Dr. McDiarmid’s Personal Injury Report for February 2014.

LAW OF INERTIA – HEAD INJURIES (Part 1) AND HOW THEY RELATE TO REAR-IMPACT COLLISIONS
Good Morning: The April 4-5, 2014 TLABC conference, in Vancouver, on brain injury, fast approaching, I thought I would give you an introduction to the topic and specifically how it relates to the rear-impact collision. The list of speakers is excellent, but the program does not appear to cover mTBI and whiplash. It is well established a person does not have to contact the interior of the vehicle’s cabin in order to have a mTBI, and they do occur in Low Speed Rear-Impact Collisions (LOSRICs).

**Mild Traumatic Brain Injury, Not So Mild After All**

*ScienceDaily (Feb. 22, 2010) — Douglas Smith, MD, director of the Center for Brain Injury and Repair and professor of Neurosurgery at the University of Pennsylvania School of Medicine, presented findings on the molecular mechanism at play in mild traumatic brain injury (mTBI), commonly known as concussions, recently at the 2010 American Association for the Advancement of Science meeting in San Diego.*

Although mTBI affects over 1.7 million people each year in the United States, it is generally ignored as a major health issue. However, this "mild" form of injury induces persisting neurological and cognitive problems in many of these patients, exacting an enormous emotional and financial toll on society.

Despite the prevalence and impact of mTBI, little is known about how mTBI affects nerve cells and connections in the brain, and therefore clinical outcomes after injury. Smith and colleagues have begun to amass data from human and animal studies on mTBI at 2-4 days after injury using advanced neuroimaging techniques. They have found distinct changes throughout the white matter in the brain. Also, protein markers of brain pathology were identified after mTBI in the blood of mTBI patients.

Concussion, from the Latin concutere ("to shake violently")[1] or concussus ("action of striking together"),[2] is the most common type of traumatic brain injury. The terms mild brain injury, mild traumatic brain injury (MTBI), mild head injury (MHI), minor head trauma, and concussion may be used interchangeably,[3][4] although the last is often treated as a narrower category.[5] Although the term "concussion" is still used in sports literature as interchangeable with "MHI" or "MTBI", the general clinical medical literature now uses "MTBI" instead.[6] In this article, "concussion" and "MTBI" are used interchangeably. Frequently defined as a head injury with a temporary loss of brain function, concussion causes a variety of physical, cognitive, and emotional symptoms, which may not be recognized if subtle.

Treatment involves monitoring as well as physical and cognitive rest (reduction of such activities as school work, playing video games and text messaging).[7] Symptoms usually resolve within three weeks, though they may persist or complications may occur for month or years.[8]
Those who have had one concussion seem more susceptible to another, especially if the new injury occurs before symptoms from the previous concussion have completely resolved.[9] There is also a negative progressive process in which smaller impacts cause the same symptom severity.[7] Repeated concussions may increase the risk in later life for dementia, Parkinson's disease, and/or depression.[9]

A variety of signs accompany concussion including somatic (such as headache), cognitive (such as feeling in a fog), emotional (such as emotional changeability), physical signs (such as loss of consciousness or amnesia), behavioral changes (such as irritability), cognitive impairment (such as slowed reaction times), and/or sleep disturbances.[7] A 2010 Pediatrics review article focusing on children and adolescents noted that fewer than 10% of sports-related concussions had associated loss of consciousness.[10]

Due to varying definitions and possible underreporting, the rate at which concussion occurs annually is not accurately known, but is estimated to be more than 6 per 1,000 people.[11] Common causes include sports injuries, bicycle accidents, motor vehicle collisions (remember, they haven’t been called ‘accidents’ >10 years), and falls, the latter two being the most frequent among adults.[12] In addition to a blow to the head, concussion may be caused by acceleration forces without a direct impact, and on the battlefield, MTBI is a potential consequence of nearby explosions.[13]

It is not known whether the brain in concussion is structurally damaged or whether there is mainly a loss of function with only physiological changes.[14][needs update] Cellular damage has reportedly been found in concussed brains, but it may have been due to artifacts from the studies.[15] It is now thought that structural and neuropsychiatric factors may both be responsible for the effects of concussion.[16]

**Rear Impact Collision and the Law of Inertia**

Rear Impact Collisions are a major cause of concussion in the US, approximately 60% to 67% of all TBI are the result of MVC.

**The Law Of Inertia**

The object in question in a whiplash trauma is a human body. The human body has two large parts that have their own separate inertia, the trunk and the head. These two large pieces of inertial mass (the head and the trunk) are connected by a thin pole, the neck.

Upon impact, the target vehicle begins to move forward into the occupant, making contact chiefly through the seat back. In accordance with Newton’s 1st law of motion, the occupant’s inertia resists this motion.
As the seat back continues to move forward, the occupant must yield. Initially, the thoracic curve is flattened by the seat back. This results in a vertical compressive force which is transmitted through the spine.

