 (allowing an expert witness to rely on DTI evidence when testifying as to the diagnosis of mTBI and its possible causation from an automobile accident as long as the expert's opinion was not based solely on DTI). Here, Dr. Benson's opinion was based on four components: the patient's history, the neurologic examination of the patient, the patient's neuropsychological results, and the patient's brain imaging including DTI. (Dr. Benson Dep. 69.) Dr. Benson's clinical assessment was based on medically accepted neurological and mental status examination techniques. (Dr. Benson Aff. ¶ 8.) In his affidavit, Dr. Benson stated:

> While DTI itself cannot diagnose the cause of white matter damage, the history of the motor vehicle accident as described by Dale Ruppel and medical records reviewed provide a solid basis to conclude that the damage shown on diffusion tensor imaging using fractional anisotrophy was caused by the motor vehicle collision of January 8, 2008.

(*Id.* ¶ 33.) Thus, like the expert in *Whilden*, Dr. Benson did not use DTI alone to diagnose diffuse axonal injury. In sum, DTI and comparative FA quantification based on DTI images are reliable methods and Dr. Benson's opinion will not be excluded under RULE 702 and *Daubert.*

2. Wording of Dr. Benson's opinion

Defendants argue that Dr. Benson's opinion is invalid because he says that the evidence "suggests" that Ruppel has a diffuse axonal brain injury and that it was caused by the accident. (DE # 56 at 10–11.) It seems that this argument goes to whether Dr. Benson's testimony is relevant and whether it would assist the trier of fact. Defendants argument appears to be that Ruppel can only present evidence of his injury if he has evidence that shows with one hundred percent certainty that he has a diffuse axonal brain injury. This is not the case. *Daubert*, 509 U.S. at 590; *United States v. Cyphers*, 553 F.2d 1064, 1072–73 (7th Cir.1977) (stating that there is no requirement that "an expert's opinion testimony must be expressed in terms of a reasonable scientific certainty in order to be admissible" and that the Seventh Circuit "adheres to the rule that an expert's lack of absolute certainty goes to the weight of his testimony, not to its admissibility"). The Seventh Circuit has stated, "we do not require utter certainty in medical opinions, nor would we expect dogmatic diagnoses from a careful scientist." *Amax Coal Co. v. Beasley*, 957 F.2d 324, 328 (7th Cir.1992).

***13** Indeed, courts regularly admit opinion evidence that falls short of a certain conclusion. *See e.g.*, *Coachmen Indus., Inc. v. Kemlite*, 3:06–cv–160, 2008 WL 4858385, at *8 (N.D.Ind. Nov.10, 2008) (admitting an expert's testimony that "specific changes made

to the MA resin values were 'most likely' responsible for the distortions"); *Hardiman v. Davita Inc.*, No. 2:05–cv–262, 2007 WL 1395568, at *6 (N.D.Ind. May 10, 2007) (finding that an expert's opinion that there was a 95% probability of causation was relevant and admissible); *Troutner v. Marten Trans., Ltd.*, No. 2:05–cv–40, 2006 WL 3523542, at *4 (N.D.Ind. Dec.5, 2006) (admitting an expert's testimony when the conclusion in his expert report was that inadequate maintenance was "the most likely root cause of the failure and injury to" the plaintiff). Further, an expert may meet *Daubert's* relevancy requirement by offering a "hypothetical explanation of the possible or probable causes of an event [that] would aid the jury in its deliberations." *Smith*, 215 F.3d at 719.

In the summary of findings section of his report, Dr. Benson stated that DTI revealed a low FA in the white matter regions of Ruppel's brain "suggesting axonal injury from trauma." (Dr. Randall Benson, "Report of Findings of TBI Research Protocal," Defs.' Exh. I, DE # 56–9.) However, Dr. Benson did not only use the word "suggest" in providing his opinion. He also stated:

> The absence of focal injury (contusion) and the presence of bilaterally symmetric axonal injury to deep white matter structures suggests that the mechanism of injury was acceleration/deceleration rather than direct impact to the skull. His history of motor vehicle accident is consistent with the findings on his MRI study.

(*Id.*) Thus this excerpt of his report, by stating that axonal injury to the white matter of Ruppel's brain was present, more definitively stated Ruppel's injury. Also, in his report Dr. Benson wrote that Ruppel "appears to have suffered a close head injury as a result of being rear-ended." (*Id.*)

Further, in his deposition, Dr. Benson explained that while he used the word "suggest" in his report, at the time he "really felt strongly that all the evidence pointed to diffuse axonal injury ." (Dr. Benson Dep. 67.) Dr. Benson's "certainty is an issue for the jury and does not affect admissibility." *Stutzman v. CRST, Inc.*, 997 F.2d 291, 296 (7th Cir.1993). Thus under federal evidentiary rules, Dr. Benson's opinion may be admitted under RULE 702. Importantly, Dr. Benson's language in presenting his opinion does not render it inadmissible when it is based on reliable methods. The Seventh Circuit has concluded that "the Federal Rules do not contain any threshold level of certainty requirement. As long as a medical expert's qualifications are proper and the expert relies on appropriate types of information under RULE 703, the district court does not abuse its discretion by admitting the medical expert's testimony." *Id.* Dr. Benson's testimony is not speculation because, as determined above, he used scientifically reliable methods to reach his conclusion.

***14** In sum, defendants' motion to exclude Dr. Benson's opinion as to diffuse axonal injury will be denied. Defendants' primary arguments for exclusion of Dr. Benson's testimony were his reliance on DTI to reach his result and his use of the word "suggest" for his diagnosis. As discussed above, DTI is a reliable method especially when used in conjunction with the other medically accepted methods relied upon by Dr. Benson. Beyond these two issues, defendants have not questioned Dr. Benson's qualifications to testify as to Ruppel's diagnosis and its causation and he appears qualified to do so. (*See* Dr. Benson Aff. ¶ 19; Dr. Benson Curriculum Vitae, DE # 58–1.) Dr. Benson may testify as to Dr. Ruppel's diagnosis of diffuse axonal injury and as to its causation.

II. SUMMARY JUDGMENT

Summary judgment should be granted "if the movant shows that there is no genuine dispute as to any material fact and the movant is entitled to a judgment as a matter of law." FED. R. CIV. P. 56(a). The party seeking summary judgment "bears the initial responsibility of informing the district court of the basis for its motion, and identifying" those materials

listed in RULE 56(c) which "demonstrate the absence of a genuine issue of material fact." *Celotex Corp. v. Catrett,* 477 U.S. 317, 323, 106 S.Ct. 2548, 91 L.Ed.2d 265 (1986).

Once the moving party has met its burden, the nonmovant may not rest upon mere allegations. Instead, "[t]o successfully oppose a motion for summary judgment, the nonmoving party must come forward with specific facts demonstrating that there is a genuine issue for trial." *Trask–Morton v. Motel 6 Operating L.P.,* 534 F.3d 672, 677 (7th Cir.2008). "It is not the duty of the court to scour the record in search of evidence to defeat a motion for summary judgment; rather, the nonmoving party bears the responsibility of identifying the evidence upon which he relies." *Harney v. Speedway SuperAmerica, LLC,* 526 F.3d 1099, 1104 (7th Cir.2008). Furthermore, when evaluating a motion for summary judgment, the court views the record and makes all reasonable inferences in a light most favorable to the nonmovant. *Popovits,* 185 F.3d at 731. If the non-moving party cannot establish an essential element of its claim, RULE 56(a) requires entry of summary judgment for that claim. *Massey v. Johnson,* 457 F.3d 711, 716 (7th Cir.2006) (citing *Celotex,* 477 U.S. at 322–23).

Defendants' summary judgment argument is that because all evidence of Ruppel's diagnosis of diffuse axonal injury and its causation are excluded under *Daubert* or for failure to comply with FEDERAL RULE OF CIVIL PROCEDURE 26(a)(2), he has no evidence to survive a motion for summary judgment.

The court will now address defendants' arguments related to FEDERAL RULE OF CIVIL PROCEDURE 26(a)(2). In their response to defendants' motion for summary judgment, the Ruppels presented affidavits of four physicians, Dr. Robert Ward, Dr. Bradley Sewick, Dr. Patrick Casey, and Dr. Pareigis, who treated Ruppel. (Pls.' Exhs. 3, 5, 6, DE57–3, 57–5, 57–6.) In reply, defendants argue that the first three physicians' proposed testimony, as set forth in their affidavits, extends beyond what the plaintiffs had outlined in their reports and summaries pursuant to FEDERAL RULE OF CIVIL PROCEDURE 26(a)(2). Defendants, citing to *Doe v. Johnson,* 52 F.3d 1448, 1464 (7th Cir.1995), appear to be arguing that these doctors' testimony should be limited to the statements made in their medical records because anything beyond that was not disclosed under RULE 26 and should be excluded under RULE 37.

***15** RULE 26.2 of the LOCAL RULES OF THE UNITED STATES DISTRICT COURT FOR THE NORTHERN DISTRICT OF INDIANA provides that if a party seeks relief under RULE 37, copies of the portions of the disclosures in dispute "shall be filed with the court contemporaneously with any motion filed under" that RULE. Defendants did not file a copy of plaintiffs' RULE 26 disclosures with their response. While this may not have been required since they did not move under RULE 37 separately, it certainly would have assisted the court in evaluating their argument. Instead defendants argue that Dr. Ward's, Dr. Casey's, and Dr. Sewick's testimony is inconsistent with the statements made in their medical records. In a sur-reply, plaintiffs contend that Dr. Ward, Dr. Casey, and Dr. Sewick, as well as Dr. Pareigis, were "properly disclosed" in their RULE 26 disclosures and their medical charts were provided to defendants with updates sent as Ruppel's treatment continued. (DE # 62 at 2.) They state that Dr. Ward, Dr. Casey, Dr. Sewick, and Dr. Pareigis are all treating physicians and none of them were retained or specially employed for this litigation. (*Id.*)

First, it appears that these witnesses were only required to give statements under RULE 26(a)(2)(C) and not expert reports under RULE 26(a)(2)(B). RULE 26(a)(2)(B) states that the disclosure of expert testimony must be accompanied by a written report when the witness is "one retained or specially employed in the case or one whose duties as the party's employee regularly involve giving expert testimony." Effective December 1, 2010, RULE 26 was amended

to add section 26(a)(2)(C). This section provides that expert witnesses who are not required to submit a report under 26(a)(2)(B) must submit a statement that provides a summary of the facts and opinions to which the witness expects to testify. The commentary to this amendment states that it will frequently apply to "physicians or other health care professionals." They also provide that under this subsection "[c]ourts must take care against requiring undue detail, keeping in mind that these witnesses have not been specially retained and may not be as responsive to counsel as those who have." Defendants do not argue that Dr. Ward, Dr. Pareigis, Dr. Sewick and Dr. Casey were not Ruppel's treating physicians, or more importantly, that they were specially retained or employed for this litigation. Thus, they were only required to comply with RULE 26(a)(2)(C). *See Coleman v. Am. Family Mut. Ins. Co.* No. 2:10–cv–167, 2011 WL 2173674, at *4 (N.D.Ind. June 2, 2011).

Second, the court has no reason to think that the proposed testimony is so inconsistent with the RULE 26(a)(2)(C) disclosures that it should be struck down under RULE 37. Defendants have not pointed to plaintiffs' RULE 26(a)(2)(C) disclosures, so the court cannot compare them to the proposed testimony and has no basis for excluding the testimony for noncompliance with RULE 26. Defendants argue that Dr. Ward, Dr. Pareigis, and Dr. Sewick cannot testify that Ruppel has diffuse axonal injury because in their medical records for Ruppel they only stated that he had closed head injury. Defendants, without pointing to any evidence from their expert medical witnesses or otherwise, assert that what the physicians have done is similar to "a doctor who makes a diagnosis of a broken bone, tenders x-rays and information relative only to a broken foot for 2 or 3 years, then later argues that the diagnosis should have covered diagnosis of a broken hand as well because they are both broken bones." (DE # 61 at 2.)

***16** In contrast, all five of plaintiffs' expert witness physicians offer testimony that a diffuse axonal injury is a type of closed head injury. (Dr. Robert C. Ward. Aff. ¶ 4, Pls.' Exh. 3, DE # 57–3; Dr. Pareigis Aff. ¶ 7; Dr. Patrick Casey Aff. ¶¶ 5, 8, Pls.' Exh. 5, DE # 57–5; Dr. Bradley Sewick Aff. ¶ 5–6, Pls.' Exh. 6, DE # 57–6; Dr. Benson Aff. ¶ 5). Dr. Sewick's explanation is representative: "A diffuse axonal brain injury is often caused by a closed head injury or traumatic brain injury. A diagnosis of closed head injury and traumatic brain injury without evidence of focal injury is suggestive of diffuse axonal injury." (Dr. Sewick Aff. ¶ 5.) Accordingly, the difference between statements of closed head injury in the medical records and a diagnosis of diffuse axonal injury may not be as stark as defendants suggest. Certainly, it does not appear to provide a basis to exclude the testimony under RULE 37. Rather, this appears to be an argument that defendants can delve into during cross examination at trial. Accordingly, these witnesses can offer testimony related to diffuse axonal injury at trial.

In evaluating whether the Ruppels have sufficient evidence as to his claim of diffuse axonal injury to allow it to survive summary judgment, the court has one remaining, and familiar, argument to address. As discussed above, defendants seem to argue that Dr. Benson's opinions as to the diagnosis and causation of diffuse axonal injury will not help Ruppel survive summary judgment because Dr. Benson uses the word "suggest." While the court has already discussed that this opinion is admissible it must now address whether, under Indiana law, which applies to the substantive law questions in this case, Dr. Benson's testimony has enough probative value that Ruppel can use it towards his burden of proof for causation.

As defendants point out, in Indiana, "[w]hen the issue of cause is not within the understanding of a lay person, testimony of an expert witness on the issue is necessary." *Daub v. Daub*, 629 N.E.2d 873, 877–78 (Ind.Ct.App.1994). To have probative value, the testimony must go beyond speculation and mere possibility. *Id.* When evaluating an expert's opinion, Indiana courts tend to look at whether the expert can tes-

tify to a reasonable degree of medical certainty, but even an opinion that something is "possible" may be admitted if presented with other evidence. *Topp v. Leffers,* 838 N.E.2d 1027, 1033 (Ind.Ct.App.2005); *Colaw v. Nicholson,* 450 N.E.2d 1023, 1030 (Ind.Ct.App.1983) ("[E]xpert medical opinion couched in terms less than that of a reasonable degree of medical certainty; such as 'possible,' 'probable,' or 'reasonably certain,' are admissible and do have probative value. However, such medical testimony standing alone, unsupported by other evidence, is not sufficient to support a verdict.") Therefore, an opinion does not need to be stated in terms of "medical certainty," but to be admitted alone, it must be more conclusive than stating a "possibility." *Longardner v. Citizens Gas & Coke Util.,* No. 49A02–511, 2006 WL 3230303, at *7 (Ind.Ct.App. Nov.8, 2006); *Hardiman,* 2007 WL 1395568, at *15.

***17** Here, Dr. Benson's report stated that Ruppel "appears to have suffered a close head injury as a result of being rear-ended." (Dr. Benson Report.) He also stated in his deposition that although he used the word "suggests" in his report he "really felt strongly that all the evidence pointed to diffuse axonal injury." (Dr. Benson Dep. 67.) Further, his opinion was based on scientifically reliable methods. He based his opinion on Ruppel's history, his neurologic examination of Ruppel, Ruppel's neuropsychological results, and his analysis of Ruppel's brain imaging including DTI. Dr. Benson's opinion is based on more than speculation and creates an issue of material fact as to both the diagnosis and causation of diffuse axonal injury. *Hardiman,* 2007 WL 1395568, at *17.

Even if Dr. Benson's testimony can not be admitted alone, there is other evidence of Ruppel's diffuse axonal injury. Dr. Pareigis wrote in her initial evaluation of Ruppel on March 28, 2008, that her impression was that Ruppel had "[c]losed head injury with probable diffuse axonal injury." (Physicians Center of Physical Medicine's Medical Records for Dale Ruppel, Defs.' Exh. C, DE # 56–3 at 32.) Dr. Pareigis and the three other treating physicians all indicate that they would testify as to Ruppel's diffuse axonal injury and its causation. Defendants own expert, Dr. Peter Carney has diagnosed Ruppel with post-concussion syndrome which appears to be related to closed head injury. (Dr. Peter Carney Report Sections D and F2.1, Pl.'s Exh. 17,[FN4] DE # 64–1.) So the Ruppels have sufficient evidence to create a genuine factual dispute as to whether Ruppel suffered diffuse axonal injury and whether that injury was caused by the accident with Kucanin.

FN4. The Ruppels cite to and quote from this exhibit in their summary judgment response, but it was inadvertently omitted from that filing. The Ruppels have moved for leave to file this exhibit now. (DE # 64.) The report is from defendants' expert witness, so they have had access to it. Therefore, the motion is **GRANTED,** and the court had considered the parts of the report and deposition that were relied on in plaintiffs' response.

In conclusion, for the foregoing reasons defendants' motion to exclude evidence and motion for summary judgment (DE54–55) are **DENIED.**

SO ORDERED.

N.D.Ind.,2011.
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H

NOTE: THIS OPINION WILL NOT APPEAR IN A PRINTED VOLUME. THE DISPOSITION WILL APPEAR IN A REPORTER TABLE.

Supreme Court, New York County, New York.
Salvatore LAMASA and Ana G. Lamasa, Plaintiffs,
v.
John K. BACHMAN, Defendant.

No. 129996/93.
April 13, 2005.

MARTIN SHULMAN, J.

***1** Defendant, John K. Bachman ("defendant" or "Bachman"), moves for an order seeking the following relief in relation to a jury verdict rendered on June 7, 2004 [FN1]:

> FN1. Normally, a motion to challenge a jury verdict pursuant to CPLR § 4404(a) is governed by the 15–day time limit of CPLR § 4405. This Court permitted the parties to stipulate to extend their time to present written arguments. *See*, "(CPLR 2004; see, 4 Weinstein–Korn–Miller, N.Y. Civ Prac para. 4405.05) ..." *Brown v. Two Exchange Plaza Partners*, 146 A.D.2d 129, 539 N.Y.S.2d 889 (1st Dept.,1989).

1) dismissing the complaint; 2) setting aside the jury verdict as against the weight of the evidence (CPLR § 4404[a]); 3) alternatively, seeking remittitur; 4) seeking defense costs and fees as against the plaintiffs, Salvatore LaMasa and Ana G. LaMasa (where appropriate: "plaintiff", "Salvatore" or "plaintiffs") in connection with plaintiffs' counsel's "withdrawal of his proffer of PET and QEEG evidence following the ruling of the Court precluding said evidence during the trial and for costs in connection with plaintiff's egregious discovery abuses." Plaintiffs oppose the motion and cross-move for additur.

The motion and cross-motion are consolidated for disposition.

Salvatore initiated what had become a protracted action against the defendant in November, 1993 for injuries he purportedly sustained as the driver of the stationary, front vehicle Bachman rear-ended during the early morning hours of November 25, 1992 at the intersection of Delancey and Clinton Streets just prior to entering the Williamsburg Bridge (the "Collision"). After being marked off the calendar at least three times, this matter was restored to the trial calendar and thereafter transferred to the New York County Civil Court on November 10, 1999 (see, CPLR § 325[d]). After languishing for four years, the parties appeared at several pre-trial conferences and the case was eventually referred to the Supervising Judge of that court.[FN2]

> FN2. Due to the confusing procedural posture of the case and an inordinate number of complex *in limine* motions/issues as well as the potential value of the case (based upon a prima facie showing), the parties' counsel concurred that the matter should be re-transferred to the Supreme Court and this Court agreed to preside over the jury trial.

Jury selection began on May 4, 2004 and the trial ended on June 7, 2004. As noted on the Jury Verdict Sheet (Exhibit A to Bachman Motion), five out of the six members of the jury reached an agreement and preliminarily reported that defendant's negligence in causing the rear-end collision was a substantial factor in causing Salvatore's injuries. The same five members of the jury further reported that as a result of the Collision, plaintiff suffered a serious injury under the No–Fault Law, Insurance Law § 5102(d) (see, Jury Question Nos.: 1A–1C). Salvatore was then awarded the following damages:

a) Past pain and suffering	$240,000
b) Future pain and suffering	$400,000 (over 20 years)
c) Past Lost Earnings	$460,713
d) Future lost earnings	$774,892 (over 13 years)
e) Past medical expenses	$ 40,768

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f) Future Medical expenses	$ 95,040 (over 20 years)
g) Past loss of medical insurance	$ 38,985
h) Future loss of medical insurance	$ 95,840 (over 13 years)
i) Future loss of social security	$122,273 (over 7 years)

The jury also awarded Salvatore's spouse, Ana La-Masa, $250,000 for past loss of services (on her derivative claim for loss of consortium) and awarded an identical sum for future loss of services (the latter to cover a period of 20 years).

It should be readily apparent that both parties had a full and fair opportunity to argue and brief the court (where necessary) and make their record, *inter alia*, concerning their respective *in limine* motions, evidentiary issues and procedural and substantive trial issues (e.g., the proper jury charges, verdict interrogatories, etc.). While this Court granted Bachman's counsel leave to make this post-verdict motion, nonetheless, to avoid any redundancy, this Court expressed an unwillingness to entertain any application addressing the liability issues and/or the varied evidentiary rulings made prior to and during the jury trial. However, this Court stated it would consider whether the jury awards were excessive and unreasonable (CPLR § 5501[c]). Still, defendant took advantage of his right to move under CPLR § 4404(a) and "re-argued" almost every one his overruled objections and denied motions duly made on the record during the course of the trial and duly preserved for a potential appeal.In its post-verdict motion, defendant's counsel argues that: Salvatore's proof of injuries never met the statutory threshold to constitute a serious injury (i.e., no loss of consciousness and no complaints of pain and/or other physical or cognitive disabilities at the time of the Collision made to the police or his late brother-in-law, no loss of ambulation, no emergency room or hospital admission at the time of the Collision, no initial complaints of headaches, depression and/or anxiety at or close in time to the Collision, a normal neurological examination seven weeks post-Collision, no evidence of either temporary or permanent traumatic brain injury ("TBI") at or close in time to the Collision and no objective findings of injuries to Salvatore's neck and back); plaintiff's proof was insufficient to show a causal connection between the Collision and Salvatore's alleged injuries (*viz.*, all of plaintiff's experts failed to opine on causation and any and all purported positive findings of TBI, post-traumatic stress disorder ["PTSD"] and neck and back injuries were reported years after the collision by medical experts retained by plaintiffs' counsel solely for trial); and plaintiffs' discovery abuses warranted the extreme sanction of dismissal of the plaintiffs' complaint.

***2** Defendant's post-verdict motion further took issue with various court rulings he deemed erroneous such as permitting plaintiff's expert neuroradiologist, Dr. Michael Lipton, to testify with respect to an innovative MRI modality utilizing Diffusion Tensor Imaging ("DTI") [FN3] as this modality is not generally accepted in the field of radiology or neuroradiology to diagnose TBI or diffuse axonal injury; precluding defendant's expert neurologist from testifying concerning Evoked Potential testing [FN4] which plaintiff argued was not addressed in defendant's expert witness disclosure notice; granting plaintiff a directed verdict on the issue of negligence; overruling certain objections to references about insurance made by various plaintiffs' witnesses; denying defendant's request for a missing witness charge with respect to various witnesses such as Dr. Wiseman (pain management specialist who treated Salvatore), Dr Leo J. Shea III (psychologist who treated Salvatore) and Mariusz Ziejewski, Ph.D. (accident reconstruction engineer); granting plaintiffs' counsel's application to modify certain no-fault interrogatories on the verdict sheet to eliminate the phrase, "[a]s a result of the accident" but otherwise accurately reciting the text of these no-fault questions in accordance with PJI 2:88E, 2:88F and 2:88G; and granting plaintiffs' counsel application to amend certain damages questions on the verdict sheet after completion of instructions to the jury to include a claim for loss of past and future medical insurance and future loss of social security benefits (or payments) and furnishing the jury with a supplementary charge with respect thereto.

FN3. DTI is an imaging technique used to study the random motion of hydrogen atoms within water molecules in biological tissue (e.g., brain white matter) and spatially map this diffusion of water molecules, *in vivo*. DTI provides anatomical information about tissue structure and composition. Changes in these tissue properties can often be correlated with processes that occur, among other causes, as a result of disease and trauma.

FN4. Evoked Potentials sometimes called

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evoked responses are tests that record the brain's responses to sound, touch and light. These tests help to evaluate a number of neurological conditions.

After the foregoing challenges, Bachman's motion then raises the issue of remittitur urging the court to either set aside or reduce the jury awards for past lost earnings ($460,713) and future lost earnings ($774,892) [FN5], reduce the jury award for past medical expenses from $40,780 to $25,000, set aside the jury award for past and future medical insurance as being duplicative, set aside the jury award for future loss of social security retirement benefits as being totally speculative or alternatively reduce the $122,273 award to $80,700 and reduce the jury awards for loss of past and future services to Ana LaMasa from $500,000 to $50,000.

FN5. Specifically, defendant contends that Salvatore's pre-accident employment history reflects a patchwork of short-term jobs, that plaintiff's most recent employment before the accident at Ogden Allied was only for two and a half years, that Salvatore intended to leave Ogden Allied to become a Con Edison meter reader rendering plaintiff's expert economist's projections and calculations uncertain and speculative, that the calculation of the past and future lost earnings on an annualized basis erroneously utilized an increase rate of 3.5% rather than the union contract increase rate, that the economist failed to consider plaintiff's pre-accident health condition (i.e., scoliosis and degenerative disc disease), that the jury ignored testimonial evidence proffered by Dr. Remling, Salvatore's treating chiropractor, to the effect that plaintiff could return to work at a less demanding job or seek part time work, and that plaintiff's expert recognized that the rate of increase for future lost earnings could have been 3.5% rather than 4.5% justifying a reduction of this award by approximately $50,000 or $60,000.

Finally, due to plaintiff's purportedly frivolous efforts to seek the admission of QEEG [FN6] and PET scan [FN7] evidence, Bachman should be awarded attorney's fees pursuant to 22 NYCRR § 130–1.1 as well as defense expert witness expenses totaling approximately $50,000.

FN6. EEG is the recording of electrical patterns at the scalp's surface showing cortical electrical activity or brain waves. This recording is called an electroencephalograph, commonly referred to as an EEG. As a diagnostic tool, Quantitative EEG or QEEG provides a digital recording of the EEG which is apparently utilized to perform a comparative analysis of many EEG tracings of a patient suffering from brain disease or trauma against a normative data base of EEG tracings.

FN7. Positron Emission Tomography (“PET”) is a medical imaging technique which scans a body's chemistry and function to detect cancer, Alzheimer's and other medical conditions.

Plaintiff's cross-motion seeks additur and through the following arguments tells a different story:

Testimonial and documentary evidence presented before the jury preponderated in favor of Salvatore establishing that he suffered serious injury (Insurance Law § 5102) including, but not limited to, neck and back injury, TBI [FN8], post-traumatic stress disorder (“PTSD” [FN9]) and a non-permanent, medically determined injury, *viz.*, non-performance of customary and daily activities for 90 of 180 days after the Collision. Each of these conditions standing alone, plaintiffs argue, would satisfy the statutory serious injury threshold;

FN8. Plaintiffs contend that treating specialists Dr. Lewis Weiner (Salvatore's treating neurologist), Dr. Steven Stein (neuropsychologist), Dr, Daniel Kuhn (Salvatore's treating psychiatrist) and Dr. Joshua Greenspan (pain management specialist), Dr. Rachel Yehuda (neuroendocrinologist/psychologist) and experts Dr. Nils Varney (neuropsychologist) and Dr. Lipton jointly and severally opined that LaMasa suffered TBI as a result of the Collision. Their findings, impressions and conclusions, counsel argues, were based on hundreds of clinical examinations performed and duly reported, treatment regimens (i.e, series of drug treatments administered for over 12 years, all proven unsuccessful), medically accepted batteries of neuropsychological tests, MRI and/or DTI studies (the latter imaging studies revealed anatomical damage such as frontal lobe, hippocampus and para hippocampal atrophy and hemocitarin residue [from internal bleeding] consistent with frontal lobe injury).

FN9. Plaintiffs similarly contend that the severity of Salvatore's PTSD defies text book analysis.

> Salvatore's counsel, drawing from Dr. Yehuda's testimony, starkly captures a singular feature of what this specialist diagnosed as one her worse cases of this disorder: "[A]s a result of the immense psychological barriers inflicted by his PTSD, LaMasa remains psychologically frozen in time. He really has no present or future, since his PTSD holds him captive in a perpetual state of fear and terror, stuck in the moments surrounding the [Collision] ..." (Flomenhaft Aff. In support of Cross–Motion at ¶ 37 paraphrasing from the Yehuda trial transcript at pp. 16 and 42–45).

***3** Unrefuted testimonial and documentary evidence presented before the jury established that as a result of the Collision, Salvatore suffered, and continues to suffer, from panic disorder, severe depression accompanied by suicidal ideation and bouts of violence, electrical dysfunction of the brain, epilepsy, chronic severe headaches, sleep cycle disorder/insomnia [FN10];

> FN10. Studies done at Mt. Sinai Medical Center Sleep Laboratory revealed "abysmally abnormal qualities in Salvatore's sleep cycles and sleep oxygenation." (Flomenhaft Aff. in support of Cross–Motion at ¶ 32).

Defendant unnecessarily reiterates his objections to the many discovery issues fully argued and briefed prior to and during the trial, which the court ruled upon on the record [FN11] and requires no serious rebuttal. Moreover, defendant conveniently overlooked his counsel's own discovery "abuses" during the course of the trial;

> FN11. To illustrate, plaintiff's counsel acknowledged defendant's understandable concern about the "eleventh hour" proffer of Grahme Fisher, an accident reconstruction specialist. Exercising its discretion to ameliorate any perceived prejudice and surprise, this Court afforded defendant's counsel ample opportunity to depose Mr. Fisher during the course of the trial and obtain all relevant data he relied upon to not only conduct effective cross-examination, but also to furnish an appropriate defense to the effect that the Collision was low-impact in nature and incapable of causing the mixed bag of injuries Salvatore claims to have suffered therefrom. In this context, plaintiffs' counsel retorted that the court ruling precluding defendant's neurologist from testifying about Evoked Potentials testing was proper because the relevant CPLR § 3101(d) notice made no mention of this subject for testimony.

References to the word, "insurance", during the testimony of some of plaintiffs' witnesses were benign in context and non-prejudicial as most of the references to insurance were made in the context of discussing the payment of plaintiff's medical bills and did not warrant a mistrial;

This Court correctly granted plaintiffs a directed verdict on the issue of negligence, correctly denied defendant's request for a missing witness charge, vis-a-vis, Drs. Weissman,, Shea and Ziejewski; correctly permitted the semantic changes to the no-fault interrogatories eliminating the introductory phrase, "[a]s a result of the accident", while retaining the text of each question in accordance with the PJI. After determining if plaintiff suffered a serious injury by responding affirmatively to the three no-fault questions, the jury properly determined the issue of causation by answering Question No.2, namely, "Was the collision involving the plaintiff and defendant a substantial factor in causing any of the injuries alleged by plaintiff?" (Exhibit A to Bachman Motion at p. 2)

Contrary to defendant's confusing assertions, the jury awards for past and future medical insurance costs were not duplicative of the awards for medical expenses, but rather awards for loss of income, that is to say, the replacement costs of heath insurance Salvatore ostensibly would have to purchase in lieu of free union health care coverage he would have otherwise received had he continued working at Ogden Allied (Exhibit B–4 to Bachman Motion; Leiken trial transcript at pp. 24–30) [FN12];

> FN12. In explaining his calculation of this loss, the expert economist determined an annualized cost of health insurance for an individual to be $5000 from 1995 (after the Collision, Salvatore's union continued to provide him with health insurance coverage for a few years) through age 65 and factored in an annual 6% increase thereto for a total cost of $134, 796 (past medical insurance cost of $38,985 and future medical insurance cost of $95,840).

Dr. Leiken similarly projected the loss of social security retirement benefits as an additional component of lost income to be $170,000 (see, Exhibit B–4 to Bachman

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motion at pp. 26–30) and the jury further reduced this sum to $122,273 over a seven year period. Defendant's counsel blurs this item of income loss with Bachman's right to pursue adjustments of the judgment at a post-verdict collateral source hearing;

Without proffering any economist to refute Dr. Leiken's assumptions, calculations and projections on behalf of plaintiffs, defendant's challenges to the past and future lost earnings awards rest on a selective and skewed analysis of the testimony, expert and other [FN13], thus, the jury awards were fair and reasonable;

> FN13. Counsel contends it was reasonable for Dr. Leiken to assume that LaMasa would have remained at Ogden Allied, because the Con Edison position, if taken, would have been in addition to his porter work at New York University. Counsel further argues that LaMasa's work history reflected plaintiff's ongoing desire to work regularly, that no part time work was available after the Collision and that even assuming some incremental improvement of his neck and back through chiropractic treatment, LaMasa still suffered from TBI and its concomitant psychiatric problems rendering him disabled from the time of the Collision.

***4** Plaintiffs agree that the past medical expense award should be reduced from $40,768 to $25,000 based upon the evidence of record; and

The aggregate award of $500,000 to Ana LaMasa for loss of services was fair and reasonable based upon her credible testimony (Mrs. LaMasa had to replace Salvatore as the head of the household raising their two sons and constantly had to care for her husband since the Collision and must continue to do so for the rest of his life).

Counsel's cross-motion further addressed the mean-spirited nature of defendant requesting costs referable to the potential proffer of testimony concerning QEEG and PET testing performed on Salvatore finding said request to be without merit as a matter of law.

Finally, plaintiffs seek additur to increase the total awards for past and future pain and suffering from $640,000 to an appropriate seven-figure number. Counsel finds support from appellate case law involving similarly situated plaintiffs who suffered from TBI and PTSD. (Flomenhaft Aff. in support of Cross–Motion at pp. 34–41).

In reply, defendant's counsel factually distinguishes the case law plaintiffs rely upon for additur, reiterates her objection to the trial testimony of Salvatore's treating specialists questioning the value of their testimony due to purported gaps in time and in treatment (i.e., Dr. Greenspan did not see Salvatore until eleven years after the Collision, etc), and reiterates defendant's position as to the lack of record evidence of causation and serious injury. For ease of reference, defendant's counsel prepared a chart as part of his “wherefore” relief. Bachman therefore seeks an order vacating the jury award *in toto* and granting a new trial or, alternatively, reducing plaintiff's total lost earnings award to $60,000, reducing plaintiff's past medical expenses award to $25,000, reducing plaintiff's total past and future loss of medical insurance costs award to $0, reducing plaintiff's future loss of social security benefits award to $80,700 and reducing Ana LaMasa's total loss of services award to $50,000.

Discussion

Preliminarily, this Court grants the unopposed branch of defendant's motion reducing the past medical expense award from $40,768 to $25,000.

Having otherwise carefully reviewed the relevant portions of the trial transcript furnished by the parties, this Court finds the jury verdict is supported by sufficient evidence as a matter of law. Stated differently, the verdict is not utterly irrational and there was sufficient evidence to raise issues of fact (i.e., causation and serious injury) for the jury to resolve. *Garricks v. City of New York*, 1 NY3d 22, 769 N.Y.S.2d 152 (2003). Further, there were valid lines of reasoning and permissible inferences for the jury to draw upon that would lead these rational jurors to reach their conclusions based upon the testimonial and other admitted evidence presented at trial and decide the triable issue of whether Salvatore suffered serious injury causally related to the Collision. *Cohen v.. Hallmark Cards, Inc.*, 45 N.Y.2d 493, 410 N.Y.S.2d 282 (1978). This ample trial record does not justify a judgment notwithstanding the verdict dismissing the complaint without re-submission of the action to another jury.

***5** Having found sufficient evidence in the trial record to support the verdict, this Court must then inquire as to whether the conflicting medical and other expert testimonial evidence presented by the parties and which resulted in “a verdict for the plaintiff[s] ... so preponder-

ate[d] in favor of the defendant that [the verdict] could not have been reached on any fair interpretation of the evidence ..." *Moffat v. Moffatt*, 86 A.D.2d 864, 447 N.Y.S.2d 313 (2nd Dept., 1982) and quoted with approval with bracketed matter added in *Lolik et al., v. Big v. Supermarkets, Inc.*, 86 N.Y.2d 744, 631 N.Y.S.2d 122 (1995). In conducting a factual inquiry of the trial record, this Court further finds no basis to set aside the verdict as against the weight of the evidence and direct a new trial.

The facts of the Collision are essentially undisputed, i.e., a rear-end collision of a stationary vehicle waiting for a light change which occurred on a wet roadway. And the issue of Bachman's negligence was resolved as a matter of law in favor of Salvatore when this Court granted plaintiffs' application for a directed verdict on the question of negligence.

This Court digresses to discuss the merits of that branch of Bachman's post-verdict motion rearguing his opposition to plaintiffs' application for a directed verdict on this issue. Bachman again makes reference to a pretrial decision and order of the Hon. Joan A. Madden issued January 13, 1998 (Exhibit C to Bachman Motion) which denied plaintiffs' motion for summary judgment finding defendant's purported negligence to be a triable issue of fact. For reasons fully stated on the record at the close of the entire case and prior to summations, this Court made it clear that Justice Madden's decision and order did not mandate that the jury decide the issue of Bachman's negligence. It must be emphasized that "[a] denial of a motion for summary judgment is not necessarily *res judicata* or the law of the case that there is an issue of fact in the case that will be established at trial ..." *Sackman–Gilliland Corporation v. Senator Holding Corp.*, 43 A.D.2d 948, 351 N.Y.S.2d 733 (2nd Dept., 1974). Further, the "proof offered to defeat a motion for summary judgment does not meet the standard of proof required to resolve an issue of fact at trial ..." *Cushman & Wakefield, Inc., v. 214 East 49th Street Corp.*, 218 A.D.2d 464,468, 639 N.Y.S.2d 1012,1015 (1st Dept., 1996). Bachman's testimony and other supporting evidence in his defense neither included any non-negligent explanation for the Collision nor rebutted the presumption of negligence under all of the circumstances underlying the Collision. Defendant's excuse that the roadway was wet preventing him from stopping sufficiently in time to avoid the impact was wholly unavailing. *Mitchell v. Gonzalez*, 269 A.D.2d 250, 703 N.Y.S2d 124 (1st Dept., 2000). Thus, plaintiffs were not foreclosed from obtaining a directed verdict on the issue of negligence. See, *Gubala v. Gee*, 302 A.D.2d 911, 754 N.Y.S.2d 504 (4th Dept., 2003).

***6** As to the issues of causation and the precise physical injuries Salvatore suffered from as a result of the Collision, the parties had numerous expert witnesses testifying and "in considering the conflicting testimony fo the parties' respective expert witnesses, the jury was not required to accept one expert's testimony over that of another, but was entitled to accept or reject either expert's position in whole or in part ..." *Mejia v. JMM Audubon, Inc.*, 1 AD3d 261, 767 N.Y.S.2d 427 (1st Dept., 2003). To reiterate, the verdict as to the Collision being a substantial factor in causing Salvatore "serious injury" as defined under the Insurance Law § 5102(d) was not against the weight of the evidence and will not be disturbed.[FN14]

> FN14. In answering Question # 2 on the verdict sheet (Exhibit A to Bachman Motion), the jury deliberated on the precise issue of causation and the wording of the question made it clear that it had to determine whether the Collision was a substantial factor in causing *any* of Salvatore's injuries. The Jury's answers to Questions1A, 1B and 1C determined the no-fault threshold issue of whether Salvatore's injuries constituted a "serious injury". This Court does not find that the deletion of the phrase, "[a]s a result of the accident", from these three threshold questions prejudiced defendant in any way or ran afoul of the applicable "serious injury" PJI charges underlying these jury questions. In short, the jury squarely disposed of the separate and discrete issues of causation and serious injury under the no-fault statute.

Defendant's disguised reargument of certain *in limine* motions this Court denied and which defendant perceives, if granted, would have otherwise either resulted in a judgment of dismissal notwithstanding the verdict or its vacatur and a directive to conduct a new jury trial is without merit.

As to defendant's charge of discovery abuses [FN15], it is essentially admitted that raw EEG epochs contained in the treatment records of Dr. Kuhn were belatedly turned over and similar records of Dr. Weiner were purportedly destroyed in the ordinary course of that physician's business. Yet, this Court ruled that Dr. Weiner could not testify about any alleged objective findings of TBI noted on such EEG data. As noted in the trial transcript, defendant

was able to have an expert witness, Dr. Marc Nuwer, testify concerning Dr. Kuhn's data at trial, who offered a contrary interpretation of such data and, for that matter, a contrary opinion concerning the collision not being a competent producing cause of Salvatore's deteriorating physical condition. Defendant's motion stridently argues about the severe prejudice in belatedly receiving the respective CPLR § 3101(d) notices and reports/data of plaintiff's experts in the fields of neuropsychology (Nils Varney, Ph.D.), sleep medicine (Dr. Stasia Wieber) and accident reconstruction/engineering (Grahme Fisher, P.E.).

> FN15. Defendant claims plaintiff failed to produce and/or timely produce raw EEG data from certain treating physicians and laboratories, failed to produce neuropsychological testing records from psychologists and untimely served expert witness notices reflecting changes in the theory of Salvatore's case (i.e., mild TBI changed to "moderate to severe" TBI and a low speed collision changed to a moderate to high speed collision).

Nonetheless, this Court afforded defendant sufficient time and opportunity prior to, and during, the trial to review such notices, reports and data and consult with and produce their own expert witnesses in these respective fields for purposes of mounting an appropriate defense; all borne out by the extensive trial record. Moreover, this Court issued rulings which tailored certain of the plaintiffs' expert witnesses' testimony after considering certain defense arguments.[FN16]

> FN16. In written communications to this Court after the motion and cross-motion became *sub judice*, Plaintiff's counsel urged this Court to resolve an issue concerning the unanticipated costs plaintiffs incurred in obtaining the printout of raw data EEG data of Salvatore taken at the New York University School of Medicine, Department of Psychiatry as well as Dr. Wieber's raw sleep study data collected at Mt. Sinai School of Medicine which were ordered to be produced and turned over to defendant prior to and during the course of the trial. Consistent with this Court's discussions with respective counsel on this matter, this Court directs that these costs incurred in this data production should be shared by the parties.

Counsel has also reargued certain adverse rulings concerning the merits of defendant's *in limine* motions to preclude due to plaintiffs' failure to timely turn over and/or not turn over records of Dr. Leo J. Shea (neuropsychologist-treatment records), Dr. Charles Wetli (pathologist), Dr. Kenneth Alper (neurologist—QEEG records),

Dr. Monte Buchsbaum (psychiatry—PET scan data). Neither the potential testimony of these witnesses nor their records, reports and data were proffered during the course of the trial based on this Court's rulings and/or other considerations. Revisiting these issues again appears to be pointless. All of defendant's remaining challenges to this Court's rulings on the admission of evidence and/or at the formal charge conference are without merit and require no additional discussion.[FN17]

> FN17. However, one example should suffice. The mere mention of the word, "insurance", during the course of testimony and the context of how insurance was discussed was not prejudicial to defendant. No testimony was elicited which publicly noted that Bachman had liability insurance and the resources to satisfy any potential judgment. In this vein, this well-educated jury evidently could not have lost sight of the fact that Bachman was represented by two prominent law firms from New York *and* Washington D.C. with no less than three attorneys at the defense table each day of trial. Since Bachman was a retired airline pilot, the jury had ample reason to speculate where the source of funds for the enormous defense costs of this lengthy trial was coming from even if no witness ever mentioned the word insurance.

***7** In continuing the requisite analysis as to the correctness of the verdict, CPLR § 5501(c) states, in relevant part:

> In reviewing a money judgment in an action in which an itemized verdict is required in which it is contended that the award is ... inadequate and that a new trial should have been granted unless a stipulation is entered to a different award, the appellate division shall determine that an award is ... inadequate if it deviates materially from what would be reasonable compensation.

Trial courts may also apply this material deviation standard in overturning jury awards but should exercise

its discretion sparingly in doing so. *Shurgan v. Tedesco*, 179 A.D.2d 805, 578 N.Y.S.2d 658 (2nd Dept., 1992); *Prunty v. YMCA of Lockport*, 206 A.D.2d 911, 616 N.Y.S.2d 117 (4th Dept., 1994); see also, *Donlon v. City of New York*, 284 A.D.2d 13, 727 N.Y.S.2d 94 (1st Dept., 2001) (implicitly approving the application of this standard at the trial level). For guidance, a trial court will typically turn to prior verdicts approved in similar cases, but must undertake this review and analysis with caution not to rigidly adhere to precedents (because fact patterns and injuries in cases are never identical) and/or substitute the court's judgment for that of the jurors whose primary function is to assess damages. *Po Yee So v. Wing Tat Realty, Inc.*, 259 A.D.2d 373, 374, 687 N.Y.S.2d 99, 101 (1st Dept., 1999).

With the exception of the conceded reduction for past medical expenses, this Court finds that the jury were able to assess the severity of Salvatore's physical injuries, his physical and mental disorders, his historic and current treatment therefor and his poor prognosis. Accordingly, the pain and suffering and medical expenses awards did not deviate materially from what would be reasonable compensation under the circumstances. *Barrowman v. Niagara Mohawk Power Corp.*, 252 A.D.2d 946, 675 N.Y.S.2d 734 (4th Dept., 1998). Thus, the branches of Bachman's post-verdict motion for remittitur and plaintiffs' cross-motion for additur as to these awards are respectively denied.

Plaintiffs' expert's *per se* calculations of Salvatore's past loss of earnings ($460,713) and future loss of earnings ($774,892) were essentially unchallenged. Plaintiff had sufficient job continuity as a porter for Dr. Leiken to properly rely on Salvatore's 1992 annualized salary of $32,380 and it was perfectly reasonable for this economist to utilize a conservative rate of interest of 3.5% set by the U.S. Department of Labor to calculate annual salary increases (after 25 years, the U.S. Department of Labor set an increase rate of 4.5% which Dr. Leiken utilized for the year 2005 and going forward) to compute these losses. Bachman submitted no evidence of negotiated union contracts covering Salvotore's job title which contained annual salary increases which were lower than the percentage increases Dr. Leiken relied upon for his calculations. All of defendant's challenges to the loss of earnings awards are meritless and unsupported by trial evidence (e.g ., Salvatore would have left his job as a porter to become a full-time Con Edison meter reader, etc.). In short, the expert's reliance on certain facts as well as certain fair and reasonable assumptions and his calculations based thereon are fully supported by the extensive trial record. *Diaz v. West 197th Street Realty Corp.*, 290 A.D.2d 310, 736 N.Y.S.2d 361 (1st Dept., 2002).

***8** Concerning the jury's awards to Ana LaMasa for loss of services, the trial record amply established that since the Collision in 1992 and during the ensuing years, Salvatore's physical and mental condition precipitously declined and Ms. LaMasa was forced to assume his familial duties in addition to her own and to provide for her family's financial welfare. The jury has had the opportunity to assess her trial testimony and the corroborating testimony of her children as to the diminished quality of her life with Salvatore. And as borne out by expert testimony, Ana LaMasa must continue to spend the rest of her life providing "24/7" care to a spouse with, *inter alia*, severe psychiatric/psychological disorders, a role which renders her a "captiv[e][to] her marital responsibilities ..." (Flomenhaft Aff. in support of Cross–Motion at ¶ 94). Therefore, the $500,000 total award to Ana LaMasa for loss of services similarly does not deviate from what would be reasonable compensation under her circumstances. *Cf.*, *Dooknah v. Thompson*, 249 A.D.2d 260, 670 N.Y.S.2d 919 (2nd Dept., 1998).

In addition, the cost of medical insurance is a component of lost income and in Salvatore's case constituted a "soft dollar" benefit he had been receiving under his union contract and potentially would have been receiving had he continued working as a porter until age 65. The costs for obtaining medical insurance coverage and unreimbursed medical expenses are clearly not one and the same (see, *Schlachet v. Schlachet*, 176 A.D.2d 198, 574 N.Y.S.2d 320 [1st Dept ., 1991]). Accordingly, the expert's calculation of medical insurance costs were fair and reasonable and the jury awards based thereon do not constitute a double recovery for past and future medical expenses.

As noted earlier, Bachman took issue with this Court's somewhat novel ruling to amend the verdict sheet to add two additional categories of damages for past and future loss of medical insurance and future loss of social security benefits as components of lost earnings/income. Plaintiffs' counsel's request for this change was made immediately after summations and completion of the jury charge and just prior to deliberations. While conceding this amendment was unorthodox, nonetheless, Bachman has failed to show how the amendment to the verdict sheet prejudiced defendant's substantive and due process rights. First, defendant did not proffer his own expert

economist to take issue with any of Dr. Leiken's testimony and particularly the calculations of these components of lost income. Second, defendant's counsel's closing argument did not even address any deficiencies, vis-a-vis, Dr. Leiken's trial testimony including his calculation of the past and future loss of earnings and their sub-categories. It cannot be said that Bachman's counsel relied on the pre-amendment version of the jury verdict sheet to structure his summation and therefore had been prejudiced by the inclusion of these new sub-categories of loss of earning damages on the verdict sheet ultimately introduced to, and considered by, the jury with additional jury instructions. Finally, defendant has neither shown that this verdict sheet amendment violated any trial rule or procedure nor constituted an abuse of this Court's discretion.[FN18]

> FN18. Unlike the sub-category of loss of medical insurance, defendant's counsel apparently recognized some merit to the jury award for loss of social security benefits when, in the alternative, counsel requested the court to reduce this award from $122,273 to $80,700. (Murphy Aff. at ¶ 98 annexed to Bachman Motion).

9** To conclude this discussion, it is necessary to address defendant's requests for costs and attorneys' fees in mounting a vigorous defense opposing the potential admissibility of expert testimony about QEEG and PET scan studies plaintiff was relying upon to corroborate Salvatore's TBI caused by the Collision. While this Court ruled that the QEEG and **PET scan** studies did not meet the ***Frye standard to warrant their admission and granted Bachman's *in limine* motions to preclude such testimony with respect thereto, plaintiffs' counsel's trial strategy to proffer such data as evidence of TBI in low to moderate impact collisions was not beyond the pale and certainly not frivolous. Nor can QEEG and PET data be viewed as junk science. In addition, counsel's withdrawal of certain expert witnesses who would otherwise have testified utilizing QEEG and PET studies was directly due to this Court's bench colloquy and rulings on the record. Parenthetically, defendant's counsel overlooks the fact that this Court conducted a *Frye* inquiry relying on dueling expert affidavits and respective supporting scientific literature as well as dueling affirmations and memoranda of law; all without the need for either party to incur the exorbitant cost of producing experts for a formal *Frye* hearing. While this Court concluded expert testimony relying on these tests did not meet the *Frye* standard at this time; still, these tests and related research are "works in progress" as to their potential, broad-based applications in the diagnosis and treatment of disease. Thus, there is simply no legal/factual basis to invoke any 22 NYCRR § 130–1.1 sanction against plaintiffs and their counsel for attempting to proffer evidence of Salvatore's TBI utilizing QEEG and PET studies to support their case.

For the foregoing reasons, this Court grants the unopposed branch of defendant's post-verdict motion reducing the award for past medical expenses from $40,768 to $25,000. In all other respects, the remaining branches of defendant's motion and plaintiffs' cross-motion are respectively denied. Plaintiffs shall submit a proposed money judgment, on notice, for signature consistent with this Court's Decision and Order. This constitutes the Decision and Order of this Court. Courtesy copies of same have been provided to counsel for the parties.

N.Y.Sup.,2005.
Lamasa v. Bachman
Slip Copy, 8 Misc.3d 1001(A), 2005 WL 1364515 (N.Y.Sup.), 2005 N.Y. Slip Op. 50882(U)

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(MD)

RICHARD ZAWASKI,
Plaintiff

v.

GIGS, LLC AND WENDELL LEE ZORMAN,
Defendants

MEMORANDUM AND ORDERS ON THE PARTIES' DAUBERT/LANIGAN II MOTIONS

On September 13, 2010, the Court held a Daubert/Lanigan II hearing on various motions concerning the admissibility or non-admissibility of various testing, test results and expert testimony. The parties did not request an evidentiary hearing on any of these motions but submitted all matters to the Court on affidavits, medical articles, and memoranda at a non-evidentiary hearing. The documents submitted to the Court were in a sixteen inches high pile and numbered approximately 3000 pages. The trial of this case has been assigned within the next month.

In Commonwealth v. Lanigan, 419 Mass. 15, 26 (1194), the Supreme Judicial Court adopted in part the basic reasoning of the United States Supreme Court in Daubert v. Merrell Dow, 509 U.S. 579, 593-594 (1993). In Case of Canavan, 432 Mass. 304, 310-311,

(2000), the Supreme Judicial Court held as follows:

> "Prior to our decision in Commonwealth v. Lanigan, supra, we required that in most circumstances "the community of scientists involved [must] generally accept[] the theory or process" for it to be admitted in evidence. The general acceptance test, or *Frye* test, often proved to be useful because, if there is a general acceptance of a theory or process in the relevant scientific community, the theory or process in question is likely reliable. However, we recognized that "strict adherence to the *Frye* test" could result in reliable evidence being kept from the finder of fact. *Id.* For example, a new theory or process might be "so logically reliable" that it should be admissible, even though its novelty prevents it from having attained general acceptance in the relevant scientific community.
>
> In order to account for this circumstance, we adopted in part the United States Supreme Court's reasoning in Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579, (1993), and held that "a proponent of scientific opinion evidence may demonstrate the reliability or validity of the underlying scientific theory or process by some other means, that is, without establishing general acceptance." Commonwealth v. Lanigan, supra at 26, 641 N.e.2d 1342. ...Thus, we have concluded that a party seeking to introduce scientific evidence may lay an adequate foundation either by establishing general acceptance in the scientific community or by showing that the evidence is reliable or valid through an alternate means. Commonwealth v. Sands, 424 Mass. 184, 185-186, 675 N.E.2d 370 (1997).

Therefore in Daubert and Lanigan II, the respective courts announced a new test to govern the admissibility of expert testimony based on scientific, technical and other specialized knowledge. A party seeking to introduce scientific evidence may

lay a foundation either by showing that the underlying scientific theory is generally accepted within the relevant scientific community or by showing the theory is reliable or valid through other means. Canavan's Case, supra at 310. In the event that a party cannot prove that the underlying scientific theory is generally accepted, the proponent of scientific opinion evidence may demonstrate the reliability or validity of the underlying scientific theory or process by some other means, that is, without establishing general acceptance. Commonwealth v. Patterson, 445 Mass. 626, 640-641 (2005).

The judge must play the role of "gatekeeper" in determining whether to admit such evidence. In doing so, the judge must preliminarily assess whether that reasoning or methodology underlying the expert testimony is scientifically valid and whether that reasoning or methodology properly can be applied to the facts in the case...The Court in Daubert described several factors bearing on this assessment, namely:

1. Whether the scientific theory or technique "can be [and has been] tested";

2. Whether the theory or technique has been subjected to peer review and publication;

3. The known or potential rate of error...and the existence and maintenance of standards controlling the technique's operation; and

4. General acceptance.

In Daubert, the Court indicated that the above four factors are not meant to provide a definitive checklist or test Daubert,. supra at 592-593.

However, it must be remembered that the determination of the judge whether some expert testimony will be admitted is only a preliminary determination. When the expert evidence is presented to the jury, it is subject to the jury's determination on whether to reject it or to accept it, in whole or in part. The expert witness may be cross-examined in all the usual areas. The jury eventually will be the sole decisionmakers of the expert testimony and all other facts.

Basically, what the two parties have done here is present to the Court a pile of approximately 3000 pages (including many very technical articles) on very complicated and highly contested medical matters and ask the Court, on its own, to determine the reliability or non-reliability of various esoteric experts and theories. The parties have neither provided nor offered to provide any "live" expert testimony to support their respective positions. There are obvious partisans on each side of the issues as to whether the medical or scientific opinions should be admitted - depending on who has hired them and on which party their testimony supports.

Without having live evidence and witnesses to be examined by the parties and the Court, this Court stands in a difficult

position to make the very important decisions as to which evidence may be "junk" expert testimony and which expert testimony is or may be legitimate medical science. Such decisions could or may well be outcome determinative in this case.

Apart from the Court's order of exclusion on Motion #5 below, that is, Plaintiff's motion to exclude any reference to the 1997 AAN position paper and to exclude any testimony which relies on said position paper (which motion has been allowed), the Court has crafted the balance of the orders individually to the particular motion involved. The parties are expected of course to rely on their cross-examination of the expert witnesses to limit or discredit their testimony. The Court will prepare and file a decision later that gives the reasons for the Court's decision in this matter.

Therefore, based on this Court's review of all documents submitted by both parties, the Court makes the following orders on the subject six motions:

(1) Motion in Limine of the Defendants, GIGS, LLC and Wendell Lee Zorman to Preclude Murdo Dowds, Ph.D. from Offering Medical Opinion Testimony Concerning the Plaintiff's Alleged Acute Concussive Brain Injury, with Plaintiff's Opposition.

ORDER: The testimony of Doctor Murdo Dowds, Ph.D. depends on her experience and expertise. There is no hard and fast rule that only a physician may give medical testimony. In this day and time, science, including medical science, is advancing so

fast that different areas of expertise overlap. It will depend on the witness' expertise. From the Court's reading of her education, training and experience, and from the substance of her opinion, it appears that the expert witness is qualified to render opinions in the areas discussed in the plaintiff's opposition to this motion, and the motion is DENIED.

(2) Motion of the Defendants, GIGS, LLC and Wendell Lee Zorman, to Preclude any and all Evidence, Testimony, Reports and/or References Relating to Diffusion Tensor Imaging because DTI is Neither Reliable Nor Accepted in the General Medical Community as Required Under Daubert and Lanigan Standards, and Opposition.

ORDER: Defendant's Motion to Preclude Evidence (et cetera) relating to Diffusion Tensor Imaging is DENIED.

(3) Plaintiff's Motion for Limited Time (One Hour Each) Attorney Conducted Voir Dire and Opposition.

ORDER: ALLOWED. The Court will hold a conference to set up the ground rules.

(4) Plaintiff's Motion in Limine to Exclude the Result of the TOMM Test and Opposition.

ORDER: The Court will hold a hearing (evidentiary) out of the presence of the jury to determine whether the TOMM test's administration followed proper procedure.

(5) Plaintiff's Motion to Exclude Any Reference to the 1997 AAN Position Paper and to Exclude any Testimony Upon Which Relies on the Position Paper and Opposition.

ORDER: ALLOWED. This Court has closely reviewed all the submissions concerning this paper written some 13 years ago. It is replete with errors as pointed out in the many other articles submitted.

(6) Plaintiff's Motion to Exclude the Purported Tractor Inspection Report dated March 20, 2006 and Opposition.

ORDER: The defendants will be expected to lay a proper foundation for the introduction of said report by the person who authored said report and performed said inspection.

By the Court,

Thomas E. Connolly
Justice of the Superior Court

Date: November 4, 2010

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89 A.D.3d 42, 929 N.Y.S.2d 264, 2011 N.Y. Slip Op. 06475
(Cite as: 89 A.D.3d 42, 929 N.Y.S.2d 264)

Supreme Court, Appellate Division, Second Department, New York.
Jacob LUGO, etc., et al., appellants,
v.
NEW YORK CITY HEALTH AND HOSPITALS CORPORATION, etc., respondent.

Sept. 13, 2011.

Background: After concluding that infant plaintiff's and his mother's expert testimony regarding causation was inadmissible, the Supreme Court, Kings County, Allen Hurkin–Torres, J., granted defendant hospital's motion for summary judgment dismissing the medical malpractice complaint based on hospital's alleged failure to timely diagnose and treat the hypoglycemia of both newborn patient and his mother, and plaintiffs appealed.

Holdings: The Supreme Court, Appellate Division, Covello, J., held that:
(1) patient's experts demonstrated that their theory of causation was reasonably permitted by a synthesis of the medical literature, and
(2) genuine issue of material fact existed as to whether patient's brain damage was caused by his episode of neonatal hypoglycemia.

Reversed.

West Headnotes

[1] Evidence 157 555.2

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k555 Basis of Opinion
157k555.2 k. Necessity and sufficiency. Most Cited Cases

Expert testimony based on scientific principles or procedures is admissible but only after a principle or procedure has gained general acceptance in its specified field.

[2] Evidence 157 555.2

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k555 Basis of Opinion
157k555.2 k. Necessity and sufficiency. Most Cited Cases

Frye decision governing admissibility of expert opinion testimony is not concerned with the reliability of a certain expert's conclusions, but instead with whether the expert's deductions are based on principles that are sufficiently established to have gained general acceptance as reliable; limited purpose of the *Frye* test is to ascertain whether the expert's conclusion is based upon accepted scientific principles, rather than simply the expert's own unsupported beliefs.

[3] Evidence 157 555.5

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k555 Basis of Opinion
157k555.5 k. Cause and effect. Most Cited Cases

It is not necessary that the underlying support for an expert's theory of causation consist of cases or studies considering circumstances exactly parallel to those under consideration in the litigation; it is sufficient if a synthesis of various studies or cases reasonably permits the conclusion reached by the plaintiff's expert; fact that there is no textual authority directly on point to support the expert's opinion is relevant only to the weight to be given the testimony, but does not preclude its admissibility.

[4] Evidence 157 555.10

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts

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157k555 Basis of Opinion
157k555.10 k. Medical testimony. Most Cited Cases

Evidence 157 556

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k556 k. References to authorities on subject. Most Cited Cases

In medical malpractice action based on allegations that hospital's failure to timely diagnose and treat the hypoglycemia of both newborn patient and his mother caused patient's brain damage and cerebral palsy, patient's experts demonstrated that their theory of causation was reasonably permitted by a synthesis of the medical literature; although none of the articles, read in isolation, provided conclusive support for the theory of causation, when considered in the aggregate for the limited purpose of applying the *Frye* test, and against the backdrop of the undisputed generally accepted principles concerning hypoglycemia set forth at the hearing, those articles established that the theory was properly based upon far more than theoretical speculation or a scientific "hunch," and the absence of medical literature directly on point with the circumstances of patient's case pertained to the weight to be given to the opinion testimony, but did not preclude its admissibility.

[5] Evidence 157 555.5

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k555 Basis of Opinion
157k555.5 k. Cause and effect. Most Cited Cases

Evidence 157 556

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k556 k. References to authorities on subject. Most Cited Cases

When *Frye* test is applied to an expert's theory of causation, court's concern must be limited to making sure that within the scientific field in question, there is a substantive, demonstrable, objective basis for the expert's conclusion, and that the focus of the inquiry in such an instance should not be upon how widespread the theory's acceptance is, but should instead consider whether a reasonable quantum of legitimate support exists in the literature for the expert's views; purpose of the *Frye* test is not to preclude expert opinion testimony based upon reasonable extrapolations from conceded legitimate empirical data.

[6] Evidence 157 555.2

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k555 Basis of Opinion
157k555.2 k. Necessity and sufficiency. Most Cited Cases

Frye inquiry into reliability of expert opinion is separate and distinct from the admissibility question applied to all evidence, whether there is a proper foundation, to determine whether the accepted methods were appropriately employed in a particular case; focus moves from the general reliability concerns of *Frye* to the specific reliability of the procedures followed to generate the evidence proffered and whether they establish a foundation for the reception of the evidence at trial.

[7] Evidence 157 555.10

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k555 Basis of Opinion
157k555.10 k. Medical testimony. Most Cited Cases

Evidence 157 556

157 Evidence
157XII Opinion Evidence
157XII(D) Examination of Experts
157k556 k. References to authorities on subject. Most Cited Cases

Patient's experts proffered sufficient foundational

evidence to support the admissibility of their testimony concerning their theory of causation in medical malpractice trial; experts made specific reference to the contents of numerous articles documenting brain MRI abnormalities in patients who had experienced hypoglycemia to support their opinion that there was a causal connection between patient's episode of hypoglycemia and the brain abnormalities later observed on his MRI film.

[8] Judgment 228 181(33)

228 Judgment
228V On Motion or Summary Proceeding
228k181 Grounds for Summary Judgment
228k181(15) Particular Cases
228k181(33) k. Tort cases in general. Most Cited Cases
(Formerly 170Ak2515)

Genuine issue of material fact existed as to whether patient's brain damage was caused by his episode of neonatal hypoglycemia, precluding summary judgment in favor of hospital on patient's medical malpractice claim based on hospital's failure to timely diagnose and treat the hypoglycemia of both newborn patient and his mother.

****266** Fitzgerald & Fitzgerald, P.C., Yonkers, N.Y. (John E. Fitzgerald, John M. Daly, Eugene S.R. Pagano, Mitchell L. Gittin, and John R. Langdell of counsel), for appellants.

Michael A. Cardozo, Corporation Counsel, New York, N.Y. (Edward F.X. Hart and Jane L. Gordon of counsel), for respondent.

REINALDO E. RIVERA, J.P., JOSEPH COVELLO, ANITA R. FLORIO, and PLUMMER E. LOTT, JJ.

COVELLO, J.

****43** Introduction*

New York courts apply the rule of *Frye v. United States*, 293 F. 1013 that expert testimony based on scientific principles ***44** or procedures is admissible, but only after a principle or procedure has gained general acceptance in its specified field. In this medical malpractice action, the principal question presented on this appeal is whether the Supreme Court, in applying the *Frye* test, properly determined that the opinion testimony of the plaintiffs' experts that the infant plaintiff's brain injuries were caused by an episode of severe neonatal hypoglycemia lasting 81 minutes was inadmissible. For the reasons set forth below, we answer this question in the negative.

Factual and Procedural Background

Factual Background

In 2001, the plaintiff Brenda Almodovar (hereinafter the mother), who was pregnant with the infant plaintiff, Jacob Lugo, began receiving prenatal care at Woodhull Hospital (hereinafter Woodhull), a facility owned and operated by the defendant. On August 11, 2001, at 31 weeks of gestation, the mother was admitted to Woodhull for signs of preterm labor. During that admission, her blood glucose level was measured at 26 mg/dL, an abnormally low level, but was subsequently measured at a normal****267** level. The mother was discharged on August 13, 2001.

On September 2, 2001, at 34 weeks of gestation, the mother, who had a history of seizures dating back to childhood, was brought to Woodhull by emergency medical services (hereinafter EMS) personnel after experiencing a grand mal seizure. On that date, she was evaluated but not admitted.

On October 5, 2001, the mother gave birth to Lugo at Woodhull by normal spontaneous vaginal delivery at 11:39 A.M. Lugo's Apgar scores, 9 at one minute, and 9 at five minutes, were “excellent,” and he initially appeared normal. However, by the time Lugo was 40 minutes old, he was experiencing tremors and, at 12:25 P.M., he was admitted to the neonatal intensive care unit.

According to the deposition testimony of Dr. Frantz Brea, the director of neonatology at Woodhull, tremors are a sign of hypoglycemia [FN1] in a newborn. At 12:25 P.M., when Lugo was admitted to the neonatal intensive care unit, his blood glucose level was measured, through a “heel stick” test, at less than 20 mg/dL, and laboratory testing of blood drawn from Lugo at that time later measured a glucose level of 3 mg/dL. According to Dr. Brea, a normal glucose level for an infant approximately 40 minutes old is about 40 mg/dL. Lugo was given a “glucose IV push” and a glucose infusion, and at 1:00 P.M., his blood glucose ***45** level was measured at 71 mg/dL, within normal

89 A.D.3d 42, 929 N.Y.S.2d 264, 2011 N.Y. Slip Op. 06475
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limits. Thereafter, Lugo's blood glucose level remained within normal limits until he was discharged from Woodhull on October 7, 2001.

FN1. Hypoglycemia means low blood sugar.

In 2002, Lugo was referred to Woodhull for evaluation due to his delays in reaching certain developmental milestones. On April 29, 2003, Lugo underwent a brain magnetic resonance imaging (hereinafter MRI) examination at Brookdale Hospital, and the resulting MRI report set forth a finding of "nonspecific white matter loss in parietal and occipital lobes with dilation of the occipital horn ... which suggests periventricular leukomalacia, as can be seen with perinatal ischemia." [FN2] Ultimately, Lugo was diagnosed with cerebral palsy (spastic diplegia type).

FN2. According to expert testimony presented in this matter, perinatal ischemia-in the context of the instant action-is a decrease in the flow of blood and/or oxygen to the brain of a fetus.

Commencement of this Action

Lugo, by his mother, and the mother, suing derivatively, commenced this action, inter alia, to recover damages for medical malpractice. In their verified bill of particulars, the plaintiffs alleged that the defendant had departed from good and accepted medical practice by, among other things, failing to timely diagnose and treat the hypoglycemia of both the mother and Lugo. They alleged that Lugo's hypoglycemia had caused, among other things, his brain damage and cerebral palsy.

The Defendant's Motion for Summary Judgment or a Frye Hearing

By notice of motion dated May 15, 2007, the defendant moved for summary judgment dismissing the complaint or, in the alternative, for a *Frye* hearing in the event that the plaintiffs, in opposition to the motion, proffered a sworn statement from an expert opining that Lugo's injuries were caused by the "possible transient episode" of maternal hypoglycemia on August 11, 2001, or the "transient episode" of hypoglycemia on October 5, 2001. As relevant here, the defendant supported its motion with the expert affirmation of Dr. Armando Grassi, who opined that Lugo's ****268** episode of neonatal hypoglycemia did not cause his alleged injuries. According to Dr. Grassi, the white matter loss shown on Lugo's April 2003 MRI was in the periventricular area and was a typical lesion resulting from a decrease in oxygenation or perfusion to the brain. In contrast, he affirmed, lesions typical of hypoglycemia are "diffuse lesions" in the brain and are not found in the periventricular area. Dr. Grassi opined that Lugo's brain injury, as depicted on his MRI, was a result of decreased oxygenation to his brain at 32–34 weeks gestation, and was not caused by the "transient hypoglycemic episode" at his birth. Dr. Grassi asserted that it was not accepted in the ***46** medical profession that "a short and promptly treated" episode of hypoglycemia in a newborn could cause brain damage in the periventricular area, as seen on Lugo's MRI film, and that Dr. Grassi had "never heard or read of a single case of periventricular leukomalacia caused by hypoglycemia."

In opposition, the plaintiffs argued, inter alia, that summary judgment was improper because there were triable issues of fact concerning, among other things, the nature and cause of Lugo's periventricular leukomalacia (hereinafter PVL) and cerebral palsy. As relevant here, they submitted the expert affirmation of Dr. Rosario Trifiletti. Dr. Trifiletti opined that Lugo had been born with "profound hypoglycemia," and that the delay in diagnosis and treatment from 11:39 A.M. to 1:00 P.M. was a substantial factor in causing his brain damage. Dr. Trifiletti disagreed with Dr. Grassi's conclusion that the mother's seizure had caused Lugo's brain injuries. According to Dr. Trifiletti, Lugo's normal appearance and good Apgar scores at birth, and the delay of the onset of his tremors until approximately 40 minutes after birth, were consistent with depletion of glucose stores after birth rather than a primary hypoxic injury. Dr. Trifiletti characterized Lugo's post-birth tremors as "subtle seizures" as defined in Volpe's Neurology of the Newborn (hereinafter the Volpe textbook), and he opined that Lugo's "tremors" or "subtle seizures" had been caused by his profound hypoglycemia at birth.

In Dr. Trifiletti's opinion, Lugo's MRI report was "essentially accurate" in its finding of PVL about the posterior (occipital) horns of the lateral ventricles, and he disagreed with Dr. Grassi's assertion that the pattern of injury it depicted was not characteristic of lesions caused by hypoglycemia. Dr. Trifiletti affirmed that there is "substantial overlap" in the lesions resulting from hypoxia and from hypoglycemic

injury. Citing Arie L. Alkalay, *et al.*, *Brain Imaging Findings in Neonatal Hypoglycemia: Case Report and Review of 23 Cases*, 44 Clin Pediatr 783–790 (2005), an article published in the November/December 2005 edition of the journal Clinical Pediatrics, Dr. Trifiletti asserted that there was a tendency towards occipital injury (as was seen in Lugo's case) with hypoglycemia. He saw nothing on Lugo's MRI film that excluded hypoglycemia as the etiology of the "obvious white matter loss and occipital horn dilation" and, in his experience of reviewing brain MRIs as part of his clinical practice over the years, he had seen "similar patterns of brain injury in comparable instances of perinatal hypoglycemia."

***47** In its reply papers, the defendant proffered the expert affirmation of Dr. Steven Pavlakis. Dr. Pavlakis affirmed, among other things, that after performing a search on "Pub Med," he found no evidence that the white matter damage seen on Lugo's MRI film could be caused by "short lived transient hypoglycemia," and that it was not generally accepted that a period of transient neonatal hypoglycemia such as that suffered by Lugo could cause ****269** his clinical outcome. Dr. Pavlakis disagreed with Dr. Trifiletti's opinion that Lugo had suffered from "subtle seizures" as defined in the Volpe textbook, and he asserted that the Alkalay article cited by Dr. Trifiletti did not discuss any patients who had experienced an episode of hypoglycemia similar to that experienced by Lugo.

In an order dated November 5, 2007, the Supreme Court granted that branch of the defendant's motion which was for a *Frye* hearing and held in abeyance that branch of the defendant's motion which was for summary judgment dismissing the complaint. The Supreme Court determined that the plaintiffs' experts had provided "scant reference" to medical or scientific literature to support their opinions, and that a *Frye* hearing should be held to determine whether their deductions were based on principles which were sufficiently established to have gained general acceptance.

The Frye Hearing

After additional motion practice not at issue on this appeal, the Supreme Court conducted a *Frye* hearing in April and May 2009. The first expert to testify for the plaintiffs was Dr. Michael Katz, a private practitioner who was board-certified in pediatric neurology and neurodevelopmental disabilities. As background, Dr. Katz testified that the normal blood glucose range for newborns is between 40 and 60 mg/dL, that a level below 40 mg/dL is considered hypoglycemia, that Lugo's measured blood glucose level of 3 mg/dL was "[p]rofoundly low," and that hypoglycemia is a medical emergency which must be treated immediately because it is a toxic state which causes brain damage. Dr. Katz's working hypothesis was that Lugo's blood glucose level was 3 mg/dL from 11:39 A.M., when he was born, until 1:00 P.M., when his blood sugar was normalized. In Dr. Katz's opinion, Lugo's brain injury was caused by this episode of hypoglycemia.

Dr. Katz testified that his opinion that an episode of hypoglycemia at a level of 3 mg/dL lasting 1 hour and 21 minutes could cause neurologic damage of the type sustained by Lugo was ***48** based on the following generally accepted scientific principles: (1) hypoglycemia causes brain injury; (2) certain infants are more susceptible than others to neurologic injury secondary to hypoglycemia; (3) hypoglycemia is a toxic and dangerous state; and (4) there is no safe level of hypoglycemia. Dr. Katz testified that his opinion that hypoglycemia caused Lugo's brain injury was based on the fact that Lugo's MRI film showed a brain injury, that Lugo had suffered from a period of proven and profound hypoglycemia, and that there appeared to be nothing else in the record or around the time of Lugo's birth suggesting that anything besides hypoglycemia caused Lugo's injury. Dr. Katz did not believe that the mother's seizure at 34 weeks of gestation had injured Lugo in the nature of a hypoxic ischemic event resulting in brain MRI abnormalities because Dr. Katz had difficulty visualizing a mechanism by which a seizure during pregnancy could cause a decrease in blood flow in the infant's brain.

Dr. Katz addressed, at length, the medical literature upon which his theory of causation was based. He noted that the Volpe textbook indicated that hypoglycemia causes brain injury and brain damage. In addition, the Volpe textbook discussed neuropathic studies indicating that hypoglycemia is a precedent of PVL and that both perinatal ischemia and hypoglycemia could cause an identical brain injury: namely, PVL. Dr. Katz explained that PVL is an injury to the white brain matter in the distribution around the ventricles.

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****270** Next, Dr. Katz discussed Arie L. Alkalay, *et al., Plasma Glucose Concentrations in Profound Neonatal Hypoglycemia*, 45 Clin Pediatr 550 (2006), an article published in the July 2006 edition of the journal Clinical Pediatrics (hereinafter the Alkalay article). He explained that the authors had compiled 16 different studies in an attempt to define low thresholds of plasma glucose concentrations constituting treatable or profound hypoglycemia, and they had concluded that plasma glucose levels of less than 25 mg/dL of several hours' duration may increase the relative risk for adverse neurologic outcome. Dr. Katz testified that a plasma glucose level is essentially the same as a whole blood glucose level, and that a plasma glucose level of 25 mg/dL is "much higher" than a whole blood glucose level of 3 mg/dL.

Dr. Katz acknowledged that one of the studies reviewed in the Alkalay article, Anne Kinnala, *et al., Cerebral Magnetic Resonance Imaging and Ultrasonography Findings After *49Neonatal Hypoglycemia*, 103 Pediatrics 724–729 (1999) (hereinafter the Kinnala article), published in the April 1999 edition of the journal Pediatrics, had excluded infants who had experienced only one episode of hypoglycemia before six hours of age. However, he did not believe that this fact affected the overall conclusion of the Alkalay article, which had examined 15 other studies besides the Kinnala article. Dr. Katz noted that the Kinnala article included a patient who had shown evidence of neurologic injury on an MRI after experiencing a hypoglycemic episode lasting two hours where the lowest glucose level was 32 mg/dL, a level "dramatically" higher than Lugo's glucose level of 3 mg/dL.

Finally, Dr. Katz discussed Burns, *et al., Patterns of Cerebral Injury and Neurodevelopmental Outcomes After Symptomatic Neonatal Hypoglycemia*, 122 Pediatrics 65 (2008) (hereinafter the Burns article), an article published in the journal Pediatrics in 2008. He explained that the authors had studied 35 term infants and had attempted to limit their study to symptomatic neonatal hypoglycemic patients, meaning those who had suffered from tremors, and to exclude brain injuries from other causes such as hypoxic ischemic encephalopathy. Sixty-three percent of the patients studied in the Burns article had experienced only one episode of hypoglycemia which had resolved promptly with treatment, and 94% of all of the patients studied had shown evidence of MRI abnormalities. The article also examined neurodevelopmental outcomes and determined that six of the subjects had developed cerebral palsy and three had developed mild motor delays.

Dr. Katz acknowledged that it was "unclear" exactly what duration and level of hypoglycemia causes neurologic injury in humans, and that there was no specific article, report, or study stating, in unambiguous terms, that an episode of hypoglycemia lasting 1 hour and 21 minutes at a level of 3 mg/dL had caused, or could cause, neonatal brain injury. However, he testified that there was not a "whole lot" of medical literature on hypoglycemia because "it is really an impossible task to prospectively look at hypoglycemia in children." Dr. Katz also acknowledged that there are a number of potential causes of PVL in addition to hypoglycemia, including hypoxic ischemia, and that it was possible that Lugo had sustained his injury during the mother's seizure and been asymptomatic at the time of birth. Dr. Katz stressed, however, that Lugo had been symptomatic for hypoglycemia, that Lugo's MRI results were consistent with ***50**hypoglycemia , that the medical literature indicates that low blood sugar causes brain damage, and that his opinion was based on ****271** the "confluence" of the medical information he had discussed.

Dr. Robert Peyster, the chief of neuroradiology at Stony Brook University Medical Center, also testified for the plaintiffs. Dr. Peyster explained that PVL is not a specific term, but, rather, refers to damage to the deep white brain matter next to the ventricles that appears as an abnormality on a CT scan or an MRI, and that PVL can be caused by both hypoglycemia and perinatal asphyxia. At the hearing, Dr. Peyster reviewed Lugo's MRI films in detail and testified that they depicted PVL. Based on Lugo's measured profound hypoglycemia and high Apgar scores, Dr. Peyster opined that the cause of Lugo's PVL was his episode of hypoglycemia and not perinatal asphyxia. Although he acknowledged that a seizure during pregnancy could potentially be severe enough to damage the brain of a fetus by reducing blood flow across the placenta, he was unaware of any reported cases where a child who had experienced such an event had received normal Apgar scores at birth.

Like Dr. Katz, Dr. Peyster addressed relevant medical literature at length. He agreed with Dr. Katz

that the Volpe textbook supported the position that hypoglycemia leads to PVL. Dr. Peyster testified that the Burns article was significant because it was the largest series to date addressing MRI findings and other issues in neonatal hypoglycemia, because it had excluded patients who might have had hypoxic ischemia, and because 94% of the patients had shown white matter abnormalities on their MRI brain scans. He considered the Burns article to be a "good paper" and the best available article addressing generalized principles regarding hypoglycemia and injuries to infants. However, Dr. Peyster conceded that the Burns article had not been designed to test the relationship between the severity or duration of hypoglycemia and neurodevelopmental outcomes and had not found any such relationship, and that the subjects studied in the Burns article had received MRI brain scans at a much earlier age than Lugo had.

Dr. Peyster acknowledged that he had not located any articles or reports specifically addressing a patient who had experienced an episode of hypoglycemia of the same level and duration as Lugo's episode, but he testified that this fact did not change his opinion that Lugo's injuries were caused by hypoglycemia because the literature he had reviewed had studied cases representing a wide range of duration times, Lugo had PVL, and ***51** Lugo's glucose level had been measured at close to zero. Dr. Peyster testified that there was no threshold of duration and severity, generally accepted by most physicians, below which hypoglycemia could *not* cause abnormalities like those seen on Lugo's MRI.

After the plaintiffs' experts testified, the defendant presented the testimony of Dr. Caren Jahre, a private practitioner and an assistant professor of radiology at New York University School of Medicine. Dr. Jahre testified that Lugo's MRI films depicted a "classic pattern" of PVL seen in the context of hypoxic encephalopathy or perinatal ischemia at 26 to 34 weeks of gestation, and that the literature she had reviewed did not associate this specific pattern with neonatal hypoglycemia. According to Dr. Jahre, medical literature indicated that the "hallmark" of brain damage resulting from hypoglycemia is cortical involvement, and some of that literature reported white matter damage caused by hypoglycemia either "out in the periphery" or against the ventricles, but limited to certain areas. In contrast, according to Dr. Jahre, the brain damage on Lugo's MRI film had a diffuse pattern tracking ****272** along the ventricles and no cortical involvement. However, she acknowledged that she and Dr. Peyster disagreed on the precise appearance of the pattern depicted on Lugo's MRI film.

In Dr. Jahre's opinion, the Burns article was flawed because, based upon the medical records of the patients it had studied, the authors had failed to exclude patients who had suffered from health issues other than neonatal hypoglycemia, including hypoxic ischemic encephalopathy. Additionally, according to Dr. Jahre, none of the MRI images in any of the literature discussed at the *Frye* hearing looked "anything close to what [Lugo's] brain looks like."

The defendant also presented the testimony of Dr. Steven Pavlakis, a professor of neurology and pediatrics at Mt. Sinai School of Medicine and the director of pediatric neurology at Maimonides Hospital. Dr. Pavlakis had performed a search and had found no literature on MRI changes resulting from hypoglycemia in newborns lasting less than two hours. He agreed that hypoglycemia can cause MRI abnormalities, that severe hypoglycemia can cause brain damage, and that Lugo's measured glucose level of 3 mg/dL was very low. In addition, he acknowledged that the scientific community does not recognize any specific level or duration of hypoglycemia which would *not* cause brain damage and that it was a generally accepted medical principle that individual susceptibility to toxic states varies.

***52** According to Dr. Pavlakis, it was "relatively common" for newborns to have hypoglycemia, low blood sugar was a common cause of tremors such as those experienced by Lugo, and such tremors were distinguishable from seizures and did not correlate to an underlying condition or particular outcome. Based on Lugo's normal appearance at birth and recovery with sugar infusions, Dr. Pavlakis did not believe that his episode of hypoglycemia had caused his brain damage. Dr. Pavlakis also excluded hypoglycemia as a cause of Lugo's injuries because "there's no case like him" of which Dr. Pavlakis was aware in the literature or in his practice.

According to Dr. Pavlakis, decreased oxygen or blood flow to a fetus between the ages of 28 to 40 weeks is the cause of PVL in "99.99 percent" of cases. He testified that PVL could be caused by anything that decreases oxygen or blood supply to a fetus un-

der 40 weeks of gestation, including, hypothetically, a seizure like the one experienced by the mother. However, like the plaintiffs' expert Dr. Katz, Dr. Pavlakis was unaware of any instance in which such a seizure had actually resulted in PVL, and he could not opine, to a reasonable degree of medical certainty, that Lugo's PVL had been caused by the mother's seizure.

When asked whether the positions taken in the Burns article were "generally accepted in the scientific community," Dr. Pavlakis responded by asserting that Lugo was not like the patients in the Burns article, who had "a lot of other issues going on," and had not experienced a short episode of hypoglycemia lasting even 1 1/2 hours. Like Dr. Jahre, Dr. Pavlakis testified that the Burns article had not been entirely successful in selecting a group of patients suffering purely from hypoglycemia, but he opined that the authors had done a good job of setting up their study and that he was not sure if a better study was possible.

Dr. Pavlakis testified that the medical literature discussed at the hearing, when considered in the aggregate, did not demonstrate that a child like Lugo who had a glucose level of 3 mg/dL for 1 hour and 21 minutes would develop PVL as a result, since none of the patients discussed in the ****273** literature had experienced a relatively short period of hypoglycemia before being discharged from the hospital without further problems. Therefore, according to Dr. Pavlakis, the theory of causation offered by the plaintiffs' experts was not scientifically accepted.

A running theme throughout the *Frye* hearing was whether the experts considered the medical literature they had reviewed ***53** to be " authoritative." Although both Dr. Katz and Dr. Peyster testified that they did not consider any of the literature they had discussed to be "authoritative," Dr. Katz testified that the Volpe textbook and the articles he had addressed were the sources he would consult for the current science in the areas discussed at the hearing. Dr. Peyster testified that he did not consider *any* medical literature, including his own book, to be "authoritative" because that term implied that everything in the article or study was correct and was not subject to any further changes. Dr. Peyster's reluctance to apply this label to medical literature was echoed by the defendant's expert Dr. Jahre, who agreed that this term was not used frequently to describe medical literature and that doctors relied upon articles not considered to be "authoritative" to assess the state of the science.

The Order and the Judgment Dismissing the Complaint

In an order entered December 15, 2009, the Supreme Court granted that branch of the defendant's motion which was for summary judgment dismissing the complaint after concluding that the plaintiffs' expert testimony regarding causation was inadmissible. In the order, the Supreme Court framed the issues to be resolved as: (1) whether the scientific community generally accepts that a short episode of hypoglycemia can cause PVL such as that shown on Lugo's MRI; and (2) whether the plaintiffs' experts could reasonably opine that Lugo's episode of hypoglycemia actually caused his injury. With respect to the first issue, the Supreme Court concluded that the plaintiffs had failed to demonstrate that it is generally accepted that hypoglycemia can cause PVL "as suffered by [Lugo]." In arriving at this determination, the Court highlighted the testimony of the defendant's experts that the patients studied in the Burns article could have suffered from hypoxic ischemic encephalopathy, and noted that the Volpe textbook stated that the topography of injuries associated with PVL differed "somewhat" from that observed with hypoxic ischemic injury. In addition, the Supreme Court concluded that Dr. Peyster's inability to label any of the medical literature he had reviewed as authoritative ran "counter" to a conclusion that the findings set forth therein were generally accepted in the scientific community.

With respect to the second issue, the Supreme Court asserted that "even if it were generally accepted that a hypoglycemic episode could cause [PVL], [the] plaintiff[s'] evidence fails to demonstrate a factual issue as to whether the hypoglycemic ***54** episode suffered by [Lugo] caused his brain injury." Addressing the factors Dr. Katz cited in support of his conclusion that Lugo's episode of hypoglycemia caused his injury, the Supreme Court concluded that, based on the testimony of the plaintiffs' experts, although Lugo's MRI did not exclude hypoglycemia as the cause of his injury, it also did not rule out other possible causes, such as hypoxia or ischemia. In addition, the Supreme Court concluded that nothing in the plaintiffs' evidence "address[ed]" Dr. Pavlakis's testimony that hypoxia and/or ischemia are the predom-

inant causes of PVL. The Supreme Court noted that none of the articles relied upon by the plaintiffs' experts addressed an episode****274** of hypoglycemia lasting 1 hour and 21 minutes, like that suffered by Lugo, and that Dr. Katz had conceded that the question of what duration and severity of blood glucose levels caused neurologic injury in humans is unclear. The Supreme Court acknowledged that, according to the Volpe textbook, the presence of seizures is a major indicator that an episode of hypoglycemia will result in neurological damage, but it rejected the assertion of the plaintiff's expert Dr. Trifiletti, set forth in his affirmation, that Lugo's post-birth tremors were consistent with subtle seizures as defined in the Volpe textbook, and that the seizures or tremors constituted evidence that the hypoglycemia caused neurological damage.

Addressing Dr. Katz's testimony that it was generally accepted that susceptibility to brain injury at a certain blood sugar level varies from individual to individual, the Supreme Court determined that Dr. Katz had provided "no indication" that Lugo was particularly susceptible to suffering such an injury from hypoglycemia. Additionally, the Supreme Court reasoned that although Dr. Katz testified that hypoglycemia is a toxic state that requires treatment regardless of the duration or blood sugar level, that testimony was inadequate to demonstrate causation in this matter. Finally, in response to Dr. Katz's testimony that there were no other possible causes of Lugo's injury, the Supreme Court noted Dr. Katz's concession that there were other possible causes of PVL, and that it was possible for Lugo to have been born with normal Apgar scores if the injury occurred in utero.

Based on the foregoing analysis, the Supreme Court concluded that the plaintiffs' experts had failed to demonstrate a foundation for their opinion that Lugo's episode of hypoglycemia caused his injury "in light of the evidence that perinatal ischemia or hypoxia is the overwhelming cause of [PVL]."

> ***55** "At best, even if [the] plaintiff[s'] experts have raised the possibility that hypoglycemia caused his injury, their testimony fails to sufficiently rule out other more likely possible causes, such as perinatal ischemia or hypoxia. It cannot be said, therefore, that [Lugo's] injury was, more likely than not, caused by the episode of hypoglycemia."

Thus, the Supreme Court reasoned that a jury verdict in favor of the plaintiffs would be "nothing more than speculation and guesswork," and the defendant was entitled to summary judgment dismissing the complaint because the plaintiffs had failed to raise a triable issue of fact regarding causation.

In a judgment entered February 1, 2010, upon the foregoing order, the Supreme Court dismissed the complaint. For the reasons that follow, we reverse the judgment.

Discussion

The Frye Test

[1] In determining the admissibility of expert testimony, New York follows the rule of *Frye v. United States*, 293 F. 1013 "that expert testimony based on scientific principles or procedures is admissible but only after a principle or procedure has 'gained general acceptance' in its specified field" (*People v. Wesley*, 83 N.Y.2d 417, 422, 611 N.Y.S.2d 97, 633 N.E.2d 451, quoting *Frye v. United States*, 293 F. at 1014; *see People v. Wernick*, 89 N.Y.2d 111, 115, 651 N.Y.S.2d 392, 674 N.E.2d 322; *Lipschitz v. Stein*, 65 A.D.3d 573, 575, 884 N.Y.S.2d 442; *Nonnon v. City of New York*, 32 A.D.3d 91, 101, 819 N.Y.S.2d 705, *affd. on other grounds* 9 N.Y.3d 825, 842 N.Y.S.2d 756, 874 N.E.2d 720; *Zito v. Zabarsky*, 28 A.D.3d 42, 44, 812 N.Y.S.2d 535; *see also* ****275***Giordano v. Market Am., Inc.*, 15 N.Y.3d 590, 601, 915 N.Y.S.2d 884, 941 N.E.2d 727). In *Frye*, the United States Court of Appeals for the District of Columbia Circuit concluded that expert testimony as to the results of a "systolic blood pressure deception test" was inadmissible because the test had not yet gained general acceptance and scientific recognition among physiological and psychological authorities (*Frye v. United States*, 293 F. at 1014). In so concluding, the *Frye* court articulated the following holding concerning expert opinion testimony based upon deductive reasoning:

> "Just when a scientific principle or discovery crosses the line between the experimental and demonstrable stages is difficult to define. Somewhere in this twilight zone the evidential force of the principle must be recognized, and while courts will go a long way in admitting expert testimony de-

duced from a ***56** well-recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs" (*id.*).

[2] In accordance with this holding, a *Frye* inquiry is directed at the basis for the expert's opinion and does not examine whether the expert's conclusion is sound. " *Frye* is not concerned with the reliability of a certain expert's conclusions, but instead with 'whether the experts' deductions are based on principles that are sufficiently established to have gained general acceptance as reliable' " (*Nonnon v. City of New York*, 32 A.D.3d at 103, 819 N.Y.S.2d 705, quoting *Marsh v. Smyth*, 12 A.D.3d 307, 308, 785 N.Y.S.2d 440; *see Lipschitz v. Stein*, 65 A.D.3d at 576, 884 N.Y.S.2d 442; *Alston v. Sunharbor Manor, LLC*, 48 A.D.3d 600, 602, 854 N.Y.S.2d 402; *DieJoia v. Gacioch*, 42 A.D.3d 977, 979, 839 N.Y.S.2d 904; *see also Ellis v. Eng*, 70 A.D.3d 887, 892, 895 N.Y.S.2d 462). Put another way, "[t]he court's job is not to decide who is right and who is wrong, but rather to decide whether or not there is sufficient scientific support for the expert's theory" (*Gallegos v. Elite Model Mgt. Corp.*, 195 Misc.2d 223, 225, 758 N.Y.S.2d 777). " '[G]eneral acceptance does not necessarily mean that a majority of the scientists involved subscribe to the conclusion. Rather it means that those espousing the theory or opinion have followed generally accepted scientific principles and methodology in evaluating clinical data to reach their conclusions' " (*Zito v. Zabarsky*, 28 A.D.3d at 44, 812 N.Y.S.2d 535, quoting *Beck v. Warner–Lambert Co.*, 2002 N.Y. Slip Op. 40431[U], *6–7, 2002 WL 31107923).

Thus, the limited purpose of the *Frye* test is to ascertain whether the expert's conclusion is based upon accepted scientific principles, rather than simply the expert's own unsupported beliefs (*see DieJoia v. Gacioch*, 42 A.D.3d at 980, 839 N.Y.S.2d 904; *Zito v. Zabarsky*, 28 A.D.3d at 46, 812 N.Y.S.2d 535; *see also Rowe v. Fisher*, 82 A.D.3d 490, 491, 918 N.Y.S.2d 342). As Justice Catterson of the Appellate Division, First Department, stated in his concurrence in *Styles v. General Motors Corp.*, 20 A.D.3d 338, 799 N.Y.S.2d 38, "[t]he *Frye* 'general acceptance' test is intended to protect [ ] juries from being misled by expert opinions that may be couched in formidable scientific terminology but that are based on fanciful theories" (*id.* at 342, 799 N.Y.S.2d 38 [internal quotation marks omitted]). Similarly, as stated by Justice Saxe of the Appellate Division, First Department, in his concurrence in *Marsh v. Smyth*, 12 A.D.3d 307, 785 N.Y.S.2d 440, "[t]he appropriate question for the court at ... a [*Frye*] hearing is the somewhat limited question of whether the proffered expert ***57** opinion properly relates existing data, studies or literature to the plaintiff's situation, or whether, instead, it ****276** is 'connected to existing data only by the ipse dixit of the expert' " (*id.* at 312, 785 N.Y.S.2d 440, quoting *General Elec. Co. v. Joiner*, 522 U.S. 136, 146, 118 S.Ct. 512, 139 L.Ed.2d 508).

Since 1923, when *Frye* was decided, New York courts have applied the *Frye* test to the results of scientific testing or measurement procedures (*see e.g. People v. Angelo*, 88 N.Y.2d 217, 644 N.Y.S.2d 460, 666 N.E.2d 1333 [polygraph test results]; *People v. Wesley*, 83 N.Y.2d 417, 611 N.Y.S.2d 97, 633 N.E.2d 451 [DNA profiling evidence]; *People v. Middleton*, 54 N.Y.2d 42, 444 N.Y.S.2d 581, 429 N.E.2d 100 [bite mark identification procedure]; *People v. Magri*, 3 N.Y.2d 562, 170 N.Y.S.2d 335, 147 N.E.2d 728 [use of radar device to measure speed]; *Styles v. General Motors Corp.*, 20 A.D.3d 338, 799 N.Y.S.2d 38 [procedure combining two separate automobile roof-stress tests]). In addition, the *Frye* test has been applied to assess the reliability of psychological or physiological theories or syndromes (*see e.g. People v. LeGrand*, 8 N.Y.3d 449, 835 N.Y.S.2d 523, 867 N.E.2d 374 [expert testimony on the reliability of eyewitness identifications]; *People v. Wernick*, 89 N.Y.2d 111, 651 N.Y.S.2d 392, 674 N.E.2d 322 [neonaticide syndrome]; *People v. Taylor*, 75 N.Y.2d 277, 552 N.Y.S.2d 883, 552 N.E.2d 131 [rape trauma syndrome]; *Oppenheim v. United Charities of N.Y.*, 266 A.D.2d 116, 698 N.Y.S.2d 144 [multiple chemical sensitivity syndrome]).

[3] New York courts have also applied the *Frye* test to assess the reliability of an expert's theory of causation in a particular case. For this category of expert opinion testimony, "it is not necessary 'that the underlying support for the theory of causation consist of cases or studies considering circumstances exactly parallel to those under consideration in the litigation. It is sufficient if a synthesis of various studies or cases reasonably permits the conclusion

reached by the plaintiff's expert' " (*Zito v. Zabarsky*, 28 A.D.3d at 44, 812 N.Y.S.2d 535, quoting *Marsh v. Smyth*, 12 A.D.3d at 312–313, 785 N.Y.S.2d 440 [Saxe, J., concurring]; *see* *DieJoia v. Gacioch*, 42 A.D.3d at 979, 839 N.Y.S.2d 904). "The fact that there [is] no textual authority directly on point to support the [expert's] opinion is relevant only to the weight to be given the testimony, but does not preclude its admissibility" (*Zito v. Zabarsky*, 28 A.D.3d at 46, 812 N.Y.S.2d 535; *see* *DieJoia v. Gacioch*, 42 A.D.3d at 979, 839 N.Y.S.2d 904).

Accordingly, this Court has affirmed the preclusion of expert testimony as to causation in circumstances where there was a complete absence of any literature or studies supporting the particular causation theory espoused by the expert. For example, in *Cumberbatch v. Blanchette*, 35 A.D.3d 341, 825 N.Y.S.2d 744, the plaintiff's expert could cite to no relevant scientific data or studies to support his causation theory that fetal distress resulting***58** from the compression of the infant plaintiff's head due to labor contractions, augmented by Pitocin, resulted in ischemia, which, in turn, resulted in an infarction, and he could cite to no instance when this type of injury had previously occurred in that manner (*id.* at 342, 825 N.Y.S.2d 744). Thus, this Court concluded that the opinion of the plaintiff's expert was scientifically unreliable (*id.* at 342–343, 825 N.Y.S.2d 744). Similarly, in *Lewin v. County of Suffolk*, 18 A.D.3d 621, 795 N.Y.S.2d 659, the plaintiffs' experts conceded that no scientific organization or national board has expressly recognized a causal relationship between in utero exposure to the pesticide Malathion and birth defects, and the peer-reviewed scientific articles and textbooks relied upon by the plaintiffs' experts did not establish the existence of such a relationship ****277**(*id.* at 622, 795 N.Y.S.2d 659). Under those circumstances, this Court concluded that the methodology employed by the plaintiffs' experts in correlating such exposure to birth defects was "fundamentally speculative" and that the Supreme Court had properly precluded the plaintiffs' experts from testifying (*id.*). And in *Hooks v. Court St. Med., P.C.*, 15 A.D.3d 544, 790 N.Y.S.2d 679, the plaintiff's expert could not cite to any relevant scientific data or studies showing a causal link between the misuse of an electric muscle-stimulating unit and glossopharyngeal neuralgia to support his theory that the improper placement of electrodes of an electrical muscle-stimulating unit on the anterior neck of a patient can cause permanent nerve damage, and he could cite to no instance when that type of injury had previously occurred in that manner (*id.* at 545, 790 N.Y.S.2d 679). Accordingly, this Court determined that the expert's opinion was scientifically unreliable (*id.*).

Standing in sharp contrast are cases in which the expert's opinion satisfied the *Frye* test because it was deduced from generally accepted scientific principles and supported by existing data or literature, although the expert could not point to a case or study involving circumstances exactly parallel to those at issue in the litigation to support his or her theory of causation. For instance, in *DieJoia v. Gacioch*, 42 A.D.3d 977, 839 N.Y.S.2d 904, the Appellate Division, Fourth Department, concluded that the Supreme Court had applied the *Frye* test too restrictively in precluding the plaintiff's experts from testifying that a cardiac catheterization in the plaintiff's groin was the cause of the plaintiff's aortic thrombosis, which led to an acute spinal cord infarct and paralysis (*id.* at 977–978, 839 N.Y.S.2d 904). Although the experts did not produce medical literature documenting a prior case study in which cardiac catheterization through the groin was the cause ***59** of aortic thrombosis that led to an acute spinal cord infarct and paralysis or linking a cardiac catheterization in the groin to these injuries, the conclusions of the plaintiff's experts were nonetheless deemed admissible under *Frye* because they were based on accepted scientific principles involving medicine and the vascular system and were not based solely upon the experts' own unsupported beliefs (*id.* at 979–980, 839 N.Y.S.2d 904). Similarly, in *Zito v. Zabarsky*, 28 A.D.3d 42, 812 N.Y.S.2d 535, the opinion testimony of the plaintiff's expert that there was a causal connection between an allegedly excessive dose of Zocor, a cholesterol-lowering drug, and the onset of polymyositis, was precluded by the Supreme Court, which concluded that the *Frye* test could not be satisfied without medical literature expressly reporting a connection between an excessive dose of Zocor and the onset of the disease (*id.* at 44–45, 812 N.Y.S.2d 535). This Court concluded that the Supreme Court's application of the *Frye* test was "overly restrictive" because the plaintiff's experts had supported their theory of a causal nexus between an excessive dose of Zocor and polymyositis with generally accepted scientific principles and existing data, including a case study documenting a patient who had been diagnosed with polymyositis after being prescribed a generic form of Zocor at a dosage different than that prescribed to the plaintiff (*id.* at 45, 812 N.Y.S.2d 535). This Court held that the theory of

causation of the plaintiff's experts "was based upon more than theoretical speculation, or a scientific 'hunch,' " and that the lack of textual authority directly on point pertained to the weight to be given to the experts' testimony, but did not preclude its admissibility (*id.* at 46, 812 N.Y.S.2d 535).

****278** [4] Here, too, the plaintiffs demonstrated that their experts' theory of causation was based upon generally accepted scientific principles, as was their burden (*see Del Maestro v. Grecco*, 16 A.D.3d 364, 791 N.Y.S.2d 139), and in concluding that this opinion testimony was inadmissible, the Supreme Court applied the Frye test too restrictively. At the *Frye* hearing, the plaintiffs' expert Dr. Katz explained that his conclusion that an episode of hypoglycemia lasting 81 minutes at a level of 3 mg/dL could cause neurologic damage of the type sustained by Lugo, i.e., PVL, was based on several generally accepted scientific principles: namely, that hypoglycemia causes brain injury, that certain infants are more susceptible than others to neurologic injury, and that hypoglycemia is a toxic and dangerous state with no safe level. The defendant's experts did not dispute the general acceptance of the foregoing scientific principles. To the contrary, the defendant's ***60** expert Dr. Pavlakis confirmed that it was generally accepted that hypoglycemia can cause brain damage, that the scientific community does not recognize any level or duration of hypoglycemia considered safe and incapable of causing brain damage, and that individual susceptibility to toxic states varies among newborns.

In addition, the plaintiffs' expert Dr. Peyster explained that PVL was simply a term that refers to damage to the deep white brain matter next to the ventricles which appears as an abnormality on an MRI brain **scan**, and the evidence presented at the ***Frye*** hearing established general acceptance of the scientific principle that hypoglycemia can cause PVL. Both Drs. Katz and Peyster testified that their opinion that hypoglycemia can cause PVL was supported by the Volpe textbook, which discusses neuropathic studies indicating that hypoglycemia is a precedent of PVL. Dr. Katz characterized the Volpe textbook as a "well written outline" of certain neonatal neurologic principles, although he acknowledged that not everyone agreed with all of its conclusions, and Dr. Peyster characterized the Volpe textbook as the best text he knew of on the topic of pediatric neurology. These assessments of the Volpe textbook were not challenged by the defendant's experts. In addition, Dr. Jahre's testimony that hypoglycemia can cause brain damage in the form of white matter damage against the ventricles provided further evidence of the acceptance of the general principle that hypoglycemia can cause PVL. Although the defendant's expert Dr. Pavlakis opined that PVL is almost always caused by a decrease of blood flow or oxygen to a baby between 28 and 40 weeks of age, he cited to no medical literature or case studies to support this specific assertion, and even he acknowledged that hypoglycemia can cause brain abnormalities discernable on an MRI film.

Concededly, the plaintiffs' experts failed to produce a case or study reporting an occurrence of PVL in circumstances exactly parallel to those at issue here—i.e., after a single episode of neonatal hypoglycemia at a level of 3 mg/dL lasting 81 minutes, or any literature expressly supporting their theory that such an episode of hypoglycemia could result in PVL. Nevertheless, the plaintiffs demonstrated that their theory of causation was reasonably permitted by a synthesis of the medical literature discussed at the hearing (*see DieJoia v. Gacioch*, 42 A.D.3d at 979, 839 N.Y.S.2d 904; *Zito v. Zabarsky*, 28 A.D.3d at 44, 812 N.Y.S.2d 535; *Marsh v. Smyth*, 12 A.D.3d at 312–313, 785 N.Y.S.2d 440). Although the Burns article was not designed to test the relationship between the severity or duration of ***61**hypoglycemia and neurodevelopmental outcomes, it limited its study to patients who had experienced neonatal hypoglycemia and excluded those who had ****279** suffered from other conditions, such as hypoxic ischemia, and it determined that 94% of the subjects studied, 63% of whom had only experienced one episode of hypoglycemia, had evidence of white matter abnormalities on their MRI brain scans. Although the Kinnala article had excluded infants who had experienced only one episode of hypoglycemia prior to six hours of age, it also documented a patient who had experienced an episode of hypoglycemia at seven hours of age which lasted two hours at a minimum glucose level of 32 mg/dL, a level "dramatically" higher than Lugo's glucose level of 3 mg/dL during his episode of hypoglycemia. That patient had shown evidence of neurologic injury on an MRI, although that abnormality had subsequently resolved. Finally, the Alkalay article, which reviewed the Kinnala article and 15 others, concluded that plasma glucose levels of less than 25 mg/dL of several hours' duration—again, a level far higher than that experienced by Lu-

go—may increase the relative risk for adverse neurologic outcome.

To be sure, none of the foregoing articles, read in isolation, provides conclusive support for the theory of causation espoused by the plaintiffs' experts. However, when considered in the aggregate for the limited purpose of applying the *Frye* test, and against the backdrop of the undisputed generally accepted principles concerning hypoglycemia set forth at the hearing, those articles establish that this theory was properly based upon far more than theoretical speculation or a scientific "hunch" (*see Zito v. Zabarsky*, 28 A.D.3d at 46, 812 N.Y.S.2d 535). Synthesized, the materials produced by the plaintiffs' experts at the *Frye* hearing provided an objective basis for their opinion that a period of severe hypoglycemia of relatively short duration can cause neurologic injury reflected as PVL on a MRI brain scan. The absence of medical literature directly on point with the circumstances at bar pertains to the weight to be given to this opinion testimony, but does not preclude its admissibility (*see DieJoia v. Gacioch*, 42 A.D.3d at 979, 839 N.Y.S.2d 904; *Zito v. Zabarsky*, 28 A.D.3d at 46, 812 N.Y.S.2d 535).

In concluding that the opinion testimony of the plaintiffs' experts did not satisfy the *Frye* test, the Supreme Court emphasized the fact that those experts were unable to characterize the literature upon which they relied as "authoritative." Seemingly, the Supreme Court ascribed significance to the experts' willingness to apply this label while disregarding the ***62** hearing testimony that the term "authoritative" is not generally applied to medical literature and that the materials discussed at the hearing represented the current science with regard to brain injuries resulting from neonatal hypoglycemia.

[5] We agree with Justice Saxe that when the *Frye* test is applied to a theory of causation, "the court's concern must be limited to making sure that within the scientific field in question, there is a substantive, demonstrable, objective basis for the expert's conclusion," and that "[t]he focus of the inquiry in such an instance should not be upon how widespread the theory's acceptance is, but should instead consider whether a reasonable quantum of legitimate support exists in the literature for the expert's views" (*Marsh v. Smyth*, 12 A.D.3d at 312, 785 N.Y.S.2d 440). In this case, the plaintiffs' experts amply demonstrated the existence of such a basis for their theory of causation, and in precluding their opinion testimony, the Supreme Court applied the *Frye* test in an overly restrictive manner. Both the plaintiffs' experts and the defendant's experts agree that an episode of severe glucose deprivation in a newborn can cause neurologic****280** damage; the principal dispute between them, which was emphasized by the testimony at the *Frye* hearing, is over how long such an episode must last before neurologic damage results. This factual disagreement should not have been resolved as a matter of law by the Supreme Court in the course of its *Frye* inquiry.

The purpose of the *Frye* test is not to preclude expert opinion testimony based upon reasonable extrapolations from conceded legitimate empirical data. It would be as unreasonable to preclude a 45–year smoker from seeking recovery if the only available empirical data addressed 50–year smokers as it was to preclude the instant plaintiffs' experts from testifying, based on their reasonable extrapolations from existing legitimate empirical data, that Lugo's severe episode of neonatal hypoglycemia caused his brain injuries.

Foundation

[6] In addition, we disagree with the Supreme Court's conclusion that the theory of causation espoused by the plaintiffs' experts lacked an adequate foundation for admissibility. "The *Frye* inquiry is separate and distinct from the admissibility question applied to all evidence—whether there is a proper foundation—to determine whether the accepted methods were appropriately employed in a particular case" (*Parker v. Mobil Oil Corp.*, 7 N.Y.3d 434, 447, 824 N.Y.S.2d 584, 857 N.E.2d 1114; *see People v. Wesley*, 83 N.Y.2d at 428–429, 611 N.Y.S.2d 97, 633 N.E.2d 451; *Jackson v. Nutmeg Tech., Inc.*, 43 A.D.3d 599, 601, 842 N.Y.S.2d 588). ***63** "The focus moves from the general reliability concerns of *Frye* to the specific reliability of the procedures followed to generate the evidence proffered and whether they establish a foundation for the reception of the evidence at trial" (*People v. Wesley*, 83 N.Y.2d at 429, 611 N.Y.S.2d 97, 633 N.E.2d 451). "The foundation ... should not include a determination of the court that such evidence is true. That function should be left to the jury" (*id.* at 425, 611 N.Y.S.2d 97, 633 N.E.2d 451).

[7] Here, the level (3 mg/dL) and duration (81 minutes) of Lugo's hypoglycemia episode were precisely quantified by the plaintiffs' experts at the *Frye* hearing (*cf. Parker v. Mobil Oil Corp.*, 7 N.Y.3d at 449–450, 824 N.Y.S.2d 584, 857 N.E.2d 1114), and the Supreme Court did not conclude that these measurements were unreliable. In addition, the plaintiffs' experts made specific reference to the contents of numerous articles documenting brain MRI abnormalities in patients who had experienced hypoglycemia to support their opinion that there was a causal connection between Lugo's episode of hypoglycemia and the brain abnormalities later observed on his MRI film (*see Jackson v. Nutmeg Tech., Inc.*, 43 A.D.3d at 602, 842 N.Y.S.2d 588). Under these circumstances, we conclude that the Supreme Court improvidently exercised its discretion in concluding that the plaintiffs' experts failed to proffer sufficient foundational evidence to support the admissibility of their testimony at trial.

The Supreme Court's conclusion that the opinion of the plaintiffs' experts lacked an adequate foundation rested largely on its findings that the evidence presented at the *Frye* hearing established that perinatal ischemia or hypoxia is the overwhelming cause of PVL and that the testimony of the plaintiffs' experts did not eliminate other "more likely possible causes" of Lugo's PVL. In relying upon such reasoning, the Supreme Court, in effect, rendered an assessment as to the ultimate merit of the opinion testimony of the plaintiffs' experts (*see People v. Wesley*, 83 N.Y.2d at 425, 611 N.Y.S.2d 97, 633 N.E.2d 451). Clearly, numerous factual disagreements between the parties' experts were highlighted****281** at the *Frye* hearing, including, but not limited to, the specific appearance of Lugo's brain MRI abnormalities and their cause. However, these factual disagreements go to the weight to be accorded to the testimony of the plaintiffs' experts by the trier of fact, and not the admissibility of such testimony (*see Jackson v. Nutmeg Tech., Inc.*, 43 A.D.3d at 602, 842 N.Y.S.2d 588).

Summary Judgment

[8] Finally, in light of our determination that the theory of causation espoused by the plaintiffs' experts is admissible at trial, we conclude that the Supreme Court improperly granted that ***64** branch of the defendant's motion which was for summary judgment dismissing the complaint. Briefly, although the defendant's expert submissions established, prima facie, that Lugo's brain damage was not caused by his episode of neonatal hypoglycemia, the plaintiffs, in opposition, raised a triable issue of fact on this point through the submission of admissible expert opinion evidence (*see generally Alvarez v. Prospect Hosp.*, 68 N.Y.2d 320, 324, 508 N.Y.S.2d 923, 501 N.E.2d 572; *Zuckerman v. City of New York*, 49 N.Y.2d 557, 562, 427 N.Y.S.2d 595, 404 N.E.2d 718). Thus, under the particular circumstances of this case, the Supreme Court should have denied that branch of the defendant's motion which was for summary judgment dismissing the complaint.

The appeal from the intermediate order must be dismissed because the right of direct appeal therefrom terminated with the entry of judgment in the action (*see Matter of Aho*, 39 N.Y.2d 241, 248, 383 N.Y.S.2d 285, 347 N.E.2d 647). The issues raised on the appeal from the order are brought up for review and have been considered on the appeal from the judgment (*see* CPLR 5501[a][1]).

Accordingly, the judgment is reversed, on the law, that branch of the defendant's motion which was for summary judgment dismissing the complaint is denied, and the order is modified accordingly.

ORDERED that the appeal from the order is dismissed; and it is further,

ORDERED that the judgment is reversed, on the law, that branch of the defendant's motion which was for summary judgment dismissing the complaint is denied, and the order is modified accordingly; and it is further,

ORDERED that one bill of costs is awarded to the appellants.

RIVERA, J.P., FLORIO and LOTT, JJ., concur.

N.Y.A.D. 2 Dept.,2011.
Lugo v. New York City Health and Hospitals Corp.
89 A.D.3d 42, 929 N.Y.S.2d 264, 2011 N.Y. Slip Op. 06475

END OF DOCUMENT

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University of Rochester
1966-1967

TEACHING RESPONSIBILITIES:

University of Medicine and Dentistry/Rutgers Medical School
Second Year Medical Interview Course
Approximately four hours a week
(From 1992 through the present)

Preceptor of "The Chronically Ill and Dying Patient,"
Course Co-Sponsored by Yale Schools of Medicine, Law, Public Health and Divinity
Approximately four hours a week
(From 1974-1977)

University of Medicine and Dentistry of New Jersey
Preceptor, Mock Psychiatry Board Examination
April 11, 2003 and others

PUBLICATIONS:

A. Refereed Original Articles in Journals:

1. Brown, Jeffrey A., "How Recent Legislation Will Affect the Future of C.R.N.A. Professionalism." 44(1) *AANA Journal* 54, 1976.

2. Brown, Jeffrey A., "Towards Managing Conflict on the Anesthesia Care Team," 45(1) *AANA Journal* 15, 1977.

3. Brown, Jeffrey A., "Core Issues of Unionization: Your Ten Most Frequently Asked Questions Answered," 48(1) *AANA Journal* 26, 1980.

4. Brown, Jeffrey A. and Greenfield, Daniel P., Editorial: "What About Prozac?" 89 (6) *New Jersey Medicine*: 445-446, (June) 1992.

5. Brown, Jeffrey A., Witt, Philip H., Greenfield, Daniel P., Editorial: "The Diagnosis and Management of Depression: An Overview," 89 (5) *New Jersey Medicine*, 395-400, (June) 1992.

6. Brown, Jeffrey A. and Greenfield, Daniel P., "Alcoholism and Depression: Three Case Studies," 6 (4) *Clinical Advances in the Treatment of Psychiatric Disorders*: 1-3, 11, (October) 1992.

7. Brown, Jeffrey A. and Greenfield, Daniel P., "What to Expect from a Psychiatric Consultation," 90 (2) *New Jersey Medicine*: 139-141 (February) 1993.

8. Brown, Jeffrey A. and Greenfield, Daniel P., "Psychological Aspects of Hysterectomy: A Case Study," 2 (2) *Women's Psychiatric Health:* 1-2, 12, (Spring) 1993.

9. Brown, Jeffrey A. and Greenfield, Daniel P., "The Use of Triazolam," 7 (2) *Clinical Advances in the Treatment of Psychiatric Disorders*: 4-6 (April) 1993.

10. Brown, Jeffrey A. and Greenfield, Daniel P., "Medicolegal Aspects of Treating Drug and Alcohol Addiction," *New Jersey Medicine*: 11 (90), (November) 1993.

11. Brown, Jeffrey A. and Greenfield, Daniel P., "Physician Compensation: What Doctors Want," *The Journal of Medical Practice Management* 12(6):1-7 (May/June) 1997.

12. Mahalick, David M., Carmel, Peter W., Greenberg, John P., Molofsky, W., Brown, Jeffrey A., Heary, Robert F., Marks, David, Zampella, Edward, Hodosh, Richard, and von der Schmidt, Edward, “Psychopharmacologic Treatment of Acquired Attention Disorders in Children with Brain Injury,” *Pediatric Neurosurgery*, 29(3):121-126 (September) 1998.

13. Brown, Jeffrey A. and Scott-Roiter, Alexis E., "Physician Practice Management Companies: Should Physicians Be Scared?" *The Journal of Medical Practice Management,* 14(5):245-249, March/April 1999.

14. Brown, Jeffrey A. and Dayle, Randy, "The ISSAC Cognitive Prosthetic System and Its Usefulness in Neurofunctional Rehabilitation," 15(1) *Rehab Pro*: 32-33 (2007).

B. Books, Monographs, and Chapters:

1. Brown, Jeffrey A., Roseman, Cyril, Kaufman, S. Joel, and Savitsky, Elaine R., State Legislative Action for Promoting Systematic Change in Health Care Delivery, Sacramento, California, Assembly Office of Research, 1971.

2. Brown, Jeffrey A., "Diagnosing and Rehabilitating the Medical Marketplace," Bennett Political Science Prize-winning research paper on the "Business and Politics of Health Care in America," University of California (Berkeley) Archives, May 1971.

3. Brown, Jeffrey A., Public Utility Regulation of Health Maintenance Organizations in Connecticut, New Haven, Connecticut, Yale Legislative Services, 1974.

4. Brown, Jeffrey A., "Interprofessional Conflict and Cooperation," Seymour Lustman Research Prize-winning paper, Yale Medical School Department of Psychiatry, May 1975.

5. Brown, Jeffrey A., "Towards Managing Conflict on the Anesthesia Care Team," 45(1) AANA Journal 15, 1977.

6. Brown, Jeffrey A. and Greenhouse, Lorrie, Approaching the Bench: A Practice Book for Connecticut Protective Services, Storrs, Connecticut: University of Connecticut Press, 1978.

7. Brown, Jeffrey A. and Greenfield, Daniel P., "Current Medicolegal Status of Prescribing Benzodiazepines: A Special Case," in Greenfield, Daniel P., Prescription Drug Abuse and Dependence: How Prescription Drug Abuse Contributes to the Drug Abuse Epidemic (Springfield, Illinois: Charles C. Thomas, 1995).

8. Brown, Jeffrey A. and Greenfield, Daniel P., "Interviewing the Difficult Patient," in Greenfield, Daniel P. (ed.), Prescription Drug Abuse and Dependence: How Prescription Drug Abuse Contributes to the Drug Abuse Epidemic (Springfield, Illinois: Charles C. Thomas, 1995).

9. Brown, Jeffrey A. and Greenfield, Daniel P., "Psychopharmacology," published in Price, David R. (ed.), The Insurer's Handbook of Psychological Claims (Washington, D.C.: Insurance Week Publications, 1995).

10. Boston, Gerald W., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 1998).

11. Boston, Gerald W., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 1999-2000 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2000).

12. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2002 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2002).

13. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2003 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2003).

14. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2004 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2004).

15. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2005 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2005).

16. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2006 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2006).

17. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2006).

18. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2007 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2007).

19. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries: 2007 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2007).

20. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2008 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2008).

21. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries: 2008 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2008).

22. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2009 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2009).

23. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries; 2009 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2009).

24. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2010 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2010).

25. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries; 2010 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2010).

26. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2011 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2011).

27. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries; 2011 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2011).

28. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2012 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2012).

29. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries; 2012 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company).

30. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2013 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2013).

31. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries; 2013 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2013).

32. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2014 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2014).

33. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries; 2014 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2014).

34. Dotson, Mark A., Kline, David B. and Brown, Jeffrey A., Emotional Injuries: Law and Practice: 2015 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2015).

35. Stern, Bruce and Brown, Jeffrey A., Litigating Brain Injuries; 2015 Supplement (Eagan, Minnesota: Thomson-Reuters West Publishing Company, 2015).

C. Other articles (review, editors, etc.) in Journals: Chapters, Books; other Professional Communications:

1. Brown, Jeffrey A., Letter to the Editor, 286 *The New England Journal of Medicine* 491, 1972.

2. Brown, Jeffrey A., Book Review of Contemporary Issues in Bioethics, 6(12) *Legal Aspects of Medical Practice* 39, 1978.

3. Brown, Jeffrey A. and Greenfield, Daniel P., "Weighing Psychiatric Claims in P.I. Cases," 124 *New Jersey Law Journal* 1344, 1989.

4. Brown, Jeffrey A. and Greenfield, Daniel P., "Managing Expert Psychiatric Testimony," 8(2) New Jersey Defense Association Newsletter 1, 1989.

5. Brown, Jeffrey A. and Sarno, John, "Let the Community Health Law Project Live," 25 *New Jersey Law Journal* 759, 1990.

6. Brown, Jeffrey A., Greenfield, Daniel P. and Miller, David, "Guest Editorial: National Mental Health Month," 89 (10) *New Jersey Medicine*: 741-2, (October) 1992.

7. Brown, Jeffrey A. and Greenfield, Daniel P., "Viewpoint: Financial Opportunities in Alternative Mental Health Delivery Systems and the `O/E' Model For Monitoring," *American Hospital Association News:* 6, (November) 1993.

8. Brown, Jeffrey A. and Greenfield, Daniel P., "Editorial: The Three Mile Island Health Alliance Company," *New Jersey Medicine* 91(3): 153-54, 1994.

9. Brown, Jeffrey A. and Kruszewski, Stefan, “Front Page Pill Pushers: How the Media Are Complicit in Drug Marketing,” 331 *British Medical Journal* 410 (13 August 2005).

PROFESSIONAL PRESENTATIONS

1. Brown, Jeffrey A. and Cohen, Ian M., "Women on Methadone," presented at the Convocation of the New York City Health Department, 18 August 1968.

2. Brown, Jeffrey A., "Managing Madness," simulation presented to the citizens Advisory Council of the Local Mental Health Advisory Boards, Sacramento California, 14 May 1971.

3. Brown, Jeffrey A., "C.R.N.A. Licensure: Pros and Cons," presented at the New York State Association of Nurse Anesthetists' Annual Meeting, 14 December 1975.

4. Brown, Jeffrey A., "The Hearsay Rule: Its Use and Abuse in Child Abuse Proceedings," presented at the Yale-New Haven Hospital's Departments of Medical and Surgical Social Services, 13 May 1976.

5. Brown, Jeffrey A., "Using Role Playing to Clarify Role Ambiguities," presented to the Yale-New Haven Hospital's Departments of Medical and Surgical Social Services, 13 May 1976.

6. Brown, Jeffrey A., "Evaluating Evidence in Child Neglect and Abuse Cases," presented at the New Haven Regional Office, State of Connecticut Departments of Welfare and of Children and Youth Services, 7 June 1976.

7. Brown, Jeffrey A., "Interprofessional Conflict," presented at the American Association of Nurse Anesthetists' 43rd Annual Meeting, Clinical Session, and Graduate Course, San Francisco, California, 25 August 1976.

8. Brown, Jeffrey A., "Legal and Psychiatric Issues in Child Protection," Grand Rounds, Mt. Sinai Hospital, Hartford, Connecticut, 15 December 1976.

9. Brown, Jeffrey A., "Child Protection and the Psychotic Parent," Grand Rounds, Mt. Sinai Hospital, Hartford, Connecticut, 15 December 1977.

10. Brown, Jeffrey A., "Psychiatry in Court: Indications and Contraindications," presented at the Psychiatry Clinic Community Conference, Norwalk Hospital, Norwalk, Connecticut, 25 April 1978.

11. Brown, Jeffrey A., "Evaluating Child Abuse," presented at the Mid-Fairfield Child Guidance Clinic, 22 December 1978.

12. Brown, Jeffrey A., "Unionization: Indications and Contraindications," presented at the New York State Association of Nurse Anesthetists' Annual Meeting, Albany, New York, 28 April 1979.

13. Brown, Jeffrey A., "Approaching the Mentally Retarded: Stereotypes, Symptoms and Solutions," presented at the Society to Advance the Retarded, Norwalk, Connecticut, 16 July 1979.

14. Brown, Jeffrey A., "Interviewing the Mentally Retarded," Workshop for C.E.T.A. Trainees, presented at the Society to Advance the Retarded, Norwalk, Connecticut, 20 August 1979.

15. Brown, Jeffrey A., "Evaluating Emotional Problems of the Mentally Retarded," presented to the Society to Advance the Retarded, Norwalk, Connecticut, 22 January 1980.

16. Brown, Jeffrey A., "Geriatric Psychiatry: Depression and the Aged," presented at WSTC, 10 May 1982.

17. Brown, Jeffrey A., "Stresses of Relocation: Psychiatric and Legal Complications for Realtors and Clients," presented at the William Pitt Real Estate Symposium, New Canaan, Connecticut, 7 February 1984.

18. Brown, Jeffrey A., "Crisis Intervention: Overview and Applications for Hostage Negotiation, Child Abuse, and Prison Management," presented at the Connecticut Justice Academy, East Haddam, Connecticut, 26 March 1984.

19. Brown, Jeffrey A., "Drug Abuse as Escape," presented at the First Congregational Church of Darien, Darien, Connecticut, 14 October 1984.

20. Brown, Jeffrey A., "Possible Proposed Legislation for `Step-Down' Facilities," presented to the New Jersey Drug Abuse Advisory Council of the New Jersey State Department of Health, Princeton, New Jersey, 13 September 1988.

21. Brown, Jeffrey A., "Managed Health Care, EAPs and Addiction Services: Surviving the 1990's," presented at Muhlenberg Regional Medical Center, Plainfield, New Jersey, 15 March 1989.

22. Brown, Jeffrey A., "Psychiatry and the Law," presented to the New Jersey Defense Association, Woodbridge, New Jersey, 18 March 1989.

23. Brown, Jeffrey A., "The Violent Teen: Psychiatric, Legal and Administrative Issues," presented to the Morris County Youth Services Advisory Committee, Parsippany, New Jersey, 6 June 1989.

24. Brown, Jeffrey A. and Greenfield, Daniel P., "Arguing and Defending Against Psychiatric Claims," presented to the Middlesex County Trial Lawyers and Bar Associations, Edison, New Jersey, 20 September 1989.

25. Brown, Jeffrey A., Hagovsky, Mathias R, Harper, John J., Ryan, Sharon W., Simon Sheldon M., and Strober-Lovett, Lynne, "Visitation and Custody After Divorce," presented to the New Jersey State Bar Association Family Law Section, Morristown, New Jersey, 11 October 1989.

26. Brown, Jeffrey A., "Managed Mental Health Quality Assurance, Utilization Review, and Risk Management," presented to New Jersey Blue Cross, Newark, New Jersey, 19 December 1989.

27. Brown, Jeffrey A., Greenfield, Daniel P. and Ryan, Sharon W., "Child Abuse and Substance Abuse," presented to the New Jersey Bar Association, Family Law Section, Paradise Island, Nassau, 17 January 1990.

28. Brown, Jeffrey A. and Greenfield, Daniel P., "Roving Symposium; Psychiatry, Medicine and the Law," presented to the New Jersey Academy of Medicine, Passaic, New Jersey, 23 January 1990.

29. Brown, Jeffrey A. and Greenfield, Daniel P., "Psychiatry and the Juvenile Offender," presented on TV-32, East Orange, New Jersey, 2 February 1990.

30. Brown, Jeffrey A., "Forensics for Psychologists: Uses and Limitations of Psychological Tests in Personal Injury and Family Relations Matters," presented to the Morris County Psychologists Association, Morristown, New Jersey, 14 February 1990.

31. Brown, Jeffrey A., Grecian, Andrea, and Hodes, Robert D., "Divorce Custody Disputes," presented on TV-32, East Orange, New Jersey, 26 February 1990.

32. Brown, Jeffrey A. and Horowitz, Philip N., "A Child Custody Primer for Neophyte Attorneys," presented at the New Jersey Institute for Continuing Legal Education Family Law Course, Newark, New Jersey, 3 March 1990.

33. Brown, Jeffrey A., "Psychiatric Evaluation of Child Custody Issues," presented to the Women Lawyers of Union County, Mountainside, New Jersey, 12 March 1990.

34. Brown, Jeffrey A. and Greenfield, Daniel P., "Medical and Neuropsychiatric Diagnostics: Neuropsychiatric Resources for Proof of Etiology and Causation of Serious Injuries," presented to the New Jersey Trial Lawyers Association, Atlantic City, New Jersey, 20 April 1990.

35. Brown Jeffrey A. and Greenfield, Daniel P., "Law and Psychiatry Grand Rounds: Recent Clinical Trends in Evaluating Testamentary Capacity, Alcohol-Influenced Behavior, and Post-Divorce Child Custody Disputes," presented at St. Clare's Hospital, Denville, New Jersey, 5 May 1990.

36. Brown, Jeffrey A., "Psychiatry and the Law: Case Discussion of Chemical Dependency, Child Abuse, and Competency," Grand Rounds presented at Elizabeth General Medical Center, Elizabeth, New Jersey, 20 November 1990.

37. Brown, Jeffrey A. and Greenfield, Daniel P., "CDS Prescribing Practices: Legal/Illegal," New Jersey Academy of Medicine Roving Symposium presented at the Essex County Hospital Center, Cedar Grove, New Jersey, 13 March 1991.

38. Brown, Jeffrey A., "Involuntary Medication: Clinical and Legal Issues," presented to the Elizabeth General Hospital Department of Psychiatry Clinical Conference, Elizabeth, New Jersey, 15 March 1991.

39. Brown, Jeffrey A. and Greenfield, Daniel P., "Somatization, Hysteria, and Faking," presented to the Jersey Association of Rehabilitation Professionals in the Private Sector, Jamesburg, New Jersey, 20 March 1991.

40. Brown, Jeffrey A., "The Uses and Misuses of Psychiatry in Court," presented to the Camden County Bar Association, Camden, New Jersey, 26 March 1991.

41. Brown, Jeffrey A., "Parental Alienation & `Brainwashing,'" presented to the Essex County Bar Association, Montclair, New Jersey, 3 April 1991.

42. Brown, Jeffrey A. and Greenfield, Daniel P., "Why Life is Toxic: Medical Causation Analysis in Toxic Tort Cases," presented to the New Jersey Trial Lawyers Association Annual Meeting, Atlantic City, New Jersey, 27 April 1991.

43. Brown, Jeffrey A., "Involuntary Medication II: Cases and Competency," presented to Elizabeth General Hospital Department of Psychiatry Clinical Conference, Elizabeth, New Jersey, 3 May 1991.

44. Brown, Jeffrey A. and Greenfield, Daniel P., "Head Injury and Psychiatric Cases," presented at the New Jersey Bar Association Annual Meeting, Atlantic City, New Jersey, 17 May 1991.

45. Brown, Jeffrey A., "Analyzing the Analysts: Experts' Uses and Limitations in Domestic Relations Matters," presented to the Ocean County Bar Association, Ocean County Justice Complex, 1 June 1991.

46. Brown, Jeffrey A., "Managed Care and the Future of Psychiatric Practice," presented to the St. Barnabas Hospital Department of Psychiatry, Livingston, New Jersey, 26 June 1991.

47. Brown, Jeffrey A., "Risk Management and the Public Sector," presented to the State of Hawaii Department of Mental Health, Kaneohe, Hawaii, 19 August 1991.

48. Brown, Jeffrey A. and Barry, Alan D., "Avoiding Bankruptcy," presented at the Association of Mental Health Administrators 1991 Annual Meeting, San Diego, California, 22 September 1991.

49. Brown, Jeffrey A. and Greenfield, Daniel P., "Stress Management for Senior Executives," presented at the Uniglobe Northeast Owners Meeting, Southampton, Bermuda, 18 October 1991.

50. Brown, Jeffrey A. and Greenfield, Daniel P., "Head Injury, Depression and Cognitive Hysteria," presented to Rehabilitation Specialists, Hawthorne, New Jersey, 31 January 1992.

51. Brown, Jeffrey A., Sherer, Arlene, and Wilson, George, "Managed Health Care," presented to the Tri-County Chapter of the New Jersey Psychiatric Association, Summit, New Jersey, 12 February 1992.

52. Brown, Jeffrey A. and Greenfield, Daniel P., "The Clinical and Administrative Management of Head and Body Injury Cases," presented to Travelers Insurance Company, Parsippany, New Jersey, 12 March 1992.

53. Brown, Jeffrey A., "`I Hate You' -- Dealing with the Alienated Child," presented to the New Jersey Trial Lawyers Association Annual Meeting, Atlantic City, New Jersey, 9 April 1992.

54. Brown, Jeffrey A. and Greenfield, Daniel P., "Understanding Closed Head Injury," presented to the New Jersey Trial Lawyers Association Annual Meeting, Atlantic City, New Jersey, 9 April 1992.

55. Brown, Jeffrey A., "`I Hate You' -- Dealing with the Alienated Child," presented to the New Jersey Trial Lawyers Association Annual Meeting, Atlantic City, New Jersey, 9 April 1992.

56. Brown, Jeffrey A. and Greenfield, Daniel P., "Understanding Closed Head Injury," presented to the New Jersey Trial Lawyers Association Annual Meeting, Atlantic City, New Jersey, 9 April 1992.

57. Brown, Jeffrey A. and Greenfield, Daniel P., "Rescuing Clinically and Financially Troubled Hospitals," presented to the American College of Physician Executives, San Francisco, California, 7 May 1992.

58. Brown, Jeffrey A. and Sica, Robert B., "Diagnosis and Treatment of Somatization, Cognitive Hysteria and Faking: Clinical and Legal Aspects," presented to the Professional Council of the Brain Injury Association of New Jersey, Inc., Edison, New Jersey, 1 June 1992.

59. Brown, Jeffrey A. and Greenfield, Daniel P., "Steps to Financial Recovery," presented to the American Hospital Association's Section for Psychiatric and Substance Abuse Services, Seattle, Washington, 13 June 1992.

60. Brown, Jeffrey A., "Up and Coming Syndromes: Thoracic Outlet Syndrome, Reflex Sympathetic Dystrophy and Fibromyalgia," presented to the Prudential Insurance Company Regional Claims Office In-Service Organization, Marlton, New Jersey, 6 August 1992.

61. Brown, Jeffrey A., "Differential Diagnosis of Malingering," presented to the Central Rehabilitation Associates, Cranford, New Jersey, 9 September 1992.

62. Brown, Jeffrey A. and Greenfield, Daniel P., "Psychiatric Issues in Occupational Medicine," presented to the American College of Occupational and Environmental Medicine, Manhattan, New York, 10 October 1992.

63. Brown, Jeffrey A., "Presenting Psychiatric Data in Child Custody Disputes," presented to the ATLA Second Annual Family Law Trial Academy, 24 October 1992.

64. Brown, Jeffrey A., "Psychiatric Managed Care: Vertical Integration -- Or Disintegration?" presented to Elizabeth General Medical Center Department of Psychiatry Grand Rounds, Elizabeth, New Jersey, 31 August 1993.

65. Brown, Jeffrey A. and Greenfield, Daniel P., "Avoidable Catastrophes: Shutdowns, Cramdowns, and Meltdowns," presented to the N.A.P.H.S. National Convention, San Diego, California, 24 January 1994.

66. Brown, Jeffrey A. and Greenfield, Daniel P., "Hospital Rescue: The Clinical Prescription," presented at the Strategic Research Institute, New York, New York, 22 March 1994.

67. Brown, Jeffrey A. and Kutner, Kenneth C., "Cognitive and Somatic Hysteria, Somatization, and Faking," presented to Comprehensive Rehabilitation Associates, Freehold, New Jersey, 12 April 1994.

68. Brown, Jeffrey A., "Parental Alienation and `Brainwashing,'" presented at the Elizabeth General Medical Center's Department of Psychiatry's Grand Rounds, 10 May 1994.

69. Brown, Jeffrey A. and Russo, Rose, "Barrier Free Travel," presented at the Kessler Institute/Northern Technology Assistance Resource Center 2nd Annual Conference, Iselin, New Jersey, 5 November 1994.

70. Brown, Jeffrey A. and Greenfield, Daniel P., "Professional Autonomy, Unionization and Antitrust: Incentives to Collaborate," presented at the Strategic Research Institute's conference on "Strategies to Effectively Integrate Physician Group Practices Into Hospital Systems," Laguna Niguel, California, 10 January 1995.

71. Brown, Jeffrey A. and Greenfield, Daniel P., "The Physician View of Network Building," presented at the Strategic Research Institute's conference on Physician Group Practices, Amelia Island, Florida, 16 March 1995.

72. Brown, Jeffrey A. and Greenfield, Daniel P., "Uncovering Hidden Value: The Physician Perspective," presented at the Strategic Research Institute's conference on "Strategies and Opportunities for Working with Distressed Health Care Organizations," New York, New York, 25 April 1995.

73. Brown, Jeffrey A., Foley, Henry A., and Nagle, Thomas B., "Three Views of Successful Network Building," presented to the Strategic Research Institute, San Francisco, California, 29 February 1996.

74. Brown, Jeffrey A., "Organic Brain Syndromes: Cognitive and Affective Elements," presented to the Trial Lawyers Association of British Columbia, 19 May 1995.

75. Brown, Jeffrey A., "Malingering, Hysteria, Somatization and Factitious Disorders: A Neuropsychiatric Perspective," presented to the Trial Lawyers Association of British Columbia, 20 May 1995.

76. Brown, Jeffrey A. and Greenfield, Daniel P., "What Drives Docs: The Doctors' Views of Compensation and Incentives," presented at the Strategic Research Institute's conference on "Physician Compensation and Productivity," New York, New York, 22 May 1995.

77. Brown, Jeffrey A., "Dealing with Claims of Post-Traumatic Stress Disorder," presented to the Insurance/Defense Network, Atlanta, Georgia, 24 August 1995.

78. Brown, Jeffrey A., "Psychiatric Assessment," presented to the Insurance/Defense Network, Atlanta, Georgia, 25 August 1995.

79. Brown, Jeffrey A., "Parental Alienation and the Hospital Clinician," Grand Rounds presented at Muhlenberg Hospital, Plainfield, New Jersey, 8 September 1995.

80. Brown, Jeffrey A., "What Drives Physicians: The Doctors' Views of Compensation and Incentives," presented to the Strategic Research Institute, San Francisco, California, 15 September 1995.

81. Brown, Jeffrey A. and Busch, Leonard R., "The Three Faces of Domestic Violence," presented to the Unity Group, Millburn, New Jersey, 19 September 1995.

82. Brown, Jeffrey A. and Price, David R., "Posttraumatic Stress Disorder, Mild Head Trauma, Work Place Harassment: Neuropsychiatric Disorders of the '90's," presented to the New York City Transit Authority, Brooklyn, New York, 20 September 1995.

83. Brown, Jeffrey A., "Post-Traumatic Stress Disorder," presented to the Insurance/Defense Network, Dallas, Texas, 2 November 1995.

84. Brown, Jeffrey A., "Neuropsychiatric Assessment," presented to the Insurance/Defense Network, Dallas, Texas, 3 November 1995.

85. Brown, Jeffrey A., "Physician Fears, Hopes, and Needs in Vertically Integrated Health Networks," presented to the Strategic Research Institute, Atlanta, Georgia, 14 December 1995.

86. Brown, Jeffrey A., "Chronic and Mental Illness Management in the Twenty-First Century," presented to the International Health Network Society, Southampton, Bermuda, 4 May 1996.

87. Brown, Jeffrey A., "Physician Hopes and Fears About Compensation," presented to the Strategic Research Institute, Chicago, Illinois, 15 May 1996.

88. Brown, Jeffrey A., "Psychiatric Assessment and Post-Traumatic Stress Disorder," presented to the Insurance/Defense Network, Breckenridge, Colorado, 19 July 1996.

89. Brown, Jeffrey A., "Executive Function Deficits and the Neuropsychiatric Sequelae of Traumatic Brain Injury," presented to the Insurance/Defense Network, Atlanta, Georgia, 22 November 1996.

90. Brown, Jeffrey A., "Going to Court in Domestic Violence Matters," presented to the Unity Group, Millburn, New Jersey, 21 January 1997.

91. Brown, Jeffrey A., "Caretaker or Custodian: an "Expander's View of Custody and Visitation Trends for the Next Century," presented to the American Trial Lawyers Association Boardwalk Seminar, Atlantic City, New Jersey, 18 April 1997.

92. Simring, Steven and Brown, Jeffrey A., "Medicating the Unruly Patient," presented to the Elizabeth General Hospital Department of Psychiatry and Behavioral Health, Elizabeth, New Jersey, 23 June 1997.

93. Brown, Jeffrey A. and Greenberg, John P., "How to Read the Medical Chart and How to Determine What Additional Information is Necessary for Your Case," UMDNJ - New Jersey Medical School Forensic Symposium, Cherry Hill, New Jersey, 11 September 1997.

94. Brown, Jeffrey A., "Kindling, Sensitization and Plasticity: Emerging Concepts in Traumatic Brain Injury," UMDNJ - New Jersey Medical School Forensic Symposium, Cherry Hill, New Jersey, 11 September 1997.

95. Brown, Jeffrey A. and Mahalick, David M., "Traumatic Brain Injury: Avoiding Traumatic Damages," presented to the New York Defense Association, New York, New York, 21 October 1997.

96. Brown, Jeffrey A., "Posttraumatic Stress Disorder, Malingering, Chronic Pain and Brain Injury: Case Management and Litigation Issues," presented to the Atlantic Mutual Insurance Company, Madison, New Jersey, 4 November 1997.

97. Brown, Jeffrey A., "Posttraumatic Stress Disorder, Malingering, Chronic Pain and Brain Injury: Case Management and Litigation Issues," presented to the Atlantic Mutual Insurance Company, Madison, New Jersey, 4 November 1997.

98. Brown, Jeffrey A., "After the World Trade Center Bombing: The Differential Diagnosis of Cognitive Complaints," presented to the New York City Port Authority, New York, New York, 17 November 1997.

99. Brown, Jeffrey A. and Segal, Vincent J., J.S.C., "When All Seems Lost: Coping with the Most Difficult Judicial Assignment," presented to the New Jersey State Judicial College, Teaneck, New Jersey, 26 November 1997.

100. Brown, Jeffrey A., Greenberg, John, and Mahalick, David M., "Understanding Traumatic Brain Injury and Executive Function Disorders," presented to the CNA Insurance Company Litigation Division, Mellville, Long Island, 1 December 1997.

101. Brown, Jeffrey A., "Neuropsychiatric Assessment of Executive Function Disorders," presented to the Insurance Defense Network Symposium, Charleston, South Carolina, 5 December 1997.

102. Brown, Jeffrey A. and Mahalick, David M., "Chronic Pain, Traumatic Brains, and Hysteria," presented to the CNA Insurance Company Claims Department, Mellville, Long Island, 12 January 1998.

103. Brown, Jeffrey A., "Competency, Concussions, and Custody Controversies," Grand Rounds presented at Saint Barnabas Hospital, Livingston, New Jersey, 25 February 1998.

104. Brown, Jeffrey A., "Traumatic Brain Injury, Malingering, and Hysteria: Differential Diagnosis and Fair Case Appraisal," presented to the CNA Insurance Company Law Department, Manhattan, New York, 3 March 1998.

105. Brown, Jeffrey A. and Scott-Roiter, Alexis E., "Hospital Buy-Outs of Physician Practices: Behavioral Barriers and Incentives," presented to the Strategic Research Institute Conference on Restructuring Hospital Acquired Physician Groups, New Orleans, Louisiana, 10 March 1998.

106. Brown, Jeffrey A., "Neutral Neuropsychiatric Assessment of Traumatic Brain Injury," presented to the Joint U.S. Attorney - New Jersey State Attorney General Office Conference on Traumatic Brain Injury Litigation, Newark, New Jersey, 17 March 1998.

107. Brown, Jeffrey A., "The Damaged Criminal Mind: Mens Rea and Litigation for the Brain Injured Defendant," presented to the New Jersey State Public Defenders Association's Annual Meeting, Trenton, New Jersey, 15 April 1998.

108. Brown, Jeffrey A. and Foley, Henry A., "Creating Compensation Plans that Motivate Physicians," presented to the Strategic Research Institute Conference on Physician Compensation and Productivity, San Francisco, California, 7 May 1998.

109. Brown, Jeffrey A. and Mahalick, David M., "Cognitive Hysteria in Children and Adults," presented to the Insurance Defense Network, Lake Tahoe, Nevada, 7 August 1998.

110. Brown, Jeffrey A., "The Objective Assessment and Fair Treatment of Brain Injured Workers," presented to the Fireman's Fund Insurance Company, Mellville, Long Island, New York, 28 October 1998.

111. Brown, Jeffrey A., "`Hysteria," Malingering, Stress, Medication, and Other Non-Traumatic Causes of Cognitive Deterioration,'" presented to the Selective Insurance Company, Sparta, New Jersey, 16 December 1998.

112. Brown, Jeffrey A., Mahalick, David M., and Burke, William H., "Distinguishing Real from Imagined Traumatic Brain Injury," presented to Selective Insurance, Sparta, New Jersey, 16 December 1998.

113. Brown, Jeffrey A., "A Decision Tree for Evaluating Traumatic Brain Injury," presented to the Chubb Insurance Company, Florham Park, New Jersey, 27 January 1999.

114. Brown, Jeffrey A., "Dealing with Lawyers in the Twenty-First Century," ZENECA Pharmaceuticals lecture presented to the North Jersey Psychiatric Society, Hackensack, New Jersey, 10 February 1999.

115. Brown, Jeffrey A., "Medical/Legal Oxymoron? - A Fair Assessment of Neuropsychiatric Claims," presented to the New Jersey Institute for Continuing Legal Education, New Brunswick, New Jersey, 17 April 1999.

116. Brown, Jeffrey A., "Taking the Trauma Out of Traumatic Brain Injury Evaluations," presented to the New York City Defense Association, New York, New York, 13 April 2000.

117. Brown, Jeffrey A., Jacoby, Jacob H., Mahalick, David M., "The Differential Diagnosis of Symptom Exaggeration in TBI, PTSD, and Chronic Pain," presented to the New York City Port Authority, Manhattan, New York, 4 August 2000.

118. Brown, Jeffrey A. and Kantor, Ruth B., "Proving Psychological Injuries," presented to the American Academy of Matrimonial Lawyers, Atlantic City, New Jersey, 29 September 2000.

119. Brown, Jeffrey A., "Competency and Neurobehavioral Impairment: Clinical and Legal Issues," presented to the Brain Rehabilitation Unit, Chilton Memorial Hospital, Pompton Plains, New Jersey, 8 March 2001.

120. Brown, Jeffrey A., Dayle, Randy A. and Gordon, Stephen L., "New Health Ventures for the New Millennium," presented on "New Jersey Business," News 12 New Jersey, Edison, New Jersey, 10 May 2001.

121. Brown, Jeffrey A., "The Neuropsychiatric Analysis and Presentation of Complex 'Pain and the Brain' Cases," presented to the Port Authority of New York/New Jersey, New York, New York, 25 May 2001.

122. Brown, Jeffrey A., "Psychologists and the Legal System," guest lecture presented to the Drew University Seminar in Forensic Psychology, Madison, New Jersey, 11 September 2001.

123. Brown, Jeffrey A., "Disability: Genuine or Disingenuine?" presented to the Prudential Insurance Company, Livingston, New Jersey, 9 January 2002.

124. Brown, Jeffrey A., "Litigating the Closed Head Injury Case: The Use and Abuse of Neurobehavioral Experts," presented to the Camden County Bar Association, Voorhees, New Jersey, 25 February 2002.

125. Brown, Jeffrey A., "How Neuropsychologists and Neuropsychiatrists Best Work Together Clinically and Legally," presented to the New York University Department of Psychology Clinical Neuropsychology Course, 28 March 2002.

126. Brown, Jeffrey A., "Assessing Functional Psychiatric Impairments," presented to the United States Social Security Administration and the New Jersey Department of Labor, Division of Disability Services at Saint Barnabas Hospital, Livingston, New Jersey, 19 June 2002.

127. Brown, Jeffrey A., "Emerging Clinical Trends in Neuropsychiatry and Their Applicability in Court," presented to Touro University School of Health Sciences, Bayshore, New York, 24 March 2003.

128. Brown, Jeffrey A., "Uses and Limitations of Neuropsychological Tests in Brain Injury Litigation," presented to the New York University Department of Psychology Clinical Neuropsychology Course, 10 April 2003.

129. Brown, Jeffrey A., "Neuropsychiatric Disability: The Struggle for Objectivity," presented to the United States Social Security Administration and New Jersey Department of Labor, Division of Disability Services at Community Hospital, Toms River, 25 June 2003.

130. Brown, Jeffrey A., "Medical Legal Issues in Brain Injury: The Defense Perspective," presented to the Brain Injury Association of America, Amelia Island, Florida, 19 September 2003.

131. Brown, Jeffrey A., "A Neuropsychiatric Perspective on the Uses and Limitations of Neuropsychological Tests," presented to the Texas Psychological Association, Dallas, Texas, 8 November 2003.

132. Brown, Jeffrey A., "Dealing with TBI Claims: Separating Fact, Fantasy and Fiction," presented to the New Jersey Institute of Continuing Legal Education, Cherry Hill, New Jersey, 13 December 2003.

133. Brown, Jeffrey A., "Dealing with Stress, Pain, and TBI Claims," presented to the PMA Insurance Company, Mount Laurel, New Jersey, 13 May 2004.

134. Brown, Jeffrey A., "Defense 'Tactics' in Traumatic Brain Injury Clinical Evaluation and Litigation," presented to The North American Brain Injury Society, Beaver Creek, CO, 22 September 2004.

135. Brown, Jeffrey A., "Civil Forensics: Competency, Custody, and Brain Catastrophes," presented to the Beth Israel Hospital - Albert Einstein Medical School Post-Graduate Forensic Psychiatry Program, Manhattan, New York, 7 December 2004.

136. Brown, Jeffrey A., "For the Defense: Punch and Counterpunch," presented to the Brain Injury Association of America, Amelia Island, Florida, 24 September 2005.

137. Brown, Jeffrey A., "Dealing with Defenses: Avoiding Predictable Blunders," presented to the Brain Injury Association of America, Miami Beach, Florida, 16 September 2006.

138. Brown, Jeffrey A., "Dealing with Plaintiffs and Treating Testifiers in Traumatic Brain Injury Cases," presented to Crum and Foster Insurance Company, Morristown, New Jersey, 23 February 2007.

139. Brown, Jeffrey A., "Respecting the Defense: Objective Pathways to Settlement," presented to the North American Brain Injury Society, New Orleans, Louisiana, 2-4 October 2008.

140. Brown, Jeffrey A., "Malingering and Misperception in Traumatic Brain Litigation," presented to French & Casey, LLP, New York, NY, 01April 2009.

141. Brown, Jeffrey A., "The Coming Great Synthesis of Neuropsychiatry and the Law," presented to the 2009 North American Brain Injury Society Medical-Legal Conference on Brain Injury, Austin, Texas, 16 October 2009.

142. Brown, Jeffrey A., "Medication Adherence and Cognitive Assistive Technology for the 21st Century," presented to the International Health Network Society, Southampton, Bermuda, 07 November 2009.

143. Brown, Jeffrey A., "Separating the Wheat from the Chaff in TBI Litigation: When to Fight and How to Settle," presented to the Nassau/Suffolk County Trial Lawyers Association, Westbury, New York, 25 March 2010.

144. Brown, Jeffrey A., "The Pleasures – and Pitfalls – of Being an Expert Witness," presented to the University of Medicine and Dentistry of New Jersey's second, third, and fourth year resident groups, Newark, New Jersey 27 August 2010.

145. Brown, Jeffrey A., "Being Caught in Child Custody Disputes: A Primer for Child Psychiatrists," presented to the University of Medicine and Dentistry of New Jersey, child psychiatry fellows and senior psychiatry residents, Newark, New Jersey, 1 September 2010.

146. Brown, Jeffrey A., "Ten Blunders Plaintiff Attorneys Make in Litigating Brain Injury Cases" presented at Mass Torts Made Perfect, Las Vegas, Nevada, 14 April 2011.

147. Brown, Jeffrey A., "How 21st Century Neuroscience Will Transform TBI Litigation From The Molecular Level Up," presented to the Central Florida Trial Lawyers Association, Orlando, Florida, 7 September 2011.

148. Brown, Jeffrey A., "How Cutting Edge Neuroscience Will Transform Traumatic Brain Injury Litigation," presented to the North American Brain Injury Society, New Orleans, LA, 15 September 2011.

149. Brown, Jeffrey A., "Understanding Causation and Maximizing Damages by Proving Critical Clinical Interactions in Mild Brain Injury Cases," presented to the 360 Advocacy Institute, Las Vegas, Nevada, 24 October 2011.

150. Brown, Jeffrey A., "Ten Blunders Plaintiff Attorneys Make in Litigating Brain Injury Cases," presented to the Law Firm of Edward Garfinkel, Brooklyn, New York, 3 December 2011.

151. Brown, Jeffrey A., "The Future of Brain and Emotional Injury Litigation," presented to the Traumatic Brain Injury and Emotional Injury Summit: Winning With 21st Century Neuroscience, Denver, Colorado, 4 December 2011.

152. Brown, Jeffrey A., "Predicting and Defeating Future Malingering Defenses," presented to the Traumatic Brain Injury and Emotional Injury Summit: Winning With 21st Century Neuroscience, Denver, Colorado, 6 December 2011.

153. Brown, Jeffrey A., "Using 21st Century Ethics and 21st Century Neuroscience to Cross Examine Defense Experts" presented to the Florida Justice Association, Orlando, Florida, 22 March 2012.

154. Brown, Jeffrey A. and DeVito, William N., "Wielding the Cutting Edge: Welding 21st Century Brain Injury Medicine and the Law," presented to the Chartis Insurance Company's In-House Counsel, Jericho, New York, 27 August 2012.

155. Brown, Jeffrey A., "Neuropsychiatry and the Law: Psychiatric Essentials for Future Board Examinees," presented to the University of Medicine and Dentistry of New Jersey Psychiatry Resident Seminar, Newark, New Jersey 29 August 2012.

156. Brown, Jeffrey A., "Tarasoff and Duty to Warn: Hot Off the Presses Issues," presented to the University of Medicine and Dentistry of New Jersey Psychiatric Resident Seminar, Newark, New Jersey 29 August 2012.

157. Brown, Jeffrey A. and DeVito, William N., "Wielding the Cutting Edge: Welding 21st Century Brain Injury Medicine and the Law," presented to the Chartis Insurance Company's Senior Adjustors and Staff Counsel, Manhattan, New York 13 September 2012.

158. Brown, Jeffrey & Wu, Joseph, "Psychiatric Injury and Neurobehavioral Science in Gas Drilling-Toxic Tort Cases – Brain Injury and Methane/Fracking Chemicals," presented to the Gas Drilling/Fracking Litigation Project Group, Las Vegas, Nevada, 10 October 2012.

159. Brown, Jeffrey & Wu, Joseph, "Objectifying Toxic Exposure: Neuropsychiatric Injuries and Damages," presented to Mass Torts Made Perfect, Las Vegas, Nevada, 11 October 2012.

160. Brown, Jeffrey A. and DeVito, William N., "Wielding the Cutting Edge: Welding 21st Century Brain Injury Medicine and the Law," presented to the Law Offices of Alan I. Lamer, Elmsford, New York, 17 October 2012.

161. Brown, Jeffrey A. and DeVito, William N., "Wielding the Cutting Edge: Welding 21st Century Brain Injury Medicine and the Law," presented to the Law Offices of Edward Garfinkel, Brooklyn, New York, 22 October, 2012.

162. Brown, Jeffrey A., "Predicting and Preventing Homicide, Suicide and Posttraumatic Stress Disorder: Clinical Interventions and Post Tarasoff Legal Obligations," presented to the University of Medicine and Dentistry of New Jersey's Psychiatric Residency Program, Newark, New Jersey 23 January 2013.

163. Brown, Jeffrey A. and Jacoby, Jacob H., "Conducting Neuropsychiatric Fact Investigations in Will Contest Cases," presented at Rutgers University Law School, Newark, New Jersey, 12 March 2014.

164. Brown, Jeffrey A., DeVito, William N., Jacoby, Jacob H., and Rothenberg, Alan L., "Truth and Self-Deception in Brain Injury Cases: Ethical Challenges for Both Attorneys and Medical Experts in Traumatic Brain Injury Cases," presented at Rutgers University Jewish Law Students Association, Rutgers University Law School, Newark, New Jersey, 12 March 2014.

165. Brown, Jeffrey A., "Deciding Who Should Be On Your Team," presented at the Defense Association of New York seminar, "The Cutting Edge 2014: Understanding Brain Injuries & Building the Best Defense," Manhattan, New York, 20 May 2014.

166. Brown, Jeffrey A., "Deciding What Your Adversaries and Their Experts Will Do," presented at the Defense Association of New York seminar, "The Cutting Edge 2014: Understanding Brain Injuries & Building the Best Defense," Manhattan, New York, 20 May 2014.

167. Brown, Jeffrey A., "Deciding How to Diffuse Diffusion Tensor Imaging," presented at the Defense Association of New York seminar, "The Cutting Edge 2014: Understanding Brain Injuries & Building the Best Defense," Manhattan, New York, 20 May 2014.

168. Brown, Jeffrey A., "Deciding How to Counterattack with Functional Resilience" presented at the Defense Association of New York seminar, "The Cutting Edge 2014: Understanding Brain Injuries & Building the Best Defense," Manhattan, New York, 20 May 2014.

169. Brown, Jeffrey A., "Deciding Potential Exposure and How Hard to Fight," presented at the Defense Association of New York seminar, "The Cutting Edge 2014: Understanding Brain Injuries & Building the Best Defense," Manhattan, New York, 20 May 2014.

170. Brown, Jeffery A. and Kardos, Mark, "How to Overcome Defenses in Traumatic Brain Injury Cases," presented at the National Business Institute's Continuing Legal Education Seminar, "Traumatic Brain Injury Cases: Doctor and Attorney Perspectives," Philadelphia, Pennsylvania, 30 October 2014.

171. Brown, Jeffrey A. and Bruderle, Stephen, "Defense Tactics Unique to Brain Injury Cases," presented at the National Business Institute's Continuing Legal Education Seminar, "Traumatic Brain Injury Cases: Doctor and Attorney Perspectives," Philadelphia, Pennsylvania, 30 October 2014.

172. Brown, Jeffrey A. and Mahalick, David M., "Investigating Closed Head Brain Injuries," presented at the National Business Institute's Continuing Legal Education Seminar, "Traumatic Brain Injury Cases: Doctor and Attorney Perspectives," Philadelphia, Pennsylvania, 30 October 2014.

173. Brown, Jeffrey A., "Winning Defense Expert Approaches," presented at the Defense Association of New York's Continuing Legal Education Seminar, "The Cutting Edge 2015: Cutting Deeper into TBI Law and Science," New York, NY 12 March 2015.

174. Brown, Jeffrey A., Key Note Address for Basic Science Graduates: "The Pleasures and Challenges of Coming to America to Practice Medicine," presented to The American University of Integrative Sciences, St. Maarten School of Medicine, Cole Bay, St. Maarten, 15 April 2015.

175. Brown, Jeffrey A., "Six Ethical Questions Every Brain Injury Expert Must Ask," presented to the AIG Group, Jericho, Long Island, 09 June 2015.

176. Brown, Jeffrey A., "The Emerging Role of Resilience and Its Relationship to Diffusion Tensor Imaging Studies," presented to the AIG Group, Jericho, Long Island, 09 June 2015.

177. Brown, Jeffrey A., "Misperception, Specificity, Localization Limits, and Resilience: The New TBI Defense Frontiers," presented to the AIG Insurance Company (Luxington Group), Boston, Massachusetts, 27 July 2015.

    (Note: The audience was 50% percent plaintiff attorneys and 50% defense attorneys who were all present at all talks.)

178. Brown, Jeffrey A., DeVito, William N., Mahalick, David M., "New 21st Century Neuroscience Implications for the Future of Brain Injury Litigation," presented to the AIG Insurance Company, Brooklyn, New York 16 September 2015.

179. Brown, Jeffrey A., DeVito, William N., Mahalick, David M., "New 21st Century Neuroscience and Behavioral Implications for Traumatic Brain Injury Litigation," presented to the AIG Insurance Company, Westchester, New York, 24 September 2015.

180. Brown, Jeffrey A., DeVito, William N., "Proving Psychiatric and Neurological Aspects of Traumatic Brain Injuries," presented at the National Business Institute Audio Seminar, Aventura, FL, 28 January 2016.

181. Brown, Jeffrey A., "Emerging Defenses and Trojan Horses in Trucking Cases," webinar presented to trucking defense attorneys & senior claims personnel, 8 June 2016.

182. Brown, Jeffrey A., "How to Use The Latest Science and Your Understanding of Brain Injuries to Help You Work Constructively with Your Adversary to Settle Cases," to be presented to the New York Defense Association, New York, New York, 22 September 2016.

CURRICULUM VITAE:

DATE: April 26, 2016

NAME: David M. Mahalick, PhD, ABPN

PRESENT TITLE: Pediatric & Adult Clinical Neuropsychologist

OFFICE ADDRESS:

2066 Millburn Avenue
Suite 201
Maplewood, NJ 07040

TELEPHONE NUMBER/E-MAIL INTERNET:

Telephone: (973) 313-9393
Facsimile: (973) 313-1666
e-mail: braindoc1@comcast.net

EDUCATION:

A. Undergraduate and Professional:
Alfred University
Alfred, New York
Bachelor of Arts- Applied Psychology 6/1982

B. Graduate and Professional:
California School of Professional Psychology
San Diego, California
Ph.D. - Clinical Psychology 12/1987

POSTGRADUATE TRAINING:

A. Internship and Residencies:

1. Pre-doctoral Internship
Clinical Psychology Internship
Escondido Community Mental Health Center
San Diego County Mental Health
July 1, 1983- June 30, 1984

2. Pre-doctoral Internship
Clinical Neuropsychology (Adult) Internship
University of California- San Diego Medical Center
Department of Neurological Surgery
25 Dickinson Street

San Diego, California
July 1, 1984- June 30, 1985

3. Pre-doctoral Internship
   Pediatric Clinical Neuropsychology (Pediatric) Internship
   University of California-San Diego Medical Center
   Department of Neurology (Peds.)
   Center for Language and Communicative Disorders
   25 Dickinson Street
   San Diego, California
   July 1, 1985- June 30, 1986

4. Residency
   Pediatric and Adult Clinical Neuropsychology Residency
   Hahnemann University Hospital Medical School
   Department of Neurology
   230 North Broad Street
   Philadelphia, Pennsylvania 19102
   July 1, 1986- June 30, 1988

B. Research Fellowships: N/A

C. Postdoctoral Appointments: N/A

MILITARY: N/A.

ACADEMIC APPOINTMENTS:

Department of Pediatrics
Robert Wood Johnson Medical School
Clinical Associate Professor of Pediatrics
April, 2016- present

Department of Pediatrics
Robert Wood Johnson Medical School
Clinical Assistant Professor of Pediatrics
Sept., 1994- June, 2002

Department of Neurology
Robert Wood Johnson Medical School
Clinical Assistant Professor of Neurology
April, 1991- Sept., 1994

Departments of Pediatrics and Surgery (Division of Neurological Surgery)
University of Medicine and Dentistry-New Jersey Medical School
Clinical Assistant Professor of Pediatrics and Surgery
June, 1993-2012.

Departments of Pediatrics and Surgery (Division of Neurological Surgery)
University of Medicine and Dentistry-New Jersey Medical School
Clinical Instructor of Pediatrics and Surgery
June, 1991-1993

Department of Psychiatry
University of Medicine and Dentistry-New Jersey Medical School
Assistant Clinical Professor of Psychiatry
January, 1994- September, 1997

Department of Applied Psychology
New York University-Steinhardt School of Education
Department of Applied Psychology
Adjunct Associate Professor of Clinical Psychology
January, 2001-2010

HOSPITAL APPOINTMENTS:

Children's Specialized Hospital
February, 1989- August, 1994.

Department of Neurosurgery
Children's Hospital of New Jersey (closed)
Neuropsychology-Consulting Staff
April, 1990- August, 1995

Department of Pediatrics
Beth Israel Medical Center
Neuropsychology-Consulting Staff
April, 1990- August, 1997

Department of Pediatrics
Robert Wood Johnson-University Hospital
Neuropsychology-Consulting Staff
December, 1991- present.

Departments of Pediatrics and Neurological Surgery
University of Medicine and Dentistry -University Hospital
Neuropsychology-Consulting Staff
June, 1991-2012.

Department of Psychiatry
Clara Maas Medical Center
Neuropsychology-Consulting Staff
June 1996- December, 2008

Department of Psychiatry
Somerset Medical Center
Neuropsychology-Consulting Staff
December, 1997- present.

Department of Psychiatry
Morristown Memorial Hospital
Neuropsychology-Consulting Staff
September, 1998- present.

Department of Psychiatry
Muhlenberg Regional Medical Center
Neuropsychology-Consulting Staff
September, 1997- April, 2009 (closed)

Department of Psychiatry
Overlook Hospital Medical Center
Neuropsychology-Consulting Staff
September, 2001- present.

Department of Neurology
Beth Israel Medical Center-North (Closed)
Neuropsychology-Professional Staff
September, 2003-August, 2005

OTHER EMPLOYMENT OR MAJOR VISITING APPOINTMENTS:

Hahnemann University Hospital
Department of Neurology
Division of Neuropsychology
Chief Neuropsychology Fellow
July, 1987-June-1988

DATHR-Comprehensive Rehabilitation Program
Brookfield, CT
Staff Clinical Neuropsychologist
July, 1988-February 1989

Children's Specialized Hospital
Director, Department of Psychology/Neuropsychology
February, 1989-August, 1994.

University of Medicine and Dentistry-New Jersey Medical School
Department of Psychiatry
Director, Neuropsychology Service

August, 1994-July, 1997.

University of Medicine and Dentistry-New Jersey Medical School
Department of Neurosurgery
Neuropsychology Consulting Staff (in house, private practice)
July, 1997- December, 2006.

President and Chief Executive Officer
Director of Neuropsychology
Neurobehavioral Institute of New Jersey
January, 2000-December, 2009

President and Chief Executive Officer
The Isabel & David M. Mahalick Foundation
April, 2000-present.

PRIVATE PRACTICE:

2066 Millburn Avenue
Suite 201
Maplewood, NJ 07040

1771 Springdale Avenue
Cherry Hill, New Jersey 08003

5 Penn Plaza
19th Floor
New York, NY 10020

LICENSURE:

New Jersey License # SI 02582
Clinical Psychology (Specializing in Neuropsychology)
February 2, 1989-present
Expiration date: 6/30/2017

New York License #013948
Clinical Psychology (Specializing in Neuropsychology)
October 10, 2000-present
Expiration date: 4/30/2017

NPI: 1962617811

DRUG LICENSURE:

CDS: N/A

DEA: N/A

CERTIFICATION:

Diplomate, American Board of Professional Neuropsychology
October 21, 2001- present.
Expiration date: N/A

MEMBERSHIPS, OFFICES, AND COMMITTEE ASSIGNMENTS IN PROFESSIONAL SOCIETIES:

International Neuropsychological Society
Member
1988-present

National Academy of Neuropsychology
Member
1988-present

New Jersey Neuropsychological Society
Member of the Board Of Trustees
1989-2002

New Jersey Neuropsychological Society
Member
1989- present

Chairman, Membership Committee
N.J. Society of Neuropsychologists
1989-1993.

National Head Injury Association
Member
1989-1997

New Jersey Head Injury Association
Member
1989-1997

Chairman, Steering Committee of the Professional Council
New Jersey Head Injury Association, Inc.
1990-1992.

American Psychological Association
Member
1988-present

Division 40 (Clinical Neuropsychology of the APA)
Member
1988-present

New Jersey Psychological Association
Member
1989-present

New York Academy of the Sciences
Member
1990- 1994

New Jersey Academy of Psychologists
Member (merged with NJPA)
1988-2010

American Congress of Rehabilitation Medicine
Member
1990-1999

HONORS AND AWARDS:

Psi Chi
American Psychological Association National Honor Society
1982.

Phi Kappa Phi
National Honor Society
1982

Distinguished Service Award
NJ Head Injury Association
1991.

Recognition Award
NJ Academy of Psychologists
1994.

Fellow
American College of Professional Neuropsychology
2001.

Fellow
American Board of Forensic Medicine
2000.

BOARDS OF DIRECTORS OR TRUSTEES POSITIONS:

Board of Trustees
New Jersey Society of Neuropsychologists
1989-1993

Board of Trustees
New Jersey Academy of Psychology
1990-1992.

Board of Trustees
Perspectives Network Spring, Texas
1990-1992.

SERVICE ON NATIONAL GRANT REVIEW PANELS, STUDY SECTIONS, COMMITTEES:

Scientific Reviewer
National Institute of Health (NIH)
National Institute of Child Health and Human Development
Special Emphasis Panel ZHD1 DSR-L 24R
March, 2000-

SERVICE ON MAJOR COMMITTEES:

A. International: N/A
B. National: N/A
C. State:
   Chairman, Membership Committee
   N.J. Society of Neuropsychologists
   1989-1993.

   Chairman, Steering Committee of the Professional Council
   New Jersey Head Injury Association, Inc.
   1990-1992.

D. Medical School/University: N/A
E. Hospital: N/A
F. Department: N/A
G. Editorial Boards: N/A
H. Ad Hoc Reviewer: N/A

SERVICE ON GRADUATE SCHOOL COMMITTEES: N/A

SERVICE ON HOSPITAL COMMITTEES: N/A

SERVICE TO THE COMMUNITY:

President and Chief Executive Officer
The Isabel & David M. Mahalick Foundation
April, 2000-present.

CLINICAL RESPONSIBILITIES:

Hahnemann University Hospital
Department of Neurology
Division of Neuropsychology
Chief Neuropsychology Fellow
July, 1987-June-1988

DATHR-Comprehensive Rehabilitation Program
Brookfield, CT
Staff Clinical Neuropsychologist
July, 1988-February 1989

Children's Specialized Hospital
Director, Department of Psychology/Neuropsychology
February, 1989-August, 1994.

University of Medicine and Dentistry-New Jersey Medical School
Department of Psychiatry
Director, Neuropsychology Service
August, 1994-July, 1997.

University of Medicine and Dentistry-New Jersey Medical School
Department of Neurosurgery
Neuropsychology Consulting Staff (in house, private practice)
July, 1997- December, 2006.

President and Chief Executive Officer
Director of Neuropsychology
Neurobehavioral Institute of New Jersey
January, 2000-December, 2009

GRANT SUPPORT: N/A

PUBLICATIONS:

A. Refereed Original Article in Journal:

1. Mahalick DM, Ruff RM, U HS (1991) Neuropsychological Sequelae of Arteriovenous Malformations. Neurosurgery 29:351-357.

2. Mahalick DM, Ruff RM, U HS, Heary RF (1993) Pre-operative versus Postoperative Neuropsychological Sequelae of Arteriovenous Malformations. Neurosurgery Vol. 33:4 pp. 563-572.

3. Mahalick DM, McDonough M, Levitt J (1995) Head Injuries in Adults and Children. Trauma 37:4 pp. 27-38.

4. Mahalick DM, Koller CJ, Pleim ET. Pediatric Trauma and head injury. Trauma 38:1 pp 39-56 April 1996.

5. Mahalick DM & Hahn G. Cognitive sequelae of electroconvulsive therapy. Trauma 38:5 pp 45-50 February 1998.

6. Mahalick DM, Carmel PW, Greenberg JP, Molofsky W, Brown JA, Heary RF, Marks D, Zampella E, Hodosh R (1998) Psychopharmacological Treatment of Acquired Attention Disorders in Children with Brain. Pediatric Neurosurgery; 29: 121-126.

7. Schulder M, Sernas TA, Adler RJ, Mahalick DM, Cook S: Thalamic stimulation in patients with multiple sclerosis. Stereotact Funct Neurosurg 72: 196-201, 1999.

B. Books, Monographs, and Chapters:

1. Mahalick DM (1989) The Neuropsychological Sequelae of Arteriovenous Malformations. Ann Arbor: UMI.

2. Mahalick DM & Ryan T V (Eds) Pediatric Brain Injury: Diagnosis and Rehabilitation. San Diego: Singular Publishing (in prep).

3. Behrens F, Schwappach, Swan K, Levy A, Barbieri R, Forster R, Mahalick DM & Chowchuvech G. Injury and Repair (chap.1.7.1 viz., Head injuries-presentations and outcomes) in Buckwalter J, Bustrode C, Carr A, Fairbank J, Marsh L, Wilson-MacDonald L. (Eds.) Oxford Textbook of Orthopeadics and Trauma. Oxford University Press (2002).

C. Patents Held: N/A

D. Other Articles:

1. Mahalick DM, Savage J (1990) Neuropsychological Assessment of the Pediatric Population. NJ Psychologist Vol. 40. pg 14.

2. Mahalick DM (1991) Pediatric Brain Injury. The Perspective Network IV:18-19.

E. Abstracts

1. Peer Reviewed Abstracts:

Mahalick DM, Ruff RM, U HS, Heary R F (1994) Pre-versus Postsurgical Sequelae of Arteriovenous Malformations. Abstracts of the 13th Annual Meeting. Archives of Neuropsychology Vol. 9: 2 pp. 159-160.

Mahalick DM Molofsky W, Bartlett JA, (1996). Psychopharmacological treatment of Children with Attention Disorders Secondary to Brain Injury. Vol. 9: 2 pp 159-160. Abstracts of the Ninteenth Annual Meeting of the International Neuropsychological Society Mid-Year Conference. J International Neuropsychological Vol 2: 3 pp 208.

Mahalick DM, McDonough M, Greenberg JP, (1996) Psychopharmacological treatment of Pediatric Traumatic Brain Injury Abstract of the Twenty -Fifth Annual International Neuropsychological Society Conference. J International Neuropsychological Vol 3: 1 pp 63.

McDonough M, Mahalick DM, Greenberg JP, (1997) Malingering on neuropsychological assessment is more often a case of individual presentation than a litigation group phenomena. Abstracts of the 17th Annual Meeting. Archives of Clinical Neuropsychology. Vol.13, Number 1: pp 60.

Mahalick DM, Hohn GE, Hunt CD, Schulder M, Carmel PW (1997): Intracarotid Sodium Amytal Testing on Patients With AVM's: Its Utility a Function of the Size and Shunt Value of the AVM. Abstracts of the 17th Annual Meeting. Archives of Clinical Neuropsychology. Vol.13, Number 1: pp 60-61.

Mahalick DM, Carmel, PW Molofsky W, Bartlett JA, McDonough M, Greenberg JP, (1998) Psychopharmacological Treatment of Pediatric Brain Injury. Abstracts of the Annual Meeting of the American Association of Neurological Surgeons. J Neurosurgery. Vol. 88: 2 pp 412A.

Mahalick DM, Greenberg JP, McGinley J (2003) Neuropsychological and Neurological Sequelae of Toxic Anhydrous Ammonia. Abstracts

of the 23rd Annual Meeting. Archives of Clinical Neuropsychology. Vol. 18: pp 727.

2. Non Peer Reviewed Abstracts: N/A

E. Reports:

1. Mahalick DM, Yalamanchi K , Ruzicka PO, Bowen M. "Spontaneous Recovery Following Pediatric Traumatic Brain Injury" Presented at the National Head Injury Foundation's Annual Conference, November, 1990, New Orleans, LA.

2. Mahalick DM & Yalamanchi K "Neuropsychological and Medical Recovery Following Pediatric Traumatic Brain Injury" Symposia Presentation presented at the NJ Head Injury Association's Annual Conference. November 1990.

3. Mahalick DM, Ruff RM, U HS "Neuropsychological Sequelae of Arteriovenous Malformations" Presented at the annual meeting of the International Neuropsychological Society. February 1991. San Antonio, Texas.

4. Mahalick DM, Yalamanchi K, Mehta U, Webb T "Psychopharmacological Treatment of Acquired Attentional Disorders in Children with Traumatic Brain Injury" Recovery Presented at the National Head Injury Foundation's Annual Conference, November, 1993, Orlando, FLA.

5. Mahalick DM, Ruff RM, U HS, Heary RF "Pre-operative versus Postoperative Neuropsychological Sequelae of Arteriovenous Malformations" Presented at the Congress of Neurosurgeons Annual Conference October 1993, Vancouver, B.C.

6. Mahalick DM, Ruff RM, U HS, Heary RF "Pre-operative versus Postoperative Neuropsychological Sequelae of Arteriovenous Malformations" Presented at the National Academy of Neuropsychologists 13th Annual Conference. October, 1993, Phoenix, AR.

7. Mahalick DM, Manniker A & Yalamanchi K "Pediatric Traumatic Brain Injury: Medical Considerations and Community/Academic Reintegration New Jersey Head Injury Association 12th Annual Seminar April 30, 1994.

8. Mahalick DM, Yalamanchi, K, Mehta U, Webb T "Psychopharmacological treatment of acquired attentional disorders secondary to pediatric traumatic brain injury" Platform presentation. Medical Conference of Virginia Annual Symposium. Williamsburg, VA. May 25, 1994.

9. Mahalick DM, McDonough M Assessing treatment efficacy in pediatric traumatic brain injury. Platform Presentation. 14th Annual National Symposia of the National Head Injury Foundation. San Diego, CA December 3, 1995.

10. McDonough M, Mahalick DM Challenges to notions of rapid spontaneous recovery in mild head trauma. Platform Presentation. 14th Annual National Symposia of the National Head Injury Foundation. San Diego, CA December 3, 1995.

11. Mahalick DM, Bartlett JA, Molofsky W Psychopharmacological treatment of acquired attentional disorders in pediatric traumatic brain injury. Poster Presentation. 14th Annual National Symposia of the National Head Injury Foundation. San Diego, CA, December 3, 1995.

12. Mahalick DM, Molofsky W, Bartlett JA, (1996) Psychopharmacological treatment of Children with Attention Disorders acquired Secondary to Brain Injury. Nineteenth Annual International Neuropsychological Society Mid-Year Conference. Veldhoven, The Netherlands, June 22, 1996.

13. McDonough M, Mahalick DM, Greenberg JP. Malingering on neuropsychological assessment is more often an individual presentation than a litigation group phenomenon. Poster Presentation. National Academy of Neuropsychology. New Orleans, LA. November 2, 1996.

14. McDonough M, Mahalick DM, Greenberg JP. MRI confirmation of neuropsychological impairment of carbon monoxide toxicity. Poster Presentation. National Academy of Neuropsychology. New Orleans, LA. November 2, 1996.

15. Mahalick DM, McDonough M, Molofsky W, Greenberg JP. Psychopharmacological treatment of Pediatric Traumatic Brain Injury. Presentation. Twenty -Fifth Annual International Neuropsychological Society Conference. Orlando, FLA. February 5-8, 1997.

16. Mahalick DM, McDonough M, Molofsky W, Greenberg, JP. Psychopharmacological treatment of Pediatric Traumatic Brain Injury. Presentation. Eight Annual Meeting of the American Neuropsychiatric Association. Orlando, FLA. February 2-4, 1997.

17. Mahalick DM, McDonough M, Greenberg JP. Neuropsychological and neuropsychiatric presentation of a patient exposed to severe electrocution injury. Presentation. Eight Annual Meeting of the American Neuropsychiatric Association. Orlando, FLA. February 2-4, 1997.

18. McDonough M, Small M, Mahalick DM. Malingering on neuropsychological assessment is more often an individual presentation than a litigation group phenomenon-part II. Poster Presentation. Eight Annual Meeting of the American Neuropsychiatric Association. Orlando, FLA. February 2-4, 1997.

19. Mahalick DM, Hohn GE, Hunt CD, Schulder M, Carmel PW: Intracarotid Sodium Amytal Testing on Patients With AVM's: Its Utility a Function of the Size and Shunt Value of the AVM. Poster Presentation at the 17th Annual meeting of the National Academy of Neuropsychology, Las Vegas, Nevada. November 12, 1997.

20. Mahalick DM, Schulder M, Cathcart CS. Neuropsychological Findings After Stereotactic Radiosurgery for AVM's. LINAC Radiosurgery Conference Sponsored by the Department of Neurosurgery and the Department of Radiation Oncology, University of Florida, Gainsville, FLA. Paper # 030. Orlando, Florida. December 13, 1997.

21. Mahalick DM, Carmel PW, Molofsky W, Bartlett JA, McDonough M, Greenberg JP, (1998). Psychopharmacological Treatment of Pediatric Brain Injury. Annual Meeting of the American Association of Neurological Surgeons. Paper #817. Philadelphia, PA. April 1998.

22. Mahalick DM. (2004). Medication and Children with Brain Injury. Children and Brain Injury: Navigating Life. Brain Injury Association of New York State. Symposium 3 C. New York, NY. March 11, 2004.

William N. DeVito

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WORK EXPERIENCE

ATTORNEY **2000 to 2016**
AIG STAFF COUNSEL / LITIGATION MANAGEMENT **NEW YORK, NY**

Senior Trial Attorney and Assistant Managing Attorney handling complex and high exposure cases for AIG Staff Counsel managing litigation and acting as defense counsel for all aspects of litigation including coverage and risk transfer issues from inception through trial. Currently leads team of six attorneys, reviews their files and provides guidance. Responsible for thought leadership and developing CLE and training programs for AIG.

- Supervise six attorneys, three paralegals and two secretaries
- In 2013 developed CLE to train all claims professionals and Staff Attorneys to better defend insured contractors
- Responsible for managing and defending all litigation for AIG's new Private Client Group Unit (personal lines)
- Developed training program on traumatic brain injury cases
- Excellent Interpersonal skills and contacts with all major Staff Counsel Clients
- Highly Rated Linkedin Profile with 1500 plus contacts and many endorsements from both inside and outside AIG
- 2015 Developed Premises Liability Training Course for Key Client and also to train all NY Staff Counsel attorneys and Claims Offices
- January 28, 2016 Presented National Version of my course on Traumatic Brain Injury for the National Business Institute

From October 2006 to December 2007 worked as National Coordinating Counsel for AIG Personal Lines for bad-faith cases around the country. Responsible for hiring and managing outside counsel, monitoring strategy, auditing and financial reserves. Successfully mediated and resolved numerous cases in several states including New Jersey, West Virginia, Florida, Louisiana and Texas.

- Responsible for reporting reserve information to Senior Executives
- Reserves required for reporting under Sarbanes-Oxley Act
- Served as mediation trainer for Staff Counsel Mediation program.
- Managed and updated company expert witness database
- Used written and oral advocacy skills to close hundreds of files

CURRENT PROJECTS

- Selected by NBI to teach National CLE on Traumatic Brain Injury (2016)
- Presented Lecture on TBI for New York Defense Association (2014 and 2015)

- Prepare CLE and CE Training on Biomechanical Defenses
- CLE Speaker for National Business Institute (NBI) on TBI October 2014
- Currently handling Special Account for NYC Brownstones (2015, 2016 and ongoing) including developing training for underwriters on the program
- Teaching DANY CLE on Traumatic Brain Injury September 22, 2016

ATTORNEY **1996-2000**
GEICO STAFF COUNSEL **WOODBURY, NY**
Responsible for all aspects of litigation defending GEICO's insureds. Seven defense verdicts in Kings County. Created and managed motion department and managed four attorneys preparing dispositive motions on behalf of claims professionals. During the first year the group prepared over one hundred motions that lead to the dismissal of dozens of cases.

ATTORNEY **1994-1996**
LAW FIRM OF FRANK MANGIATORDI **NEW YORK, NY**
Responsible for all aspects of plaintiff personal injury litigation including handling complex construction and medical malpractice cases. Handled all pre-trial aspects of medical malpractice cases for senior partner including drafting all motions and attending all discovery issues.

ATTORNEY **1993-1994**
STATE OF CONNECTICUT SUPERIOR COURT **NEW HAVEN, CT**
Responsible for research and writing legal memoranda and decisions for several Judges of the Superior Court in Stamford and Danbury, Connecticut. Several published decisions in the State Reporter.

Education:
JURIS DOCTOR **1990-1993**
PACE LAW SCHOOL **White Plains, NY**
Graduated top 25%, GPA 3.21. Internships in Federal District Court, Southern District of New York and State Appellate Division. I wrote decisions that were published in the Federal and State Reporters.

BACHELOR OF ARTS **1986- 1990**
ADELPHI UNIVERSITY **Garden City, NY**
Graduated Cum Laude from Honors Program. Defended thesis on posttraumatic stress disorder in Vietnam Veterans

INTERESTS AND ACTIVITIES
Assistant Scoutmaster Boy scout Troop 150 Sparta, NJ, merit badge counselor for Citizenship in the Nation, Law, Family and three other merit badges. Order of the Arrow member (Scouting's Honor Society). Fluent in Spanish. Blackbelt First Degree in Tae Kwon Do studying for Second Degree Black belt, testing November 2016. Proud father of an Eagle Scout.