So far, we have not been able to determine to what degree—if any—the lumbar spine also flattens. As the vertical compressive force \((-z)\) continues up the spine, some rise of the torso also occurs. This is called ramping and is halted after 1-3 inches of vertical displacement, usually because of the restraining effect of the seat belt and the weight of the torso. Meanwhile, as the torso now is undergoing both a \(z\) acceleration vertical and an \(x\) linear acceleration, the head—also acting in accordance with Newton’s 1st law of motion—attempts to remain at rest. As the vertical force extends upwards into the neck it initiates flexion of the upper cervical segments and hyperextension of the lower segments.

As the torso continues to move forward, the neck begins to pull the head along with it. This has the effect of further flexing the upper cervical spine and hyperextending the lower cervical spine (primarily the C5-6 segments) and the spine assumes an s-shaped configuration, Figure below. The head also is induced to extend along with the neck as the head takes up the backset distance during the head lag phase.

Depending on specific head restraint geometry (occupant's position relative to the restraint), head restraint contact will usually occur in about 100 msec at which time head translational acceleration will peak. Any stored energy in the seat back from its deflection (usually about 5-15 degrees) will be released as the occupant begins to more forward into the re-entry phase. This effectively increases the torso and head speed (overspeed).

As this change of phase occurs, the direction of horizontal shear reverses rapidly and the rearward bending moment quickly gives way to a forward bending moment. Depending on the initial position of the occupant with respect to safety harnesses, the lap and shoulder portions will eventually restrain the forward moving torso which will effectively aggravate the neck’s bending moment since the forward moving head’s inertia again is behaving in accordance with Newton’s 1st law of motion—this time in the \(x\) direction with some angular momentum and acceleration. The weight of the human head (10-12 lbs.) plus the force of a moving vehicle that may carry 10,000 to 20,000 pounds of force is not a good combination. Below is the example of the head striking which is also the same mechanism of the head striking the head rest.
Loss of Consciousness (LOC). Inquire whether LOC occurred or was observed and the length of time the patient lost consciousness. (Note: Research indicates that up to 90% of concussions do not involve LOC.)\textsuperscript{19,20}
Post-traumatic amnesia is better predictor of outcome after MTBI than loss of consciousness; recent research has indicated that amnesia may be up to 4-10 times more predictive of symptoms and cognitive deficits following concussion than is LOC (less than 1 minute). CDC Toolkit, “Heads Up: Brain Injury in Your Practice,” Acute Concussion Evaluation (ACE), Instructions, p. 2

This is a troubling trend, this also indicate that most general medical practices, family practices, primary care physicians, urgent care facilities and chiropractic offices are also missing at least 56% of the injuries.

Neuropathophysiology of MTBI

Unlike more severe TBIs, the disturbance of brain function from MTBI is related more to dysfunction of brain metabolism rather than to structural injury or damage. The current understanding of the underlying pathology of MTBI involves a paradigm shift away from a focus on anatomic damage to an emphasis on neuronal dysfunction involving a complex cascade of ionic, metabolic and physiologic events. Clinical signs and symptoms of MTBI such as poor memory, speed of processing, fatigue, and dizziness result from this underlying neurometabolic cascade.¹⁷

The purpose of the Personal Injury Report is to keep you updated on relevant academic concepts pertaining to side/rear impact whiplash injury patients. I hope that the information is useful in terms of enhanced understanding, as well as helpful for the personal injury attorneys to deal with insurance claim adjusters, healthcare providers and adverse medical experts.
This spring is the final leg in my journey to be able to qualify as an expert witness in crash reconstruction. Previously you would have to hire an ACR, which for the most part really did nothing when it came to relating the injuries to the crash. As one of only a very small handful of health care providers, in Canada, certified in Crash Reconstruction I am able to testify regarding issues related to the crash itself, the biomechanics of how the person(s) were injured, and of course the clinical, all in one sitting and all from one person, something never before made available to counsel on Vancouver Island, let alone the lower mainland.

As we all know only 3-5% of these cases over make it to trial, and the number one reason these cases fail (let alone get minimal settlements) is failure to link the specifics of the crash to the specifics of the injuries. Having an IME report, including all three components, is proving most ‘enlightening’ when talking settlement amounts and the other side claims your client could not have been injured. Because the average medical doctor has as much training in these injuries as they do in dentistry, your clients own doctors lack of findings is typically used against your client. Those days are now over!!!!

Let’s start to introduce some actual science into these cases and get away from so many of those tired, and outdated, arguments. My hope is that this, and earlier PI Reports, will help us on the road to less reliance on Cultural Authority and more reliance on peer reviewed literature.

Additionally, your law firm has access to daily phone consultation (or email) with me, to discuss any pertinent issues you might face, on a particular case. That applies as much to defense as plaintiff counsel.

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References:


