

COMPARISON AND CHARACTERIZATION OF DIFFERENT CONCUSSIVE BRAIN INJURY EVENTS

Marshall Kendall

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University of Ottawa

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“The measure of who we are is what we do with what we have.”

Vince Lombardi

Abstract

Concussions are debilitating injuries affecting the short and long-term health of those who suffer from them. While an increased awareness of the injury has helped lead to a better understanding of the importance of better monitoring and treatment protocols, concussive injuries continue to occur at an alarming rate. Current injury risk thresholds exist and are used in the development of better equipment to protect athletes in high impact sports, however much of this data is based on simulations and/or cadaveric and animal model data from falls. Thus, there is a lack of data from concussive injuries resulting from a multitude of injury events within different sports, including collisions, falls and punches. The purpose of this thesis was to use dynamic response characteristics and brain tissue response to compare four different injury events from reconstructions of real life concussive injury events. This research was designed to provide information related to brain trauma characteristics specific to four common concussive brain injury events.

Seventy-two (72) injury reconstructions were used in this study involving four injury events; collisions, helmeted falls, unhelmeted falls and punches. The results from the first study revealed that while all injury events produced similar MPS and Von Mises stress values, the injury events produced different peak linear and rotational accelerations. In terms of risk for concussive injury, differences were also found between percent risk for concussion and the specific injury events, questioning the validity of current concussive thresholds' applicability to across all types of concussive injury events. The second study aimed to characterize each concussive injury event by means of specific kinematic characteristics unique to that particular event. The results showed that dynamic response variables that accounted for the most variance changed dependant on the concussive producing event. The third study compared maximal principle strain and strain fields within the brain by the specific injury event. The results showed that global strain within specific regions of the brain were significantly different between the different injury events. Furthermore, unique strain fields within the cerebrum were found between the four concussive injury events.

The three studies in this research program characterize four common concussive injury events found in sport. It aimed to describe the unique dynamic response characteristics for each injury event that may have significant influence on protective equipment development and standards testing. Finally, though the aim of this study was not to correlate location of strains within the brain with onset of concussive symptoms and duration, this study demonstrated that the concussive producing event can have an effect on location of peak strain and strain field within regions of the cerebrum associated with concussive symptoms.

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PART I

CHAPTER 1

Introduction

Brain injury is a debilitating problem that affects over 1.5 million people annually in the United States alone [CDC, 1999; Sosin et al., 1996]. The effects of brain injury can vary significantly dependant on the severity of the injury. Traumatic (TBI) and mild traumatic (mTBI) brain injury typically result in significant injuries affecting the patients' quality of life, livelihood and could even result in death. The annual direct and indirect cost of traumatic brain injury in Canada has been estimated at \$3 billion, with over 11,000 associated fatalities. While mTBI type injuries can occur to anyone, a recent rise of concussive injuries in high impact sports, such as football and hockey, has captured the attention of brain injury researchers. Recent studies have shown that mTBI type injuries may be associated with more long-term conditions, such as chronic traumatic encephalopathy (CTE), which can have significant effects on the quality of life of the individual suffering the repercussions (i.e. Parkinson's, Dementia, Alzheimer's) [McKee et al., 2009 ; Thurman et al., 1998]. As a result, research aiming to better understand the incidence of concussion has increased.

Head and brain injuries occur due to either a direct or an indirect impact to the head [Viano et al., 1989]. The inbound energy from these impacts may result in a traumatic brain injury (TBI) or mild traumatic brain injury (mTBI). These injuries can be divided into two groups; focal or diffuse injuries. Contrary to traumatic brain injuries, where MRI and other medical scans can help determine the severity and location of a particular brain injury, mild

traumatic brain injuries, such as concussions, rarely show up in MRI or other types of scanning technologies. This is problematic in that it limits the ability to determine injury severity and area within the brain affected by mTBI. While there has been virtual extinction of traumatic brain injuries in hockey and football, mild traumatic brain injuries (mTBI) have increased. Currently, there is lack of research defining the different impact events leading to mTBI. While the force of the impact plays an important role in the mechanics of brain injury, other factors, such as impact location and impact duration, add to the complexity of head injuries. Thus, the relationship between kinetic input and the resultant head injury is not as simple as cause and effect [Hardy et al., 1994]. Previous researchers attempted to create concussion thresholds based on kinematic data, such as peak linear acceleration. However, peak linear acceleration did not correlate well with the incidence of actual brain injury [Oeur et al., 2011]. Finite element modeling (FEM) of the brain was developed in order to aid in simulating and quantifying brain deformation metrics [Horgan and Gilchrist, 2003; Willinger and Baumgartner, 2003a/b; Zhang et al., 2004; Kleiven, 2007]. Injury risk thresholds for concussion have been created using injury reconstructions in conjunction with FEM [Zhang et al., 2004; Kleiven, 2007]. This method proved to correlate better since it uses the acceleration time histories to determine the resulting brain deformations rather than a single peak acceleration value [Post and Hoshizaki, 2012]. The shape of acceleration time-history curves has been shown to change the output of brain deformation metric values within the brain from the FEM [Post et al., 2012]. It is still, however, unclear which characteristics within the kinematic variables are most influential in creating these changes for mTBI injury levels. For instance, head injuries involving a fall to the ground versus someone receiving a punch to the head are parametrically quite different even though both may result of a concussion. Symptoms resulting from the concussive injury may have some

similarities; however, injuries that affect deeper brain tissues may give some indication of injury severity and possibly correlate back to the duration of the given concussive symptoms or duration of symptoms. The variance in symptoms and time out of the game is likely a result of the manner in which the concussive injury occurred. Understanding the differences between the different concussive injury events has yet to be well defined.

The purpose of this research was to compare four concussive injury events by peak dynamic response and brain tissue stress/strain resulting from reconstructions of diagnosed concussive injuries. Furthermore, this thesis aimed to uniquely characterize each injury event through specific characteristics of the dynamic response time histories curves (i.e. peak, slope, duration) which accounted for the most variance. Finally, the location of the peak strain within the brain was compared between the different concussive injury events. The result of this research should provide important information related to brain trauma characteristics related to the different types of concussive injury events.

Epidemiology

Brain injury is a growing problem that significantly affecting ones' ability to function normally in day-to-day activities. Reports show that 75%-90% of the 1.4 million TBI related deaths; hospitalization and emergency room visits each year are mTBI or concussive type injuries [CDC, 2003; Kraus & Nourjah, 1988; Luerssen et al., 1988; Lescohier & DiScala, 1993]. The direct medical costs and indirect costs such as lost productivity from mTBI totaled an estimated \$12 billion in the United States in 2000 [Finklestein et al., 2006]. While brain injuries can occur doing daily tasks, sports provide an increased risk to children and adults alike. An estimated 38 million children and adolescents participate in organized sports in the United States

[National council for youth sports, 2001]. Alternatively, 170 million adults participate in physical activities, including sports [Exercise CDC, 2006]. The Centers for Disease Control estimates that 1.6 to 3.8 million concussions occur in sports and recreational activities annually [Langlois et al., 2006]. Among the youth, the incidence of concussion in sports is second only to motor vehicle accidents [Sosin et al., 1996]. While head injuries can occur to any age group within the population, the recent attention concerning concussions brought forward by the media in sports, such as ice hockey and football, has increased awareness of the injury. These two sports in particular have recently been associated with increases in the incidence of concussive type injuries (mTBI). The sport of football has been shown to have the highest concussion rate (6.4 per 10,000 Athlete Exposures (AE)), followed by boys' ice hockey (5.4 per 10,000 AE) and boys' lacrosse (4.0 per 10,000 AE). Interestingly, this study also showed that concussions represented a greater proportion of total injuries among boys' ice hockey (22.2%) than all other sports studied (13.0%) [Marar et al., 2012].

According to the International Ice Hockey Federation (2010), Canada has over 577,000 registered hockey players competing at a variety of levels. The British Columbia Injury Research and Prevention Unit (2010) reports that in 1999, 3.8% of all sport-related emergency room visits in Canada for 1999 were head injuries in ice hockey. Recent epidemiology studies have identified different impact event leading to concussive type injury in ice hockey. Hutchison et al. (2012) reported that player-to-player contact accounted for 88% of concussion occurrence in professional hockey players during a 3-year timeframe. These findings are slightly higher than previous findings [Benson et al., 2002; Marar et al., 2012, Gerberich et al., 1987; Agel et al., 2007] that further broke down the player-to-player contact into specific categories where the injury likely occurred. They reported head injuries occurred with player-player contact (38% -

70%) and player-playing surface contact (17 - 39%). Identifying the different impact events creating the injury is an important first step in working to prevent them from occurring. Without proper knowledge, surrounding the characteristics of these different impact events, rule changes and the development of new testing standards for optimization of protective equipment to protect athletes may face significant challenges.

Types of Brain injuries

In sports, most concussive brain injuries occur from direct contact with players, immovable structures or the ground. Two main groups of brain injuries typically ensue from the damaging energy of these impacts; focal or diffuse type injuries.

Focal injuries are usually associated with severe head and brain injuries such as skull fractures or skull deformation and intracranial bleeds (subdural, epidural and intracerebral) haematomas which occur mostly at the location where the impact occurred. These injuries tend to occur when the brain motion inside the skull, due to both linear and rotational accelerations, causing contusions from contact with bony protuberances inside the skull and bleeds from arterial/venal tears inside the brain (Viano et al., 1989).

Diffuse brain injuries have been associated primarily with high head rotations causing diffuse strains to brain tissue over a larger area of the brain. These diffuse effects of the rotational component of inertial loading are produced by a centripetal progression of strains, which increase from the outer surface of the brain and move towards the core of the brain dependant on the level of inertial loading [Ommaya and Gennarelli, 1974]. Two forms of diffuse injuries have been identified in literature; diffuse axonal injury (DAI) and concussion or mTBI. As stated above, these injuries have the distinct characteristic of affecting a large area of the

brain due to the propagation of the impact energy throughout the brain tissue. Current theories state that this energy results primary from the rotational component of the impact. While various levels of severity of DAI can be diagnosed through fMRI, lower level mTBI (concussion) cannot, thus making it very difficult to diagnose or determine the severity of a concussive injury.

Mild traumatic brain injury (mTBI)

Mild traumatic brain injury (mTBI) has been defined as any trauma-induced combination of symptoms, signs and clinical laboratory tests that raise a reasonable suspicion of brain damage resulting from the traumatic impact [Margulies, 2000]. The severity of a concussive injury can vary quite extensively from one individual to another. Current methods for evaluating these injuries relate mostly to the presence of symptoms. While the symptoms associated with concussions tend to be temporary, some individuals continue to have symptoms for prolonged durations (persistent concussive symptoms (PCS)). It has been hypothesized to be associated with severity of concussion; however no research to this date has shown a correlation between PCS and concussion. As with DAI, concussions are believed to primarily result from the rotational component of an impact. These rotational components of the impact have been shown to create strains and stresses to specific locations within the brain, leading to specific physiological responses (symptoms) of concussion [Holbourn, 1943].

Diffuse axonal injury (DAI)

DAI affects the white matter of the brain. In particular, the axons of the white matter are damaged from shearing forces within the brain tissue as a result primarily of rotational components of a given impact. These strains and stresses can affect all areas of the brain depending on severity of the injury and thus can result in injury outcomes varying on a

continuum from loss of consciousness to death [Bandak, 1994]. Diffuse tensor imaging (DTI) has recently provided a means to track fluids movements within the white matter tracts.

Summary

The focus of this research will be on the mTBI/concussive injuries. While the material characteristics of the brain are complex and its' physiological response are still not completely understood, the kinetics and kinematics creating concussive injuries are also unique and complex by nature. Head protection in sports continues to evolve, however the complex nature of the concussive injury has made it difficult to identify accurate injury thresholds and appropriate injury metrics for concussive type injuries. Research isolating injury event specific dynamic response variables could lead to the development of protective headgear for specific sports, decreasing the risk of concussive injuries specific to the impact event within each particular sport.

CHAPTER 2

Anatomy of the Brain

The brain is one of the biggest and most complex organs in the human body. An adult male brain typically has a mass of approximately 1.4 kilograms made up primarily of fat and protein. The functions of this organ are seemingly never ending. With more than 100 billion nerve cells, the brain not only puts together thoughts and highly coordinated physical actions but also regulates our unconscious body processes, such as digestion and breathing. An injury to any part of the brain would likely disrupt one or more of these functions leading to, depending on the severity of the injury, disability or even death. Brain injury research measuring direct tissue damage and brain motions is very challenging. The use of cadavers, physical models and animal models, although they provide important information, are still not without their own inherent limitations. Scaling errors (animals), live tissue response versus dead tissue response to an impact is likely different from one another (cadaveric) are just a few examples of the limitations of these methods. In response to these limitations, the creation of finite element models of the human brain opened the door to a means of measuring tissue deformation.

The brain is made up of billions of nerve cells, which are known as neurons. These neurons make up the organ's grey matter. The neurons transmit and gather electrochemical signals that are communicated via a network of millions of nerve fibers called dendrites and axons. These nerve fibers make up the brain's white matter. This organ sits in a fluid known as cerebrospinal fluid (CSF) while encased with a compact type bone structure comprising the human skull for protection. The CSF is located in the brain and spinal cord and forms a protective cushion that gives some buoyancy to the central nervous system organs

Regions of the Brain

Three primary regions form the human brain; the cerebrum, cerebellum and brain stem (Figure 2-1).

Cerebrum

The cerebrum is the largest part of the brain, accounting for 85 percent of the organ's weight. The cerebrum contains two hemispheres, which is divided into four regions; the frontal lobes, the parietal lobes, occipital lobes and the temporal lobes (Figure 2-1). Each of these regions have specific roles critical to sustaining life, physical and mental functions. The frontal lobes are located behind the forehead and are involved with speech, thought, learning, emotion, and motor functions. Behind them are the parietal lobes, which process sensory information such as touch, temperature, and pain. At the rear of the brain are the occipital lobes, dealing with vision. Finally, the temporal lobes, which are located near the temples, are involved with hearing and memory. Each hemisphere of the cerebrum is composed of grey matter and white matter [Marieb, 1998]. The grey matter is the most superficial tissue and is referred to as the cerebral cortex.

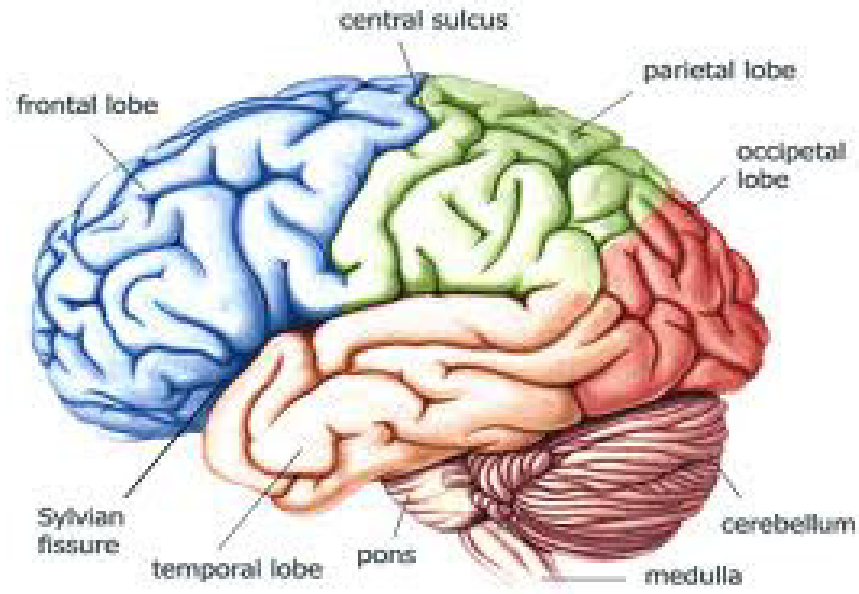


Figure 2-1. Regions of the human brain

The cerebral cortex accounts for roughly 40% of the total brain mass. The cerebral cortex contains three areas: i) motor areas; ii) sensory areas; and iii) association areas. Motor areas control voluntary motor functions, sensory areas provide conscious awareness of sensation, and association areas act to integrate diverse information for action [Marieb, 1998]. Located below the grey matter in the cerebral cortex is the cerebral white matter. The role of the white matter is to provide communication between cerebral areas, the cerebral cortex and lower central nervous system (CNS) centers. White matter is composed of myelinated fibers bundled into large tracts [Marieb, 1998]. White matter essentially connects the cortex to the rest of the nervous system, including the receptors and effectors within the body.

Cerebellum

The cerebellum is the second largest part of the brain, accounting for around 11% of the total brain mass (Figure 2-1). It is located in the posterior cranial fossa of the skull, under the

occipital lobes and is separated from them by the tentorium cerebelli and the transverse cerebral fissure. Like the cerebrum, the cerebellum has a thin outer cortex of grey matter, internal white matter and small deeply situated pockets of grey matter. The cerebellum acts to coordinate body movements, maintaining muscle tone, posture and equilibrium. It also plays a major role in skilled motions [Marieb, 1998].

Brain Stem

The brain stem is located at the base of the skull where the spinal cord enters the cranial cavity. Like the spinal cord, it consists of deep grey matter surrounded by white matter fiber tracts. The brain stem is composed of three parts: the midbrain, the pons and the medulla oblongata. The brain stem is known to produce the automatic behaviors necessary for survival, and relays messages between the spinal cord and the cerebrum [Marieb, 1998]. Concussion severity and loss of consciousness was thought to be associated with injury to this region of the brain [Ommaya & Gennarelli, 1974].

Physiological functions of the brain

Brain injury is very complex given the high complexity of the anatomical structures as described previously and their inter-connectivity between different regions. These shared connections make it challenging to understand the onset and duration of particular symptoms post concussive injury. Concussive injuries tend to exhibit a variety of symptoms that can vary in terms of intensity and duration. Research has suggested that some of the symptoms commonly associated with concussion such as, cognitive processing speed, verbal memory, visual memory and stress, may represent damage to a specific functional regions of brain tissue (Purves et al.

2001; Levin et al. 2012). Specific regions within the brain are responsible for managing these different functions (Table 2.1)

Table 2-1. Brain regions and their functionality

Region of the Brain	Functionality	Reference
Prefrontal cortex	decision-making and social behaviour	Yang et al. [28]
Dorsolateral prefrontal area	memory and organization	Yang et al. [28]
Motor association cortex and Primary motor cortex	planning and execution of movement patterns	Luppino & Rizzolatti [29]
Primary somatosensory cortex	sense of touch and proprioception	Price [30]
Visual cortex	Visual information	Braddick et al. [31]
Sensory and Visual association areas	Sensory integrations and perception of the environment	Price [30]
Auditory cortex	sound processing	Purves et al. [15]
Auditory association area	functions associated with recognition of sounds	Creutzfeldt et al. [32 – 33]

While concussive injuries are virtually undetectable via medical scanning, previous research on stroke patients has shown that physical tissue damage to a particular region of the brain could result in various levels of functional disability related to the affected area [Shumway-Cook & Olmscheid, 1990; Dault & Dugas, 2002]. A recent study using reconstructions of concussive injuries did show a link between axonal damage, regions of brain tissue and associated concussive symptoms [Wright et al. 2012].

Summary

Currently, concussive symptom onset and duration resulting from a hit to the head can vary significantly between individuals. Common symptoms such as headache, dizziness, nausea or vomiting are usually observed immediately follow the impact, while other symptoms such as persistent headache, poor attention, concentration and memory dysfunction have been reported within days to weeks after sustaining a concussion [Kelly and Rosenberg 1997]. For the most part, concussive symptoms tend to resolve within 3-7 days however some cases report symptoms lasting longer than two weeks [McClincy et al. 2006] or even up to a year [Alves, et al., 1993]. There has been some suggestion that concussive symptoms may be linked to the specific region of the brain that suffered brain tissue damage because of the impact. In theory, each concussive injury impact event could affect a specific/different region of the brain, thus creating unique symptoms and duration of the injury. Identifying the specific region of brain injury as it pertains to different concussive injury events has not yet been defined.

CHAPTER 3

Paradigm for head/brain injury

An injury mechanism is described as the mechanical and physiological change that results in anatomical and physiological damage [Viano et al., 1989]. The different injury impact events affecting these injury mechanisms can have significant effects on the brain tissue response to an impact. The head injury paradigm described by Gennarelli (1993) (Figure 3.1) relates mechanical input to biological response. While static loads are considered injurious, they are quite often focal type injuries related to the site of the applied force. Dynamic impacts not creating skull fracture tend to induce more complex and diffuse type brain injury response. If the skull fractures, the injury will most likely remain superficial and affect the area focally, causing cerebral lacerations and hemorrhages [Holbourn, 1945]. This research paper will focus primarily on the dynamic impact loads and their effects on brain tissue for closed head brain injuries causing concussions. The resultant brain injury resulting from these dynamic loads can be dependent on multiple factors such as impact location, duration, magnitude and the types of impact force applied (linear or rotational acceleration). These parameters are important to understand when associating a loading condition creating a concussive injury to actual brain tissue injury thresholds.

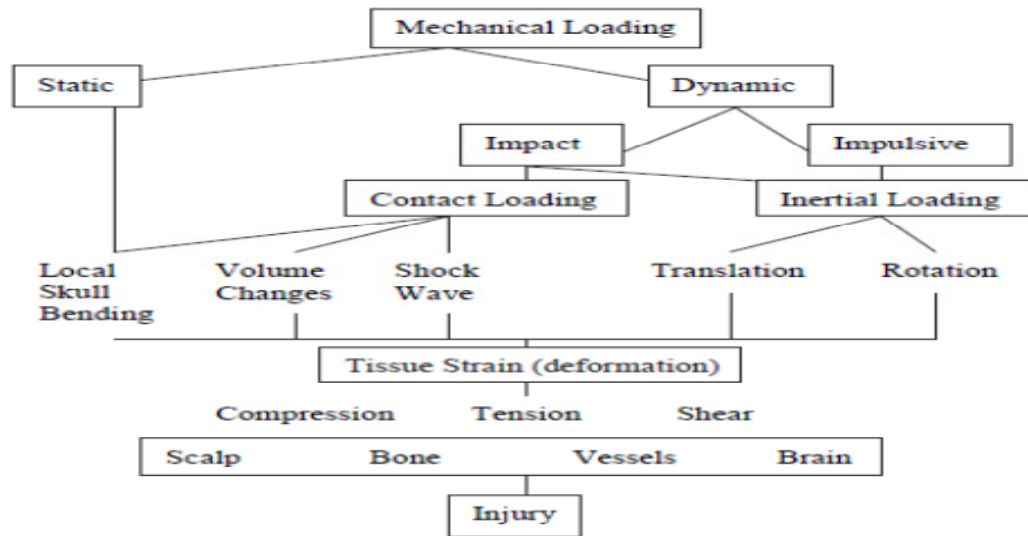


Figure 3-1. Mechanical loading to the head may lead to tissue damage through deformation or acceleration of the head. This tissue damage may lead to brain injury [Gennarelli, 1993].

Mechanism of Injury

Many theories of head/brain injury mechanisms have been proposed and developed through literature over the past decades. The most notable mechanisms presented in literature are; Pressure gradient (negative and positive), acceleration (linear and rotational) and brain deformation (relative motion, stress and strain). These will be described in greater length in the following section.

Researchers tend to agree that concussive type injuries tend to result from more diffuse type injuries including concussion (mTBI) which can be very mild to severe (PCS) and diffuse axonal injury (DAI), which would be a more severe type injury which causes injuries such as cerebral swelling, cerebral ischemia. It is understood that most collisions involving the head result in a certain proportion of linear and rotation motion, and thus create a complex cascade of physical tissue deformations and physiological responses. The principle mechanism of injury for purely

linear acceleration loadings has been reported to principally be related to pressure gradient changes within the skull, while for purely rotational acceleration loadings of the head have been associated to be shear stresses of the brain tissue [Unterharnscheidt, 1971].

Cavitation theory

The understandings of the mechanisms of brain injury have evolved significantly over the years. Linear acceleration was deemed the most important parameter in brain injury in the early studies examining head injury. Several theories were proposed as to how the brain would be injured following translational accelerations. Some of these theories included the creation of pressure waves within the brain, skull deformation, brain deformation and pressure gradients changes. Gross (1958) proposed a theory that pressure may be an indirect cause of injury since the collapse of the cavity (cavitation) is what damages the brain, not the pressure itself. The results of Gross's research sufficiently support the explanation of contre-coup injuries. The explanation for coup injuries due to negative pressures was more difficult. It was hypothesized that this injury is caused by a rapid return of the skull to its original geometry following a blow, thus creating a cavity between the area of the brain and skull (in its deformed position). Adding to this theory, Suh et al. (1972) showed that negative pressure was not located in the region opposite to the impact, which contradicted the idea of contre-coup injury due to negative pressure. Some researchers began to doubt the negative pressure theory referencing that the density of the brain materials are very different from density of the liquids being used in testing. Stalhammar (1975a, 1975b) and Stalhammar & Olsson (1975) reported, after impacting tests with rabbits, that negative pressures developed through impacts were not important to the injury mechanism. Lubock & Goldsmith (1980) also agreed that cavitation coincided with negative pressure transients; however, the formation of bubbles (release of gas or vaporization) would

more likely occur in liquid than brain tissue. Nusholtz et al. (1984, 1987) through a series of impact to macaques, stated that negative pressure is present during an impact but non-injurious. Though cavitation seems to be an important influence on focal type injuries, it is not accepted as a primary cause for injury (Horgan, 2005).

Pressure Gradient

The theory of pressure gradient changes within the brain as a mechanism of concussive injury follows closely the cavitations theory. The theory states that following an impact to the head, the brain will have a tendency to go from a high-pressure location to a low-pressure location. This tendency can create shear stresses and deforms the brain. An actual brain injury would occur when small bony protuberances resist this deformation thus damaging the tissues. An early study using strain gauges and pressure plugs to investigate the dynamic skull stress in impacted dogs [Gurdjian & Lissner; 1944], showed that compression and pressure increases at the impact site and tension and pressure decreases at the opposite site. It was deemed that dynamic stress was produced by pressure gradients. Using a methodology causing concussive events in dogs, Gurdjian et al. (1953) again monitored intra-cranial pressure and acceleration. The findings from this study would suggest that brain injuries would develop from either a short duration, high magnitude acceleration and pressure pulse or a long duration, low magnitude pulse. Thomas et al. (1967) used physical models to demonstrate that as acceleration and pressure increased, so did the pressure gradient. They concluded that pressure gradients were present post-impact and that compression and acceleration were both important to injury mechanism. Gurdjian et al. (1968) applied this line of thinking to animal models and confirmed the findings reported by Thomas et al. (1967) that acceleration and skull deformations caused by an impact caused pressure gradients. They also noted that their observations showed that the

brain has a tendency to flow from a high-pressure area to a low-pressure area, thus causing brain deformation and shear stresses. Further research by Gurdjian (1978) related acceleration to the creation of pressure inside the skull following an impact. This study produced occipital blows to Rhesus monkeys' head sliced in the medial plain and placed on a glass plate prior to the impact. With the ability to visually monitor brain motion, it was revealed that a frontal space was created during the impact, and there was movement of the brain stems following occipital impacts. Thus, the pressure gradient is believed to be formed by skull deformation at the impact site and the consequential relative motion of the intracranial contents.

Linear acceleration

Linear acceleration was initially considered to be the most important mechanism of brain injury [Gurdjian & Lissner, 1944; Gurdjian et al., 1953, Gurdjian et al., 1963]. The role of rotational acceleration, negative pressure and cavitation were not accepted early on, with researchers stating that these mechanisms had little or no significant effect on brain tissue injury. Further claims stated that acceleration and deceleration were present in typical head injuries and almost always associated with skull compression. This led to a theory that deformation of the skull as well as changes in intracranial pressure were linked to brain trauma. Ommaya et al. (1966) indicated that rotation alone could not produce the levels of injury caused by direct impact. Cerebral concussion was produced in monkeys by direct impact to the occipital area of the head and by impact to the base of a mobile chair. The analysis of the extensive data collection revealed that although concussion could be caused by whiplash, it required higher levels of acceleration than what was required by direct impact, making rotational acceleration a lesser contributor to the mechanism of brain injury. Ommaya et al., (1966) estimated that approximately twice the rotational velocity was required to produce cerebral concussion by

indirect impact. The importance of rotational acceleration began to gain ground based on further research by Ommaya and Hirsch (1971) which suggested that rotation could account for as much as 50 percent of the potential for brain injury, while the remainder was attributed to direct impact. More recently, researchers began to agree that brain injury might occur according to different impact mechanics. Studies by Gennarelli et al. (1971, 1972) showed that translation of the head in the horizontal plane would produce mainly focal effects, thus causing contusions and intracerebral hematomas. Diffuse injuries were observed only when a rotational component was involved. Unterharnscheidt (1971) verified these findings further with a study where they describe the principal mechanism resulting from linear acceleration to be pressure gradient, while shear stress, from differential motion between the skull and brain, was identified as the mechanism for rotational acceleration. Ono et al. (1980) conducted a series of experiments with monkeys and found no correlation between the occurrence of concussion and rotational acceleration. It was concluded that the concussion could be produced by linear acceleration from a direct impact.

Rotational Acceleration

Rotational acceleration have been linked to diffuse type brain injuries. This type of injury can arise with or without a direct impact to the head. Early research by Holburn (1943) referred to force and deformation of different areas of the brain following direct or indirect movements (impact) causing brain injuries. Holburn's theories were developed and adjusted to adhere to Newton's laws of motion within the shape of the human skull and brain. This theory states that all injuries could be replicated through the application of rotational acceleration alone and that linear acceleration has little influence on injury outcome. It is primarily due to the brain's inability to rotate freely in the frontal compartment, which creates strains and stresses causing

injury. This supposedly explains the predominance of anterior fossae injury regardless of frontal or occipital impacts (Hardy et al., 1994). Holbourn (1943, 1945) referenced the properties of the brain as primary factors in how the brain can be injured. Based on these properties (high bulk modulus (incompressible) and low Young's modulus (high flexibility and shape change)) it would be assumed that the brain is subject to injury through rotational acceleration rather than by linear acceleration. Holbourn also referenced the frontal and temporal regions of the brain as the most susceptible injurious regions due to rough geometry of the skull. Unterharnscheidt & Higgins (1969) successfully tested Holbourn's theory by applying rotational accelerations (no impact) to squirrel monkeys. Observations from this study showed that the monkeys suffered from subdural hematomas, torn bridging veins and lesions of the brain and spinal cord. An important study by Lowenheilm (1975) proposed rotational acceleration as the cause of gliding contusion resulting from excessive strain in cerebral vessels. It was concluded that the site of maximum shear occurred at a constant distance from the surface of the brain. This was the first study of its kind to report on location of injury within the brain resulting from different amplitudes of rotational acceleration. They identified that the deep brain could be injured while the surface was not injured and that the zone of maximum shear became deeper as the rotational acceleration pulse duration increased. A series of studies found good correlation between concussions and rotational acceleration after delivering non-deforming rotational impacts to monkeys [Gennarelli et al., 1971; Gennarelli et al., 1981; Gennarelli et al., 1983; Gennarelli and Thibault, 1982; Thibault and Gennarelli, 1985]. They also stated that rotational acceleration contributed more than linear acceleration to the generation of concussive injuries, diffuse axonal injuries, and subdural hematomas. They hypothesized that these injuries were induced by the shear strain generated by rotational acceleration. Though much of the above research seemed to

prove the importance in rotational accelerations in causing brain injury, most of these studies involved animal models. Adams et al. (1981) tried related findings in these earlier studies to clinical findings in humans. Though the findings were closely related, there were some differences regarding the location of the brain injury. For instance, while white matter was damaged in the animal models, they did not find white matter damage in the human brains. Even with this difference, they did still find subdural haematomas, which is similar to previous animal-modeled studies. While amplitude of rotational acceleration had been investigated, the duration of the pulse was not reported. Gennarelli (1983) tackled this topic with a study where they attempted to produce diffuse axonal injuries in subhuman primates. It was found that subdural haematomas were produced by short, high amplitude acceleration pulses while DAI was produced by longer, lower amplitude pulses. From this study it was concluded that virtually all types of injury can be produced by rotational acceleration applied to the head if applied appropriately.

Combination Linear/Rotational

It is unrealistic to think of a head impact with purely linear or purely rotational acceleration. Bayly et al. (2005) demonstrated that a combination of both translational and rotational brain movements were present for purely linear impacts. While the mechanisms causing brain injury are still unclear, it has been suggested by many researchers that a combination of multiple mechanisms may be at play. A study by Gurdjian & Gurdjian (1975) suggested that a combination of elastic skull deformation, positive and negative pressure as well as an inertial brain lag could present a clearer picture of head injury. This study did confirm that both linear and rotational accelerations components played a role in concussive brain injuries.

Ommaya et al. (1971) disputed the cavitation hypothesis of coup and contre-coup contusions based on real world data and their work with Rhesus monkeys. Based on their findings, they proposed that skull distortion and head rotation were the mechanisms involved and not linear dominant mechanisms such as coup, contre-coup and negative pressures. With relative brain movement within the skull resulting from both linear and rotational components of a head impact, understanding the mechanics related to how the brain is injured as a result of this had not been well understood. In response to this, a study by Ommaya and Gennarelli (1974) contributed to this lack in the literature with the development of the centripetal theory of cerebral concussion. Their research determined that some form of rotation of the head was necessary in order to achieve various categories of concussive injury, such as loss of consciousness. The basis of this theory surrounds the material properties along with the geometry of the inner skull and the brain. They claim that the diffuse effects of the rotational component of inertial loading are produced by a centripetal progression of strains, which increase from the outer surface of the brain and move towards the core of the brain dependant on the level of inertial loading. They do caution that symptoms of brainstem injury can occur in the absence of significant cortical injury, supporting the claim that the mechanisms of concussion may be more complex than a single mechanism on its own. Willinger et al. (1992a) studied the theory of multiple mechanisms further. They attempted to better understand contre-coup lesions following direct impacts to the head. Their findings noted that the particular types of injury were related to the impacted structure and the impact location and suggested three define mechanisms of brain injury; brain-skull relative motion, local bone deflection and intra-cerebral strain [Willinger et al., 1992b]. The inertial motion of the brain within the skull is a key component to concussive brain injuries. Stalnaker et al. (1977) claimed that the head does not always function as on single mass, yet can

also function as a group of individual masses. Assuming this was a correct theory, head and brain motion would not necessarily move in the same direction and in the same method one to another. A later study [Willinger et al, 1994] validated their theory using natural resonance frequency to determine if head motion was considered whole or in multiple masses. This study determined that an impact below a frequency of 120 Hz then the head and brain moved as a whole and thus diffuse axonal injury could be observed as an injury. Whereas, if the frequency was above the 120 Hz value, then the head and brain functioned as multiple masses creating relative motion of the brain within the skull which created injuries such as contusion and subdural hematomas. The theory of relative brain motion has been investigated in the past using cadaver and/or animal specimen and qualitative observation of global motions [Pudenz and Sheldon, 1946; Hodgson et al., 1966; Nusholtz et al., 1984]. While these models allow for measurement of head and brain relative motion, there are question on the transferability of the data to real life injuries. In response to these concerns, methodologies using bi-planar x-ray to follow brain motion during closed head cadaveric impacts were developed [Hardy et al., 2001]. While these impacts use cadavers that are of comparable size and tissue, differences in head response due to lack of fluids and tissue degradation has been stated as a limitation of this methodology. Bayly et al. (2005) used live human subject, subjected to very mild head deceleration (for ethical reasons), to monitor brain movements using magnetic resonance imaging. This methodology was limited to very low impacts velocities in comparison to the other methodologies, however did confirm the presence of both linear and rotational acceleration.

CHAPTER 4

Head Injury Reconstruction Models

Given the ethical challenge of performing head impact research, many different reconstruction models have been developed such as; animal, cadaver, physical and mathematical/finite element models in order to better understand head/brain injury mechanisms. Each of the reconstruction methods has their own advantages and disadvantages. Extensive research on the biomechanics of head injury has been conducted over the last century yet only a few studies have been conducted using volunteer subjects [Eiband, 1959; Bayly et al., 2005]. For ethical reasons, impacts had to remain well under injury levels, providing little data concerning traumatic brain injury (TBI) mechanisms.

Animal Model

The animal models had the advantage of being readily available and offered the possibility to test above injury thresholds allowing for a direct analysis of TBI [Pudenz & Shelden, 1946; Ommaya et al., 1966; Gennarelli et al., 1971; Gennarelli et al., 1972]. Unfortunately, the use of anesthetics, differences in brain size, inherent tolerance and skull characteristics made extrapolated data limited. Furthermore, the response of internal parameters such as stress and strain to impact could not be monitored. Physical models have also been used to predict the human head's response to impact without the variability associated with biological subjects [Gurdjian & Lissner, 1961; Suh et al., 1972].

Cadaveric Model

To reduce this gap in data scaling, cadaveric heads were used to study skull deformation and fracture because they possessed the same mass distribution, geometry and anatomical characteristics as live human heads [Gurdjian et al., 1949]. Once repressurized, they were also used to understand the development of pressure gradients [Gurdjian et al., 1966; Thomas et al., 1966]. Though very limited, some three-dimensional dynamic impact response data has been collected from cadavers [Nahum et al., 1977; Ono et al., 1980; Hardy et al., 2007]. Unfortunately, impacting cadaveric heads does not reveal information concerning injury to the soft tissues and has been shown to be highly variable. This variability is partly due to difference in head geometry/sizes, tissue density and response difference between live tissue and dead tissue. In order to reduce some of the variability, the use of surrogate headforms has become increasingly popular for impact reconstructions.

Surrogate Headform

Injury reconstructions from recorded events provide a method for better understanding the dynamics surrounding actual concussive injuries. Using estimations of impact velocities and effective mass of the impacting object, calculation of kinetic energy (J) to the head can be made and compared to established thresholds [McIntosh et al., 2000; Enouen, 1986]. Anthropometric dummies were often used to design, develop and test protective devices [Viano et al., 1989; Post et al., 2010; Post et al., 2011]. They were ideal because they behaved in a humanlike manner following an impact; were easily available, rarely damaged and provided the researchers with repeatable data. The advantage of using these surrogate headforms was that they were durable and built to similar mass, geometry and skin characteristics of a real human headform. Although

dummies were incapable of showing signs of injury, they were equipped with devices (e.g. accelerometers), allowing for the measurement of biomechanical parameters which could be correlated to human impact response [Viano et al., 1989]. The advantage of using these headforms was that they could be impacted multiple times above injurious levels.

Currently, there are many types of headforms available for impact testing each with varying degrees of biofidelity and repeatability. Rigid headforms (Half CEN EN 960, used by Head Protection technical body of the European Committee for Standardization) tend to be used in testing for the majority of helmet standards. This is in large part due to the high repeatability of rigid type materials. Headforms that are more compliant have been developed in order to deliver more realistic dynamic responses from impact reconstructions. The Bi-mass 150 and the Eindhoven are two headforms for which specific characteristic changes were made to increase the headform's biofidelity [Willinger et al., 2001; Van den Bosch, 2006]. The Bi-mass 150 can measure skull and brain dynamic impact response separately, while the Eindhoven uses a composite material shell with markers imbedded (tracked by X-ray) in a silicone gel brain. The Hybrid III and Hodgson-WSU headforms are the two most common head forms used for impact testing and were developed and validated using cadaveric peak linear acceleration impact data [Hubbard & McLeod, 1974]. Calibration of this headform is done using a single 37.6cm drop to the forehead region, where peak linear acceleration values having to fall between 225 – 275g [Mertz, 1985]. The Hodgson-WSU headform was developed mainly for helmet standards testing and was validated using load deflection curves from cadaveric head data [Hodgson, 1975]. Though these two headforms were designed to recreate dynamic impact response approximating a human cadaveric head, the validation was limited to peak linear acceleration resulting from an impact to the frontal region of the head. While the Hybrid III was not necessarily designed for

direct impacts, it was the only surrogate headform that had the ability to be instrumented to measure three-dimensional dynamic impact responses. The Hodgson-WSU headform has the advantage of being designed for direct impacts however was limited to measuring linear acceleration only; though, it has recently been fitted for measuring the three dimensional components of dynamic response which is currently being validated. The neck component also has an important influence on the dynamic impact response during impact reconstructions. Recent research has demonstrated that the Hybrid III neck may have an effect on the dynamic impact response of a headform depending on direction of the impact [Foreman & Hoshizaki, 2011]. While the Hybrid III neckform is the most commonly used in impact reconstructions, it has calibrated in the sagittal plane, limiting its use to anterior-posterior motion plane [Deng, 1989]. The current hybrid III neckform is asymmetric by design and thus creates an asymmetric response. A neutral or unbiased neckform could eliminate this bias and give new insight to three-dimensional dynamic impact responses.

Mathematical modeling / FE Models

Recently, there has been an emergence of computer models to determine risk of brain injury [Zhang et al., 2001a; Kleiven, 2007; Horgan & Gilchrist, 2003]. Finite element (FE) models were ideal because they could account for the irregular shape and multiple material compositions of the brain. Furthermore, simulations performed with a FE model had the advantage of being repeatable, reproducible and did not have the biological variability that accompanies surrogates [Zhang et al., 2001b]. The principle behind these models is that any basic function can be represented by a linear combination of algebraic polynomials [Voo et al., 1996]. Linear and rotational loading curves from physical reconstructions or simulated impact were input at the centre of gravity of the model and brain deformations are then be calculated. FE

models are used to analyze tissue deformation by decomposing the head into small elements (e.g. blocks, tetrahedrons), each having the continuum material properties of the tissue it represents. These models are extremely valuable because they calculated the internal stresses and subtle internal motions of the modeled structure to predict the failure of the tissues composing the head [Ward & Nagendra, 1985]. Although complete validation has not yet been achieved, most recent models have been partially validated [Horgan & Gilchrist, 2003; Kleiven, 2003]. These models have been validated using cadaveric intracranial and ventricular pressure data published by Nahum et al. (1977) and Trosseille et al. (1992). A limitation of these models is that they are dependent on the material properties assumed in mathematical models [Lowenhielm, 1975; Advani et al., 1975]. Thus, more complete validations of the models are required in order to establish an accurate link between tissue damage and injury type.

Brain anatomy and tissue material composition is important in the development of numerical and finite element models. Previous research on brain injury utilizing animal and cadaveric models, have had limited success due to a number of factors. For cadaveric studies, cadaveric tissue likely does not replicate tissue response that would normally be found in that of live tissue during impacts. Prange et al., (2002) stated that cadaveric tissues function mechanically different under various loading conditions due to change in their material properties [Prange et al., 2002]. Animal research, while answering the challenges investigating the response of living brain tissue, had other important challenges relating to the geometry and size of the human brain [Ommaya et al., 1967]. Because of the challenges associated with in-vivo brain injury studies on live humans the use of accurate finite element models of the brain for impact research has garnered increased importance. The use of physical head surrogates

instrumented with accelerometers has enabled researchers to correlate head motion with brain tissue response. While these metal headforms offer highly reliable data, the validity of the data has been questioned due to its lack of biofidelity.

Finite element (FE) modeling of the head/brain have given researchers the ability to simulate head and brain injuries, while maintaining proper geometry, anatomy and material properties. Early studies using FE modeling of the brain revealed important information relating to mechanisms of brain injury. The first version of the finite element model used for head and brain injury involved axisymmetrical models to compare material characteristics [Kenner and Goldsmith, 1972; Chan, 1974]. Model development progressed to the inclusion of different regions of the brain, with varying material properties. A three dimensional model of the brain containing dura folds, ventricles and brain stem was developed by Ward and Thompson (1975) for frontal impacts. Nahum et al., (1980) further developed a model that could be used for simulation of lateral impacts and quickly discovered distinct differences in pressure gradient between frontal, occipital and frontal impacts. These pressure gradient differences are likely a result of changes in the linear and rotational acceleration between the three impact locations. Further investigation into the correlation of head/brain motion and tissue response resulted in the creation of new three-dimensional FE models [Ruan et al., 1993; Zhou et al., 1995] partially validated to intracranial pressure data from Nahum et al., (1977) Ruan et al., (1993) found correlations between head acceleration and intracranial pressure while Zhou et al., (1995) demonstrated the importance of differentiating between grey and white matter. They also reported that coup and contrecoup injuries are a result of intracranial pressure gradients, and that brain stem injuries are likely caused by shear stress and strain. The importance of defining material properties of the head and brain cannot be understated in the development of an accurate

FE model. In particular, the characteristics and properties of brain tissue and the skull/brain interface definition are essential for correct simulations [Claessens et al., 1997]. Furthermore, the correct boundary conditions for the FE model are essential for accurate reproduction of cadaver-based validations [Turquier et al., 1996]. In attempts to progress FE models and make them more accurate representation of human response, more complete FE models were developed [Zhang et al., 2001; Horgan & Gilchrist, 2003; Kleiven 2006, Willinger et al., 1996]. While each of these models have their specific differences in terms of material properties, they all have been validated to both intracranial pressure [Nahum et al., 1977] and brain motion [Hardy et al., 2001] data from cadaver experiments. While they were partially validated to cadaveric data, this also is a limitation of the models. Much of the validation data comes from single impacts in a single impact direction, which would limit the applicability to all impact conditions. Material properties are also limited since these properties may vary from one individual to another combined with loading rate differences of brain tissue characteristics. Therefore, while much research remains for complete validation of the different brain models, they are still quite useful in quantifying brain tissue response. The University College Dublin Brain Trauma Model (UCDBTM) was used for this dissertation due to its availability and its wide use in the brain injury field in published peer reviewed articles. The description of the model development and material properties are summarized in the following sections.

Model development

University College Dublin Brain Trauma Model (UCDBTM) was developed based on the geometric shape of a male cadaver by means of CT and MRI imaging techniques (Horgan and Gilchrist, 2003; Horgan and Gilchrist, 2004; Horgan, 2005). The model divides the head and brain into ten sections: the scalp, skull (cortical and trabecular bone), pia, falx, tentorium,

cerebrospinal fluid (CSF), grey and white matter, cerebellum and brain stem with the properties and densities of the tissue associated with the specific regions (Tables 4.1 & 4.2).

For components involving sliding surfaces, a friction coefficient of 0.2 was used (Miller et al., 1998). All fluid within the falx, brain and between the tentorium, cerebrum and cerebellum were described using the same fluid algorithm. In total, the brain model was comprised of 26,000 elements. The depth of the CSF layer was 1.3 mm to simulate the norm for the average adult male.

Table 4.1. Finite element model material properties for UCDBTM

Material	Young's modulus (Mpa)	Poisson's ratio	Density (kg/m ³)
Scalp	16.7	0.42	1000
Cortical Bone	15000	0.22	2000
Trabecular Bone	1000	0.24	1300
Dura	31.5	0.45	1130
Pia	11.5	0.45	1130
Falx	31.5	0.45	1140
Tentorium	31.5	0.45	1140
CSF	15000	0.5	1000
Grey Matter	Hyperelastic	0.49	1040
White Matter	Hyperelastic	0.49	1040

Table 4.2. Characteristics of the brain tissue for UCDBTM

Material	Shear Modulus (kPa)		Decay Constant (GPa)	Bulk Modulus (s⁻¹)
	G₀	G_∞		
Cerebellum	10	2	80	2.19
Brain Stem	22.5	4.5	80	2.19
White Matter	12.5	2.5	80	2.19
Grey Matter	10	2	80	2.19

Material Properties

The importance of describing the material properties in FE modeling is imperative to building an accurate model [Turquier et al., 1996; Claessens et al., 1997; Bandak and Eppinger, 1994]. Understandably, biological tissues typically have nonlinear responses to loading. For example, a tissue under compression will generate larger stresses for smaller and smaller strain increments as the tissue deforms. This response is not only a mechanical response but can also be time dependant, also referred to as a tissue's viscoelasticity. While previous finite element models of the brain used linear elastic material constitutive laws [Shugar and Katona, 1975; Ward and Thompson, 1975] the UCDBTM used nonlinear viscoelastic material law under large deformation as described by Mendis et al. (1995) and Kleiven & Von Holst (2002). A

hyperelastic material model was used for the brain in shear in conjunction with a viscoelastic material property. The equation describing the hyperelastic law was given by:

$$C_{10}(t) = 0.9C_{01}(t) = 620.5 + 1930e^{-t/0.008} + 1103e^{-t/0.15} (Pa)$$

Where C_{10} and C_{01} are the temperature-dependent material parameters and t is time in seconds.

The compressive behavior of the brain was considered elastic. The shear modulus parameters were determined from a logarithmic plot (Horgan, 2005):

$$G(t) = G_{\infty} + (G_0 - G_{\infty})e^{-\beta t}$$

Where G_{∞} is the long-term shear modulus, G_0 is the short-term shear modulus and β is the decay factor (Horgan and Gilchrist, 2003).

The CSF layer within the model was developed using solid elements with low shear modulus since fluid typically has a high bulk modulus and zero resistance to shear [Ruan, 1993; Zhou et al., 1995; Kang et al., 1997; Hu et al., 1998; Gilchrist and O'Donoghue, 2000; Gilchrist et al., 2001].

The models development was not without its limitation. The characteristics of brain tissue used in this model were approximated from cadaveric and scaled animal anatomical testing. The use of animal testing models for data gather poses the risk of scaling law errors to human geometries. Another limitation, which has been addressed in the above sections relates to the use of cadaveric brain tissue properties to describe living brain tissue injury response. The last limitation refers to differences between subjects, gender and geometries of the headform which cannot be easily accounted for in FE models. Though there are a number of limitations associated with FE modeling of the brain, cadaver tissue represents the closest fit to living

human tissue characteristics (Horgan, 2005). The material properties for the bone, scalp and intracranial membranes were taken from the literature (Ruan, 1993; Kleiven and von Holst, 2002; Zhou et al., 1996; Willinger et al., 1995) with the values in table 4.1. The weight of the model was scaled to the dimensions of Nahum et al. (1977), which resulted in a mass of 1.422 kg. Inertial properties are $I_{yy} = 1795 \text{ kg.mm}^2$, $I_{zz} = 1572 \text{ kg.mm}^2$ and $I_{xx} = 1315 \text{ kg.mm}^2$, and are similar to those of Kleiven and von Holst (2002).

Validation of the Model

This model, as discussed earlier, was validated with intracranial pressure data from Nahum et al. (1977) and Trosseille et al. (1992) cadaver impact tests and brain motion against Hardy et al.'s (2001) research. The model was validated further using comparisons of real world brain injury events to assess its validity for use in injury assessment settings [Horgan, 2005; Doorly 2007]. Cadaveric head impacts [Nahum et al., 1977] were simulated using the UCDBTM. The comparison measures were selected at the same locations as Nahum et al.'s (1977) transducers: the coup (frontal) site, the parietal site, the occipital site and the contrecoup site. The results demonstrated good correlation between the cadaver experiments and the FE simulation. The maximum values, shape and duration of the response correlated well for each of the specified locations.

The model was also validated with intracranial pressure data from Trosseille et al.'s (1992) cadaver impacts that also included both linear and rotational head motion using a 12-accelerometer array affixed to its skull in the occipital region. The results showed good agreement between the simulation and cadaveric data for the shape and duration of pressure in the frontal lobe region. The final validation compares the simulation data to cadaveric brain

displacement data from Kleiven and Hardy (2002). The results produced similar brain displacement data for the simulations versus the experimental data [Horgan, 2005]. Overall, the UCDBTM correlated well to cadaveric data in all three validation procedures. The model performed best in the measurement of pressure response when compared with a translational cadaver impact; however, was less reliable in the more complex impact conditions [Horgan, 2005]. The limitations of the model as it relates geometry and description of material properties and their response characteristics is likely the cause for these lower performance results.

CHAPTER 5

Kinematic Injury Predictors

For years, researchers have attempted to describe and quantify brain injury through various equations, resulting in a single numeric value. These different injury predictors have been used to attempt to quantify brain injury. The goal of creating such predictors is to attempt to identify a threshold for injury. With the many different mechanisms of injury presented earlier in this text, it becomes quite complex to isolate one single level for brain injury tolerance. Since there is a high complexity of the different mechanisms for brain injury, it is important to develop an injury threshold measure that includes all the important variables involved in creating the injury [O'Donoghue, 1999]. The injury reconstruction variables would be defined as velocity, mass, location and impact vector. These variables would create unique kinematic loading conditions that would affect deformation of the brain tissue [Kendall et al., 2012]. Other factors affecting the brain tissue strain are; geometries of the head, constitutive properties of the tissue, as well as age, sex, health and intoxication [Horgan, 2005].

With so many variables at play, creating specific thresholds for brain injury becomes very difficult. To date, research has focused on linear acceleration as an injury predictor, due to its link to injurious intracranial pressures within animals [Gurdjian et al., 1966: Thomas et al., 1966], this variable, however, was shown to have a low correlation to brain injury in humans. Integration of the acceleration-time curve was proposed since impacts causing high accelerations for long durations, would create a high change of velocity and would be more likely to cause severe injuries than impacts with identical acceleration but occurring over a shorter period of time [Newman, 1998]. Recent research has focused on the use of finite element models to

simulate injurious events in order to estimate brain tissue deformation [King et al., 2003]. There have been a number of brain injury predictors developed in an attempt to quantify injury.

Peak Linear Acceleration

Linear acceleration has been linked to increases in intracranial pressure caused primarily by brain motion within the skull [Gurdjian et al., 1966; Thomas et al., 1966]. It was determined that these pressure changes within the brain were a result of deformation of the skull and brain tissue and their acceleration post impact. Gurdjian et al. (1961) stated that if the kinematics of an impact event could be measured, then potential risk of brain injury could be predicted. Further research on peak linear acceleration confirmed correlation of this variable to increases of intracranial pressure causing brain injury [Nusholtz et al., 1987].

Linear acceleration refers to the change of velocity of the head following an impact. Although it is not a direct measurement of brain trauma, it has been correlated to intracranial pressure [Zhang et al., 2001a; Zhang et al., 2004; Kleiven, 2007; Hardy et al., 2007] and strain [Hardy et al., 2007]. Furthermore, it is the most common parameter used to predict the occurrence of brain injury. Linear acceleration has been directly measured using piezoelectric accelerometers located in a subject's mouth [Higgins et al., 2007], attached to the skull [Yoganandan et al., 2006] or helmet [Beckwith et al., 2007] or directly inside a surrogate headform [Padagaonkar et al., 1975]. Linear accelerations of 80-95 g were associated with the probability of sustaining a concussion [Gurdjian et al., 1966, Zhang et al., 2004; Ono et al., 1980; Broglio et al., 2010].

The specific role of linear and rotational accelerations in causing injury was analyzed during a set of experimentations on anesthetized dogs. The results revealed rotational

acceleration having minimal involvement during many of the concussive events. Linear acceleration was consequently identified as the critical factor in head trauma [Gurdjian et al., 1955]. The hypothesis is supported by data comparing the severity of cerebral concussion in sub-human primates produced by either direct impact to the occipital area of the head or by impact to the base of a mobile chair [Ommaya et al., 1966; Ommaya & Hirsch, 1971]. The analysis of this extensive data collection revealed that although inertial loading could produce concussion, it required higher levels of rotational acceleration than what was required by direct impact identifying once again linear acceleration as the primary factor contributing to brain injury.

Ono et al. (1980) also observed the contribution of linear acceleration to brain injuries. The experiment compared pure translational inertial loading, pure rotational inertial loading and direct impact to a non-restrained head. The results showed that skull deformation was necessary to create cortical contusion and that concussion did not correlate with rotational acceleration. Conversely, linear acceleration was highly correlated, leading the authors to conclude that concussion was caused by linear acceleration from direct impacts.

Thresholds using peak linear acceleration as injury predictors were proposed from impact reconstructions of 12 cases involving 24 National Football League (NFL) players [Zhang et al., 2004]. The response of the players' brain was obtained by using the Wayne State University Brain Injury Model (WSUBIM). Based on logistic regression analysis, the 25 %, 50 % and 80 % probability of sustaining a concussion were estimated to be 66 g, 82 g and 106 g, respectively. Despite being widely used in the field of head protection [CSA, 1997; NHTSA, 2001; ISO, 2006; ASTM, 2007; NOCSAE, 2007; ECS, 2010], peak linear acceleration represents a limited criterion because mechanically, a single point on a pulse could not accurately define the response of a structure [Gadd, 1966; Newman, 1998].

Rotational acceleration

Goggio (1941) was the first to suggest a theory that rotational acceleration could cause brain trauma and compared the movement of the brain within the skull to a glass filled with water. Like water, the brain remained motionless during a rapid rotation of the skull, causing damage in the regions where bony protuberances were present. Holbourn (1943, 1945) elaborated this theory, by speculating that shear stress was the true cause of injury. This was due to the brain's high tolerance to compression (bulk modulus) and poor ability to resist tension (Young's modulus). He concluded by stating that rotational forces, not linear, were responsible for concussion, hemorrhage and contre-coup injuries. Rotational acceleration refers to the rate of change of rotational velocity of the head following an impact. Although it is not a direct measurement of brain trauma, it has been correlated to stress [Zhang et al., 2001a; Zhang et al. 2004], and strain [Kleiven, 2007; Hardy et al., 2001]. Padgaonkar et al. (1975) proposed a method, which requires nine uni-axial accelerometers to be positioned in an orthogonal arrangement [Padagaonkar et al., 1975]. The method used the following equations to obtain rotational acceleration:

$$\alpha_x = (A_{z1} - A_{z0})/2\rho_{y1} - (A_{y3} - A_{y0})/2\rho_{z3}$$

$$\alpha_y = (A_{x3} - A_{x0})/2\rho_{z3} - (A_{z2} - A_{z0})/2\rho_{x2}$$

$$\alpha_z = (A_{y2} - A_{y0})/2\rho_{x2} - (A_{x1} - A_{x0})/2\rho_{y1}$$

Where α was the rotational acceleration for component i (x, y, z), A_{ij} was the linear acceleration for component i (x, y, z) along orthogonal arm j ($1, 2, 3$), and ρ_{x2} was the length of orthogonal arm j acting along the component i . This method for collection rotational data has been deemed the most accurate [Newman et al., 2005].

Research investigating the role of accelerations in TBI was limited to using monkeys, physical models and FE models [Gennarelli & Thibault, 1971; Gennarelli et al., 1972; Lowenhielm, 1975; Unterharnscheidt & Higgins, 1969; Gennarelli et al., 1981; Gennarelli & Thibault, 1982; Gennarelli et al., 1982; Adams et al., 1981; Adams et al., 1983]. The results of these studies demonstrated the predominant role of rotational acceleration in the mechanism of concussion, diffuse axonal injury and subdural hematoma. This led to the conclusion that rotational acceleration alone was capable of causing sufficient shear stress to cause any type of brain injury, if applied properly.

Gadd severity index (GSI)

Current helmet standards utilize GSI as a threshold variable in determining if a helmet passes a certification or not [NOCSAE]. Early research has shown that peak linear acceleration correlates well with skull fracture and intracranial pressure. Further studies determined that brain injury would be better predicted by integrating the area under the acceleration-time pulse [Gurdjian et al., 1966; Newman et al., 1998]. The Wayne State Tolerance Curve (WSTC) was created to determine the relationship between acceleration and time for frontal impacts [Gurdjian et al., 1953; Gurdjian et al., 1955; Gurdjian et al., 1964; Gurdjian et al., 1966]. It has been shown with animal and cadaver models that impacts causing high accelerations over a long period of time would create a high change of velocity and would be more likely to cause severe injuries than impacts with identical acceleration but occurring over a shorter period of time. The curve defined the limit between fatal and non-fatal head responses to impacts and its interpretation revealed that high accelerations could be only be tolerated for short durations while low accelerations could be withstood for longer time interval [Hodgson et al., 1970; Gurdjian et al., 1955; Stapp, 1970].

The Gadd severity index (GSI) was developed to compare impacts with different acceleration-time curves which could aid in the evaluating the development of head protection in the auto industry [Gadd, 1966]. The GSI was obtained by plotting the data points of the WSTC (Figure 5.1) as well as volunteer data obtained by the NASA [Eiband, 1959] on a logarithmic plot.

Considering that the acceleration was not constant, the following expression was used:

$$GSI = \int_{t_0}^t \bar{a}^{2.5} dt$$

where $a(t)$ is the resultant linear acceleration (g) expressed as a function of time. The value of 1000 was proposed as the injury threshold based on the data from the WSTC (Figure 5-1) and NASA data. Proposed thresholds for risk of 5 %, 50 % and 95 % mTBI injury were determined to be 24, 291 and 559 respectively [Newman et al., 2002].

While the measure includes both peak linear acceleration and duration, there are some limitations. The equation produced a linear curve which is likely unrealistic given the material properties of the skull and brain. The protocol assumed that the skull was non-deformable, with the accelerometers positioned at the back of the head [Newman, 1980]. Further research determined that a non-linear relationship might be a better representation of the threshold identified by the WSTC [Versace, 1971]. All impacts used to create the WSTC were limited to acceleration response for frontal cadaveric impacts and thus may not necessarily be reproducible in living human beings for all types of impact locations [Newman, 1980; Prasad et al., 1985].

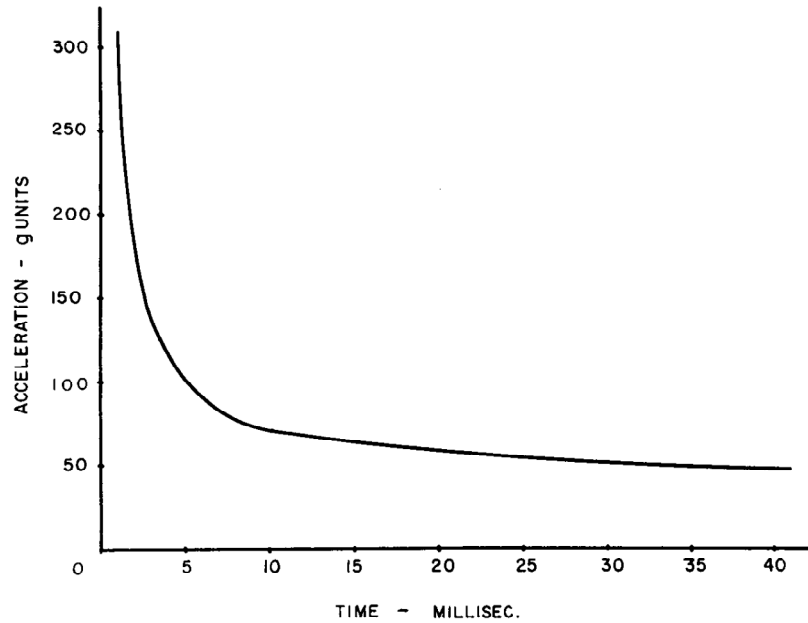


Figure 5-1. Wayne State Tolerance Curve [Zhang, 2001]

The JARI Human Tolerance Curve (JHTC) (Figure 6-1), which is similar to the WSTC, was developed using a series of experimentations on primates and human cadaveric skulls [Ono et al., 1980]. Both the WSTC and JHTC curves displayed similar trends for time durations below 10 ms and differed only slightly for longer time intervals [Ono, 1998; Schmitt et al., 2007].

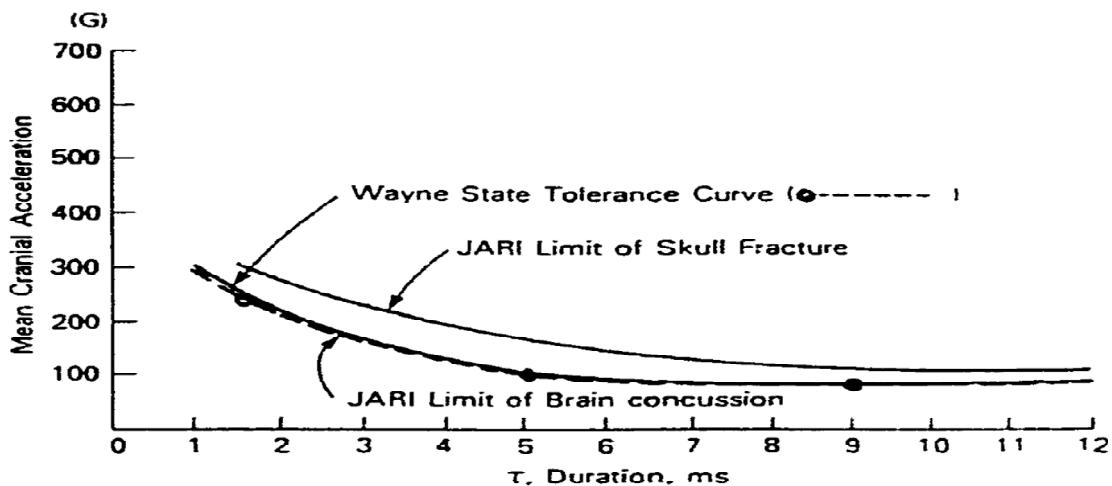


Figure 6-1. A comparison between the WSTC and the JARI tolerance curve [Zhang, 2001]

Head injury criterion

The Head injury criterion was developed in response to criticism and limitations of the GSI [Versace, 1971]. The HIC was created from an extrapolation of the WSTC similar to the GSI. The main difference between the HIC and the GSI was that the HIC is calculated by choosing a time range for the integration of the linear acceleration curve. The equation was the following:

$$HIC = (t - t_0) \left[\left(\frac{1}{t - t_0} \right) \int_{t_0}^t \vec{a}(t) dt \right]^{2.5}$$

Where t and t_0 (referred to as the HIC interval) were the initial and final times (in seconds) during which HIC attained a maximum value.

The tolerance value for the HIC calculation was set to 1000. Research showed that skull fractures and brain injuries occurred when the pulse was below 13.7 ms and 10 ms, respectively [Nusholtz et al., 1984]. Thus, a limit of 15 ms imposed within the criterion by the international standards organization [Prasad & Mertz, 1985]. Current recommendations for time interval limits are set to 15ms (ISO) and 36ms (NHTSA). Thresholds for concussive injury for a HIC15 were estimated to be between 150 and 400 [Prasad & Mertz, 1985].

As was the case with the GSI, certain limitations were found with the HIC. Although a HIC threshold of 1000 was set for life threatening injuries, it was never actually validated with injurious reconstructions at the time it was created [Prasad & Mertz, 1985]. The HIC has been critiqued due to limitations associated with the WSTC data. Moreover, it would seem that the HIC might underestimate the severity of an injury for impacts lasting less than 5 ms [Ward et al.,

1980]. Finally, since HIC was never validated to injury data, it was found not to correlate strongly to either skull fractures or brain vascular injury [Viano, 1988].

GAMBIT

The GAMBIT was the first algorithm to combine both linear and rotational accelerations in an attempt to create a threshold for concussive brain injury [Newman, 1986]. This injury predictor was validated against all known head injury databases where three-dimensional head kinematics were collected. The algorithm used for the calculation is as follows;

$$G(t) = \left[\left(\frac{a(t)}{a_c} \right)^n + \left(\frac{\alpha(t)}{\alpha_c} \right)^m \right]^{1/S}$$

Where $a(t)$ and $\alpha(t)$ refer to instantaneous value of linear and rotational accelerations respectively and n , m and S are constant.

Unlike other algorithms, the GAMBIT does not set a particular threshold related to a certain ASI value. Instead, the algorithm imposes a particular value for linear and rotational acceleration where brain injury would typically occur. A brain injury would be predicted when a value for G goes above 1.

Head impact power

The head impact power (HIP) was developed from actual NFL head impacts causing concussive injuries [Newman et al., 1999, 2000a, 2000b]. Similar to the GAMBIT, the HIP also uses rotational accelerations within its algorithm. Where the two differ, was that the HIP separated the resultant accelerations into six main components. It also took into consideration acceleration, mass, velocity change and direction. It was obtained with the following equation:

$$\begin{aligned} \text{HIP} = & (ma_x \int a_x dt) + (ma_y \int a_y dt) + (ma_z \int a_z dt) \\ & + (I\alpha_x \int \alpha_x dt) + (I\alpha_y \int \alpha_y dt) + (I\alpha_z \int \alpha_z dt) \end{aligned}$$

Where m is the mass of the head, I_i is the moment of inertia of the head, a_i is the peak linear acceleration and α_i is the peak rotational acceleration.

The 5 %, 50 % and 95 % probability of sustaining a concussion were estimated to be 4.70 kW, 12.79 kW and 20.88 kW, respectively and were lower than the ones proposed by Marjoux (2008). HIP has been shown to correlate better to mTBI than peak linear acceleration, peak rotational acceleration, GSI, HIC₁₅ and GAMBIT [Newman et al., 2000a]. The theory behind the HIP was that if the rate of change of kinetic energy passes a critical value, a brain injury would occur [Post & Hoshizaki, 2012]. The problem with the HIP is that it assumes the angle and direction of the impact vector. While Newman reported errors of up to 16%, further research has shown that small variations in impact vector (amplitude and duration) can have significant effects on dynamic head response (Walsh & Hoshizaki, 2010).

Direct brain injuries metrics

The brain is an incompressible substance enclosed in a rigid container. Brain tissue has been described as a soft viscoelastic inhomogeneous and anisotropic material [Parker, 2012]. Brain tissue damage will occur when it exceeds its tolerance levels [Viano et al., 1989]. Stress and strain are two parameters that have been related to intracranial pressure as a result of relative brain motion. These two parameters have been linked to different types of brain injury; cerebral contusion [Gurdjian & Gurdjian, 1967; Miller et al., 1998], intracranial haematoma [Willinger & Baumgartner, 2003; Margulies et al., 1990], diffuse axonal injury [Willinger & Baumgartner,

2003; Margulies et al., 1990; Zhou et al., 1995; Bain & Meaney, 2000], and concussion [Zhang et al., 2001a; Zhang et al., 2004; Willinger & Baumgartner, 2003; Gurdjian et al., 1963]. Direct measurements of brain tissue strain during dynamic impacts have been limited to animal and cadaveric tissue. Maximal principal strain tolerance levels for axonal type tissue were determined using squid and pig axons [Bain & Meaney, 2000; Thibault, 1993]. The mechanical (structural) and physiological (functional) tolerance levels of materials were characterized using the relationship between stress and strain [Goldsmith & Plunkett, 2004]. For example, an artery subject to tensile stress greater than its tensile strength would tear (mechanical failure) and cause intracranial hematoma [Goldsmith & Plunkett, 2004]. Physiological failures would occur at lower levels and may temporarily disturb nerve activity [Galbraith et al., 1993].

Strain

There are three principal types of strain for which a tissue can be exposed; tensile, shear and compressive. The first two are associated with lacerations, fractures, ruptures and avulsions, while the third is associated with crushing injuries [Viano et al., 1989]. While strain is typically caused by an application of force or stress, shear strains are commonly associated with impacts inducing high rotation accelerations [Ommaya and Gennarelli, 1974; Meaney and Smith, 2011]. While modern models have been validated against pressure and displacement [Horgan & Gilchrist, 2003; Horgan & Gilchrist, 2004; Kleiven, 2002], it was demonstrated that FE models validated for pressure can give a wide range of shear responses for the same impact [Bradshaw & Morfey, 2001].

Tensile strains occur when tissues are stretched along their axis. Research has shown that interruptions in signal transfer along nerve tissue can occur even without mechanical failure of

the tissue [Bain and Meaney, 2011]. This is important, specifically for mild traumatic brain injuries (mTBI) and concussive injuries [Meaney and Smith, 2011]. Recent research has identified the calculation of maximum principal strain to correlate well with brain tissue injury and failure. This calculation essentially defines the principal axes where the highest elongation of tissue relative to its original length occurs [Silva, 2006]. It is often used to quantify brain deformation in finite element modeling simulations for head injury [Kleiven, 2007; Zhang et al., 2004; Post et al., 2012]. The limitations to this measurement parameter are that it takes the largest value along the principal axis as its only measurement [Post, 2013]. This could result in maximum principle strain not being sensitive enough for a particular type of injury mechanism.

Stress

Intracranial stress is generally reported using von Mises stress, which combined the three-dimensional stresses. The calculation was developed to give a better representation of the stresses experienced by the tissue [Zhang et al., 2004, Kleiven, 2007; Horgan & Gilchrist, 2003; Willinger & Baumgartner, 2003; Horgan & Gilchrist, 2004]. This calculation is used to sum the various tensors involved in the deformation of a structure and reports it in units of pressure. This has been widely used by researchers as it accounts for the different directions of deformation upon an element [Post, 2013].

Intracranial pressure

The skull and brain were reported to move as a unit and relative to each other upon impact [Hodgson et al., 1966; Gurdjian et al., 1968; Bayly et al., 2005; Gosh et al., 1970]. The differential movement was suggested to be caused by the different inertia of the brain and the skull, which influence their reaction to applied forces [Pudenz & Shelden, 1946; Gurdjian et al.,

1968; Viano et al., 1989]. Observations revealed that intracranial pressure may rise at the impact site as the skull pressed against the brain and reduce at the opposite site as the skull separated from the slower moving brain [Thomas et al., 1967; Gurdjian & Gurdjian, 1975].

Peak pressure

Pressure is the result of a force applied on the surface of a body over a given area and has been linked to cerebral contusion [Gurdjian & Gurdjain, 1967] and concussion [Zhang et al., 2001a; Gurdjian et al., 1966; Kleiven, 2007; Gurdjian et al., 1955; Hadad et al., 1955; Stalhammar, 1975b]. A positive pressure implied that the applied forces were compressive while a negative pressure implied that the forces were tensile. Past research has measured pressure directly using pressure transducers [Gurdjian et al., 1955; Denny-Brown & Russell, 1941; Thomas et al., 1966] and Finite element models [Horgan & Gilchrist, 2003; Zhou et al., 1995; Horgan & Gilchrist, 2004; Kleiven, 2002]. The first attempt at predicting TBI using peak pressure was performed by Ward and colleagues [Ward et al., 1980], who used a finite element model to reproduce head impacts in animal and cadaver tests. A tolerance limit was set at 265 kPa for severe injury and 173 kPa for moderate injuries. Recent studies, using injury data from the NFL, suggested that peak pressures of 60-90 kPa were likely to cause concussion [Zhang et al. 2004; Kleiven, 2007]. While the pressure ranges reported seem to depict a rather large threshold window, a linear relationship between acceleration and pressure gradient was confirmed [Nahum et al., 1977].

Although peak pressures were correlated with concussion and contusions, the locations associated with peak pressures did not match the observed injury pattern [Kleiven, 2007; Mao & Yang, 2010]. Furthermore, experimentation on animals as well as clinical observations reported

changes in intracranial pressure upon impact; however, it had no influence on the TBI [Stalhammar, 1975b; Denny-Brown & Russell, 1941; Stalhammar, 1975a; Stalhammar & Olsson, 1975; Nusholtz et al., 1987; Van Gijn, 2009]. Although the stresses and strains calculated with an FE model were believed to be the most accurate method for predicting TBI [Zhang et al., 2004; Kleiven, 2007; Mao & Yang, 2010], the results are highly dependent of the material properties chosen for the FE brain tissues. Recent data showed that modification in brain tissue properties when using a FE model did not affect intracranial pressures [Kleiven, 2007], shedding more doubt on its predictive ability.

Cumulative Strain Damage Measure (CSDM)

The cumulative strain damage measure (CSDM) was developed by Bandak and Eppinger (1994) to account for the large areas of deformation within the brain tissue. Based on maximum principal strain which has been associated with the mechanism of injury producing DAI (Meaney and Thibault, 1990), this accumulation of strain damage was calculated by measuring the volume of the brain that had experienced strain above a predetermined threshold. This measurement is based on the theory that diffuse type brain injuries occur following the exposure of a volume of tissue to injurious levels of strain. Research using FE brain models found that (CSDM) was highly influenced by rotational acceleration. Since this unit of measure considers the volume of brain tissue injured, it may be a better interpretation for brain tissue injury rather than metrics describing single peak characteristics of one specific brain tissue element; as is done with maximum principal strain and Von Mises stress parameters [Post et al., 2013].

Strain rate

Strain rate is the rate of change of strain with respect to time. Due to the viscoelastic nature of brain tissue, it has been proposed that this variable could be an important parameter related to brain tissue injury [King et al., 2003]. Research describing the characteristics of brain tissue under different loading rates, have to date reported that certain structure are strain rate dependent while other structures are not [Monson et al., 2003; Darvish and Crandall, 2001; Hrapko et al., 2006]. They reported that denser type materials are more prone to strain rate loads than less dense materials. With the brain being comprised of organic tissue of different density, it is reasonable to assume that certain areas of the brain may be more prone to strain rate than other structures. To date, there has been little data relating strain rate to brain injury.

Product of Strain over Strain Rate

Research by King et al. (2003) proposed the use of the product of strain over strain rate as viable parameters to quantify brain tissue injury. This metric was based upon work done by Hardy et al. (2001) and Newman et al (1999). Further research relating this metric to reconstructions of actual brain injuries showed that the product of strain over strain rate had the highest correlation to mTBI followed by strain rate. Kleiven (2007) supported these findings using world reconstructions to assess the correlation of the product of strain and strain rate.

CHAPTER 6

Concussive Injury Reconstructions

Measuring actual injurious events in live humans is extremely difficult, if not impossible to do largely due to ethical issues. Thus, correlating indirect metrics to risk of injury thresholds would be greatly beneficial to better understand mechanism of brain injury. The following section will describe some indirect measures and how they relate to brain tissue injury prediction.

Relationship between kinematic parameter and brain injury

Impact reconstructions are a method for using live humans in order to better understand the dynamics of concussive type injuries. Anthropomorphic dummies were often used to design, develop and test protective devices [Viano et al., 1989; Post et al., 2010; Post et al., 2011]. They were ideal because they behaved in a humanlike manner following an impact; were easily available, rarely damaged and provided the researchers with repeatable data. Although dummies were incapable of showing signs of injury, they were equipped with devices (e.g. accelerometers), allowing for the measurement of biomechanical parameters which could be correlated to human impact response [Viano et al., 1989]. Anthropometric dummies were also being used for impact reconstructions [Smith et al., 1988]. Zhang et al. (2004) used surrogates to reconstruct impacts from video of NFL football games. Linear and rotational responses were fed into a finite element model to establish stress and strains to the brain and concussive injury. Recently, the kinematic outputs have been used to run finite element models in an attempt to correlate stress and strains to traumatic brain injuries in the human [Zhang et al., 2004; Kleiven, 2007; Willinger & Baumgartner, 2003; Viano et al., 2005; Waliko et al., 2005].

Reconstruction of injurious events can produce very useful information regarding brain injury mechanisms. The outcome of injury can be associated with kinematic information collected with video. This being said, reconstruction errors for linear and rotational acceleration as high as 17 % and 25 % have been reported [McIntosh et al., 2000; Newman et al., 2005]. Recent studies looking specifically at kinematics and brain deformation metrics of traumatic brain injury events used a methodology of corridor of response [Post et al., 2012; Post, 2013]. In short, they estimated independent variables for falls (velocity and location of impact) using Madymo. They created a corridor for velocity for which the injury may have occurred (worst-case scenario = high velocity and best-case scenario = lower velocity). From this corridor, kinematic and brain tissue response was estimated for the injury obtained [Post et al., 2012; Post, 2013].

Dynamic response and Brain tissue damage

Assessing the potential tissue damage resulting from impacts or impulses to the head has been a key point of research for many years. In an ideal setting, live humans volunteers would be used to link head impacts to resultant injury. For ethical reasons however, this is not possible. The use of animal, cadaver and physical models have enabled researcher to assess the parameters and mechanisms involved in head impact and relate these to potential damage to brain tissue as a result of momentum and energy transfer. The limitation with these techniques is that large errors may exist with respect to exact thresholds of tissue tolerances due to scaling data from animal models and extrapolate sub-concussive data from volunteers [Ewing et al., 1975] to the adult human brain injuries [Ommaya & Hirsch, 1971; Ommaya et al., 1967]. Cadaveric models do offer relative size and weight of a human head, however, lack active fluid flows that may affect tissue response along with high variability in size and geometry of individual skulls. The recent

emergence of computer models [Zhang et al., 2001a; Kleiven, 2006; Horgan & Gilchrist, 2003] has enable researchers to input material properties of the skull and brain, from cadaveric tissue samples, into a computerized model and simulate different impact conditions. Linear and rotational loading curves have been shown to correlate well to brain deformation. Maximal principal strain (MPS) and Von Mises stress (VMS) are two brain deformation metrics that have had previous anatomical and reconstructive research showing correlations to brain injury [Zhang et al., 2004; Kleiven, 2007; Miller et al., 1998; Willinger & Baumgartner, 2003]. FE modeling of the brain during impact allows for the examination of the effect of complex loading curves on brain tissue deformations. The characteristics of these loading curves can then be used to predict brain deformation metrics that have a higher significance in predicting nervous system tissue damage [Yoganandan et al., 2008; Post et al., 2012]. The relationship between the dynamic characteristics (linear and rotational loading curves) and potential brain tissue injury (MPS & VMS) have been reported in terms of brain tissue tolerance and injury risk (Table 6.1).

Table 6-1. Relationship between dynamic impact parameters and tolerance levels for potential brain tissue injury [Zhang et al., 2004 ; Kleiven, 2007]

Injury risk	Linear acceleration	Rotational acceleration	Maximal principal strain	Von Mises stress	Intracranial pressure
25%	66 g	4600 rad/s ²	-	-	-
50%	82 g	5900 rad/s ²	0.21 MPS	7.8 – 8.4 kPa (Brain stem & Corpus callum)	65.8 – 90.0 kPa (Grey Matter)
80%	106 g	7900 rad/s ²	0.26 MPS	-	-

Summary

Head injuries are caused by changes in momentum and kinetic energy associated with a non-elastic collision. This process creates forces that modify the head's motion (linear and rotational accelerations) and deform the scalp, skull and brain. Current neurological tissue strain measurements have been limited to direct measure in cadavers [Schreiber et al., 1997], animals [Bain & Meaney, 2000; Thibault, 1993] and using FE models [Zhang et al., 2004; Kleiven, 2007; Willinger & Baumgartner, 2003]. Research in the mechanics of neurotrauma has long been limited to measuring indirect mechanical parameters such as linear and rotational acceleration. These dynamic parameters have a direct link to brain deformation metrics. Using these loading curves with finite element models has expanded the knowledge concerning impact mechanics by allowing researchers to observe the stresses and strains occurring within the cranial cavity upon impact. Continuous validation of these models is required in order to ascertain accurate brain deformation metrics as they relate to actual injury events.

CHAPTER 7

Brain and tissue Injury threshold

Identifying thresholds for brain injury remains a priority among the head impact research community. Like any structure, blood vessels, bones and neural tissue behave a certain way under loading and have unique mechanical and physiological failure thresholds to stress and strain. Mechanical failure occurs when the stress applied to a tissue is sufficiently high to disrupt its structure, e.g. fractures. Physiological failure occurs when stress is sufficiently high to disrupt a tissue's functions, i.e. concussions. Pressure gradient has been shown to be an important mechanism for coup, contrecoup and brain deformation type injuries due to cavitation collapse or relative brain motion.

Thresholds developed through animal and cadaveric research seem to have high variance that puts into question its predictability of brain injury. Threshold values for intracranial pressure related to brain injury from physical, animal or cadaveric impacts, though limited, are presented in Table 7-1. While the pressure ranges reported seem to depict a rather large threshold window, a linear relationship between acceleration and pressure gradient was confirmed [Nahum et al., 1977]. This knowledge provides the possibility of using kinematic parameters to evaluate risk of brain injury.

Table 7-1. Proposed pressure thresholds/tolerance levels for brain injury

Author	Method	Measurement	Threshold/Tolerance	Injury
Gurdjian et al. (1961)	Cadavers – linear acceleration	Average pressure	ranges of 2 to 144 kPa	Brain tissue shearing
Unterharnscheidt and Sellier (1966)	Animal models	Peak Negative Pressure	-101 kPa	Brain contusions
Ward et al. (1980)	Finite element models	Created blood pressure tolerance using acceleration time histories	234 kPa	brain contusions
Zhang et al., 2004	FE model	Peak Pressure	65.8 kPa (50 % risk)	Concussion Grey matter
Kleiven, 2007	FE model	Peak Pressure	90.0 kPa (50 % risk)	Concussion Grey matter

Acceleration of the head has been linked to increased intracranial pressure leading to stress and strain of brain tissue. As a result, thresholds for linear and rotational acceleration as they relate to brain tissue injury have been proposed (Table 7-2). The reasoning behind the high range of threshold can be explained by limitations associated with the scaling of animal data to the adult human [Ommaya & Hirsch, 1971; Ommaya et al., 1967], the material properties assumed in mathematical models used [Lowenhielm, 1975; Advani et al., 1975] and the extrapolations of sub-concussive data obtained using volunteers [Ewing et al., 1975].

Table 7-2. Proposed acceleration threshold/tolerance levels for head and brain injury

Authors	Method	Measurement	Threshold	Brain injury
Hodgson et al. (1971)	Cadavers	Linear acceleration and force	300 g +	Skull fracture
Gurdjian et al. (1966)	Animal and Cadaver	Linear acceleration	90 g	concussions
Ono et al. (1980)	Animal	Linear acceleration	90g – 9ms contact time 220 g – 2ms contact time	Concussions + contusions
Zhang et al. (2004)	Live human impacts + reconstruction	Linear and rotational accelerations	Lin - 66 g (25% risk), 82 g (50% risk) & 106g (80% risk) Ang – 4,600 (25%), 5,900 (50%) & 7,900 rad/s ² (80%)	concussions

Stress and strain on brain tissue as a result of intracranial pressure, acceleration and brain deformation has been well documented. Maximal principal strain and Von Mises stress are two brain deformation metrics that have had previous anatomical and reconstructive research showing correlations to brain injury [Kleiven, 2007; Zhang et al., 2004; Miller et al., 1998; Willinger & Baumgartner, 2003]. Maximal principal strain is defined as the maximal value of the principal strain that corresponded to stretching of the tissue. Von Mises stress is defined as the combination of the three-dimensional stresses of a given tissue and is considered to provide a better representation of the strains experienced by the tissue. Zhang et al. (2004) reported that no significant relationship existed between maximum Von Mises stress and peak linear acceleration;

however, maximum Von Mises stress was highly sensitive to rotational acceleration. Tolerance values for animal, cadaver and finite element models are shown in Table 7-3.

Table 7-3 Proposed brain deformation (MPS and VMS) tolerance levels for brain injury

Author	Method	Measurement	Tolerance limit	Brain injury
Margulies et al. (1990) & Meaney et al. (1995)	Gel inside cadaver and animal skulls	Shear strain	0.2 to 0.3 shear strain	Diffuse axonal injury
Thibault et al. (1993)	Squid axons	Maximal principal strain (MPS)	0.1 MPS	Conservative threshold for tissue damage causing concussion
Shreiber et al. (1997) Bain et al. (2000)	Cadaver and Pig optical nerve	Maximal principal strain (MPS) & Von mises stress (VMS)	MPS - 0.18 - 0.19 VMS - 6-16 kPa	Diffuse axonal injury, concussion
Willinger & Baumgartner, 2003	FE model	Maximal principal strain (MPS) & Von mises stress (VMS)	VMS - 6-16 kPa	probability of sustaining concussion, contusion and DAI
Zhang et al. (2004)	FE model	Maximal principal strain (MPS) & Von mises stress (VMS)	MPS - 0.225 (50% risk) & 0.244 (80% risk) VMS in upper brainstem - 7.8 kPa (50% risk)	Risk of concussion
Kleiven, 2007	FE model	Maximum	MPS – 0.21 (50%	Risk of

	principal Strain & Von Mises stress (VMS)	risk) VMS-8.4 kPa (50 % risk) =Corpus callosum	concussion
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While cadaveric and animal models provide direct measures on tissue deformation during impacts, extrapolating these measures directly to live humans is not practical. Measuring brain tissue deformation using finite element models of the human brain is considered the most effective method in evaluating risk of sustaining an mTBI [King et al., 2003]. The use of finite element (FE) models to create brain injury thresholds was done in response to the difficulties and impracticalities in measuring mechanical parameters directly associated with brain injuries [Zhang et al., 2001, Zhou et al., 1995; Willinger et al., 1995; Ruan et al., 1997; Horgan & Gilchrist, 2003; Kleiven, 2006]. The validation of these models was found to be in agreement with intracranial pressure and brain motion data taken from select cadaver experiments [Hardy et al., 2001; Nahum et al., 1977]. While it is understood that acceleration does not represent the diverse injury mechanisms of brain trauma, it has been shown that the characteristics of these loading curves can then be used to predict brain deformations, which have a higher significance in predicting nervous system tissue damage [Zhang et al., 2004; Yoganandan et al., 2008; Post et al., 2012]. Accurate thresholds have not yet been established for the human brain tissue. Tolerance levels for von Mises stress of 6-16 kPa were associated with the probability of sustaining concussion, contusion and DAI [Zhang et al. 2004; Kleiven, 2007; Willinger & Baumgartner, 2003; Schreiber et al., 1997]. Tolerance levels for maximal principal strain of 0.1-0.2 were associated with the probability of suffering from a concussion [Kleiven, 2007; Thibault, 1993]. Using FE modeling Kleiven (2007) confirmed the risk of injury thresholds developed by Zhang et al. (2004) stating that a maximal principal strain of 0.21 in the corpus callosum (or 0.26

in the gray matter) and a Von Mises stress of 8.4 kPa in the corpus callosum correlate best with a 50 % probability of concussion.

Summary

In summary, head and brain injuries are a result of the direct or indirect application of force to the human head. Having different inertial properties, the skull, blood vessels and brain tissue react differently to the force loading and are subject to varying degrees of injury risk; accelerations, pressures and deformation. If the load is sufficiently high, the tissues will deform, causing injury if certain thresholds are surpassed. Injuries such as fractures, haemorrhages and strains occur when deformations are above a tissue's mechanical threshold while injuries such as concussions occur when deformations are above a tissue's physiological threshold. In the past, physical, animal and cadaveric models were used to help characterize brain injury mechanism and determine brain tissue thresholds but had limited success. Impact reconstructions using more progressive finite element analysis has helped establish tolerance limits of brain tissue as a result of dynamic response values from the impact (loading curves). Current tolerance limits for 50% risk of injury are reported as; 82g (peak linear acceleration, 5 900 rad/s² (peak rotational acceleration) producing with MPS and VMS levels of 0.225 and 7.8 kPa respectively [Kleiven 2007; Zhang et al., 2004]. FE modeling is considered a very valuable tool; however, it is limited to measuring mechanical parameters. Injury levels can only be estimated since validation of these models using injury data has not been completed. Methodological improvements in impact reconstruction as well as a validation of a finite element model will help increase the understanding of the mechanisms and thresholds for mild traumatic brain injuries.

PART II

The incidence of concussion in sports has seen dramatic increases over the past decade. While some head impacts in sport result in significant injuries and time out of the game, other seemingly more damaging impacts, do not result in concussive injury. There are many different ways players receive head injuries in the sports arena. Some of the most common ways to receive a concussive injury include collisions, falls and punches to the head. These different methods of loading likely create different brain tissue responses that result in a completely different type of concussive brain injuries. For instance, hitting ones head on the ice likely results in a more complete transfer of energy than a collision between a player's elbow and another player's head. For collisions and punches, the head will tend to move out of the way during the impact whereas, in a fall it cannot. These differences could be attributed to the independent variables associated with the different impact conditions. In the case of a fall, the mass of the ground is higher than the mass of the head, whereas in the case of a punch, the mass of the head may be equal or larger than the effective mass of the punch. The aim of this dissertation to compare and characterize differences between different types of concussive head impact injuries by means of dynamic impact response characteristics and brain tissue responses within the cerebrum. Comparisons between four predominant concussive injury producing events (collisions, helmeted falls, unhelmeted falls and punches) were analyzed by way of the components of the dynamic response of the head, the effect of these components on different variables of brain deformation metrics and how these injury types influence injury in particular regions of the brain. The results of this study provide important information on differences in concussive injury per a specific impact events leading to injury. This information can lead to help improve the safety of the players playing the sports through rule changes and/or

development of protective headwear geared towards technologies that manage specific dynamic response components, influencing unique brain tissue responses dependant on the specific injury event type.

Study #1

BIOMECHANICAL COMPARISON OF FOUR SPECIFIC IMPACT EVENTS CREATING CONCUSSIVE BRAIN INJURY

INTRODUCTION

Fast paced and high impact sports, continue to grow in popularity among the youth. The incidences of traumatic brain injury (mTBI) in these sports are also a growing concern among these athletes. Concussions are one of the most common injuries sustained by athletes across all levels of play and age groups in ice hockey [Pashby et al., 2001; Marchie et al., 2003; Flik et al., 2005; Goodman et al., 2001; Agel et al., 2007; Cantu, 1996; Kelly et al., 1991]. Similar rates of concussive incidence are seen in professional football and Australian Rules football [Casson et al., 2010; Australian National Health and Medical Research Council, 1994; McCrory, 1998; Seward et al., 1993; Victorian Football League Medical Officer's Association, 1986]. The risk associated with concussive injuries is difficult to ascertain, largely due to the complexity of the impacts resulting in the injury. In 2011, the leading cause of concussion were identified as falls (32.5%), followed by sports and recreational activities (18.0%) and struck by or against an object (9.4%) [Rajabali et al., 2013]. Recent research identified specific impact events of injury within ice hockey where most concussive injuries occur [Hutchison et al., 2013]. They identified collisions between players, falls and fighting among the highest risks for injury. The mechanics behind these different injury typologies, however, are still not well defined.

Concussions in high impact sports, including ice hockey, combative sports (boxing and mixed martial arts) and American football (NFL), can occur from a number of different impact patterns. The most common impacts causing concussive injury involve falls to the ground and body segment hits to the head [Cantu, 1996; Powell, 1998]. Falls can occur in a variety of different environments, not solely sports. In sports, like football and ice hockey, concussive injuries resulting from falls occur with the player wearing a helmet. Conversely, falls to the ground in everyday life involve an unprotected head (unhelmeted). This results in differences in compliance between the two conditions resulting in differences in the dynamic response loading curves. Collisions in sports like ice hockey and football are common occurrences and typically involve a shoulder or elbow impact to the head [Cusimano et al., 2011; Emery et al., 2010; Hagel et al., 2006; Honey, 1998; Hutchison et al., 2013]. Ice hockey is also unique in that the rules permit fighting at the junior and professional levels. Recent findings suggest that fighting in ice hockey increases the likelihood of concussive injuries [Donaldson et al., 2013]. Combative sports, such as boxing and MMA, with one of the goals is to “knockout” their opponent, are also considered high risk sports for concussive injury [Zazryn et al., 2003; Hutchison et al., 2014]. Differences in both mass and compliance characterize the punch as a unique impact events for concussive injury. The differences in mass, compliance and energy transfer specific to each of these impact conditions create unique loading characteristics, which contribute to the risk of concussive injury.

Head and brain injuries result from either a direct or an indirect impact to the head [Viano et al., 1989]. Early research identified the acceleration components of either peak linear [Gurdjian et al., 1944, Gurdjian et al., 1955, Gurdjian & Lissner, 1961, Gurdjian et al., 1963] or peak rotational acceleration as capable of producing a variety of brain tissue injuries [Holbourn,

1943; Ommaya and Hirsh, 1971; Gennarelli et al., 1971, Gennarelli et al., 1972]. More recent research has described brain injuries as occurring through a combination of elastic deformation of the skull, intracranial pressures and brain motion relative to the skull from a combination of peak linear and peak rotational acceleration [Gurdjian and Gurdjian, 1975; Gennarelli et al., 1983; Bayly et al., 2005]. While peak kinematic response as it related to brain injury had been described, injuries continued to occur at lower magnitudes. Gennarelli et al., (1983), described the importance of impact duration as it relates with the peak dynamic response through experimental research in animals. The Wayne State tolerance curve established high peak response with short impulse duration trending towards low peak dynamic response with longer impulse durations [Gurdjian et al., 1970]. Differences in these dynamic loading curves may have significant effects on the brain tissue response related to risk for concussive injury [Kendall et al., 2012].

The influence of the dynamic response loading curves, created by the different impact conditions, on the resulting brain tissue response is not well understood. In order to relate brain tissue response to injury, reconstructions of injuries from motorcycle, football and pedestrian impacts, using surrogate head and neckforms, mathematical models calculating impact velocities and FE modeling was performed [Willinger and Baumgartner, 2003; Kleiven ,2006; Post et al., 2012]. Injury thresholds were determined from these studies for brain deformation metrics; such as MPS and Von Mises stress. Further reconstructions, using finite element simulations, were examined using American Football concussion impacts and thresholds for risk of concussive injury were proposed in relation to the dynamic response of the head [Willinger and Baumgartner, 2003; Zhang et al., 2004]. Peak linear acceleration values of 66 g, 82 g and 106 g and peak rotational values of 4600, 5900 and 7900 rad/s² corresponded to 25 %, 50 % and 80 %

probability of sustaining a concussion [Zhang et al., 2004; Kleiven, 2007]. Maximum principal strain and Von Mises stress thresholds obtained from the injury reconstruction simulations produced a 50% probability of concussion resulting in strain values of 0.21 in the corpus callosum and 0.26 in the grey matter and a stress value of 7.8 kPa [Zhang et al., 2004; Kleiven, 2007; Willinger & Baumgartner, 2003]. While thresholds for peak dynamic response and brain tissue response have been established, these thresholds were developed from American football injury data, which represented helmet-to-helmet concussive injury event. Recent research examining concussions from shoulder/elbow to head impacts in ice hockey revealed much lower dynamic response values than those proposed for 25% risk of concussive injury [Rousseau et al., 2014]. Patton et al. (2013) also observed differences in MPS values for unhelmeted head to shoulder impact in rugby impacts. This might suggest that unique impact conditions will result in differences in peak dynamic responses and tissue strain within the brain.

The purpose of this study was to compare the peak linear and rotational dynamic response variables and associated brain deformation metrics (MPS and Von Mises stress) for four (4) specific concussive injury events (collisions, unhelmeted falls, helmeted falls and punches). It was proposed that the peak dynamic response (i.e. peak linear and rotational acceleration) would be significantly different between the different impact conditions. Significant differences in MPS and Von Mises stress would also be observed at the brain tissue response level.

METHODS

Seventy-two (72) concussive impact reconstructions were obtained from hospital reports and sporting events representing four (4) unique impact events; collision, helmeted and

unhelmeted falls, and punch. The reconstruction variables (velocity and location of impact) were determined by the Neurotrauma Impact Science Laboratory (NISL) team from video of the concussive sporting events (football, hockey, MMA) and through description from the hospital reports. A corridor of response for impact velocity was defined by a “best case” (-10%), “calculated” and “worst case” (+10%) scenario. The worst-case scenario was determined by adding 10% to the calculated velocity, while the best case was defined as 10% less than the calculated impact velocity. This corridor for impact velocity was created in order to frame injurious event within previously reported percent error ranges in rugby and ice hockey reconstructions using video footage of the injury events [MacIntosh et al., 2000; Post et al., 2015]. Nine (9) trials for each of the impact reconstruction were collected for each injury reconstruction totaling 648 trials. For statistical analysis, only the “best case” impacts (10% less than the calculated impact velocity) were used since this represent an under-estimation of the injury event. The concussive injury events used for reconstruction in this study were identified based on the follow criteria:

- Diagnosed by doctor or professional medical staff and identified on medical reports (this refers primarily to hospital fall data).
- Injuries identified through video footage where symptoms of loss of consciousness or presence of postural imbalance immediately after impact.
- Injured player ended up missing playing time due to head injury as identified in team injury reports.
- Impacts not showing any symptoms of concussion were excluded from the data set.

A 50th-percentile adult male Hybrid III headform was used for all injury reconstructions. An unbiased neck form was attached to the Hybrid III instead of the traditional hybrid III neckform in order to reduce directional biasness [Deng, 1989; Foreman et al., 2013]. While a

pilot study comparing the unbiased neckform to the Hybrid III neckform revealed some significant differences for peak resultant linear and rotational accelerations (at the FPE15 and SCG impact sites) the magnitude of these differences are likely not sufficient to effect the validity for the use of the unbiased neckform. In fact, the fact that the unbiased neckform performs in a similar fashion to the hybrid III neckform suggests the use of either neckform for this study.

The head form was instrumented with nine mounted single-axis (Endevco7264C-2KTZ-2-300) accelerometers (Endevco, San Juan Capistrano, CA) in a 3-2-2-2 accelerometer array [Padagonkar et al., 1975]. All the accelerometer signal data were sampled at 20 kHz and filtered with a 4th order 1650 Hz low pass Butterworth filter according to the SAE J211 convention.

Fall Events

The fall loading condition was defined as an event where the subject fell from a determined height to the ground. The injury must have occurred with the head being impacted clean and only once during the event. A total of fourteen (14) sports injury reconstruction (football & ice hockey) which involved a helmet were placed into the helmeted fall category while twelve (12) falls were placed in the unhelmeted fall category, creating two distinct fall conditions. All fall reconstructions in this study followed utilized the same impact surface and head protection as described in each of the individual reconstruction events [Post et al., 2014] (Figure S1-1).



Figure S1-1. Example of a monorail drop system used to simulate a head impact to the ice.

Collision Events

The collision loading condition was defined as impacts resulting from two players colliding, where the shoulder of the “hitter” connected with the head of the player being hit. Twenty-six (26) injury reconstructions were used in this study. The inclusion criteria for this impact event required that the injury occurred from a single impact to the head. Only injury events where location of contact on the head and impact velocity could be easily calculated from video were used in this study. Methodology for these injury reconstructions followed the steps described by Rousseau et al. (2014) (Figure S1-2).

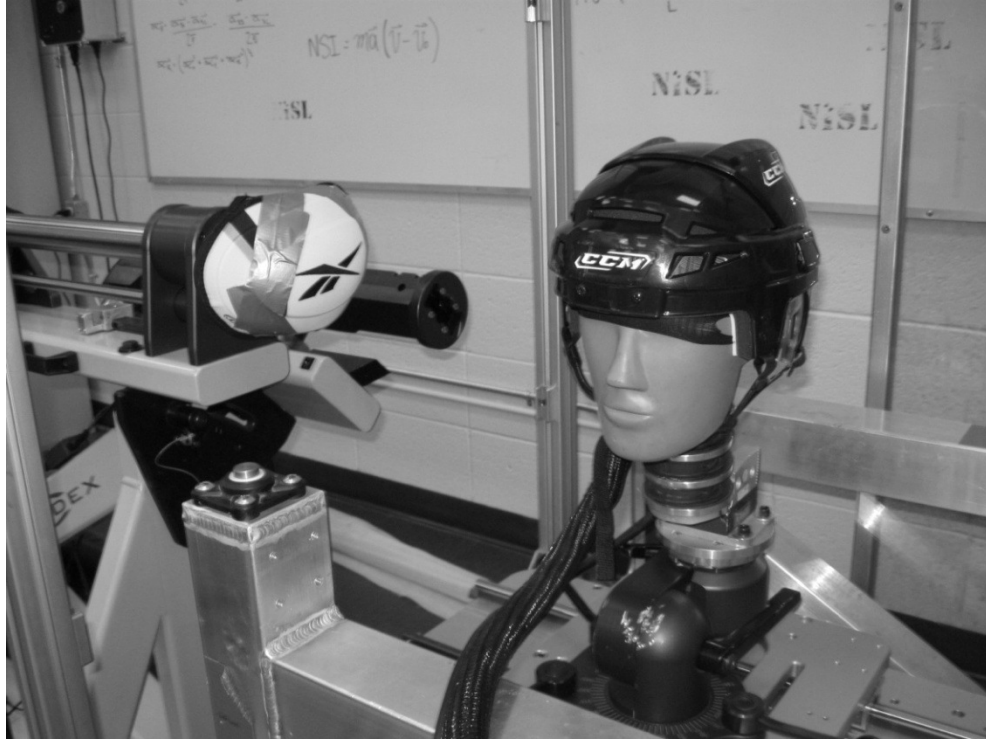


Figure S1-2. Linear impactor system used to simulate the shoulder impact condition.

Punch Events

The punch loading condition cases were collected from concussive injury events from filmed Mixed Martial Arts events. Twenty (20) injury reconstruction cases were used in this study. The inclusion criteria for the punch impact event resulting in concussive injury was described as follows; the event video allowed for good visibility of the hand impacting the head in order to calculate impact velocity and location. The final impact from a punch before the fighter lost consciousness and/or showed signs of imbalance were determined as the concussive blow. A high-speed anvil launcher was used to reconstruct concussive event resulting from a punch (Figure S1-3). The angle of the headform was adjusted to match the events at increments of 5 degrees.

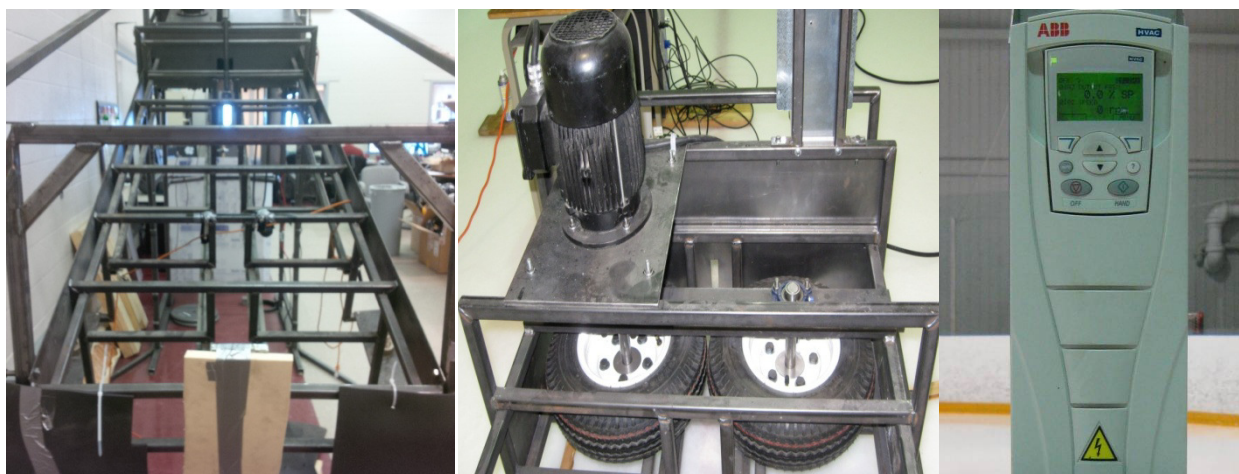


Figure S1-3. High speed impacting system used to reconstruct the punch events (left); Wheels powered by an electric motor used to accelerate the anvil (middle); Velocity will be controlled electronically (right).

Impact velocity was monitored using a High Speed Imaging PCI-512 Fastcam running at 2 kHz and Photron Motion Tools computer software. Impact velocities have been reported between 7 – 12 m/s [Atha et al., 1985; Waliko et al., 2005; Viano et al., 2005; Gullledge & Dapena, 2007]. The inbound velocity and location of impact was determined from video footage of injury event using Kinovea software and the same methodology as previous sports injury reconstruction studies [Post et al., 2014; Rousseau et al., 2014]. Effective impact mass of a punch has been estimated at 3.35 kg (+/- 1.78) for a rigid punch from boxers with weights ranging from 50kg to 108kg [Walilko et al. (2005)]. Three weight categories were created from the anthropometric data of the fighters reported by Walilko et al. (2005), with their associated average effective masses (Table S1-1).

Table S1-1. Mass of anvil used for impact reconstruction based on subject mass.

Subject Mass	Effective mass used in reconstruction	Avg. based from Walilko's data
Less than 80 kg	3.40 kg	3.35 kg (avg. subject mass of 71 kg)
80 kg – 90 kg	3.89 kg	3.68 kg (avg. subject mass of 85 kg)
Over 90 kg	4.37 kg	4.6 kg (avg. subject mass of 108 kg)

Punch anvil compliance

The impacting anvil was developed to accommodate varying masses ranging from three to nine kilograms. The end of the anvil was instrumented with varying density and layers of foam to simulate compliance to match that of a hand and MMA style glove (Figure S1-4). In order to ensure that the compliance of the impacting anvil matched that of a punch, three dimensional dynamic response data was collected from punching trials to a hybrid III headform while wearing MMA style punching gloves. Trials were completed using the anvil while changing foam layers and densities. Acceptable compliance for the punch loading condition was determined by matching the dynamic response curve shape and impact durations (Figure S1-5).



Figure S1-4. Impacting anvil used to simulate a punch. Foam layers can be seen in second photo. The final photo shows a 500 gram mass that can be added to the anvil to adjust the impact mass.

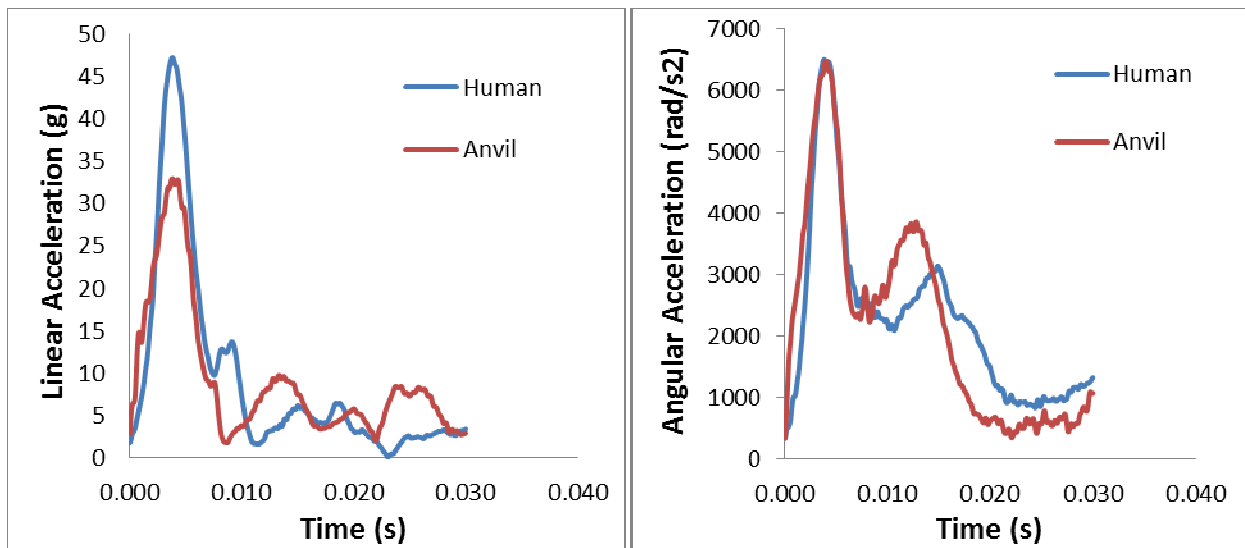


Figure S1-5. Resultant linear and rotational dynamic response curves from human punch and anvil impacts to a hybrid III headform for compliance matching.

The resulting X, Y and Z linear and rotational acceleration loading curves were applied to the center of mass of the University College Dublin Brain Trauma Model (UCDBTM) to calculate Maximal Principal Strain (MPS) and Von Mises Stress [Horgan & Gilchrist, 2003; Horgan & Gilchrist, 2004]. The geometry of the UCDBTM was resolved from computed tomography (CT) and magnetic resonance imaging (MRI) scans of a male cadaver. The version

of the model used for this research was composed of the following parts: scalp, three-layered skull (cortical and trabecular bone), dura, cerebrospinal fluid (CSF), pia, falx, tentorium, cerebral hemispheres, cerebellum and brainstem. Overall, this version of the UCDBTM has approximately 26 000 elements. The model was validated according to cadaveric pressure and brain motion data [Nahum et al., 1977; Hardy et al., 2001] as well as reconstructions of traumatic brain injuries [Doorly & Gilchrist, 2006].

Five one-way ANOVAs were used to determine main effect of the different impact events on the dependent variables; peak linear and rotational acceleration, impact duration, MPS and Von Mises stress ($p < 0.05$).

RESULTS

Comparison of all concussive injury impacts by means of different impact events revealed significant differences ($p < 0.05$) for peak linear and peak rotational acceleration between all impact events (Table S1-2). The collision events produced the lowest dynamic impact response for all the four impact events with mean values of 29.5 ± 9.5 g and 3.2 ± 1.4 krad/s² for peak linear and rotational acceleration, respectively.

Table S1-2. Comparison of dynamic impact response values and global stress and strains from four (4) concussive injury impact conditions.

Impact conditions	Average Velocity (m/s)	Impact Duration (s)	Linear Acc. (g)	Rotational Acc. (rad/s²)	MPS	VMS
Collisions	7.3 (1.4)	0.025 (0.003)	29.5 ** (9.6)	3222 ** (1379)	0.32 (0.12)	9624 (4258)
Falls (helmeted)	5.2 (1.1)	0.014 (0.003)	113.7 ** (48.5)	7783** (2957)	0.51 (0.14)	14345 (4067)
Falls (unhelmeted)	3.9 (0.5)	0.004 (0.001)	291.9 ** (84.1)	18631** (5745)	0.55 (0.13)	16530 (4579)
Punches	6.5 (0.7)	0.007 (0.001)	75.4 ** (30.4)	12228** (5344)	0.50 (0.16)	17733 (6270)

**** denotes significant differences between all impact conditions**

The highest peak linear and rotational accelerations were produced by the unhelmeted fall events with values of 291.9 ± 84.1 g and 18.6 ± 5.7 krad/s². The punch and helmeted fall events both fell in between the unhelmeted fall and collisions events, with 75.4 ± 30.4 g and 113.7 ± 48.5 g for peak linear, 12.2 ± 5.3 krad/s², and 7.8 ± 3.0 krad/s² from peak rotational respectively. Significant differences were also found for impact duration between all four (4) impact conditions. In terms of concussive injury risk, all impact conditions were above a 50% risk threshold [Zhang et al., 2004; Kleiven, 2007] except the collision events, which fell below a 25% risk of injury (Figures S1-6 & S1-7).

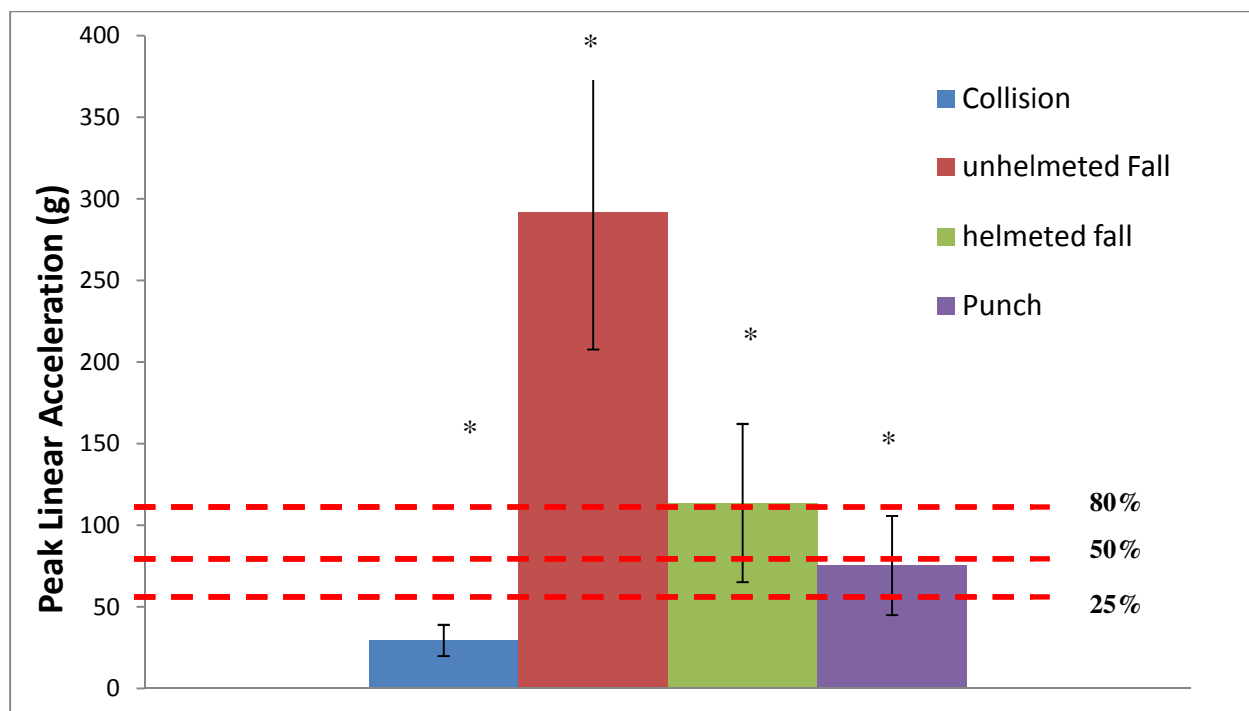


Figure S1-6. Peak resultant linear acceleration for four concussive injury impact events (Collision, Fall (helmeted), Fall (unhelmeted) and Punch) with proposed risk for concussive injury (Zhang et al., 2004).

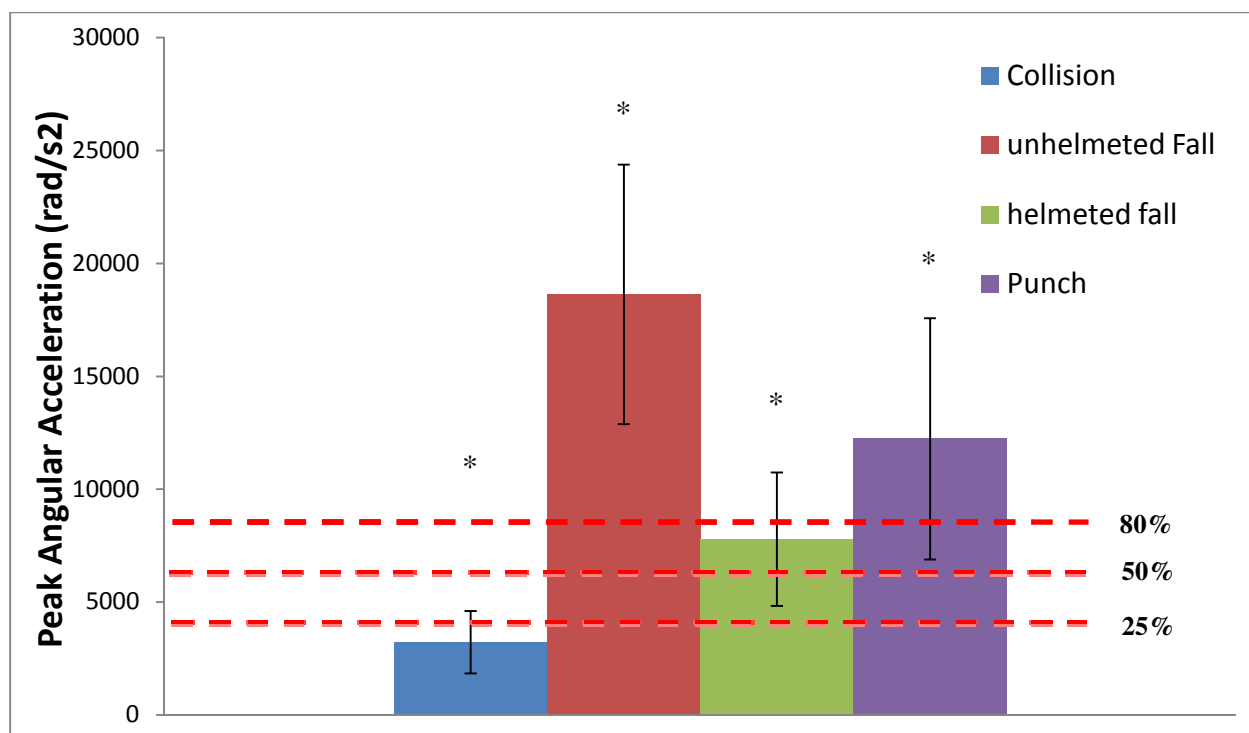


Figure S1-7. Peak resultant rotational acceleration for four concussive injury impact events (Collision, Fall (helmeted), Fall (unhelmeted) and Punch) with proposed risk for concussive injury (Zhang et al., 2004).

No significant differences were found between the different impact events for MPS and Von Mises Stress (Table S1-2). A similar trend however, was found where the highest values are produced by the unhelmeted fall events followed by the punch, helmeted fall and collision impact events. MPS results ranged from 0.32 – 0.55 global strain, with the collision events producing the lowest value and the unhelmeted fall producing the highest. This trend was also evident with VMS where a range of 9.6 – 17.7 kPa was produced from all injury events with the collisions producing the lowest value. In term of concussive injury risk, all four impact conditions produced MPS and Von Mises stress values above the 80% risk for concussive injury threshold (Figures S1-8 & S1-9) [Zhang et al., 2004].

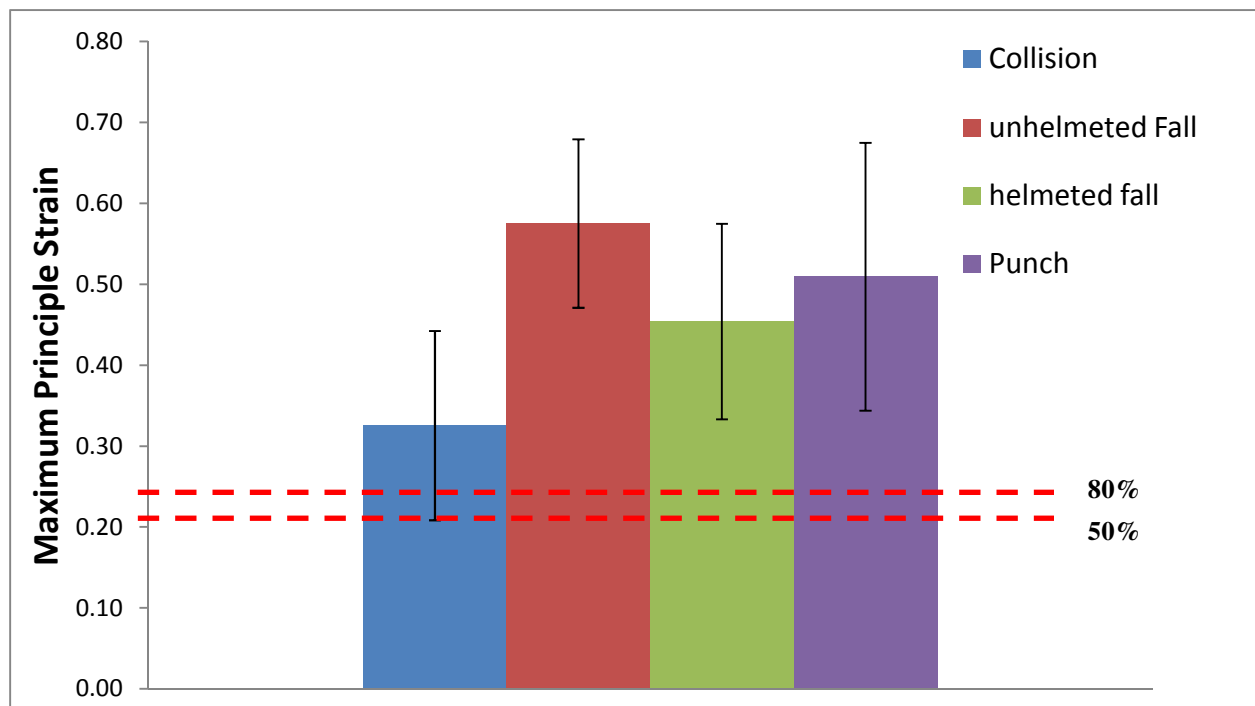


Figure S1-8. MPS values for four concussive impact conditions (Collision, Fall (helmeted), Fall (unhelmeted) and Punch) with proposed 50% and 80% risk of concussion thresholds (Zhang et al., 2004).

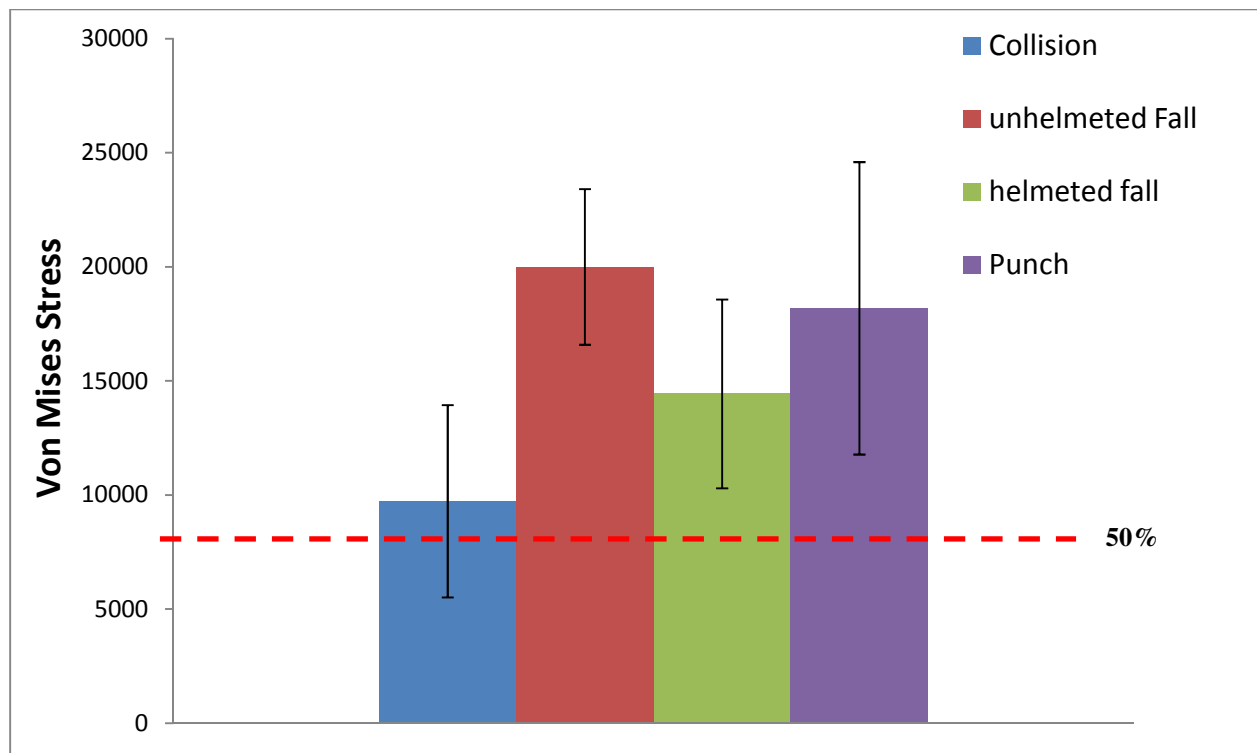


Figure S1-9. Von Mises Stress values for the four concussive impact conditions (Collision, Fall (helmeted), Fall (unhelmeted) and Punch) with proposed 50% risk of concussive threshold (Kleiven, 2007).

DISCUSSION

Head and brain injuries can be both devastating and life changing events for all who are affected. While a head injury can occur in normal everyday activities, high impact sports such as football and ice hockey, provide environments that produce higher risks of injury. Not all concussive injuries are the same. The onset of symptoms and duration of injury increase the difficulty in preventing and predicting injury rehabilitation. Previous research has identified different impact events which result in concussive injuries [Hutchison et al., 2013]. The premise of this study runs on the assumption that a unique concussive injury would occur from different impact conditions causing the injury. This study compared four (4) distinct impact conditions creating concussive injury through reconstruction of injury cases from ice hockey, American

football, mixed martial arts (MMA) and hospital fall data sets. The hypothesis was that each unique impact condition causing the concussive injury would produce differences in terms of dynamic response and resulting brain tissues deformation metrics (MPS and Von Mises stress) unique to that impact event.

The dynamic response results of this study produced significant differences in both mean peak linear and rotational acceleration between all four concussive injury impact events. Trends and patterns were observed with the relationship of the amplitudes of peak linear and peak rotational accelerations depending on the impact conditions. Each of these injury events possesses distinct differences in mass, velocity, impact direction and compliance that influence the peak dynamic response due to differences in how impact energy is transferred. The two fall event (helmeted and unhelmeted) reconstructions involve head forms impacting large masses (i.e. ground) and thus results in almost complete transfer of kinetic energy of the impact to the head and brain. The collision (13.1 kg) and punch (4.0 kg) event reconstructions involve smaller effective masses than the fall events. These reconstructions also differ from the fall events in that the headform is stationary prior to an impact from a moving anvil representing a shoulder or fist [Kendall et al., 2013]. Therefore, energy transfer would be dependent on location of the impact and the motion of the head post impact (moving out of the way of the anvil). Collision and punch reconstructions on the other hand, can have more off-centered impacts. This is due to angle change of both the head and the impacting surface. The punch reconstructions can have impacts to the side of the chin, with an impact direction coming from the side. This location and direction of the impact would prevent the vector from passing through the center of the head, and a higher lever arm, which changes the relative levels of linear and rotational accelerations for these types of impacts. Finally, impact compliance differences between the four injury impact conditions

contributed to the significant differences reported in mean peak linear and rotational accelerations. The collision and helmeted fall injury events primarily involved impacts with a helmet, which result in some impact energy transfer being absorbed by compliant material. Conversely, the unhelmeted fall and punch events involved impacts without helmets.

In terms of proposed injury risk reported in the literature, all impact events except the collision event (less than 25% risk), resulted in peak dynamic response values around 80% risk for concussive injury. The resulting dynamic response values for the fall (unhelmeted and helmeted) and punch events were higher than proposed thresholds of 73.6 – 103.4 g for peak linear acceleration and $5.0 - 7.9 \text{ krad/s}^2$ for peak rotational acceleration [Naunheim et al., 2000; Newman et al., 1999; Newman et al., 2000; Newman et al., 2005; Duma et al., 2005 ; Broolinson et al., 2006; Funk et al., 2006; Guskiewicz et al., 2007; Schnebel et al., 2007; McAllsiter et al., 2012; Rowson et al., 2012; Broglio et al., 2010; McInstosh et al., 2014]. Contrarily, the collision events produced lower peak linear and rotational accelerations than the proposed concussive injury ranges. The collision data from this study supports previous collision impact event data where concussive injuries occurred from peak linear and rotational accelerations of $28.7 \pm 7 \text{ g}$ and $3.6 \pm 1.1 \text{ krad/s}^2$, respectively [Rousseau et al., 2014]. As a result, peak dynamic response may not be the best indicator of concussive injury risk for this type of concussive injury impact condition. The higher compliance condition created with the shoulder pad and helmet resulted in greater impact energy absorption from the added layers of compliance, thus reducing the peak dynamic response to levels below injury risk thresholds. The concussive injury risk data primarily comes from professional football helmet to helmet reconstructions [Newman et al., 1999; Newman et al., 2000; Newman et al., 2005]. Helmet to helmet impacts represent another loading condition for concussive injury and thus the injury risk thresholds would be unique to

that particular loading condition. The effect of compliance not only reduces the peak dynamic response, but also increases the duration of impact. Impact duration has been suggested as a factor differentiating between no concussive injury and concussive injury for collision type impacts [Rousseau, 2014]. Significant differences were found in this study for impact duration between the four impact conditions. The shortest duration resulted from the unhelmeted fall condition, which offered the least amount of compliance. The longest impact duration was found with the collision events. The link between head injury risk and impact duration is supported in the literature [Gadd, 1966; Versace, 1971, Gurdjian et al., 1970, Post et al., 2014]. Rather than focus solely on peak dynamic response, it is more likely that a link between peak linear/peak rotational acceleration and impact duration plays a role in the influence concussive injury risk.

The resulting brain tissue response (MPS and Von Mises) produced no significant differences between the four concussive injury events. This result is not necessarily surprising, given that all impacts used in this study represented diagnosed concussive injuries. It is interesting however, since the dynamic response data did produce differences between loading conditions. An explanation for the lack of significance between loading conditions for the brain tissue response may be attributed to factors associated with compliance, direction of impact, velocity and mass. For example, the punch impact event cases involved primarily side impacts, which may increase stress on brain tissue in the transverse direction of the brain tissue fibers [Prange et al., 2002], while the falls were primarily to the rear location of the head. Comparatively to previous research using finite element models simulations to model impact reconstructions, the MPS and Von Mises stress results of this current study were higher than the reported threshold for 80% risk for concussive injury across all injury impact events (Kleiven, 2007; Zhang et al., 2004). Interestingly, the collision events reported higher than 80% risk for

concussive injury for MPS and Von Mises stress, however less than 25% risk in peak linear and rotational accelerations. It is likely that the shape and specific characteristics (i.e. impact duration) of the loading curves, played an important role in created these higher strains in the brain tissue [Post et al., 2012a; Post et al., 2012b].

Limitations

A Hybrid III headform was used in the reconstruction of the injury events. The surrogate headform has limitations associated with biofidelity which are well documented [Deng, 1989; Mertz, 1985; Hubbard and McLeod, 1974]. The data presented in this study are specific to shoulder collisions, punches and falls from professional ice hockey, professional football, professional MMA and hospital concussive injury cases. All concussions were diagnosed at the discretion of medical and team doctors. Injury reconstruction data was gathered from team or hospital injury reports and thus were completed to best of their knowledge. The injury events were reconstructed from video of the event or estimated from detailed hospital injury reports. The UCD brain trauma model was used to quantify brain tissue responses and possesses similar limitation to other finite element brain models. This model was developed as an approximation of the materials comprising brain tissue and their response characteristics, therefore brain tissue response should be interpreted appropriately.

CONCLUSION

The purpose of this study was to compare the peak dynamic impact response and brain tissue metrics, for four concussive injury loading conditions. While no significant differences were found for brain tissue deformation values (MPS and VMS), significant differences for mean peak linear and rotational accelerations between the four impact conditions were found. These differences in peak dynamic responses also produced differences in concussive injury risks between the different impact events, bringing to question the validity of using peak dynamic response values alone to determine risk for concussive injury encompassing all impact events. Additionally, significant differences in impact duration also occurred between the different loading conditions. The unique impact characteristics of these four concussive injury events likely contribute to differences in dynamic impact loading curves that influence tissue response within the brain reflecting concussions. Since a number of impact factors make each loading condition unique, it is likely that other characteristics related to the three-dimensional dynamic loading curves and not just peak response alone, influence brain tissue response and injury risk.

REFERENCES

1. Pashby, T., Carson, J.D., Ordogh, D., Johnston, K.M., Tator, C.H., Mueller, F.O. (2001). Eliminate head-checking in ice hockey. *Clin J Sport Med.* Oct; 11(4):211-213.
2. Marchie, A., Cusimano, M.D. (2003). Bodychecking and concussions in ice hockey: Should our youth pay the price? *CMAJ.* Jul 22; 169(2):124-128.
3. Flik, K., Lyman, S., Marx, R.G. (2005). American collegiate men's ice hockey: an analysis of injuries. *American Journal of Sports Medicine.* Feb; 33(2):183-187.
4. Goodman, D., Gaetz, M., Meichenbaum, D. (2001). Concussions in hockey: there is cause for concern. *Med Sci Sports Exerc.* Dec; 33(12):2004-2009.

5. Agel, J., Dompier, T.P., Dick, R., Marshall, S.W. (2007). Descriptive epidemiology of collegiate men's ice hockey injuries: National Collegiate Athletic Association Injury Surveillance System, 1988-1989 through 2003-2004. *J Athl Train.* Apr-Jun; 42(2):241-248.
6. Cantu RC. (1996). Head injuries in sport. *Br J Sports Med.* Dec; 30(4):289-296.
7. Kelly, J.P., Nichols, J.S., Filley, C.M., Lillehei, K.O., Rubinstein, D., Kleinschmidt-DeMasters, B.K. (1991). Concussion in sports. Guidelines for the prevention of catastrophic outcome. *JAMA.* Nov 27; 266(20):2867-2869.
8. Casson, M.D., Viano, D., Powell, J.W. and Pellman, E.J. (2010). Twelve years of National Football Leagues concussion data. *Sports Health*, vol. 2 (6): 471-483.
9. Australian National Health and Medical Research Council. (1994). *Football Injuries of the Head and Neck.* Australian Government Publishing Service, Canberra. pp. 78–79.
10. McCrory, P. (1998). Neurological injuries in Australian and rugby football. In: B. D. Jordan, P. Tsaris, and R. F. Warren (Eds.) *Sports Neurology*, 2nd Ed. Philadelphia: Lippincott-Raven, pp. 441–451.
11. Seward, H., Orchard, J., Hazard, H. and Collinson, D. (1993). Football injuries in Australia at the elite level. *Med. J. Aust.*; 159:298 –301.
12. Victorian Football League Medical Officer's Association (1986). A three-year survey of Victorian football league injuries, H. Seward and J. Patrick Melbourne, 11–19.
13. Rajabali, F., Ibrahimova, A., Turcotte, K., & Babul, S. (2013). *Concussion among children and youth in British Columbia.*
14. Hutchison, M.G., Comper, P., Meeuwisse, W.H. & Echemendia, R.J. (2013). A systematic video analysis of National Hockey League (NHL) concussions, part I: who, when, where and what? *Br J Sports Med*, doi:10.1136/bjsports-2013-092234.
15. Powell, J.W. (1998). The evolution of ice hockey related injuries in the NHL. In: Biasca N, Montag W-D, Gerber C, editors. *Safety in ice hockey: IIHF International Symposium on Medicine and Science in Ice Hockey on the 8th Ice Hockey World Championship.* Zurich: IIHF. 19–25.
16. Cusimano, M.D., Taback, N.A., McFaull, S.R., Hodgins, R., Bekele, T.M., et al. (2011). Effect of bodychecking on rate of injuries among minor hockey players. *Open Med*, 5: e57–64.

17. Emery, C.A., Kang, J., Shrier, I., Goulet, C., Hagel, B.E., et al. (2010). Risk of injury associated with body checking among youth ice hockey players. *JAMA*, 303: 2265–72.
18. Hagel, B.E., Marko, J., Dryden, D., Couperthwaite, A.B., Sommerfeldt, J., et al. (2006). Effect of bodychecking on injury rates among minor ice hockey players. *CMAJ*, 175: 155–60.
19. Honey CR. (1998). Brain injury in ice hockey. *Clin J Sport Med*, 8: 43–6.
20. Donaldson, L., Asbridge, M., Cusimano, M.D. (2013) Bodychecking Rules and Concussion in Elite Hockey. *PLoS ONE* 8(7): e69122. doi:10.1371/journal.pone.0069122.
21. Zazryn, T.R., Finch, C.F. & McCrory, P. (2003). A 16 years study of injuries to professional boxers in the state of Victoria, Australia. *Br J Sports Med*; 37:321-24.
22. Hutchison, M.G., Comper, P., Meeuwisse, W.H., Echemendia, R.J. (2014). An observational method to code concussions in the National Hockey League (NHL): the heads-up checklist. *Br J Sports Med*; 48:125-129. Doi:10.1136/bjsports-2012-092059.
23. Viano, D., King, A., Melvin, J. & Weber, K. (1989). Injury biomechanics research: An essential element in prevention of trauma. *Journal of biomechanics*, 22(5), 403-417.
24. Gurdjian, E.S. and Lissner, H.R. (1944). Mechanisms of head injury as studied by the cathode ray occilloscope: Preliminary report. *Journal of Neurology, Neurosurgery and Psychiatry*, 1, 393-399.
25. Gurdjian, E.S., Webster, J.E. & Lissner, H.R. (1955) Observations on the mechanism of brain concussion, contusion, and laceration. *Surgery, Gynecology & Obstetrics* 101(6): 680-690.
26. Gurdjian, E.S. & Lissner, H.R. (1961). Photoelastic confirmation of the presence of shear strains at the craniospinal junction in closed head injury. *Journal of Neurosurgery*, 18(1), 58-60.
27. Gurdjian, E.S., Lissner, H.R., & Patrick, L.M. (1963). Concussion – Mechanism and pathology. In: *Proceedings of the 7th Stapp Car Crash Conference*, Los Angeles CA, USA, 470-482.
28. Holbourn, A.H.S. (1943) Mechanics of head injury. *The Lancet* 242(6267): 438-441.
29. Ommaya, A.K. and Hirsch, A.E. (1971). Tolerance of cerebral concussion from head impact and whiplash in primates. *Journal of Biomechanics*, 4: 13-22.

30. Gennarelli, T.A., Thibault, L.E., and Ommaya, A. (1971). Comparison of translational and rotational accelerations in experimental cerebral concussion. In Proceedings of the 15th Stapp car Crash Conference.
31. Gennarelli, T.A., Thibault, L.E., and Ommaya, A. (1972). Pathophysiological responses to rotational and translational accelerations of the head. Proceedings of the 16th Stapp Car Crash Conference, SAE paper No. 720970.
32. Gurdjian, E.S. & Gurdjian, E.S. (1975). Re-evaluation of the biomechanics of blunt impact injury of the head. *Surgery, Gynecology and Obstetrics*, 140(6): 845-850.
33. Gennarelli, T. (1983). Head injury in man and experimental animals: Clinical aspects. *Acta Neurochirurgica*, 32, 1-13.
34. Bayly, P.V., Cohen, T.S., Leister, E.P., Ajo, D., Leuthardt, E.C., & Genin, G.M. (2005). Deformation of the human brain induced by mild acceleration. *Journal of Neurotrauma*, 22(8), 845-856.
35. Gurdjian, E.S., Lange, W.A., Patrick, L.M., Thomas, L.M. (1970). Impact injury and crash protection. Thomas CC, editor, Springfield; Illinois.
36. Kendall, M., Post, A., Rousseau, P., Oeur, A., Gilchrist, M.D., and Hoshizaki, T.B. (2012). A comparison of dynamic impact response and brain deformation metrics of head impact reconstructions for three mechanisms of head injury in ice hockey. Proceedings of IRCOBI, Dublin, IRL, Sept 10-14.
37. Willinger, R., & Baumgartner, D. (2003). Human head tolerance limits to specific injury mechanisms. *International Journal of Crashworthiness*, 8(6): 605-617.
38. Kleiven, S. (2006). Evaluation of head injury criteria using a finite element model validated against experiments on localized brain motion, intracerebral acceleration, and intracranial pressure. *International Journal of Crashworthiness* 11 (1): 65-79.
39. Post, A., Hoshizaki, T.B., Gilchrist, M.D. and Brien S. (2012). Analysis of the influence of independent variables used for reconstruction of a traumatic brain injury incident. Proceedings of the Institution of Mechanical Engineers, Part P: Journal of Sports Engineering and Technology 2012 226:209 originally published online 14 March. DOI: 10.1177/1754337112436629.
40. Zhang L, Yang KH, King AI. (2004). A proposed injury threshold for mild traumatic brain injury. *J Biomech Eng*; 126:226–36.
41. Kleiven, S. (2007). Predictors for traumatic brain injuries evaluated through accident reconstruction. *Stapp Car Crash Journal* 51 : 81-114.

42. Rousseau, P. (2014). Analysis of concussion metrics of real-world concussive and non-injurious elbow and shoulder to head collisions in Ice hockey. PhD thesis, University of Ottawa, Ottawa, Canada.
43. Patton, D.A., McIntosh, A.S. and Kleiven, S. (2013). The Biomechanical determinants of Concussion: Finite element simulations to investigate brain tissue deformations during sporting impacts to an unprotected head. *Journal of Applied Biomechanics*, 29: 721-730.
44. McIntosh, A.S., McRory, P. & Comerford, J. (2000). The dynamics of concussive head impacts in rugby and Australian Rules football. *Medicine and Science in Sports & Exercise*; February, p.1980 – 1984.
45. Post, A. Koncan, D., Kendall, M., Clark, M. Kosziwka, G. Chen, W. & Hoshizaki, T.B. (2015). The effect of camera angle on velocity measurement from video analysis for use in brain injury reconstruction. Submitted to *Journal of Sports Biomechanics*. (*In Review*)
46. Foreman SF & Hoshizaki TB (2011). The influence of headform geometry on the dynamic impact response of a Hybrid III headform. In: *Proceedings of the 23rd International Society of Biomechanics Congress*, Bruxelles, Belgium.
47. Padagaonkar, A.J., Krieger, K.W., & King, A.I. (1975). Measurement of acceleration of a rigid body using linear accelerometers. *Journal of Applied Mechanics*, 42, 552-556.
48. Post, A. (2014). The influence of acceleration loading curve characteristics on traumatic brain injury. *Journal of Biomechanics*. 01/2014; 47 (5).
49. Atha, J., Yeadon, M.R., Sandover, J. & Parsons, K.C. (1985). The damaging punch. *British medical journal*; 291: December: 21-28.
50. Waliko, T.J., Viano, D.C. and Bir, C.A. (2005). Biomechanics of the head for Olympic boxer punches to the face. *Br J Sports Med*; 39: 710-719.
51. Viano, D.C., Casson, I.R., Pellman, E.J., Bir, C.A., Zhang, L., Sherman, D.C. and Boitano, M.A. (2005). Concussion in professional football: comparison with boxing head impacts—part 10. *Neurosurgery*, Dec; 57(6): 1154-72.
52. Gullledge, J.K. and Dapena, J. (2007). A comparison of the reverse and power punches in oriental martial arts. *Journal of Sports Sciences*; (26)2: 189-196.
53. Horgan, T.J., & Gilchrist, M.D. (2003). The creation of three-dimensional finite element models for simulating head impact biomechanics. *International Journal of Crashworthiness*, 8(4): 353-366.

54. Horgan, T.J., and Gilchrist, M.D. (2004). Influence of FE model variability in predicting brain motion and intracranial pressure changes in head impact simulations. *IJCrash*, 9 (4): 401-418.
55. Nahum, A.M., Smith, R., and Ward, C.C. (1977). Intracranial pressure dynamics during head impact. *Proceedings 21st Stapp Car Crash Conference*. SAE paper No. 770922.
56. Hardy, W.N., Foster, C.D., Mason, M.J., Yang, K.H., King, A.I., and Tashman, S. (2001). Investigation of head injury mechanisms using neutral density technology and high-speed biplanar x-ray. *Stapp Car Crash Journal*, The Stapp Association, Ann Arbor, Michigan.
57. Doorly, M.C. and Gilchrist, M.D., (2006). The use of accident reconstruction for the analysis of traumatic brain injury due to head impacts arising from falls, *Computer Methods in Biomechanics and Biomedical Engineering*, 9(6): 371-377.
58. Kendall, M., Post, A., Zanetti, K., Gilchrist, M.D. & Hoshizaki, T.B. (2013). Comparison of dynamic and static head impact reconstruction methodologies by means of dynamic impact response and brain deformation metrics. *Proceedings of the Institution of Mechanical Engineers*, Gothenberg, Sweden, September 2013.
59. Naunheim, R.S., Standeven, J., Richter, C., et al. (2000). Comparison of impact data in hockey, football, and soccer. *J Trauma* (48): 938–41.
60. Newman, J.A., Beusenberg, M.C., Fournier, E., et al. (1999). A new biomechanical assessment of mild traumatic brain injury: part 1—Methodology. *International Research Council on the Biomechanics of Impact Conference*. Sitges, Spain: IRCOBI, 17–36.
61. Newman, J.A., Barr, C., Beusenberg, M.C., et al. (2000). A new biomechanical assessment of mild traumatic brain injury: part 2—Results and conclusions. *International Research Council on the Biomechanics of Impact Conference*. Montpellier, France: IRCOBI, 223–33.
62. Newman, J.A., Beusenberg, M.C., Shewchenko, N., et al. (2005). Verification of biomechanical methods employed in a comprehensive study of mild traumatic brain injury and the effectiveness of American football helmets. *J Biomech*, 38:1469–81.
63. Duma, S.M., Manoogian, S.J., Bussone, W.R., et al. (2005). Analysis of real-time head accelerations in collegiate football players. *Clin J Sports Med*, 15:3–8.
64. Brolinson, P.G., Manoogian, S.J., McNeely, D., et al. (2006). Analysis of linear head accelerations from collegiate football impacts. *Curr Sports Med Rep*, 5:23–8.

65. Funk, J.R., Duma, S.M., Manoogian, S.J., et al. (2006). Development of concussion risk curves based on head impact data from collegiate football players. *Injury Biomechanics Research: 34th International Workshop*. Dearborn, MI, USA, 1–15.
66. Guskiewicz, K.M., Mihalik, J.P., Shankar, V., et al. (2007). Measurement of head impacts in collegiate football players: relationship between head impact biomechanics and acute clinical outcome after concussion. *Neurosurg*, 61:1244–53.
67. Schnebel, B., Gwin, J.T., Anderson, S., et al. (2007). In vivo study of head impacts in football: a comparison of National Collegiate Athletic Association Division I versus high school impacts. *Neurosurg*, 60:490–6.
68. McAllister, T.W., Ford, J.C., Ji, S., et al. (2012). Maximum principal strain and strain rate associated with concussion diagnosis correlates with changes in corpus callosum white matter indices. *Ann Biomed Eng*, 40:127–40.
69. Rowson S, Duma SM, Beckwith JG, et al. (2012). Rotational head kinematics in football impacts: an injury risk function for concussion. *Ann Biomed Eng*, 40:1–13.
70. Broglio, S.P., Schnebel, B., Sosnoff, J.J., et al. (2010). Biomechanical properties of concussions in high school football. *Med Sci Sport Exerc*, 42:2064–71.
71. McIntosh, A.S., Patton, D.A., Fréchède, B., et al. (2014). The biomechanics of concussion in unhelmeted football players in Australia: a case–control study. *BMJ Open*, 4:e005078. doi:10.1136/bmjopen-2014- 005078.
72. Gadd, C.W. (1966). Use of a weighted impulse criterion for estimating injury hazard. In *Proc. 10th Stapp Car Crash Conference*, Holloman Air Force Base, NM, pp. 164-174.
73. Versace, J. (1971). A review of the severity index. *Proc. 15th Stapp Car Crash Conference*, Coronado, CA, pp. 771-796.
74. Prange, M.T., Meaney, D.F., and Margulies, S.S. (2002). Defining brain mechanical properties: Effects of region, direction and species. In: *Proceedings 44th Stapp Car Crash Conference*.
75. Post, A., Hoshizaki, T.B., Gilchrist, M.D., (2012a). Finite element analysis of the effect of loading curve shape on brain injury predictors. *Journal of Biomechanics* 45, 679-683.
76. Post, A., Walsh, E.S., Hoshizaki, T.B., Gilchrist, M.D., (2012b). Analysis of loading curve characteristics on the production of brain deformation metrics. *Journal of Sports Engineering and Technology* 226 (3/4), 200-207.
77. Deng, Y. (1989). Anthropomorphic dummy neck modeling and injury considerations. *Accident Analysis & Prevention*, 21(1), February, Pages 85-100.

78. Mertz, H. (1985). Biofidelity of the Hybrid III Head, Society of Automotive Engineers Inc., 851245: 1-9.
79. Hubbard, R. & McLeod, G. (1974). Definition and Development of a Crash Dummy Head, Eighteenth Stapp Car Crash Conference, December; SAE 741193.

Study #2

DETERMINATION OF DYNAMIC RESPONSE CHARACTERISTICS THAT DESCRIBE FOUR DIFFERENT CONCUSSIVE INJURY IMPACT EVENTS

INTRODUCTION

Between 1.6 and 3.8 million sports related mTBI are reported to occur annually in the United States alone [Meehan & Micheli, 2011; Guskiewicz et al., 2003; Langlois et al., 2006]. While the use of helmets have been successful in the reduction of traumatic head injuries resulting in death in sports like ice hockey and football, concussive injuries continue to rise. Helmets were not primarily developed to protect against concussive injuries, the method for which they are currently assessed for performance is limited to drops on to an anvil, simulating a fall to ground impact event. Concussive injuries in sport can occur from a variety of different impact events. The most common concussive injury event in sport have been identified as; falls, collisions and punches [Cusimano et al., 2011; Emery et al., 2010; Hagel et al., 2006; Honey, 1998; Hutchison et al., 2013]. These difference impact events create dynamic loading to the brain tissue that are unique to the given event.

In the assessment for concussive injury risk, brain tissue loading is measured by dynamic responses variables (linear and rotational acceleration) which have been correlated to strain of brain tissue [Zhang et al., 2004; Kleiven, 2007; Willinger et al., 1997]. With recent increases in the incidence of concussive injuries in sport, the use of accurate prediction variables become

more important. Currently, risk of concussive injury is assessed using algorithms that combine peak linear, peak rotational resultant accelerations and in some cases impact durations [GSI, HIC, Gambit]. While peak linear accelerations have been primarily linked to focal type head injuries (haematomas, hemorrhages), rotational accelerations have been linked to more diffuse type brain injuries, such as concussions. Peak linear and rotational accelerations have been linked to risk of concussive injury thresholds [Zhang et al., 2004; Kleiven, 2007]. Helmet test standards utilize peak linear resultant accelerations to determine if a given helmet passes or fails [CSA, NOCSAE, HECC]. These testing protocols involve impacts simulating a fall to ground and therefore do not represent dynamic response values for other types of injury loading conditions. Furthermore, while these peak resultant acceleration values have been linked to risk for concussive injuries, the data has been limited to modeled simulations of head impacts [Zhang et al., 2004; Kleiven, 2007; Willinger et al., 1997]. Recent research however has shown that peak linear and rotational accelerations vary in amplitudes dependent on the loading condition creating the injury [Kendall et al., 2012; Kendall et al., 2015]. As a result of these differences in peak dynamic response, similar concussive injury risk thresholds might not best representative for all concussive injury impact events [Kendall et al., 2015; Rousseau, 2014]. Differences in the shape of the dynamic impact response loading curves can also be observed between the different impact events of injury [Kendall et al., 2012; Kendall et al., 2015]. FE models have been used to demonstrate the effect of different shaped dynamic response loading curves on brain deformation metrics [Kleiven, 2007; Post et al., 2012a; Post et al., 2012b]. This would suggest that specific characteristics of the dynamic response loading curves may have a greater effect on brain tissue response over the peak dynamic response. While, it is well established that concussive injuries

can occur in many different ways, it is still unclear how dynamic characteristics of each impact represent injury risk.

The purpose of this study was to determine which dynamic response loading curve characteristics best described each of the four common concussive injury events (collision, helmeted fall, un-helmeted fall and punch). It was hypothesized that the uniqueness of each particular injury event would result in different dynamic response characteristics which would uniquely describe each concussive injury event.

METHODOLOGY

The three dimensional dynamic response loading curves from the seventy-two (72) head impact reconstructions collected from four (4) concussive injury events reported previously in the first study were used for analysis in this study. The four injury events were reconstructed from video and/or hospital injury reports. Each injury event was reconstructed according to impact variables that would most represent the nature of that particular loading condition. The fall events employed the monorail drop system to simulate falls to the similar impacting surfaces as per the injury events [Post et al., 2013, Kendall et al 2012; Kendall, 2015]. The collision impacts used a linear impactor with effective mass [Rousseau, 2014] and compliance matched to a shoulder impact [Rousseau et al., 2015]. Impact reconstructions used protocols developed in previous research for these types of impacts [Rousseau, 2014; Kendall, 2015]. Finally, the punch conditions used a high-speed projectile launcher for lower mass and compliance to match the punch impacts [Walilko et al., 2005; Kendall, 2015].

Curve characteristic analysis

In order to analyze differences between the three-dimensional loading curve shapes for the four concussive injury events (collisions helmeted falls, un-helmeted fall and punch), specific characteristics of the curves were identified. The peak accelerations, slope, integral, duration and time to peak on outcome mTBI have already been identified as key characteristics to help describe the different traumatic brain injuries (Figure S2-1) [Post et al., 2013]. These same characteristics were chosen as potential descriptors for different concussive injury impact events.

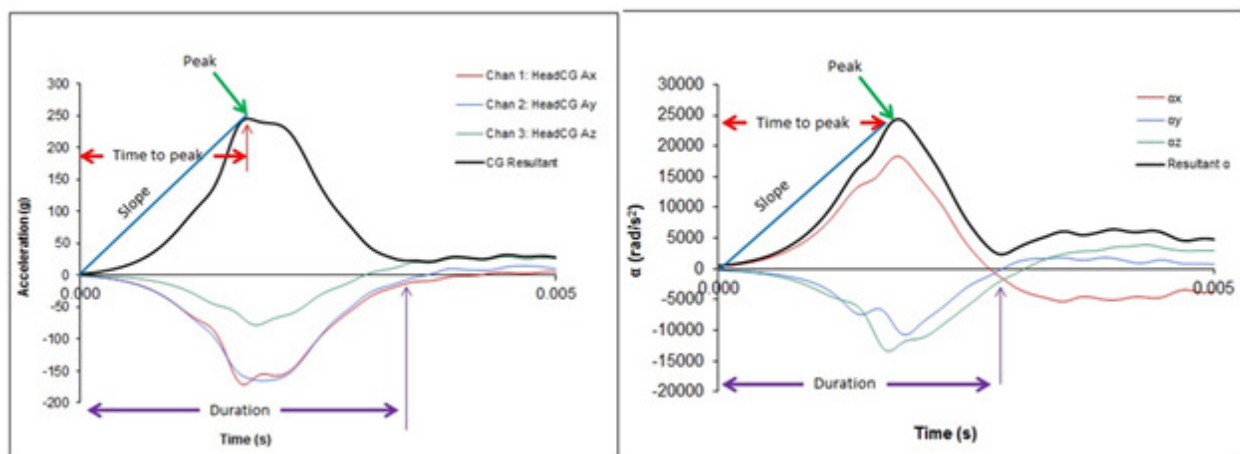


Figure S2-1. Linear (right) and rotational (left) loading curve characteristic breakdown using resultant acceleration curves as an example [From Post et al., 2013].

A factor analysis (principle components) was used to reveal the dynamic response variables that best described each of the four concussive injury events. A Kaiser-Meyer-Olkin (KMO) statistics was run for each injury event to ensure sampling adequacy for predicting if the data factor well based on correlations and partial correlations. An overall KMO of 0.6 (Mediocre) was required to proceed with the factorial analysis. The characteristic with the highest Eigen value for each factor will be identified. Other dynamic response characteristics

within each factor with Eigen values of 0.859 or lower within each factor will not be included in the analysis.

The independent variables for this study were the four (4) injury events (Collision, helmeted fall, unhelmeted fall and Punch). The dependant variables were the characteristics of the three dimensional dynamic response loading curves (peak linear & rotational acceleration (X, Y, Z and Resultant); linear and rotational time-to-peak (X, Y, Z and Resultant); linear and rotational total duration (X, Y, Z and Resultant); linear and rotational integral (X, Y, Z and resultant); linear and rotational slope (X, Y, Z and Resultant); linear and rotational time to peak (X, Y, Z and Resultant)). For point of reference, the X, Y and Z axis are defined according to the cardinal system where; X is in the sagittal plane, Y in the frontal plane and Z in the transverse plane.

Each of these dependant variables were placed in one of the following six (6) categories (Table S2-1): 1) Motion type (linear and rotational acceleration), 2) Magnitude of response, 3) Pulse duration, 4) Loading Rate and 5) Velocity 6) Direction of Motion.

Table S2-1. Categorization of dependant variables of dynamic response.

Type of Motion	Magnitude	Pulse Duration	Loading Rate	Velocity	Direction
Linear acceleration	Peak linear acceleration	Linear acceleration time-to-peak	Slope of linear acceleration	Linear acceleration Integral	Component of linear acceleration X, Y, Z
Rotational Acceleration	Peak Rotational Acceleration	Rotational acceleration time-to-peak	Slope of Rotational acceleration	Rotational acceleration Integral	Component of Rotational acceleration X, Y, Z

RESULTS

The results of this study produced qualitative differences between the resultant linear and rotational acceleration loading curves between the four concussive injury events (Figure S2-2 & S2-3). Differences involving the shape of time history curves, magnitude and duration were found between the four different loading conditions. Four principal component analyses were used in order to reduce the data from the curve time histories characteristics in order to reveal the characteristics which best describe each of the different concussive injury events (Table S2-2 – S2-5).

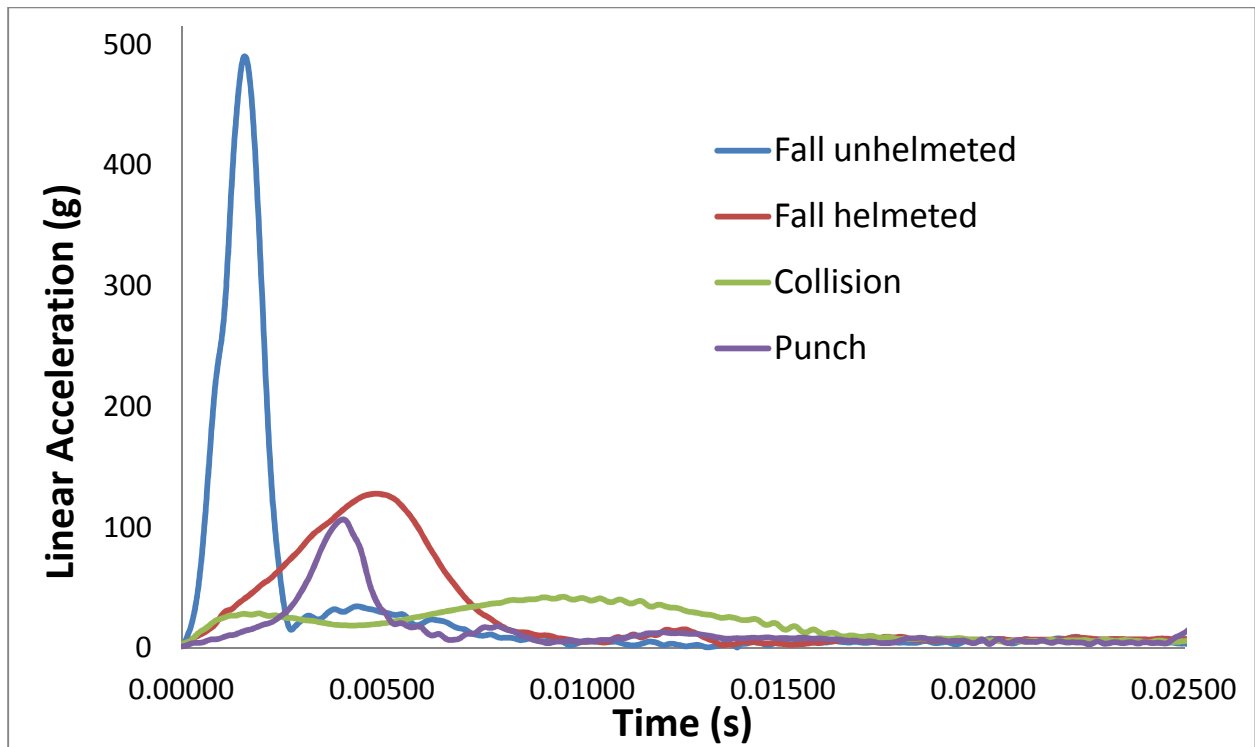


Figure S2-2. Resultant linear acceleration loading curves, from once case each, of the (4) concussive injury events (collision, unhelmeted fall, helmeted fall and punch).

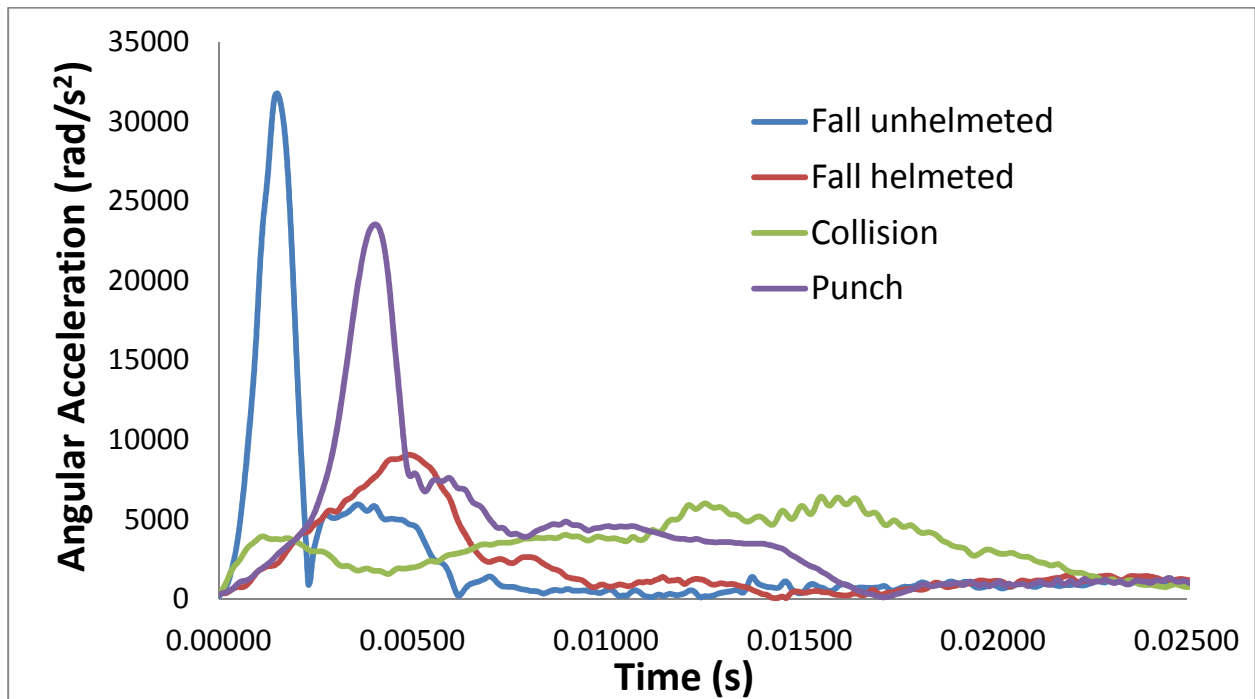


Figure S3-2. Resultant rotational acceleration loading curves, from one case each, of the (4) concussive injury events (collision, unhelmeted fall, helmeted fall and punch).

The collision injury event (Table 2) dataset comprised of 25 cases. A KMO statistic of 0.669 was achieved, which is considered “mediocre” in producing reliable factors for this event [Kaiser, 1974]. A four-factor solution was produced from the principal component analysis, which accounted for a total variance of 62.2% (Table S2-2). The peak linear resultant and peak linear Y characteristics along with linear Z integrals created the first factor that accounted for 20.8% of the total variance within this injury event. The second factor included more directional characteristics of linear and rotational integrals, accounting for 15.3% of the total variance. The third factor did not have a characteristic above the 0.860 threshold. Linear resultant slope (0.751), however, was the characteristics with the highest Eigen value within this third factor that accounted for 14.4% of the total variance. Finally, the fourth factor, accounting for 11.7% of the variance, was the integral values of resultant and Z direction rotational acceleration.

Table S2-2. Dynamic response loading curve characteristics that account for the most variance in concussive injuries resulting from the collision impact events. (Rotated Eigen values > 0.860).

Dynamic response variables	Factors			
	1	2	3	4
Linear Resultant Integral	0.893			
Peak Linear Y	0.885			
Linear Z Integral	0.860			
Peak Linear X		0.952		
Linear X Integral		0.901		
Rotational Y Integral		0.875		
Linear Resultant Slope			0.751	
Rotational Resultant Integral				0.892
Rotational Z Integral				0.873

Variance accounted for	20.8%	15.3%	14.4%	11.7%
Cumulative	20.8%	36.1%	50.5%	62.2%

The helmeted fall injury event dataset contained fourteen impact reconstructions resulting in a KMO statistic of 0.580. A three-factor solution was presented for this impact event that would account for 68.3% of the total variance (Table S2-3). The first factor produced primarily X and Y direction peak and slope characteristics, which accounted for 28.4% of the variance. Rotational resultant integral was the most correlated characteristics for the second factor accounting for 26.6% of the variance. The time to peak characteristics of the linear resultant curve accounted for 13.3% of the total variance in the helmeted fall impact event.

Table S2-3. Dynamic response loading curve characteristics that account for the most variance for concussive injuries resulting from the helmeted fall impact events. (Rotated Eigen values > 0.860).

Dynamic response variables	Factors		
	1	2	3
Linear X Slope	0.956		
Peak Linear X	0.946		
Peak Rotational Y	0.941		
Rotational Y Slope	0.940		
Rotational Y Integral	0.932		
Rotational Resultant Integral		0.880	
Linear Resultant time to peak			0.860
Variance accounted for	28.4%	26.6%	13.3%
Cumulative	28.4%	55.0%	68.3%

The un-helmeted fall injury event contained thirteen injury reconstructions for this analysis. A KMO statistic of 0.615 was achieved for this injury event dataset. Four factors were determined from the factor analysis that accounted for 84.7% of the variance (Table S2-4). Linear and rotational integrals produced the first factor, accounting for 34.8% of the total variance. The second factor included the resultant peak linear, rotational accelerations, and slope of these curves. This second factor accounted for 28.9% of the total variance. The integral of the rotational Z curve was the third factor, which accounted for 12.9% of the variance. The final factor was the linear X time to peak which account for 8.1% variance.

Table S2-4. Dynamic response loading curve characteristics accounting for the most variance for concussive injuries resulting from the unhelmeted fall impact events. (Rotated Eigen values > 0.860).

Dynamic response variables	Factors			
	1	2	3	4
Linear Y Integral	0.931			
Rotational X Integral	0.897			
Rotational Resultant Integral	0.880			
Rotational Resultant Slope		0.970		
Peak Linear Resultant		0.958		
Peak Rotational Resultant		0.940		
Linear Resultant Slope		0.935		
Rotational Z Integral			0.764	
Linear X time to peak				0.819
Variance accounted for	34.8%	28.9%	12.9%	8.1%
Cumulative	34.8%	63.7%	76.6%	84.7%

The punch impact event dataset contained twenty (20) injury reconstructions, which produced a KMO statistic of 0.737 (Table S2-5). The factorial analysis revealed three (3) factors that accounted for 72.3% of the variance within the impact event. The first factor comprised primarily of resultant rotational peak, slope and integral characteristics. These characteristics accounted for 30.3% of the total variance. The second factor included directional curve characteristics in linear X and Rotational Y (peak, integral and slope) comprising 28.3% of the variance. The rotational X time to peak characteristic formed the final factor and accounts for 13.8% of the variance.

Table S2-5. Dynamic response loading curve characteristics that account for the most variance for concussive injuries resulting from the punch impact event. (Rotated Eigen values > 0.860).

Dynamic response variables	Factors		
	1	2	3
Rotational resultant slope	0.980		
Peak rotational resultant	0.973		
Rotational Z Slope	0.866		
Rotational Resultant Integral	0.860		
Linear X Integral		0.974	
Peak Linear X		0.962	
Linear Resultant Slope		0.944	
Rotational Y Integral		0.912	
Peak Rotational Y		0.905	
Rotational Y Slope		0.862	
Rotational X time to peak			0.727
Variance accounted for	30.3%	28.2%	13.8%
Cumulative	30.3%	58.5%	72.3%

Table S2-6 presents a summary of the characteristics of the dynamic response loading curves that account for the most variance in each of the injury events by category describing the characteristics seen in Table S2-1. The Collision impact event is dominated by linear magnitude and velocities (20.8%), followed by directional magnitudes and velocities for both linear and rotational accelerations as a second factor (15.3%) and linear acceleration loading rate as a third factor (14.4%). The helmeted fall impact event was characterized primarily with directional magnitude of both linear and rotational acceleration and directional rotational velocities(28.4%), followed by resultant rotational velocity as a second factor (26.6%) and linear acceleration pulse duration for the third factor (13.3%). The unhelmeted fall impact event was characterized primarily by components of linear, rotation velocities (34.8%), followed by peak linear and rotational acceleration magnitudes and loading rates (28.9%), directional magnitudes of rotational acceleration and loading rates for the third factor (12.9%). Finally, the first factor for the punch impact event revealed rotational acceleration magnitude, velocity and loading rate characteristics (30.3%), followed by directional linear and rotational acceleration magnitudes, velocities and loading rates for the second factor (28.2%) and rotational acceleration pulse duration for the third factor (13.8%).

Table S2- 6. Summary of characteristic of dynamic response loading curve categories that best describe the four concussive injury events (collision, helmeted falls, unhelmeted falls and punch).

Factors		Impact Events							
		Collision		Helmeted Falls		Unhelmeted Falls		Punch	
		Categories	Linear	Rotational	Linear	Rotational	Linear	Rotational	Linear
1	Magnitude	X		X	X				X
	Pulse duration								
	Loading rate			X	X				X
	Velocity	X			X	X	X		X
	Direction	X		X	X	X			
Variance accounted for		20.8%		28.4%		34.8%		30.3%	
2	Magnitude	X				X	X	X	X
	Pulse duration	X							
	Loading rate					X	X	X	
	Velocity		X		X			X	X
	Direction	X	X					X	X
Variance accounted for		15.3%		26.6%		28.9%		28.2%	
3	Magnitude								
	Pulse duration			X					X
	Loading rate	X							
	Velocity						X		
	Direction						X		
Variance accounted for		14.4%		13.3%		12.9%		13.8%	
Cumulative		50.5%		68.3%		76.7%		72.3%	

DISCUSSION

Head and brain injuries, particularly in sports, have been shown to occur in a number of different ways [Hutchinson et al., 2013]. The subtle differences between the mechanics creating these concussive injuries are not easily defined through peak linear and rotational acceleration alone. Therefore, the purpose of this study was to determine the dynamic response loading curves characteristics that best characterize each of the four concussive injury events (collision, helmeted and un-helmeted fall and punch).

The dynamic response loading curves from the four concussive injury events in this study were qualitatively different; in magnitude, pulse duration and time history curve shape. Differences were also found when examining the specific characteristics of the loading curves that best described each of the loading conditions. The collision event was characterized primarily by peak linear directional magnitudes and pulse velocity. While the magnitudes for the collision events were very low relative to concussive injury risk, the duration of these pulses were quite long which may explain why integral values for the directional linear accelerations was revealed as a primary characteristic. The primary characteristics revealed for the helmeted fall events differed slightly where the loading condition was defined primarily with loading curve characteristics related to directional peak linear and rotational acceleration magnitudes and loading rate. The slopes and magnitudes are defined primarily by the front end of the loading curves, whereas with the collision impact events, the primary characteristic was identified as the integral value of the curve, which encompasses the entire loading curves. Similarities between these two injury events, by way of a second factor, were found regarding directional rotational acceleration integral, reinforcing the importance of the relationship between peak response and duration of the pulse in concussive type injuries [Versace, 1971]. The unhelmeted fall impact

event was characterized primarily by directional linear and resultant rotational acceleration magnitudes and pulse durations. The directional integrals for both linear and rotational acceleration were identified as primary characteristics for this loading condition. This demonstrates the importance of the relationship between high peak dynamic responses over very short impulse duration (5 ms). While similar characteristics were found with the collision events, the difference is observed in a reversal of the magnitudes/pulse duration relationships where a low magnitude dynamic response with prolonged pulse durations (25ms) resulted from the collision events. This is consistent with the relationship described by the Wayne State tolerance curve that defined higher risk of concussive injury for impact with high magnitude/short duration pulses compared to smaller magnitude and longer pulse durations [Versace, 1971]. Finally, the punch event was characterized primarily by resultant rotational acceleration magnitude, pulse duration and loading rate. This differed from the previous three impact events where rotational acceleration characteristics were revealed as primary characteristics for those events. Similar to the unhelmeted event, loading rate was identified as a key characteristic that may speak to the relationship between magnitude and impact duration. This loading condition is similar to the unhelmeted fall event in the sense that the impact location was to an unprotected area of the head. On the other hand, the collision and helmeted fall impact events for the most part included some level of compliant material (shoulder pad and/or helmet). The duration components with this loading condition are likely attributed to these differences in compliance between the shoulder pads and helmet materials worn by the players. Previous studies investigating collision type impacts reported significant differences for impact duration dependent the loading condition creating the injury [Kendall, 2015]. While the higher compliance within the impact condition mitigates the peak dynamic responses, it increases the duration of the time curves that

may result in high strains within the brain tissue resulting in concussion [Gilchrist, 2003]. This would also likely influence the characteristics of the loading curves that may account for the most variance within each impact event.

Concussive injuries continue to occur at a high rate in sports. Sports, such as ice hockey and football are high speed sports that require helmet protection for all participants. These helmets are required to pass standard testing protocols (NOCSAE, CSA, HECC) to reduce risk for head injury. Since helmets were primarily designed to protect against traumatic head injuries and cranial fractures, they typically use peak linear acceleration thresholds linked to cadaveric data [Gurdjian et al., 1970]. While concussions are classified as more diffuse type injuries correlating better to rotational accelerations, alternative algorithms were developed include peak rotational resultant acceleration [HIP, GAMBIT]. Interestingly, the results of this study failed to identify peak linear or rotational resultant accelerations as a primary characteristic accounting for the most variance within any of the four concussive injury events. In fact, peak linear and rotational resultant accelerations were identified in only two instances within the four loading conditions. The first was as a second factor in the unhelmeted fall events, where both peak linear and rotational resultant acceleration accounted for 28.9% of the variance. The second instance was as a primary factor in the punch events, where peak rotational resultant acceleration accounted for 30.3% of the variance. As a result, this study would suggest that assessing risk of concussive injury on peak resultant acceleration variables alone may not be sufficient. The results of the component analysis in this study showed that even in the impact events of injury where these peak resultant accelerations accounted for the most variance, the percentages accounted for were so low, that this variable would likely not be sufficient on its own to describe the injury. This would also support observations in the first study, where although all head

impact reconstruction resulted in concussive injury, differences in terms of percentage of risk for concussive injury were found between the different concussive injury events. Therefore, the use of peak resultant acceleration alone may not be sufficient to capture the risk of concussion for each of the individual loading conditions.

Due to the complexity of head/brain injuries and differences in concussive injury loading conditions, isolating one particular characteristic of the dynamic response curves to describe a particular impact event may not be realistic. While distinct differences were found between the dynamic response variables that characterize each concussive injury events, the three factor levels comprising the characteristics accounted for, at the highest level, only about 30% of the total variance. Therefore, it is more likely that a combination of characteristics across the three factors would better characterize a specific impact event. For instances, the collision events might be characterized by directional resultant linear and rotational velocities and linear acceleration loading rate, accounting for 50.5% of the total variance, while the punch events might be characterized by resultant rotation velocity, linear resultant loading rate and directional linear acceleration time to peak, accounting for 72.3% of the total variance.

Limitations

The data presented in this study are specific to shoulder collisions, punches and falls from elite ice hockey, professional football, professional MMA and hospital concussive injury cases. Physicians diagnosed all concussions. This information was gathered from team and hospital injury reports. The injury events were reconstructed using video of the event or estimated from detailed hospital injury reports. The UCDBTM possesses similar limitation to other finite element brain models. This model was developed as an approximation of the materials

comprising brain tissue and their response characteristics, therefore brain tissue response should be interpreted appropriately. The hybrid III head form has limitations associated with biofidelity which are well documented [Deng, 1989; Mertz, 1985; Hubbard and McLeod, 1974]. The unbiased neckform was chosen over the hybrid III due to the hybrid III's directionally biased design for frontal inertial loading [Foreman et al, 2011; Deng, 1989].

CONCLUSION

The objective of this study was to describe the impact kinematic characteristics for four concussive injury events commonly seen in sports. The results from this study demonstrated that differences between impact events are characterized by unique dynamic response variables. While current concussive threshold use peak resultant accelerations to determine risk or performance of protective equipment, this variable did not show up as a characteristic for any of the loading conditions in this study, except the punch event. While differences were found in the characteristics describing each impact event, the amount of variance that it accounted for was low. However, the use of a combination of variables to characterize a specific impact event would provide higher correlations due to the complexities that describe brain injuries. The use of a single peak resultant acceleration as currently employed in impact testing methodologies for helmet testing and head injury assessment may be too general and not account for injury risk from different concussive injury events. While this study describes the unique characteristics for each of the different impact events, it does not in any way describe the relationship between these descriptors and the risk for concussive injury thresholds. These specific characteristics of dynamic loading curves identified per impact event may eventually lead to the development of better concussive risk assessment protocols and head protective equipment where risks from those particular impact events creating the concussive injury are present.

REFERENCES

1. Bain, A.C., & Meaney, D.F. (2000). Tissue-level thresholds for axonal damage in an experimental model of central nervous system white matter injury. *Journal of Biomechanical Engineering*, 122(6), 615-622.
2. Doorly, M.C. and Gilchrist, M.D. (2006). The use of accident reconstruction for the analysis of traumatic brain injury due to head impacts arising from falls. *Computer Methods in Biomechanics and Biomedical Engineering*, 9(6), 371-377.
3. Gadd, C.W. (1966). Use of a weighted impulse criterion for estimating injury hazard. In *Proc. 10th Stapp Car Crash Conference*, Holloman Air Force Base, NM, pp. 164-174.
4. Galbraith, J.A., Thibault, L.E., & Matteson, D.R. (1993). Mechanical and electrical responses of the squid giant axon to simple elongation. *Journal of Biomechanical Engineering*, 115(1), 13-22.
5. Goldsmith, W. (1981). Current controversies in the stipulation of head injury criteria. Letter to the editor. *Journal of Biomechanics*, 14, 883-884.
6. Goldsmith, W., & Plunkett, J. (2004). A biomechanical analysis of the causes of traumatic brain injury in infants and children. *The American Journal of Forensic Medicine and Pathology*, 25(2), 89-100.
7. Gurdjian, E.S., Lissner, H.R., & Patrick, L.M. (1963). Concussion – Mechanism and pathology. In: *Proceedings of the 7th Stapp Car Crash Conference*, Los Angeles CA, USA, 470-482.
8. Gurdjian, E.S., & Gurdjian, E.S. (1967). Cerebral contusions: re-evaluation of the mechanism of their development. *The Journal of Trauma*, 16(1), 35-51.
9. Gurdjian ES, Lange WA, Patrick LM, Thomas LM. In: *Impact injury and crash protection*. Thomas CC, editor. Springfield; Illinois: 1970.
10. Guskiewicz KM, McCrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study. *JAMA*. 2003;290:2549.
11. Hardy, W.N., Foster, C.D., Mason, M.J., Yang, K.H., King, A.I., Tashman, S., (2001). Investigation of head injury mechanisms using neutral density technology and high-speed biplanar x-ray, *Stapp Car Crash Journal*. The Stapp Association, Ann Arbor, Michigan.
12. Horgan, T.J., & Gilchrist, M.D. (2003). The creation of three-dimensional finite element models for simulating head impact biomechanics. *International Journal of Crashworthiness*, 8(4), 353-366.
13. Horgan, T.J., and Gilchrist, M.D. (2004) Influence of FE model variability in predicting brain motion and intracranial pressure changes in head impact simulations. *International Journal of Crashworthiness* 9(4): 401-418.

14. Hutchison, M.G., Comper, P., Meeuwisse, W.H. & Echemendia, R.J. (2013). A systematic video analysis of National Hockey League (NHL) concussions, part I: who, when, where and what? *Br J Sports Med*, doi:10.1136/bjsports-2013-092234.
15. Kendall, M., Post, A., Rousseau, P., Oeur, A., Gilchrist, M.D., and Hoshizaki, T.B. (2012). A comparison of dynamic impact response and brain deformation metrics of head impact reconstructions for three mechanisms of head injury in ice hockey. *Proceedings of IRCOBI*, Dublin, IRL, Sept 10-14.
16. King, A.I., Yang, K.H., Zhang, L. & Hardy, W. (2003). Is head injury caused by linear or angular acceleration? *IRCOBI conference-Lisbon (Portugal)*, September, pages 1-12.
17. Kleiven, S. & von Holst, H. (2002). Consequences of brain size following impact in prediction of subdural hematoma evaluated with numerical techniques, *Proceedings of IRCOBI conference*, Munich Germany, 161-172.
18. Kleiven, S. (2007). Predictors for traumatic brain injuries evaluated through accident reconstructions. In: *Proceedings of the 51st Stapp Car Crash Conference*, San Diego CA, USA, 35 pages.
19. Langlois, J.A., Rutland-Brown, W., Wald, M.M. (2006). The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil.*(21): 375–8.
20. Mao, H., and Yang, K.H. (2010) Investigation of brain contusion mechanism and threshold by combining finite element analysis with in vivo histology data. *International Journal for Numerical Methods in Biomedical Engineering*, n/a. doi: 10.1002/cnm.1403.
21. Margulies, S. S., Thibault, L. E., & Gennarelli, T.A. (1990). Physical model simulations of brain injury in the primate. *Journal of Biomechanics*, 23(8): 823-836.
22. Meehan WP & Micheli LJ. Concussion results in deficits in neurocognitive functioning. *Clin Sports Med*. 2011;30:28–8.
23. Miller, R.T., Margulies, S.S., Leoni, M., Nonaka, M., Chen, X., Smith, D.H., and Meaney, D.F. (1998) Finite element approaches for predicting injury in an experimental model of severe diffuse axonal injury. In *Proc. 42nd Stapp Car Crash Conference*, Charlottesville, VA, pp. 155-167.
24. Nahum, A.M., Smith, R., Ward, C.C. (1977). Intracranial pressure dynamics during head impact, *Proceedings 21st Stapp Car Crash Conference*. Warrendale, PA, SAE paper No. 770922.
25. Newman, J. (1986). A generalized acceleration model for brain injury threshold (GAMBIT). In *Proceedings International Conf. on the Biomechanics of Impact (IRCOBI)*, 121-131.
26. Newman, J., Barr, C., Beusenberg, M., Fournier, E., Shewchenko, N., Welbourne, E., and Withnall, C. (2000a). A new biomechanical assessment of mild traumatic brain injury:

Part 2 – results and conclusions. In International IRCOBI Conference on the Biomechanics of Impact, 223-233, Montpellier, France.

27. Newman, J.A., Beusenbergh, M.C., Shewchenko, N., Withnall, C., and Fournier, E. (2005). Verification of biomechanical methods employed in a comprehensive study of mild traumatic brain injury and the effectiveness of American football helmets. *Journal of Biomechanics*, 38, 1469-1481.
28. Parker, R. (2012). *Concussive Brain Trauma: Neurobehavioral Impairment & Maladaptation*, 2nd edition, CRC press, Taylor & Francis group.
29. Post A, Hoshizaki TB, Gilchrist MD and Brien S. (2012) Analysis of the influence of independent variables used for reconstruction of a traumatic brain injury incident. *Proceedings of the Institution of Mechanical Engineers, Part P: Journal of Sports Engineering and Technology* 226:209 originally published online 14 March 2012. DOI: 10.1177/1754337112436629.
30. Post, A. and Hoshizaki, T.B. (2012). Mechanisms of brain impact injuries and their prediction: A review. *Trauma*, 14(4), 327-349.
31. Post, A., Hoshizaki, T.B., Gilchrist, M.D., (2012a). Finite element analysis of the effect of loading curve shape on brain injury predictors. *Journal of Biomechanics* 45, 679-683.
32. Post, A., Walsh, E.S., Hoshizaki, T.B., Gilchrist, M.D., (2012b). Analysis of loading curve characteristics on the production of brain deformation metrics. *Journal of Sports Engineering and Technology* 226(3/4), 200-207.
33. Ruan, J. (1994). *Impact Biomechanics of head injury by mathematical modeling*, PhD thesis, Wayne State University.
34. Shuck, L. & Advani, S. (1972). Rheological response of human brain tissue in shear, *Journal of Basic Engineering*, 905-911.
35. Thibault, L.E. (1993). Brain injury from the macro to the micro level and back again: what have we learned to date?. In: *Proceedings of the IRCOBI Conference*, Eindhoven, The Netherlands, 3-25.
36. Viano, D.C., King, A.I., Melvin, J.W., & Weber, K. (1989). Injury biomechanics research: an essential element in the prevention of trauma. *Journal of Biomechanics*, 22(5), 403-417.
37. Ward, C.C., and Nahum, A. (1979). Correlation between brain injury and intracranial pressure in experimental head impacts. *Proceedings of the international Conf. on Biomechanics of Trauma, IRCOBI*, Gotenborg, Sweden.
38. Willinger, R., Taled, L. & Kopp, C. (1995). Modal and temporal analysis of head mathematical models, *Journal of Neurotrauma*, 12, 743-754.
39. Willinger, R., & Baumgartner, D. (2003). Human head tolerance limits to specific injury mechanisms. *International Journal of Crashworthiness*, 8(6), 605-617.

40. Yogandandan, N., Li, J., Zhang, J., Pintar, F.A., and Gennarelli, T.A. (2008). Influence of angular acceleration-deceleration pulse shapes on regional brain strains. *Journal of biomechanics*, 41, 2253-2262.
41. Zhang, L., Yang, K., Dwarampudi, R., Omori, K., Li, T., Chang, K. et al., (2001). Recent advances in brain injury research: A new human head model development and validation, *Stapp Car Crash Journal*, 45, 369-393.
42. Zhang, L., Yang, K.H., & King, A.I. (2001a). Comparison of brain responses between frontal and lateral impacts by finite element modeling. *Journal of Neurotrauma*, 18(1), 21-30.
43. Zhang, L., Yang, K. H., & King, A. I. (2004). A proposed injury threshold for mild traumatic brain injury. *Journal of Biomechanical Engineering*, 126(2), 226-236.

Study #3

INFLUENCE OF CONCUSSIVE INJURY IMPACT EVENTS ON LOCATION OF PEAK TISSUE RESPONSE WITHIN DIFFERENT REGIONS OF THE BRAIN.

INTRODUCTION

A concussion is defined as a complex patho-physiological process affecting the brain, induced by biomechanical forces and resulting in the rapid onset of short-lived impairment of neurological function that resolves spontaneously [McCrory et al., 2013]. Concussion has also been described as an acute psychological experience of trauma incurred through head impact, acceleration, or both: an alteration or limited loss of consciousness [Parker, 2012]. While the definitions are slightly different, both describe trauma to the brain tissue with or without loss of consciousness. This is important since concussive injuries often occur without sufficient neurotrauma to be detected using current neuro-imaging techniques [Jantzen et al., 2004; Huisman et al., 2004]. This presents unique challenges to the medical practitioners in diagnosing and establishing a treatment for concussive injuries. As a result, many concussions go undiagnosed, allowing players to return to play too soon or even continue playing in the game where the injury occurred.

Concussive symptoms vary significantly between individuals. Common symptoms such as; headache, dizziness, nausea or vomiting are typically observed immediately following the impact, while other symptoms such as; persistent headache, poor attention, concentration and

memory dysfunction have been reported days to weeks after sustaining a concussion [Kelly and Rosenberg 1997]. For the most part, concussive symptoms tend to resolve within 3-7 days, however some cases report symptoms lasting longer than two weeks [McClincy et al. 2006] or up to a year [Alves, et al., 1993]. It is difficult to predict the duration and severity of concussive symptoms. Previous research has linked some concussive symptoms to specific functional regions of the brain [Purves et al., 2001]. It is proposed that concussive symptoms may be linked to the specific region of the brain that suffered brain tissue damage (Table S3-1). This may provide a more precise description of concussions effecting different parts of the brain.

Table S3-1. Brain regions commonly associated with concussive symptoms and their functionality

Region of the Brain	Functionality	Reference
Prefrontal cortex	decision-making and social behaviour	Yang et al., 2009
Dorsolateral prefrontal area	memory and organization	Yang et al., 2009
Motor association cortex and Primary motor cortex	planning and execution of movement patterns	Luppino & Rizzolatti, 2000
Primary somatosensory cortex	sense of touch and proprioception	Price, 2000
Visual cortex	Visual information	Braddick et al., 2001
Sensory and Visual association areas	Sensory integrations and perception of the environment	Price, 2000
Auditory cortex	sound processing	Purves et al., 2001
Auditory association area	functions associated with recognition of sounds	Creutzfeldt et al. 1989a & 1989b

While it is understood that there are many different ways people and athletes receive concussive brain injuries, the effect of these unique impact events creates differences in terms of peak dynamic head response (linear and rotational accelerations) and resulting MPS within the brain tissue [Post et al., 2012a; Kendall et al., 2012]. King and colleagues proposed that injurious brain tissue deformations are due to differences in the characteristics of the linear and rotational acceleration time history loading curves [King et al., 2003]. These differences in history curve characteristics may influence not only the severity but also affect different location of injurious brain deformations. A comparison of three different methodologies for head impacts, representing a fall, body-to-body impact and a punch, resulted in differences in peak strain of the brain tissue location within the brain [Kendall et al., 2012]. Brain tissue deformation is directionally dependent has been demonstrated in research using a number of injury models [Gennarelli et al, 1987; Hodgson et al, 1983; Prange et al, 2002]. The tissue response (bridging veins) and deformation tended to be more sensitive to antero-posterior rotational motions while translation motions had almost no effect on increases in tissue strain [Kleiven, 2003]. A recent study by Post (2013) examined different types of TBI injuries resulting from falls. He found that as the magnitude of brain deformation increased, so did the severity of the injury. While these studies used reconstruction data from traumatic brain injuries it is likely that the mechanical response of the brain tissue resulting in concussive injuries would respond similarly. These differences may result in different types of concussive injury depending on the magnitude, location and duration of brain tissue strain. Each impact event resulting in a concussive injury could affect a specific region of the brain, creating unique symptom onset and duration of the injury.

Maximal principal strain values within global sections of the brain have been reported in previous research for both concussive injury and non-injury reconstructions [Zhang et al., 2004; Viano et al., 2005; Kleiven (2007); Deck et al., 2008; Takhounts et al., 2008; Kimpara et al., 2012; McAllister et al., 2012; Patton et al., 2013, Rousseau, 2014]. These studies have reported MPS values for 50% risk of concussive injury as 0.19 to 0.38 for white matter and 0.26 to 0.47 grey matter. While research has identified the brainstem as playing an important role in concussive injury [McCrory, 2001; Guskiewicz & Mihalik, 2011; Makoshi et al., 2014], risk thresholds have been limited to animal data. Makoshi et al. (2014) reported brain stem strain values between 0.15 - 0.34 associated with varying grades of concussive injury in monkeys. A MPS value of 0.21 was found as a 50% risk for concussive injury in the brainstem. Recent research reconstructing rugby and Australian Rules football concussive injuries identified the thalamus as being the best brain deformation predictor for concussive injuries [Patton et al., 2013]. The study also reported strain values with a 50% risk for concussion for different regions of the brain of 0.13 in the thalamus, 0.14 in the brainstem, 0.26 in the white matter, and 0.27 in the grey matter. While these threshold values may be true for concussion risks reported specific to collision sports like rugby, Australian rules rugby, and American football, they may not be representative of all impact conditions which result in concussive brain injury.

The purpose of this study was to investigate differences in brain tissue response (MPS) and region of the brain affected for four specific impact events resulting in concussion (collision, unhelmeted falls, helmeted falls and punch).

METHODS

This study included the seventy-two (72) impact reconstructions from the four concussive injury impact events (falls, collision and punch) identified in the first study. The four impact events were reconstructed from video and/or hospital injury reports. Each injury event was reconstructed according to impact variables that would most represent the nature of that particular impact event. The fall events employed the monorail drop system to simulate falls to the similar impacting surfaces as per the injury events [Post et al., 2013, Kendall et al 2012]. The collision impacts used a linear impactor with effective mass [Rousseau, 2014] and compliance matched to a shoulder impact [Rousseau et al., 2015]. Impact reconstructions set-up were conducted following protocols developed in previous research for collision type impacts [Rousseau, 2014]. Finally, the punch events used a high-speed projectile launcher for lower mass and compliance to match the punch impacts [Walilko et al., 2005]. The resulting linear and rotational accelerations from the impact events were used as input for the University College Dublin Brain Trauma Model (UCDBTM) in order to obtain strain values within the brain tissue [Horgan & Gilchrist, 2003; Horgan & Gilchrist, 2004].

Research Variables

In the first analysis, the independent variables for this study were the four (4) concussive injury impact events (Collision, helmeted fall, Unhelmeted fall and Punch). The dependant variables were the maximal principle strain (MPS) in the grey matter, white matter and brain stem. The second analysis, focusing on regions typically related to onset of concussive symptoms within the cerebrum, had the impact events as independent variables and MPS within specific regions in the cerebrum (Auditory Association Area, Prefrontal Cortex, Motor

Association Area, Primary Motor Cortex, Primary Somatosensory Cortex, Sensory Area, Visual Association Area, Visual Cortex, Auditory Cortex, Dorsolateral Prefrontal Association Area) as the dependant variables.

Brain Model

The geometry of the UCDBTM was resolved from computed tomography (CT) and magnetic resonance imaging (MRI) scans of a male cadaver. The version of the model used for this research was composed of the following parts: scalp, three-layered skull (cortical and trabecular bone), dura, cerebrospinal fluid (CSF), pia, falx, tentorium, cerebral hemispheres, cerebellum and brainstem (Table S3-2; S3-3). Overall, this version of the UCDBTM has approximately 26,000 elements. The model was validated according to cadaveric pressure and brain motion data [Nahum et al., 1977; Hardy et al., 2001] as well as reconstructions of traumatic brain injuries [Doorly & Gilchrist, 2006].

The material properties (Tables S3-2 and S3-3) of the cerebrum, cerebellum and brain stem were derived from work done by Zhang et al. (2001). The remaining parts of the model (scalp, cortical and trabecular bone, and intracranial membranes) were derived from research conducted by Ruan (1994), Willinger et al. (1995), Zhou et al. (1995) and Kleiven and von Holst (2002). Work done by Zhou et al (1995) and Shuck and Advani (1972) were used in the characterization of the shear modulus of viscoelastic brain tissue:

$$G(t) = G_{\infty} + (G_0 - G_{\infty})e^{-\beta t}$$

Where, G_{∞} is the long term shear modulus, G_0 is the short term shear modulus and β is the decay factor [Horgan & Gilchrist, 2003]. The brain skull interface was modeled by modeling the CSF as brick elements with a low shear modulus, which allows it to behave like water. The contact

definition at the brain skull interface was assigned as no separation and used a friction coefficient of 0.2 [Miller et al., 1998].

Table S3-2. Properties of the materials used in the finite element model for UCDBM

Material	Poisson's Ratio	Density (kg/m³)	Young's Modulus (MPa)
Grey Matter	0.49	1060	(hyperelastic)
White Matter	0.49	1060	(hyperelastic)
Trabecular Bone	0.24	1300	1000
Dura	0.45	1130	31.5
Pia	0.45	1130	11.5
Falx	0.45	1140	31.5
Tentorium	0.45	1140	31.5
CSF	0.5	1000	-

Table S3-3. Characteristics of the brain tissue for UCDBM

Material	Shear Modulus (kPa)		Decay Constant (GPa)	Bulk Modulus (s⁻¹)
	G₀	G_∞		
Cerebellum	10	2	80	2.19
Brain Stem	22.5	4.5	80	2.19
White Matter	12.5	2.5	80	2.19
Grey Matter	10	2	80	2.19

Regions of the brain

The three-dimensional dynamic response-loading curves from the injury reconstructions of the four concussive impact events (collisions, helmeted falls, unhelmeted falls and punch) were used to run the UCDBTM brain model. The brain model was, in a first analysis, divided into global regions (grey matter, white matter and brain stem) to determine difference in MPS between these global regions. A follow up analysis divided the cerebrum into 10 regions commonly associated with concussive symptoms (Figure S3-1), in order to analyze differences in MPS per specific regions within the cerebrum [Post et al., 2012; Kendall et al., 2012].

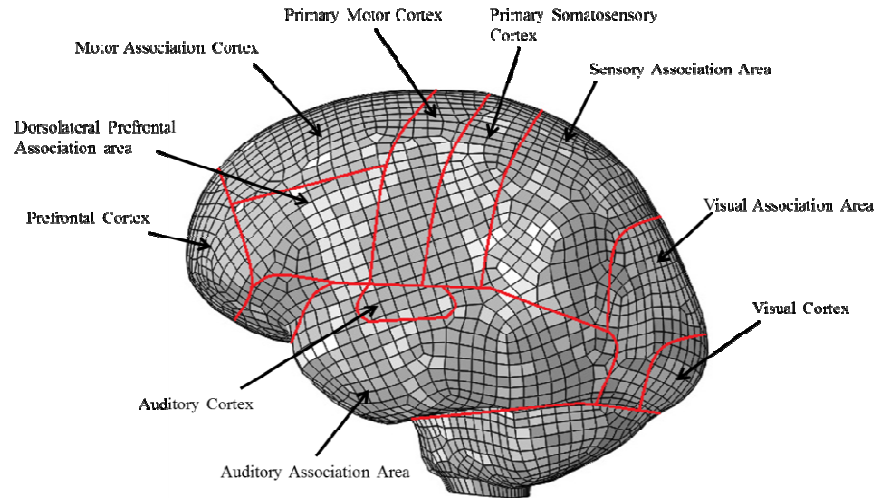


Figure S3-1. Image of the UCDBTM segmented into regions associated with symptoms of concussion.

Statistical analysis

A MANOVA followed by three (3) one-way ANOVAs were used to determine main effect of the impact events on the specific brain region; Cerebrum, Cerebellum and Brain Stem ($p < 0.05$). Further analysis will examine the strain field created by each of the four impact events. The strain field is a depiction of how strain within each of the 10 different regions of the cerebrum is different from one specific region to another. To achieve this, ten (10) one-way ANOVAs were used to determine main effect of the impact events on strain fields within regions of the brain commonly associated with concussive symptoms; Auditory Association Area, Prefrontal Cortex, Motor Association Area, Primary Motor Cortex, Primary Somatosensory Cortex, Sensory Area, Visual Association Area, Visual Cortex, Auditory Cortex, Dorsolateral Prefrontal Association Area ($p < 0.05$).

RESULTS

MPS in global regions of the brain

The MPS results for the global regions of the brain (grey matter, white matter, cerebellum and brain stem) are reported in Table S3-4. Significant differences in grey matter strains were found between all impact events except for the unhelmeted vs punch events ($p < 0.05$). Significant differences in white matter strains were found between all impact events with the exception of; the unhelmeted vs helmeted events, the unhelmeted vs punch events and the helmeted vs punch events ($p < 0.05$). Finally, significant differences in MPS were found in the brainstem region between all impact conditions except the helmeted fall and punch events ($p < 0.05$). For the unhelmeted events, only three injury (cases #5, #9, #12) cases were used in the analysis of brainstem region due to abnormally high strain values (1.885 ± 1.138).

Table S3-4. MPS values for global regions of the brain.

Brain Region	Collisions	Un-helmeted fall	Helmeted fall	Punch
Grey Matter (SD)	0.32 (0.11)	0.58 ^a (0.11)	0.41 (0.12)	0.51 ^a (0.17)
White Matter (SD)	0.29 (0.12)	0.42 ^{ab} (0.10)	0.42 ^c (0.12)	0.44 ^{abc} (0.15)
Brainstem (SD)	0.38 (0.08)	0.67 (0.19)	0.47 ^a (0.25)	0.50 ^a (0.09)

^{abc} - denotes impact events with no significant differences between them

MPS of regions between impact events

The analysis was further broken down to evaluate specific regions within the cerebrum commonly associate with concussive injury symptoms. Significant differences in MPS values for regions within the cerebrum revealed some differences between the four impact events (Figure S3-2). The top three MPS values for the collision impact events occurred in the auditory association area, followed by the dorso-lateral prefrontal cortex and primary somatosensory cortex. The un-helmeted fall impact events produced the highest MPS values in the auditory association area, the prefrontal cortex and visual association area. The highest MPS value for the helmeted impact events were found in the auditory association area, followed by the dorso-lateral prefrontal cortex and the visual association area. Finally, consistent with the previous three impact events, the punch events produced the highest MPS in the auditory association area. The primary somatosensory cortex and sensory association area rounded out the top three areas for the punch loading condition.

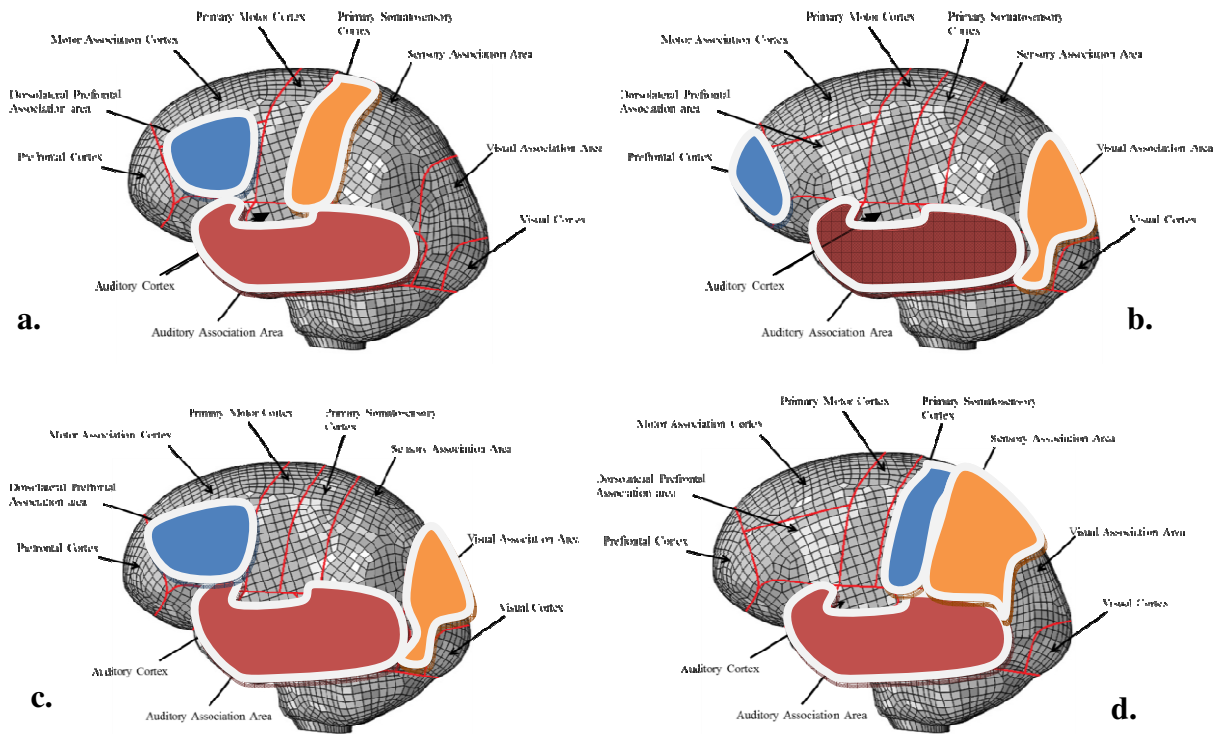


Figure S3-2. Differences in location of the three highest (red = highest, blue = 2nd highest, orange = 3rd highest) MPS within the brain per impact event (a.) Collisions, (b.) Un-helmeted fall, (c.) Helmeted fall and (d.) Punch.

The impact event and regions of the cerebrum (in bold) with the highest MPS values are depicted in Table S3-5. All four impact events produced the highest MPS value in the auditory association area. The collision event (0.32 ± 0.12) was found to be significantly different from the other three loading conditions ($p < 0.05$). The next highest region of strain for the collision event was the dorso-lateral prefrontal cortex (0.22 ± 0.09). Once again, significant differences were found between the collision event and the other three loading conditions ($p < 0.05$). The primary somatosensory cortex was found to be the region producing the third highest MPS values for the collision event (0.20 ± 0.08) and significant differences ($p < 0.05$) to all other loading conditions except the helmeted fall event were found.

Table S3-5. Location within the cerebrum of the three highest MPS values by concussive injury impact event (Bold identifies top three regions of MPS within each impact event).

Cerebrum Region	Collisions	Un-helmeted fall	Helmeted fall	Punch
Auditory Association Area (SD)	0.32[†]$\Phi$$\beta$ (0.12)	0.57*Φ (0.11)	0.45*[†] (0.13)	0.50* (0.16)
Dorso-lateral prefrontal cortex (SD)	0.22Φ (0.09)	0.42* Φ (0.08)	0.30* (0.10)	0.35* (0.12)
Prefrontal Cortex (SD)	0.16 [†] (0.06)	0.45 (0.13)	0.23 [†] (0.10)	0.24 [†] (0.08)
Visual Association Area (SD)	0.18 [†] Φ (0.05)	0.43Φ (0.07)	0.30[†] (0.11)	0.27 [†] (0.06)
Sensory Association Area (SD)	0.20 β (0.08)	0.34 (0.09)	0.25 β (0.07)	0.36 (0.16)
Primary Somatosensory Cortex (SD)	0.20β (0.08)	0.40* (0.15)	0.23 β (0.06)	0.40* (0.19)

* denotes significant differences between with Collision impact event

[†] denotes significant differences between with Unhelmeted impact event

Φ denotes significant differences between with Helmeted impact event

^B denotes significant differences between with Punch impact event

The auditory association area for the un-helmeted fall impact events (0.57 ± 0.11) was found to be significantly different from the collision and helmeted fall impact event ($p < 0.05$). The prefrontal cortex (0.449 ± 0.131) was the next highest strain value and was found to be significantly different to the three other loading conditions ($p < 0.05$). The visual association area (0.449 ± 0.074) produced the third highest MPS values for this impact event and was also found to be significantly different to the other concussive injury impact events ($p < 0.05$).

For the helmeted impact condition the highest MPS value were again found in the auditory association area (0.45 ± 0.13) and significantly different to all other impact events except the punch ($p < 0.05$). The next highest MPS values for this loading condition was found in the dorsal lateral prefrontal cortex (0.30 ± 0.10) and were deemed significantly different to all other impact events except the punch impact events ($p < 0.05$). The third highest MPS value was found in the visual association area (0.30 ± 0.11) and again found to be significantly different to all other loading conditions except the punch events ($p < 0.05$).

Similarly to the other impact conditions, the auditory association area revealed the highest MPS value for the punch impact events (0.50 ± 0.16). This value was only found to be significantly different ($p < 0.05$) to the collision loading condition. The primary somatosensory cortex was the location of next highest strain value for the punch impact event and was significantly different ($p < 0.05$) to the other impact events with the exception of the un-helmeted fall event. Finally, the third highest MPS value was found in the Sensory association area (0.36 ± 0.16) and significantly different ($p < 0.05$), to the other loading conditions with the exception of the un-helmeted fall events.

MPS of regions within each impact event

The data from all ten regions within each impact event was analyzed to define strain fields unique to each impact event. Based on the peak MPS values for each region of the cerebrum, significant differences between regions were identified through multiple one-way ANOVAs and post-hoc tests (Table S3-6.). The strain fields are also depicted in Figure S3-3 for more visual effect.

Collision impact event

The auditory association area (0.32 ± 0.12) revealed the highest strain field and was significantly different to all other regions for the collision impact event ($p < 0.05$). The second strain field was led by the Dorso-lateral prefrontal cortex (0.22 ± 0.09) found significantly different ($p < 0.05$) to the Prefrontal cortex and Visual cortex regions. While Prefrontal cortex and Visual cortex formed the third strain field, the second strain field was contained the Auditory cortex, Dorsolateral prefrontal cortex, Motor association cortex, Visual association area, Sensory association area, Visual association area, Primary somatosensory cortex and Primary motor cortex.

Table S3-6. Brain strain field within the cerebrum for each of the four concussive injury impact events (Red = 1st level of strain field (highest); Orange = 2nd level of strain field (second highest); Yellow= 3rd level of strain field (third highest); Green = 4th level of strain field (fourth highest)).

Impact events	Regions of Cerebrum									
	1	2	3	4	5	6	7	8	9	10
Collision	0.32 (0.11)	0.18 (0.07)	0.22 (0.09)	0.17 (0.06)	0.16 (0.06)	0.10 (0.03)	0.18 (0.05)	0.19 (0.08)	0.20 (0.08)	0.20 (0.08)
Un-helmeted fall	0.57 (0.11)	0.26 (0.07)	0.42 (0.08)	0.37 (0.06)	0.45 (0.13)	0.26 (0.04)	0.43 (0.07)	0.34 (0.09)	0.40 (0.15)	0.37 (0.11)
Helmeted fall	0.45 (0.12)	0.24 (0.12)	0.30 (0.09)	0.24 (0.08)	0.23 (0.10)	0.25 (0.09)	0.30 (0.10)	0.25 (0.06)	0.24 (0.05)	0.17 (0.10)
Punch	0.50 (0.16)	0.32 (0.14)	0.35 (0.12)	0.26 (0.07)	0.24 (0.08)	0.15 (0.04)	0.27 (0.06)	0.36 (0.16)	0.40 (0.19)	0.34 (0.11)

1-Auditory Association Area, 2- Auditory Cortex, 3- Dorsolateral prefrontal association area, 4- Motor Association Cortex, 5-Prefrontal Cortex 6-Visual Cortex, 7-Visual Association Area, 8-Sensory Association Area 9- Primary Somatosensory Cortex, 10- Primary Motor Cortex

Un-helmeted fall impact events

Similar to the Collision impact events, the auditory association area produced the highest MPS value (0.567 ± 0.109). The MPS results for the auditory association area were found to be significantly different ($p < 0.05$) to the auditory cortex and visual cortex only. Therefore the highest strain field for the unhelmented fall events was defined by; all regions excluding the auditory cortex and visual cortex. The visual cortex and auditory cortex defined the second strain field for this loading condition.

Helmeted fall impact events

The helmeted fall impact events also revealed the highest strains in the auditory association area (0.454 ± 0.128) and significantly different to all other regions ($p < 0.05$), forming the first strain field. The dorso-lateral prefrontal cortex was the next highest strain value and was found to be significantly different to only the primary motor cortex ($p < 0.05$). Therefore, the second highest strain field was created with all regions except the auditory association area and primary motor cortex. The primary motor cortex formed the third strain field.

Punch impact event

As with the other three impact events, the auditory association area (0.498 ± 0.158) produced the highest MPS value. The auditory association area was found to be significantly different from all regions of the cerebrum ($p < 0.05$) and thus defined the highest strain field within this region alone, similar to the collision and helmeted fall impact events. The primary somatosensory cortex (0.404 ± 0.186) and sensory association area (0.364 ± 0.164) represented the second highest region for MPS. The second highest region, the primary somatosensory cortex was found to be significantly different to all regions except the auditory cortex, dorso-lateral prefrontal cortex, motor association cortex, prefrontal cortex, visual cortex, visual association area, sensory association area and primary motor cortex ($p < 0.05$). This group created the next highest strain field. Finally, the Visual association area was found to be significantly different to all regions except the motor association cortex, prefrontal cortex ($p < 0.05$) creating the third strain field. The prefrontal cortex region formed the fourth strain field level on its own.

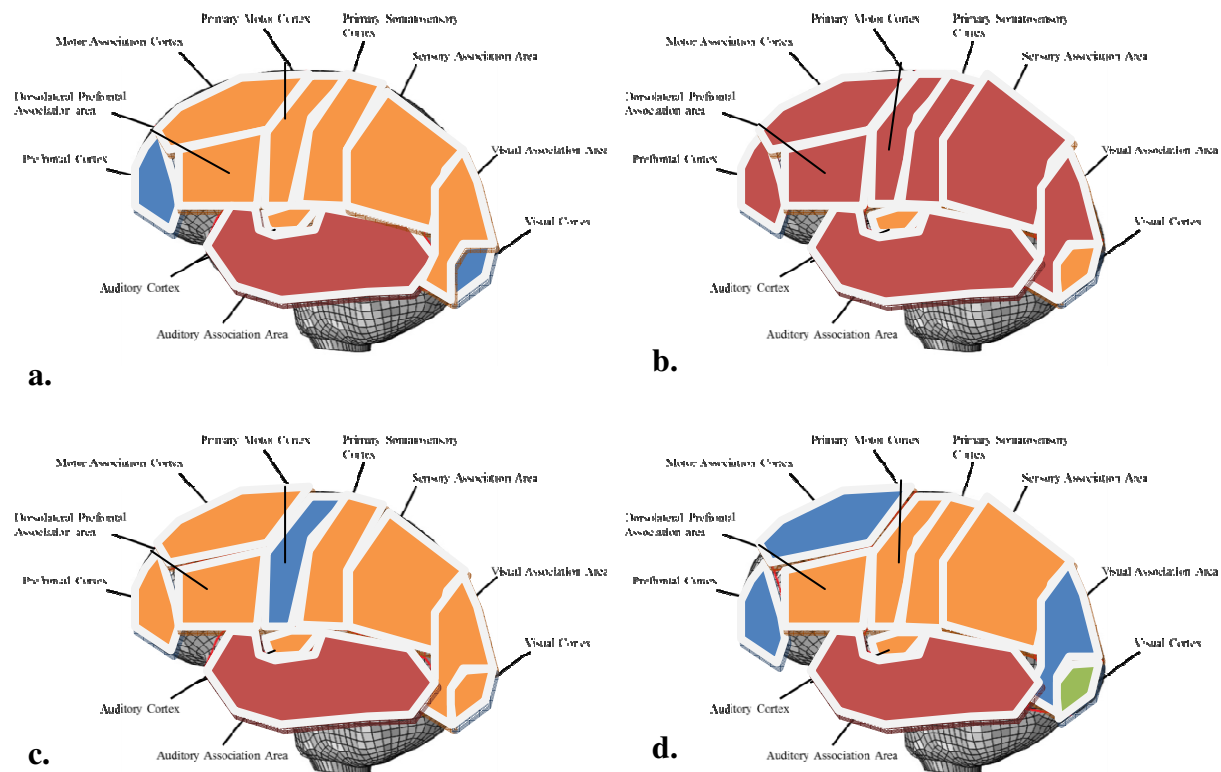


Figure S3-3. Differences in location of the strain fields (red = first level strain field, blue = 2nd level strain field, orange = 3rd level strain field, green = 4th level strain field) within the brain per impact event (a.) Collisions, (b.) Un-helmeted fall, (c.) Helmeted fall and (d.) Punch.

DISCUSSION

This study was designed to examine differences in the location of maximum principle strain (MPS) within different regions of the brain between four concussive impact conditions. While the aim of this study was not to associate particular symptoms of concussion to a specific region of the brain affected, it was hypothesized that each of these different concussive injury impact conditions create a unique type of concussive injury, that would highlight changes in location where the highest MPS values occur in the brain. These unique differences may present themselves in the form of differences in onset, and/or duration of concussive symptoms. Although symptom onset and duration were not monitored or analyzed in this study, this

hypothesis was based on the rationale that brain strain fields within the cerebrum would be different for each concussive injury impact event.

The results of this study produced MPS values within or above previous reported concussive injury range in all three of these brain regions (grey matter, white matter and brain stem) [Makoshi et al., 2014, Zhang et al., 2004, Kleiven, 2007, Patton et al., 2013]. This was expected since all reconstructions used in this study represented diagnosed concussive injuries. The more interesting results were the differences in MPS in each global region between the impact events. Differences in terms of dynamic response time histories likely contributed to these differences in location and amplitude of MPS within the brain. This result clearly demonstrates that impact events can affect strain field locations within different regions of the brain. It is important to note at this point that unrealistically high strain values (180% strain) were found in the brainstem for nine out of the twelve unhelmeted impact events. This is likely due to the high magnitude dynamic response and which essentially saturates the finite element model. Secondly, while the model has been partial validated to pressure and brain motion study, the brainstem region has not yet been validated.

When examining specific brain regions within the cerebrum commonly associated with concussive symptoms, the results showed that the impact event affected the location of the highest MPS values. While the auditory association area was identified as the location of the highest MPS value across all four loading conditions, the second and third highest values tended to change dependent on the type of injury event (Table S3-2). The commonality of the auditory association area producing the highest strain among all four impact events may support the theory surrounding the effects of skull geometry and bony prominences creating high stress points within the brain as a result of high rotational accelerations [Ommaya & Gennarelli, 1974].

This result also may explain some of the commonality in symptom onset for all concussions, while the differences in the second and third highest values could potentially explain variations in symptom onset. The impact condition in this study (Collision, Helmeted fall, punch and unhelmeted fall) can be classified at separate ends of the compliance spectrum. A compliance effect was observed with respect to MPS values within the cerebrum. The collision impact event, which was characterized by the most compliance within the system, tended to produce significantly lower MPS values across its three highest regions of strain when compared to the other concussive injury loading conditions. Conversely, the unhelmeted fall impact event, which would be classified as having the least amount of compliance within the system, tended to produce the highest strains compared to the other injury impact events. Significant differences between these regions of higher strain values between impact events tended to occur between loading conditions where greater differences in compliance were present.

Strain fields within impact events

The results of this study support a continuum effect for compliance, where increased MPS values are observed with a decrease in compliance within the impact condition for a given injury loading condition. The unhelmeted fall events would be defined as the least compliant loading condition, followed by the punch events. The helmeted fall events would be classified as having more compliance than the Punch followed by the Collision events providing the most compliance among the four loading conditions in this study. This compliance effect is further amplified in the analysis of brain strain fields within each concussive injury impact event (Table S3-6 & Figure S3-3). The unhelmeted fall events, with the least compliance produced only two levels of strain fields. The highest strain value was found in the auditory association area; however, this value was not significantly different from the other regions of the cerebrum with

the exception of the auditory and visual cortex. These two regions formed the second level strain field for this impact event. For the most part, this loading condition was populated by injury cases from hospital fall data. Though it wasn't the focus of the study, most of these injury cases resulted in multiple symptom onset with prolonged duration, commonly associated with persistent concussive syndrome (PCS). On a concussive injury continuum, this impact event might fall at the high end of the continuum closely associated with diffuse axonal injury (DAI) type injuries [Hoshizaki et al., 2014]. The strain fields for the other injury events also differ from one another. The collision event produced three strain fields significantly different from one another. After the auditory association area, the second level strain field was defined by all other regions of the brain except the prefrontal and visual cortex, which formed the third level strain field. The helmeted fall event contained three strain fields with auditory association area producing the highest strain, the dorsolateral prefrontal cortex and the primary motor cortex leading the second strain and the third strain fields respectively. The punch event was the only loading condition to contain four strain fields. These changes in strain field patterns are likely a result of the differences between the specific characteristics of the dynamic loading curves and energy transfer which is unique to each injury event seen in the second study. The characteristics creating the different concussive injury events have a significant effect on the tissue response within the different regions of the brain resulting in specific strain field patterns. While it is clear that the strain fields shown in the study differ between loading conditions, this supports the theory that concussive injuries result from diffuse energy transmitted through out the brain [Ommaya and Gennarely, 1974]. Current head impact reconstructions have not yet linked particular symptoms of concussions with damage to a specific area of the brain. Studies have reported symptoms present immediately after a concussive event are associated with the

biomechanical parameters of the collision, as well as the specifically affected brain structures [Jacobson & Marcus, 2008; Janowsky et al., 1989; Kontos et al., 2004; Ropper & Samuels, 2009; Viano et al., 2005]. The diffuse wave of energy from each of the impact conditions creates damaging strain fields within specific regions of the brain dependent the impact condition, which could be associated with specific onset of symptoms. The duration of these symptoms could be related to the magnitude of these strain fields since it has been reported that axonal strain levels of 0.12 create functionality loss of the axon; though only temporary [Galbraith et al., 1993, Thibault et al, 1985]. This could also be true for the different levels of strain fields with respect to specific regions of the brain, onset of symptoms and their duration. Specific regions of the brain affected with low strains may have symptoms present early, but resolve quickly, while higher magnitude levels of strain may have symptoms present with longer durations or even some loss of functionality [Galbraith et al. 1993; Thibault et al., 1985].

Limitations

The surrogate headform used for the injury reconstructions was the Hybrid III head form. It is commonly used as a physical model in impact reconstruction due to its reliability. While it is reliable, it does not provide a true biofidelic response due to its construction materials (i.e. steel). The UCDBTM is one of few partially validated models available for this type of research. The model makes assumptions surrounding the characteristics of the brain tissue and the interactions (anisotropic elements) between different parts of the brain and skull. As a result, the comparisons made with the UCDBTM are meant to be representative of how the brain may deform under the loading scenarios and may not represent the exact motion of the brain resulting from impacts. Furthermore, brain deformation metric analysis was limited to the cerebrum since brain stem has not yet been validated for finite element modelling.

CONCLUSION

The purpose of this study was to report and compare the locations of the highest MPS values resulting from four specific impact events (collision, unhelmeted falls, helmeted falls and punch). The results of this study identified differences in regions within the cerebrum of highest MPS commonly associated with symptoms of concussive injury for each of the impact events. Previous studies have defined concussive injuries as diffuse, this study support these claims with the identification of unique strain fields dependent on the impact event. The strain levels within each of the strain fields could help explain the onset of multiple symptoms when concussed, while the amplitude of these strains may explain the duration of their presence. The results of this thesis could help provide important information in the development of head protection and impact material to protect and test against specific impact events /loading conditions common to that sport.

REFERENCES

1. Alves, W., Macciocchi, S.N. & Barth, J.T. (1993). Post concussive symptoms after uncomplicated mild head injury. *Journal of Head Trauma Rehabilitation*, 8, 48–59.
2. Braddick, O.J., O'Brien, J.M., Wattam-Bell, J., Atkinson, J., Hartley, T. & Turner, R. (2001). Brain areas sensitive to coherent visual motion. *Perception*, 30; 61–72.
3. Creutzfeldt, O., Ojemann, G., Lettich, E. (1989a). Neuronal activity in human lateral temporal lobe. I. Responses to speech. *Exp. Brain Res.* 77:451-475.
4. Creutzfeldt, O., Ojemann, G., Lettich, E. (1989b). Neuronal activity in human lateral temporal lobe. II. Responses to subjects own voice. *Exp. Brain Res.* 77:476-489.
5. Doorly, M.C. & Gilchrist, M.D. (2006). The use of accident reconstruction for the analysis of traumatic brain injury due to head impacts arising from falls, *Computer Methods in Biomechanics and Biomedical Engineering*, 9(6), 371-377.

6. Gennarelli, T.A., Thibault, L.E., Tomel, G., Wiser, R., Graham, D., Adams, J. (1987). Directional dependence of axonal brain injury due to centroidal and non centroidal acceleration. In: Proceedings of the Stapp Car Crash Conference, New Orleans, LA, 49-53.
7. Hardy, W.N., Foster, C.D., Mason, M.J., Yang, K.H., King, A.I. & Tashman S. (2001). Investigation of head injury mechanisms using neutral density technology and high-speed biplanar x-ray, Stapp Car Crash Journal, The Stapp Association, Ann Arbor, Michigan,.
8. Hodgson, V.R., Thomas, L.M., and Khalil, T.B. (1983). The role of impact location in reversible cerebral concussion. In Proceedings of the 27th Stapp Car Crash Conference, SAE paper No. 831618.
9. Horgan, T.J. and Gilchrist, M.D. (2003). The creation of three-dimensional finite element models for simulating head impact biomechanics, International Journal of Crashworthiness, 8(4), 353-366.
10. Horgan, T.J. and Gilchrist, M.D.(2004). Influence of FE model variability in predicting brain motion and intracranial pressure changes in head impact simulations, International Journal of Crashworthiness, 9(4), 401-418,.
11. Huisman, T.A., Schwamm, L.H., Schaefer, P.W., et al. (2004). Diffusion tensor imaging as potential biomarker of white matter injury in diffuse axonal injury. AJNR; 25:370–376.
12. Jacobson, S. & Marcus, E.M. (2008). Neuroanatomy for the neuroscientist. New York, NY: Springer, Verlag.
13. Janowsky, J.S., Shimamura, A.P., Kritchevsky, M. & Squire, L.R. (1989). Cognitive impairment following frontal lobe damage and its relevance to human amnesia. Behavioral Neuroscience, 103(3), 548-560.
14. Jantzen, K.J., Anderson, B., Steinberg, F.L., et al. (2004). A prospective functional MR imaging study of mild traumatic brain injury in college football players. AJNR; 25:738–745.
15. Kelly, J.P. & Rosenberg, J.H. (1998). The development of guidelines for the management of concussion in sports. J Head Trauma Rehabil. 13:53–65
16. Kendall, M., Post, A., Rousseau, P., Oeur, A., Gilchrist, M.D., and Hoshizaki, T.B. (2012). A comparison of dynamic impact response and brain deformation metrics of head impact reconstructions for three mechanisms of head injury in ice hockey. Proceedings of IRCOBI, Dublin, IRL, Sept 10-14.
17. Kleiven, S., and Von Holst, H. (2002). Consequences of size following trauma to the human head. Journal of Biomechanics, 35, 135-160.
18. Kleiven, S. (2003). Influence of impact direction to the human head in prediction of subdural hematoma. Journal of Neurotrauma, 20(4), 365-379.

19. Kontos, A.P., Collins, M. & Russo, S.A. (2004). An Introduction to Sports Concussion for the Sport Psychology Consultant. *Journal of Applied Sport Psychology*, 16(3), 220-235.
20. Luppino, G. & Rizzolatti, G., The organization of the frontal motor cortex. *News Physiol. Sci.*, Vol. 15, October; 219-224, 2000.
21. McClincy, M.P., Lovell, M.R., Pardini, J., Collins, M.W. & Spor, M.K. (2006). Recovery from sports concussion in high school and collegiate athletes, Vol. 20, No. 1 , Pages 33-39 doi:10.1080/02699050500309817)
22. McCrory P, Meeuwisse W, Aubry M, et al. Consensus statement on Concussion in Sport – 4th International Conference on Concussion in Sport held in Zurich, November 2012. *Clin J Sport Med* 2013;23(2):89-117.
23. Miller, R., Margulies, S., Leoni, M., Nonaka, M., Chen, Z., Smith, D., and Meaney, D. (1998). Finite element modeling approaches for predicting injury in an experimental model of severe diffuse axonal injury. In *Proceedings of the 42nd Stapp Car Crash Conference*, SAE paper No. 983154.
24. Nahum, A.M., Smith, R. and Ward, C.C. (1977). Intracranial pressure dynamics during head impact, *Proceedings 21st Stapp Car Crash Conference*. Warrendale, PA, SAE paper No. 770922.
25. Parker, R. (2012). *Concussive Brain Trauma: Neurobehavioral Impairment & Maladaptation*, 2nd edition, CRC press, Taylor & Francis group.
26. Post, A., Hoshizaki, T.B., Gilchrist, M.D., (2012a). Finite element analysis of the effect of loading curve shape on brain injury predictors. *Journal of Biomechanics* 45, 679-683.
27. Post A. (2013). The influence of dynamic response characteristics on traumatic brain injury. Doctoral Thesis, University of Ottawa, Canada.
28. Practice parameter: the management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. *Neurology*. 1997;48:581–585.
29. Prange, M.T., Meaney, D.F., and Margulies, S.S. (2002). Defining brain mechanical properties: Effects of region, direction and species. In: *Proceedings 44th Stapp Car Crash Conference*.
30. Price, C.J., The anatomy of language: contributions from functional. neuroimaging. *Journal of Anatomy*. 197(3): 335-359, 2000.
31. Purves D, Augustine WJ, Fitzpatrick D, Lawrence CK, LaMantia AS, McNamara JO, Williams SM eds. *Neuroscience*, 2nd edition, Sinauer Associates, Sunderland (MA), 2001.
32. Ropper, A.H. & Samuels, M.A., *Adams and Victor’s principles of neurology*. New York, NY: McGraw Hill Medical, 2009.

33. Shuck, L. & Advani, S. (1972). Rheological response of human brain tissue in shear, *Journal of Basic Engineering*, 905-911.
34. Thibault, L.E. & Gennarelli, T.A. (1985). Biomechanics of diffuse brain injuries. Proc. 29th Stapp car crash conference, SAE paper No. 856022.
35. Viano, D.C., Casson, I.R., Pellman, E.J., Zhang, L., King, A.I. & Yang, K.H., Concussion in professional football: Brain responses by finite element analysis: Part 9. *Neurosurgery*, 57(5), 891-915, 2005.
36. Willinger, R., Taled, L., and Pradoura, P. (1995). Head biomechanics from finite element model to the physical model. Proceedings of the 1995 IRCOBI Conference.
37. Yang, Y., Raine, A., Colletti, P., Toga, A.W. & Narr, K.L. (2009). Abnormal temporal and prefrontal cortical gray matter thinning in psychopaths. *Molecular Psychiatry*, 14, 561–562; doi:10.1038/mp.2009.12.
38. Zhang, L., Yang, K.H., Dwarampudi, R., Omori, K., Li, T., Chang, K., Hardy, W.N., Khalil, T.B., and King, A.I. (2001b). Recent advances in brain injury research: a new human head model development and validation. *Stapp Car Crash Journal* 45, 369-394.
39. Zhou, C., Khalil, T.B., King, A.I. (1995). A new model for comparing responses of the homogeneous and inhomogeneous human brain. In: Proceedings of the 39th Stapp Car Crash Conference, 121-136.

PART III

Global discussion and conclusions

The purpose of this thesis was to compare and characterize four impact events creating concussive injuries by the kinematics of dynamic and brain tissue response. The first study used seventy-two (72) concussive injuries reconstructions categorized into four impact events of injury (Collisions, Helmeted falls, unhelmeted falls and punches) to compare peak dynamic response of the head and brain tissue metrics. The second study used the dynamic response time history curves from the first study to characterize the unique dynamic response elements for each of the different concussive injury impact events. The third study compared strain values within the different brain regions commonly associated with concussive symptoms per the different loading conditions.

The first study developed and used specific methodologies that accounted for mass, velocity and compliance specific to each specific impact event. Peak linear/rotational acceleration and impact duration data were gathered and compared between loading conditions. The resulting dynamic response time history curves were input into the UCDBTM to compare MPS and Von Mises stress values between impact events. All injury events fell within at least a 25% risk for concussive injury except the collision impact event [Zhang et al., 2004; Kleiven, 2007]. The collision event fell below the 25% risk of concussive injury, while other impact events resulted in higher injury risk, suggesting that current threshold may not account for all concussive injury conditions. This dataset is one the largest concussive injury reconstruction database represented by actual events specific to different impact events of its kind in literature to date. The methodologies created in this study may provide for more accurate injury risk

thresholds specific to loading conditions leading to concussive injury in future studies or for equipment safety standard development.

The second study used the reconstruction data from the first study. While the first study focused on the reconstruction methodologies for the four injury conditions compared the peak dynamic response and brain tissue stress/strain values between these impact events, the second study looked to uniquely characterize each concussive injury event through dynamic response loading curve characteristics. Using a principal components analysis, the characteristics of the linear and rotational acceleration loading curves from the injury reconstructions were analyzed for each of the four injury events [Post et al., 2013]. The results of this study showed differences in the unique characteristics that best characterized each specific impact event. However, using one primary characteristic for each of the injury events may not be sufficient to describe injury risk due to low correlations values (30%). Thus, this research suggests combinations of characteristics are necessary to describe each concussive impact event. For example, the collision events might be characterized by directional resultant linear and rotational velocities, and linear acceleration loading rate, accounting for 50.5% of the total variance, while the punch events might be characterized by resultant rotation velocity, linear resultant loading rate and directional linear acceleration time to peak, accounting for 72.3% of the total variance. These results demonstrated the importance of the characteristics dynamic response time history curves for both linear and rotational acceleration from different impact events of injury in the creation of strain fields within the brain creating concussive injuries. This information could be useful in the identification and development of specific protective equipment standards unique to different impact events in various sports. This research also promotes the importance of using FE

modeling with injury reconstructions to gain a better understanding of the effects of specific characteristics of the dynamic response loading curves as they relate to brain tissue response.

The third study identified differences in global strain between the four concussive injury events for grey matter, white matter and brain stem. It also identified strain field differences between loading conditions by regions within the cerebrum commonly associated with concussive symptoms [Yang et al., 2009; Luppino & Rizzolatti, 2000; Price, 2000; Braddick et al., 2001; Purves et al., 2001; Creutzfeld et al., 1989a; Creutzfeldt et al., 1989b]. The results of this study produced significant differences between all four injury events for magnitude of strain in grey matter, white matter and brain stem. Further analysis of strain fields within regions of the cerebrum also resulted in significant differences in locations of the highest strain values between injury events. While this study was not correlating onset and duration of particular symptoms relative the location of the highest strain values, this information does show that the components making up a particular concussive injury event (mass, velocity, location and compliance) can affect the strain fields within the cerebrum. While previous research studies have demonstrated differences in location of strain within the brain dependant on impact location, no data has demonstrated strain field changes resulting from different impact events. In addition, the results showed that location within the cerebrum producing the highest strain field is dependent on the concussive injury event. This research promotes the importance of using FE modeling with injury reconstructions in order to gain a better understanding the effects of specific characteristics of the dynamic response loading curves as they related to brain tissue response. Though the aim of this study was not to correlate onset and duration of concussive symptoms to location of the high strain, the methodology presented in this dissertation could open doors to this possibility in future research.

Conclusion

The purpose of this dissertation was to compare and characterize four specific concussive injury events (collisions, helmeted falls, unhelmeted falls and punches) by means dynamic response and brain tissue response characteristics through impact reconstructions of actual concussive injury events. The first study created the methodologies to reconstruct actual injury cases for each of the four injury events and compared peak linear and rotational accelerations and brain tissue response typically used in assessments for risk of concussive injury [Zhang et al., 2004; Kleiven, 2007]. Further analysis was completed in study two to characterize each impact event through specific characteristics of the dynamic response time histories curves (i.e. peak, slope, duration). The assumption here was that each impact event produced a different type of concussive injury through different characteristics of the dynamic response loading curves. Finally, the location of the peak strain within the brain was compared between loading conditions. The premise for this analysis was that if different loading conditions produce different types of concussive injury, then the different impact events might create different strain fields at different locations within the brain. The result of the first study demonstrated that the four concussive injury events in this study do produce significant differences in terms of peak linear and rotational accelerations. Interestingly however, when comparing these peak responses to current risk for concussion thresholds [Zhang et al., 2004; Kleiven, 2007], the collision event fell well below the 25% risk for injury, putting into question the validity of using peak dynamic response variables across all impact events. Further analysis examining the characteristics of the dynamic loading curves revealed that each impact event could be uniquely characterized by different kinematic metrics. This information is likely more valuable in describing injury risk for that specific impact event over the current peak linear and peak rotational acceleration

thresholds. Finally, with onset and duration of symptoms following a concussive impact is often very unpredictable, the hope is that the results of this study will shed some light on the importance of the specific impact event and location within the brain of high strains. The third study demonstrated significant differences in strain field between the four impact events. Though not correlated to actual symptoms, it may help open the door to possibly relate symptoms to specific regions affected for specific impact conditions.

While current helmet manufacturers are expected to have their equipment pass regulatory standard testing, these tests typically are limited to one type of injury condition (Falling) [CSA, HECC, NOCSAE]. The information in this thesis shows that there is a need for protocols that require helmets to pass testing designed for several different impact events within that particular sport. Furthermore, future helmet design may consider development of liners that are better able to manage the specific dynamic response characteristics for the specific impact events commonly seen within that particular sport.

PART IV

Statement of Contributions

List of collaborators:

Marshall Kendall (PhD student) was involved in all aspects of this thesis, from conception to data collection, analysis and writing.

T. Blaine Hoshizaki was involved as the thesis supervisor and was involved in a supervisory role in all aspects of the thesis.

Michael D. Gilchrist provided finite element modeling support and was a committee member for the thesis.

Susan Brien was involved in the acquisition of subjects for the thesis from the Hull Hospital in Hull Canada. She also provided analysis of the medical imaging results.

Shawn Marshall was involved in the acquisition of subjects for the thesis from the General Hospital in Ottawa Canada.

Micheal Cusimano was involved in the acquisition of the MMA data used in this thesis.

Part V

Complete Reference list

Adams, J.H., Graham, D.I., and Gennarelli, T.A. (1981). Acceleration induced head injury in the monkey. II. Neuropathology. *Acta Neuropathol* 7, 26-28.

Adams, J.H., Graham, D.I., and Gennarelli, T.A. (1983) Head injury in man and experimental animals: neuropathology. *Acta Neurochirurgica Supplementum* 32: 15-30.

Advani, S., Powell, W., Huston, J., & Ojala, S. (1975). Human head impact response – experimental data and analytical simulations. In: *Proceedings of the IRCOBI Conference*, Berlin, Germany, 153-163.

Agel J, Dompier TP, Dick R, Marshall SW. (2007). Descriptive epidemiology of collegiate men's ice hockey injuries: National Collegiate Athletic Association Injury Surveillance System, 1988-1989 through 2003-2004. *J Athl Train*. Apr-Jun; 42(2):241-248.

Alves, W., Macciocchi, S.N. & Barth, J.T. (1993). Post concussive symptoms after uncomplicated mild head injury. *Journal of Head Trauma Rehabilitation*, 8, 48–59.

American Society for Testing and Materials (2007) ASTM F1045-07 Standard performance specification for ice hockey helmets. West Conshohocken PA, USA, 10 pages.

Arbogast, K.B., Margulies, S.S., (1998). Material characterization of the brainstem from oscillatory shear tests. *Journal of biomechanics* 31(9), 801-807.

Atha, J., Yeadon, M.R., Sandover, J. & Parsons, K.C. (1985). The damaging punch. *British medical journal*; 291: December:21-28.

Australian National Health, and Medical Research Council. (1994). *Football Injuries of the Head and Neck*. Australian Government Publishing Service, Canberra., pp. 78–79.

Bain, A.C., & Meaney, D.F. (2000). Tissue-level thresholds for axonal damage in an experimental model of central nervous system white matter injury. *Journal of Biomechanical Engineering*, 122(6), 615-622.

Bandak, F.A. (1994). On the mechanics of impact neurotrauma: a review and critical synthesis. *Journal of Neurotrauma*, 12(4), 635-649.

Bandak, F.A., and Eppinger, R.H. (1994). A three-dimensional FE analysis of the human brain under combined rotational and translational accelerations. *Proceedings 38th Stapp Car Crash Conference*, 145-163.

- Bayly, P.V., Cohen, T.S., Leister, E.P., Ajo, D., Leuthardt, E.C., & Genin, G.M. (2005). Deformation of the human brain induced by mild acceleration. *Journal of Neurotrauma*, 22(8), 845-856.
- Beckwith, J.G., Chu, J.J., & Greenwald, R.M. (2007). Validation of a non invasive system for measuring head acceleration for use during boxing competition. *Journal of Applied Biomechanics*, 23(3), 238-244.
- Benson, B.W., Rose, M.S. & Meeuwisse, W.H. (2002). The impact of face shield use on concussions in ice hockey: a multivariate analysis, *British Journal of Sports Medicine*, 36: 27-32.
- Benson, B.W., Meeuwisse, W.H., Rigos, J., Kang, J. & Burke, C.J. (2011). A prospective study of concussions among National Hockey League players during regular season games: the NHL-NHLPA concussion program. *Canadian Medical Association Journal*, 193(8), 905-911.
- Braddick, O.J., O'Brien, J.M., Wattam-Bell, J., Atkinson, J., Hartley, T. & Turner, R. (2001). Brain areas sensitive to coherent visual motion. *Perception*, 30; 61–72.
- Bradshaw, D.R.S., and Morfey, C.L. (2001). Pressure and shear responses in brain injury models. In 17th International Technical Conference on the Enhanced Safety of Vehicles, Amsterdam, Netherlands, 10pp.
- Brain injury awareness month (2010). Centers for Disease Control and Prevention, <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5908a8.htm>
- Broglio, S.P., Schnebel, B., Sosnoff, J.J., Shin, S., Feng, X., He, X., & Zimmerman, J. (2010). The biomechanical properties of concussion in high school football. *Medicine & Science in Sports & Exercise*, Post acceptance, 25 March, 2010.
- Canadian Standard Association (1997) CAN/CSA Z262.1-M90 (R1997) – Ice hockey helmets. Mississauga ON, Canada, 38 pages.
- Cantu RC. (1996). Head injuries in sport. *Br J Sports Med*. Dec; 30(4):289-296.
- Casson MD, Viano DC, Powell JW and Pellman EJ. (2010). Twelve years of National Football Leagues concussion data. *Sports Health*, vol. 2 (6): 471-483.
- Center for Disease Control and Prevention (CDC, National Center for Injury Prevention and Control. Report to Congress on mild traumatic brain injury in the United States: steps to prevent a serious public health problem. Atlanta (GA): Center for Disease Control and Prevention; 2003.
- Clauser, C.E., McConville, J.T. & Young, J.M. (1969). Weight, volume and center of mass of segments of the human body. Technical report AMRL-TR-69-70. Dayton, OH; Wright-Patterson Air Force Base.

- Creutzfeldt, O., Ojemann, G., Lettich, E. (1989a). Neuronal activity in human lateral temporal lobe. I. Responses to speech. *Exp. Brain Res.* 77:451-475.
- Creutzfeldt, O., Ojemann, G., Lettich, E. (1989b). Neuronal activity in human lateral temporal lobe. II. Responses to subjects own voice. *Exp. Brain Res.* 77:476-489.
- Cusimano MD, Taback NA, McFaull SR, Hodgins R, Bekele TM, et al. (2011). Effect of bodychecking on rate of injuries among minor hockey players. *Open Med*, 5: e57–64.
- Daneshvar, D.H., Nowinski, C.J., McKee, A., & Cantu, R.C. (2011). The epidemiology of sport-related concussion. *Clin Sports Med.* January; 30(1): 1–17. doi: 10.1016/j.csm.2010.08.006
- Darvish, K.K. & Crandall, J.R., (2001). Nonlinear viscoelastic effects in oscillatory shear deformation of brain tissue. *Medical Engineering and Physics* 23(9), 633-645.
- Dawodu S. et al. (2011). Traumatic Brain Injury (TBI) – Definition, Epidemiology, Pathophysiology.
[http://erairways.org/ERAirways/Trauma_files/Traumatic%20Brain%20Injury%20\(TBI\)%20-%20Definition,%20Epidemiology,%20Pathophysiology.pdf](http://erairways.org/ERAirways/Trauma_files/Traumatic%20Brain%20Injury%20(TBI)%20-%20Definition,%20Epidemiology,%20Pathophysiology.pdf)
- de Koning, J.J., de Grout, G., and van Ingen Schenau, G.J. (1992). Ice friction during speed skating. *Journal of Biomechanics*, 25(6), 565-571.
- Denny-Brown, D., & Russell, W.R. (1941). Experimental cerebral concussion. *Brain*, 64(2-3), 93-164.
- Deng Y. (1989). Anthropomorphic dummy neck modeling and injury considerations. *Accident Analysis & Prevention*, 21(1), February, Pages 85-100.
- Donaldson, L., Asbridge, M., Cusimano M.D. (2013). Bodychecking Rules and Concussion in Elite Hockey. *PLoS ONE* 8(7): e69122. doi:10.1371/journal.pone.0069122
- Donnelly, B.R. & Medige, J., (1997). Shear properties of human brain tissue. *Journal of Biomechanical Engineering* 119(4), 423-432.
- Doorly, M.C. and Gilchrist, M.D., (2006). The use of accident reconstruction for the analysis of traumatic brain injury due to head impacts arising from falls, *Computer Methods in Biomechanics and Biomedical Engineering*, 9(6), 371-377,.
- Doorly, M.C. (2007). Investigations into head injury criteria using numerical reconstruction of real life accident cases. PhD thesis, University College Dublin.
- Dault, M.C. & Dugas, C. (2002). Evaluation of a specific balance and coordination programme for individuals with a traumatic brain injury. *Brain Inj.*, Mar;16(3):231-44.

Eiband, A.M. (1959) Human tolerance to rapidly applied accelerations: a summary of the literature. NASA Memorandum 5-19-59E. NASA Lewis Research Center, Cleveland, OH.

El Sayed T., Mota A., Fraternali F. & Ortiz M. (2008). Biomechanics of traumatic brain injury. *Computer Methods in Applied Mechanics and Engineering*, Volume 197, Issues 51-52, 15 October 2008, Pages 4692-4701.

Emery, C.A., Kang, J., Shrier, I., Goulet, C., Hagel, B.E., et al. (2010). Risk of injury associated with body checking among youth ice hockey players. *JAMA*, 303: 2265–72.

Enouen, S. (1986). The development of experimental head impacts procedures for simulating pedestrian head injuries, proceedings of the 37th Stapp car crash conference, Oct. 27-29, San Diego, SAW report P-189, pp. 199-218.

European Committee for Standardization (2010). CEN/TC 226, EN 1317-2 – Road restraint systems – Part 2: Performance classes, impact test acceptance criteria and test methods for safety barriers including vehicle parapets. Brussels, Belgium.

Ewing, C., Thomas, D., Lustick, L., Becker, G., Willems, G., & Muzzy, W. (1975). The effect of the initial position of the head and neck to –Gx impact acceleration. In: *Proceedings of the 19th Stapp Car Crash Conference*, San Diego CA, USA. SAE Technical Paper No. 751157.

Finkelstein E, Corso P, Miller T et al. (2006). *The Incidence and Economic Burden of Injuries in the United States*. New York (NY): Oxford University Press.

Flik K, Lyman S, Marx RG. (2005). American collegiate men's ice hockey: an analysis of injuries. *American Journal of Sports Medicine*. Feb; 33(2):183-187.

Foreman SF & Hoshizaki TB (2011). The influence of headform geometry on the dynamic impact response of a Hybrid III headform. In: *Proceedings of the 23rd International Society of Biomechanics Congress*, Bruxelles, Belgium.

Gadd, C.W. (1966). Use of a weighted impulse criterion for estimating injury hazard. In *Proc. 10th Stapp Car Crash Conference*, Holloman Air Force Base, NM, pp. 164-174.

Galbraith, J.A., Thibault, L.E., & Matteson, D.R. (1993). Mechanical and electrical responses of the squid giant axon to simple elongation. *Journal of Biomechanical Engineering*, 115(1), 13-22.

Gennarelli, T.A., Thibault, L.E., and Ommaya, A. (1971). Comparison of translational and rotational accelerations in experimental cerebral concussion. In *Proceedings of the 15th Stapp car Crash Conference*.

Gennarelli, T.A., Thibault, L.E., and Ommaya, A. (1972). Pathophysiological responses to rotational and translational accelerations of the head. In *Proceedings of the 16th Stapp Car Crash Conference*, SAE paper No. 720970.

- Gennarelli, T.A., Adams, J.H., and Graham, D.I. (1981). Acceleration induced head injury in the monkey. I. The model, its mechanical and physiological correlates. *Acta Neuropathol*, 7, 23-25.
- Gennarelli, T.A. and Thibault, L.E. (1982). Biomechanics of acute subdural hematoma. *Journal of Trauma*, 22(8), 680-686.
- Gennarelli, T. (1983). Head injury in man and experimental animals: Clinical aspects. *Acta Neurochirurgica*, 32, 1-13.
- Gennarelli, T.A., Thibault, L.E., Adams, J.H., Graham, D.I, Thompson, C.J., and Marcincin, R.P. (1983). Diffuse axonal injury and traumatic coma in the primate. *Ann Neurol*, 12, 564-574.
- Gennarelli, T.A., Thibault, L.E., Tomel, G., Wiser, R., Graham, D., Adams, J. (1987). Directional dependence of axonal brain injury due to centroidal and non centroidal acceleration. In: *Proceedings of the Stapp Car Crash Conference*, New Orleans, LA, 49-53.
- Gennarelli, T. (1993). Mechanisms of brain injury. *Journal of emergency medicine*, 11, 5-11.
- Gerberich, S.G., Finke, R., Madden, M., Priest, J.D., Aamoth, G., Murray K. (1987). An epidemiological study of high school ice hockey injuries. *Childs Nerv Syst.*, 3(2):59-64.
- Gilchrist, M.D. and O'Donoghue, D. (2000). Simulation of the development of frontal head impact injury. *Computational Mechanics*, 26(3), 229-235.
- Gilchrist, M.D., O'Donoghue, D., and Horgan, T. (2001). A two-dimensional analysis of the biomechanics of frontal and occipital head impact injuries. *International Journal of Crashworthiness*, 6(2), 253-262.
- Goggio, A.F. (1941). The mechanism of contre-coup injury. *Journal of Neurology, Neurosurgery & Psychiatry* 4(1): 11-22.
- Goldsmith, W. (1981). Current controversies in the stipulation of head injury criteria. Letter to the editor. *Journal of Biomechanics*, 14, 883-884.
- Goldsmith, W., & Plunkett, J. (2004). A biomechanical analysis of the causes of traumatic brain injury in infants and children. *The American Journal of Forensic Medicine and Pathology*, 25(2), 89-100.
- Goodman D, Gaetz M, Meichenbaum D. (2001). Concussions in hockey: there is cause for concern. *Med Sci Sports Exerc.* Dec; 33(12):2004-2009.
- Gosh, H.H., Gooding, E., & Schneider, R.C. (1970). The lexan calvarium for the study of cerebral responses to acute trauma. *Journal of Trauma*, 10(5), 370-385.
- Gross, A.G. (1958). A new theory on the dynamics of brain concussion and brain injury. *Journal of Neurosurgery*, 29, 725-732.

Gulledge, J.K. & Dapena, J. (2007). A comparison of the reverse and power punches in oriental martial arts. *Journal of Sports Sciences*, 26:2, 189-196.

Gurdjian, E.S. and Lissner, H.R. (1944). Mechanisms of head injury as studied by the cathode ray oscilloscope: Preliminary report. *Journal of Neurology, Neurosurgery and Psychiatry*, 1, 393-399.

Gurdjian, E.S. & Webster, J.E., (1947). The mechanism and management of injuries to the head. *Journal of the American Medical Association* 134(13), 1072-1077.

Gurdjian, E.S., Webster, J.E., and Lissner, H.R. (1949) Studies on skull fracture with particular reference to engineering factors. *American Journal of Surgery* 78(5): 749-751.

Gurdjian, E.S., Lissner, H.R., Latimer, F.R., Haddad, B.F., and Webster, J.E. (1953). Quantitative determination of acceleration and intracranial pressure in experimental head injury: Preliminary report. *Neurology*, 3, 417-423.

Gurdjian, E.S., Webster, J.E. & Lissner, H.R. (1955) Observations on the mechanism of brain concussion, contusion, and laceration. *Surgery, Gynecology & Obstetrics* 101(6): 680-690.

Gurdjian, E.S. & Lissner, H.R. (1961). Photoelastic confirmation of the presence of shear strains at the craniospinal junction in closed head injury. *Journal of Neurosurgery*, 18(1), 58-60.

Gurdjian, E.S., Lissner, H.R., Evans, F.G., Patrick, M.S. & Hardy, W.G. (1961). Intracranial pressure and acceleration accompanying head impacts in human cadavers. *Surg. Gyn. Obst.* 113(4):185-190.

Gurdjian, E.S., Lissner, H.R., & Patrick, L.M. (1963). Concussion – Mechanism and pathology. In: *Proceedings of the 7th Stapp Car Crash Conference*, Los Angeles CA, USA, 470-482.

Gurdjian, E.S., Hodgson, V.R., Hardy, W.G., Patrick, L.M. & Lissner, H.R. (1964). Evaluation of the protective characteristics of helmets in sports. *The Journal of Trauma* 4(3): 309-324.

Gurdjian, E.S., Roberts, V.L. & Thomas, L.M. (1966). Tolerance curves of acceleration and intracranial pressure and protective index in experimental head injury. *The Journal of Trauma* 6(5): 600-604.

Gurdjian, E.S., Lissner, H.R. & Hodgson, V.R. (1966). Mechanisms of head injury. *Clinical Neurosurgery* 12: 112-128.

Gurdjian, E.S. & Gurdjian, E.S. (1967). Cerebral contusions: re-evaluation of the mechanism of their development. *The Journal of Trauma*, 16(1), 35-51.

Gurdjian, E.S., Hodgson, V.R., Thomas, L.M. & Patrick, L.M. (1968). Significance of relative movements of scalp, skull, and intracranial contents during impact injury to the head. *Journal of Neurosurgery*, 29(1), 70-72.

Gurdjian ES, Lange WA, Patrick LM, Thomas LM. In: *Impact injury and crash protection*. Thomas CC, editor. Springfield; Illinois: 1970.

Gurdjian, E.S. & Gurdjian, E.S. (1975). Re-evaluation of the biomechanics of blunt impact injury of the head. *Surgery, Gynecology and Obstetrics*, 140(6), 845-850.

Gurdjian, E.S. (1978). Movement of the brain and brain stem from impact induced linear and angular acceleration. *Trans. Am. Neurol. Assoc.*, 99, 248-249.

Guskiewicz, K.M., McCrea, M., Marshall, S.W., et al. (2003). Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study. *JAMA*;290:2549.

Hadad, B.F., Lissner, H.R., Webster, J.E., & Gurdjian, E.S. (1955). Experimental concussion – relation of acceleration to physiologic effects. *Neurology*, 5(11), 798-800.

Hagel BE, Marko J, Dryden D, Couperthwaite AB, Sommerfeldt J, et al. (2006). Effect of bodychecking on injury rates among minor ice hockey players. *CMAJ*, 175: 155–60.

Hardy, W.N., Khalil, T.B. & King, A. I. (1994). Literature review of head injury biomechanics. *International journal of impact engineering*, 15(4):561-586.

Hardy, W.N., Foster, C.D., Mason, M.J., Yang, K.H., King, A.I., and Tashman, S. (2001). Investigation of head injury mechanisms using neutral density technology and high-speed biplanar x-ray. *Stapp Car Crash Journal*, The Stapp Association, Ann Arbor, Michigan.

Hardy W.N., Mason M.J., Foster C.D., Shah C.S., Kopacz J.M., Yang K.H., and King A.I. (2007). A Study of the Response of the Human Cadaver Head to Impact, *Stapp Car Crash J.*, October; 51: 17–80.

Higgins, M., Halstead, P.D., Snyder-Mackler, L., & Barlow, D. (2007). Measurement of impact acceleration: mouthpiece accelerometer versus helmet accelerometer. *Journal of Athletic Training*, 42(1), 5-10.

Hockey Canada. Player registration [Data file]. Retrieved from http://www.hockeycanada.ca/index.php/ci_id/23952/la_id/1.htm, 2011. ASTM (2007)

Hockey Canada. Player registration [Data file]. Retrieved from http://www.hockeycanada.ca/index.php/ci_id/23952/la_id/1.htm, 2011.

- Hodgson, V.R., Gurdjian, E.S., & Thomas, L.M. (1966). Experimental skull deformation and brain displacement demonstrated by lash x-ray technique. *Journal of Neurosurgery*, 25(5), 549-552.
- Hodgson, V.R., Thomas, L.M. & Prasad, P. (1970). Testing the validity and limitations of the severity index. In Proc. 14th Stapp Car Crash Conference, Ann Arbor, MI, SAE Paper No. 700901.
- Hodgson, V.R. & Thomas, L.M. (1971). Breaking Strength of the human skull vs. impact surface curvature. National Technical Information Service, Springfield, Virginia, Report: DOT HS 800 583.
- Hodgson, V.R. (1975). National operating committee on standards for athletic equipment football helmet certification program. *Medicine and science in sport*, 7(3): 225-232.
- Hodgson, V.R., Thomas, L.M., and Khalil, T.B. (1983). The role of impact location in reversible cerebral concussion. In Proceedings of the 27th Stapp Car Crash Conference, SAE paper No. 831618.
- Holbourn, A.H.S. (1943) Mechanics of head injury. *The Lancet* 242(6267): 438-441.
- Holbourn, A.H.S. (1945). The mechanics of brain injuries. *British Medical Bulletin*, 3(6), 147-149.
- Honey, C.R. (1998). Brain injury in ice hockey. *Clin J Sport Med*, 8: 43-6.
- Horgan, T.J., & Gilchrist, M.D. (2003). The creation of three-dimensional finite element models for simulating head impact biomechanics. *International Journal of Crashworthiness*, 8(4), 353-366.
- Horgan, T.J., and Gilchrist, M.D. (2004). Influence of FE model variability in predicting brain motion and intracranial pressure changes in head impact simulations. *IJCrash*, 9 (4), 401-418.
- Horgan, T.J. (2005). A finite element model of the human head for use in the study of pedestrian accidents. PhD Thesis, University College Dublin, Dublin Ireland.
- Hrapko, M., van Dommelen, J.A.W., Peters, G.W.M., Wismans, J.S., (2006). The mechanical behaviour of brain tissue: Large strain response and constitutive modeling. *Biorheology* 43(5), 623-636.
- Hu, H., Nayfeh, A., and Rosenberg, W. S. (1998). Modeling of human brain movability during impact. In 5th International LS DYNA UsersConference.
- Hubbard, R. & McLeod, G. (1974). Definition and Development of a Crash Dummy Head, Eighteenth Stapp Car Crash Conference, December; SAE 741193.

Huisman, T.A., Schwamm, L.H., Schaefer, P.W., et al. (2004). Diffusion tensor imaging as potential biomarker of white matter injury in diffuse axonal injury. *AJNR*; 25:370–376.

Hutchison, M.G., Comper, P., Meeuwisse, W.H. & Echemendia, R.J. (2013). A systematic video analysis of National Hockey League (NHL) concussions, part I: who, when, where and what? *Br J Sports Med*, doi:10.1136/bjsports-2013-092234.

International Standard Organization (2006) ISO TC 83/SC 5N Working draft ISO/EN 10256 Head and face protection for use in ice hockey. Ottawa ON, Canada, 65 pages.

Jacobson, S. & Marcus, E.M. (2008). *Neuroanatomy for the neuroscientist*. New York, NY: Springer, Verlag.

Janowsky, J.S., Shimamura, A.P., Kritchevsky, M. & Squire, L.R. (1989). Cognitive impairment following frontal lobe damage and its relevance to human amnesia. *Behavioral Neuroscience*, 103(3), 548-560.

Jantzen, K.J., Anderson, B., Steinberg, F.L., et al. (2004). A prospective functional MR imaging study of mild traumatic brain injury in college football players. *AJNR*; 25:738–745.

Kang, H.S., Willinger, R., Diaw, B.M., and Chinn, B. (1997). Modeling of the human head under impact conditions: A parametric study. Proceedings, 41st Stapp Car Crash Conference. SAE paper No. 973338.

Kelly JP, Nichols JS, Filley CM, Lillehei KO, Rubinstein D, Kleinschmidt-DeMasters BK. (1991). Concussion in sports. Guidelines for the prevention of catastrophic outcome. *JAMA*. Nov; 266(20):2867-2869.

Kelly, J.P. & Rosenberg, J.H. (1998). The development of guidelines for the management of concussion in sports. *J Head Trauma Rehabil.*; 13:53–65.

Kendall, M., Post, A., Rousseau, P., Oeur, A., Gilchrist, M.D., and Hoshizaki, T.B. (2012). A comparison of dynamic impact response and brain deformation metrics of head impact reconstructions for three mechanisms of head injury in ice hockey. Proceedings of IRCOBI, Dublin, IRL, Sept 10-14.

King, A.I., Yang, K.H., Zhang, L. & Hardy, W.N. (2003). Is angular acceleration more injurious to the brain than linear acceleration?. *Frontiers of biomedical engineering; Proc. World congress of Chinese Biomedical Engineers*; Taipei, Taiwan.

Kleiven, S. (2002) *Finite Element Modeling of the human head*. PhD Dissertation, Royal Institute of Technology, Stockholm, Sweden.

- Kleiven, S., and Hardy, W.N. (2002). Correlation of an FE model of the human head with experiments on localized motion of the brain – Consequences for injury prediction. In: 46th Stapp Car Crash Journal, 123-144.
- Kleiven, S., and Von Holst, H. (2002). Consequences of size following trauma to the human head. *Journal of Biomechanics*, 35, 135-160.
- Kleiven, S. (2003). Influence of impact direction to the human head in prediction of subdural hematoma. *Journal of Neurotrauma*, 20(4), 365-379.
- Kleiven, S. (2006). Evaluation of head injury criteria using a finite element model validated against experiments on localized brain motion, intracerebral acceleration, and intracranial pressure. *International Journal of Crashworthiness* 11 (1), 65-79.
- Kleiven, S. (2007) Predictors for traumatic brain injuries evaluated through accident reconstructions. *Proc. of the 51st Stapp Car Crash Conference*, San Diego, CA, pp. 81-114.
- Kontos, A.P., Collins, M. & Russo, S.A. (2004). An Introduction to Sports Concussion for the Sport Psychology Consultant. *Journal of Applied Sport Psychology*, 16(3), 220-235.
- Kraus, J.K. & Nourjah, P. (1988). The epidemiology of mild, uncomplicated brain injury. *Journal of Trauma*, 28(12):1637-43.
- Langlois J, Kegler S, Butler J, Gotsch K, Johnson R, Reichard A, et al. (2003). Traumatic Brain Injury-Related Hospital Discharges: Results from a 14-State Surveillance System, 1997. *MMWR Surveill Summ* 52 (No. SS-4):1–20.
- Langlois, J.A., Rutland-Brown, W., Wald, M.M. (2006). The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil*, 21:375–8.
- Lee, M.C. & Hault, R.C., (1989). Insensitivity of tensile failure properties of human bridging veins to strain rate: implications in biomechanics of subdural hematoma. *Journal of Biomechanics* 22(6-7), 537-542.
- Lescohier, I., DiScala, C. (1993). Blunt trauma in children: causes and outcomes of head versus intracranial injury. *Pediatrics*, 91(4):721-5.
- Lowenhielm, P. (1975). Mathematical simulation of gliding contusions. *Journal of Biomechanics*, 8, p. 351-356.
- Lubock, P. and Goldsmith, W. (1980). Experimental cavitation studies in a model head-neck system. *Journal of Biomechanics*, 13, 1041-1052.

- Luerssen, T.G., Klauber, M.R. & Marshall L.F. (1993). Outcome from head injury related to patient's age: A longitudinal prospective study of adult and pediatric head injury. *Journal of Neurosurgery*, 68(3):409-16.
- Luppino, G. & Rizzolatti, G. (2000). The organization of the frontal motor cortex. *News Physiol. Sci.*, Vol. 15, October; 219-224.
- Mao, H., and Yang, K.H. (2010) Investigation of brain contusion mechanism and threshold by combining finite element analysis with in vivo histology data. *International Journal for Numerical Methods in Biomedical Engineering*, n/a. doi: 10.1002/cnm.1403.
- Marar, M., McIlvain, N.M., Fields, S. & Comstock, R.D. (2012). Epidemiology of concussions among United States high school athletes in 20 sports.
- Marchie A, Cusimano MD. (2003). Bodychecking and concussions in ice hockey: Should our youth pay the price? *CMAJ*. Jul; 169(2):124-128.
- Margulies, S. S., Thibault, L. E., & Gennarelli, T.A. (1990). Physical model simulations of brain injury in the primate. *Journal of Biomechanics*, 23(8), 823-836.
- Margulies S & Thibault K (2000). Infant Skull and Suture Properties: Measurements and Implications for Mechanisms of Pediatric Brain Injury, *J. Biomech. Eng.*, August 2000, Volume 122, Issue 4, 364 (8 pages).
- Marieb, E.N. (1998). *Human anatomy and physiology*, 4th ed. Benjamin/Cummings Science Publishing, California.
- Marino, G.W. (1977). Kinematics of ice skating at different velocities. *Research Quarterly*, 48, 93-97.
- Marino, G.W. & Drouin, D. (2000). Effects of fatigue on forward, maximum velocity in ice hockey skating, *Proceedings of 18th ISBS Conference*, Hong Kong.
- Marjoux, D., Baumgartner, D., Deck, C., and Willinger, R. (2008) Head injury prediction capability of the HIC, HIP, SIMon and ULP criteria. *Accident Analysis and Prevention* 40(3): 1135-1148.
- McClincy, M.P., Lovell, M.R., Pardini, J., Collins, M.W. & Spor, M.K. (2006). Recovery from sports concussion in high school and collegiate athletes, Vol. 20, No. 1 , Pages 33-39 doi:10.1080/02699050500309817 .McCrory, P. (1998). Neurological injuries in Australian and rugby football. In: B. D. Jordan, P. Tsaris, and R. F. Warren (Eds.) *Sports Neurology*, 2nd Ed. Philadelphia: Lippincott-Raven, pp. 441–451.

McCrory P, Meeuwisse W, Aubry M, et al. Consensus statement on Concussion in Sport – 4th International Conference on Concussion in Sport held in Zurich, November 2012. *Clin J Sport Med* 2013;23(2):89-117.

McElhaney, J. (1976) Head Injury Criteria. *Mechanics of Composite Materials* 12(3): 411-429.

McKee, A.C., Cantu, R.C., Nowinski, C.J., et al. (July 2009). Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. *J. Neuropathol. Exp. Neurol.* 68 (7): 709–35. doi:10.1097/NEN.0b013e3181a9d503. PMC 2945234. PMID 19535999.

McIntosh, A.S., McCrory, P. & Comerford, J. (2000). The dynamics of concussive head impacts in rugby and Australian rules football. *Medecine & Science in sports & Exercise* 32(12):1980-1984.

Meaney, D., Smith, D.H., Shreiber, D.I., Bain, A.C., Miller, R.T., Ross, D.T. & Gennarelli, T.A. (1995). Biomechanical analysis of experimental diffuse axonal injury. *Journal of neurotrauma*, 12(4):689-694.

Meaney, D.F. and Smith, D.H. (2011). Biomechanics of concussion. *Clinical Sports Medicine*, 30(1), 19-31.

Meehan, W.P. & Micheli, L.J. (2011). Concussion results in deficits in neurocognitive functioning. *Clin Sports Med.*; 30:28–8.

Mendis, K., Stalnaker, R., and Advani, S. (1995). A constitutive relationship for large deformation finite element modeling of brain tissue. *Journal of Biomechanical Engineering*, 117(4), 279-285.

Mertz H. (1985). Biofidelity of the Hybrid III Head, Society of Automotive Engineers Inc., 851245 : 1-9.

Miller, R., Margulies, S., Leoni, M., Nonaka, M., Chen, Z., Smith, D., and Meaney, D. (1998). Finite element modeling approaches for predicting injury in an experimental model of severe diffuse axonal injury. In *Proceedings of the 42nd Stapp Car Crash Conference*, SAE paper No. 983154.

Monson, K.L., Goldsmith, W., Barbaro, N.M., Manle, G.T., (2003). Axial mechanical properties of fresh human cerebral blood vessels. *Journal of Biomedical Engineering* 125(2), 288-294.

Mueller, F.O. & Cantu, R.C. (2009). *Catastrophic Football Injuries Annual Report*. National Center for Catastrophic Injury Research.

Nahum, A.M., Smith, R., and Ward, C.C. (1977). Intracranial pressure dynamics during head impact. Proceedings 21st Stapp Car Crash Conference. SAE paper No. 770922.

National Operating Committee on Standards for Athletic Equipment (2007) Standard drop test method and equipment used in the evaluating the performance characteristics of protective headgear. Washington DC, USA, 26 pages.

National Highway Traffic Safety Administration (2001) Standard No. 218 FMVSS 218 – 49 Motorcycle helmets. Washington DC, USA, 10 pages.

Newman, J.A. (1980). Head injury criteria in automotive crash testing. In Proc. 24th Stapp Car Crash Conference, Troy, MI, pp. 703-747.

Newman, J.A. (1986). A generalized model for brain injury threshold (GAMBIT). In Proc. International Conference on the Biomechanics of Impacts, Zurich, Switzerland.

Newman, J.A. (1998). Kinematics of head injury. In *Frontiers in Head and Neck Trauma: Clinical and Biomechanical*, ed. N. Yoganandan, F.A. Pintar, S.J. Larson and A. Sances Jr., pp. 200-214. IOS Press, Amsterdam, Netherlands.

Newman J, Barr C, Beusenberg M, et al. (2000a). A new biomechanical assessment of mild traumatic brain injury: Part 2 – results and conclusions. In: International IRCOBI Conference on the Biomechanics of Impact, Montpellier, France, pp. 223–233.

Newman, J.A., Shewchenko, N., and Welbourne, E. (2000b). A proposed new biomechanical head injury assessment function: The maximum power index. In Proc. 44th Stapp Car Crash Conference, Atlanta, GA, pp. 215-247.

Newman, J.A. (2002). *Biomechanics of Head Trauma: Head Protection*. Accident Injury. Springer New York p-303-323.

Newman, J.A., Beusenberg, M.C., Shewchenko, N., Withnall, C., & Fournier, E. (2005). Verification of biomechanical methods employed in a comprehensive study of mild traumatic brain injury and the effectiveness of American football helmets. *Journal of Biomechanics*, 38(7), 1469-1481.

Nusholtz, G.S., Lux, P., Kaiker, P.S., & Janicki, M.A. (1984). Head impact response – skull deformation and angular accelerations. In: *Proceedings of the 28th Stapp Car Crash Conference*, Chicago IL, USA, 41-74.

Nuscholtz, G.S, Kaiker, P.S. and Gould, W.S. (1987). Two factors critical in the pressure response of the impacted head. *Aviation, Space and Environmental Medicine*, 58(12), 1157-1161.

O'Donoghue, D. (1999). Biomechanics of frontal and occipital head impact injuries: A plane strain simulation of coup and contrecoup contusion. Master's thesis, University College Dublin, Belfield, Dublin 4, Ireland.

Oeur, A., Post, A., Hoshizaki, T.B., and Gilchrist, M. (2011). The Correlation Between Brain Deformation Metrics and Acceleration Using a Linear and Non-Linear Impact Protocol. XXIIIrd International Society of Biomechanics Congress, Brussels, Belgium.

Ommaya, A.K., Hirsch, A.E., & Martinez, J.L. (1966). The role of whiplash in cerebral concussion. In: Proceedings of the 10th Stapp Car Crash Conference, New Mexico, USA, 314-324.

Ommaya A.K., Yarnell P., Hirsch A.E. and Harris E.H. (1967). Scaling of experimental data on cerebral concussion in sub-human primates to concussion threshold for man, Stapp Car Crash Conference, Oct. 10-11, report 2640.

Ommaya, A.K. and Hirsch, A.E. (1971). Tolerance of cerebral concussion from head impact and whiplash in primates. *Journal of Biomechanics*, 4, 13-22.

Ommaya, A.K., Grubb, R.L., and Naumann, R.A. (1971). Coup and contrecoup injury: Observations on the mechanics of visible brain injuries in the rhesus monkey. *Journal of Neurosurgery*, 35, 503-516.

Ommaya, A.K., & Gennarelli, T.A. (1974). Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations on blunt head injuries. *Brain*, 97(4), 633-654.

Ono, K., Kikuchi, M., Nakamura, H., Kobayashi, H., and Nakamura, N. (1980). Human head tolerance to sagittal impact reliable estimation deduced from experimental head injury subhuman primates and human cadaver skull. In Proceedings of the 24th STAPP Car Crash Conference, SAE paper No. 801303.

Ono, K. (1998). Human head impact tolerance. In *Frontiers in Head and Neck Trauma: Clinical and Biomechanical*, ed. N. Yoganandan, F.A. Pintar, S.J. Larson and A. Sances Jr., pp. 183-199. IOS Press, Amsterdam, Netherlands.

Padagaonkar, A.J., Krieger, K.W., & King, A.I. (1975). Measurement of angular acceleration of a rigid body using linear accelerometers. *Journal of Applied Mechanics*, 42, 552-556.

Parker, R. (2012). *Concussive Brain Trauma: Neurobehavioral Impairment & Maladaptation*, 2nd edition, CRC press, Taylor & Francis group.

Pashby, T., Carson, J.D., Ordogh, D., Johnston, K.M., Tator, C.H. & Mueller, F.O. (2001). Eliminate head-checking in ice hockey. *Clin J Sport Med*. Oct; 11(4):211-213.

Pellman, E.J., Viano, D.C., Tucker, A.M., Casson, I.R. & Waeckerle, J.F. (2003). Concussion in professional football: reconstruction of game impacts and injuries. *Neurosurgery*. Oct; 53(4):

Post A, Hoshizaki TB and Gilchrist M. (2010). Evaluation of American football helmet performance using finite element modelling. In: VIIIth Conference of the International Sports Engineering Association, Vienna, Austria.

Post A, Oeur A, Hoshizaki TB and Gilchrist MD. (2011). Examination of the relationship of peak linear and angular acceleration to brain deformation metrics in hockey helmet impacts. *Comput Meth Biomech Biomed Eng*: in press.

Post, A. & Hoshizaki, T.B. (2012). Mechanical properties describing brain impact injuries: A review. *Trauma* 14 (4), 327-349.

Post A, Hoshizaki TB, Gilchrist MD and Brien S. (2012) Analysis of the influence of independent variables used for reconstruction of a traumatic brain injury incident. *Proceedings of the Institution of Mechanical Engineers, Part P: Journal of Sports Engineering and Technology* 226:209 originally published online 14 March 2012. DOI: 10.1177/1754337112436629.

Post, A., Hoshizaki, T.B., Gilchrist, M.D., (2012a). Finite element analysis of the effect of loading curve shape on brain injury predictors. *Journal of Biomechanics* 45, 679-683.

Post, A., Walsh, E.S., Hoshizaki, T.B., Gilchrist, M.D., (2012b). Analysis of loading curve characteristics on the production of brain deformation metrics. *Journal of Sports Engineering and Technology* 226(3/4), 200-207.

Post, A. (2013). The influence of dynamic response characteristics on traumatic brain injury. PhD thesis, University of Ottawa, Canada.

Powell, J.W. (1998). The evolution of ice hockey related injuries in the NHL. In: Biasca N, Montag W-D, Gerber C, editors. *Safety in ice hockey: IIHF International Symposium on Medicine and Science in Ice Hockey on the occasion of the 8th Ice Hockey World Championship*. Zurich: IIHF. 19–25.

Powell JW. (2001). Cerebral Concussion: Causes, Effects, and Risks in Sports. *J Athl Train*. Sep; 36(3):307-311.

Practice parameter: the management of concussion in sports (summary statement) (1997). Report of the Quality Standards Subcommittee. *Neurology* ; 48: 581–585.

Prange, M.T., Meaney, D.F., and Margulies, S.S. (2002). Defining brain mechanical properties: Effects of region, direction and species. In: *Proceedings 44th Stapp Car Crash Conference*.

Prasad, P., and Mertz, H.J. (1985). The position of the United States Delegation to the ISO working group 6 on the use of HIC in the automotive environment. *SAE Paper No. 851246*.

Prasad, P., Melvin, J.W., Hulke, D.F., King, A.I., and Nyquist, G.W. (1985) Head. In Review of Biomechanical Impact Response and Injury in the Automotive Environment, ed. J.W. Melvin and K Weber, pp. 1-43. (Contract No. DTNH22-83-C-07005) National Highway Traffic Safety Administration, Washington, DC.

Price, C.J. (2000). The anatomy of language: contributions from functional. neuroimaging. *Journal of anatomy*. 197(3): 335-359.

Pudenz, R.H., & Shelden, C.H. (1946). The lucite calvarium – A method for direct observation of the brain. II. Cranial trauma and brain movement. *Journal of Neurosurgery*, 3(6), 487-505.

Purves D, Augustine WJ, Fitzpatrick D, Lawrence CK, LaMantia AS, McNamara JO, Williams SM eds. *Neuroscience*, 2nd edition, Sinauer Associates, Sunderland (MA), 2001.

Rashid, B., Destrade, M., Gilchrist, M.D., (2012a). Mechanical characterization of brain tissue in compression at dynamic strain rates. *Journal of the Mechanical Behavior of Biomedical Materials* 10, 23-38.

Rashid, B., Destrade, M., Gilchrist, M.D., (2012b). Mechanical characterization of brain tissue in tension at dynamic strain rates. *Journal of the Mechanical Behavior of Biomedical Materials*, in press.

Ropper, A.H. & Samuels, M.A., Adams and Victor's principles of neurology. New York, NY: McGraw Hill Medical, 2009.

Ruan, J.S., Khalil, T.B., and King, A.I. (1993). Finite element modeling of direct head impact. *Proceedings of the 37th Stapp Car Crash Conference*, San Antonio, Texas, USA, SAE 933114.

Ruan, J. (1994). *Impact Biomechanics of head injury by mathematical modeling*, PhD thesis, Wayne State University.

SB Nation (2010). <http://www.pensionplanpuppets.com/2010/10/15/1751273/sizing-up-the-nhl-by-height-weight-and-age> .

Schmitt, K.-U., Niederer, P., Muser, M., and Walz, F. (2007). *Trauma biomechanics: Accidental injury in traffic and sports* – 2nd edition, pp. 55-81. Springer, New York.

Seward, H., Orchard, J., Hazard, H., and Collinson, D. (1993). Football injuries in Australia at the elite level. *Med. J. Aust.*, 159: 298 –301.

Shojaati, M. (2003). Correlation between injury risk and impact severity index ASI. In *Proc. 3rd Swiss Transport Research Conference*, Monte Verità, Switzerland, 10 pages.

Shreiber, D.I., Bain, A.C., & Meaney, D.F. (1997). In vivo thresholds for mechanical injury to the blood-brain barrier. In: Proceedings of the 41st Stapp Car Crash Conference, Lake Buena Vista FL, USA, 177-1990.

Shuck, L. & Advani, S. (1972). Rheological response of human brain tissue in shear, *Journal of Basic Engineering*, 905-911.

Shugar, T. and Katona, M. (1975). Development of a finite element head injury model. ASCE, *Journal of Engineering mechanics*, EM3 (E101/173), 223-239.

Shumway-Cook A & Olmscheid R. (1990). A systems analysis of postural dyscontrol in traumatically brain-injured patients. *J Head Trauma Rehabil.*;5:51-62. 39

Silva, V.D. (2006). *Mechanics and Strength of Materials*. New York, NY, Springer Berlin Heidelberg.

Smith, T.A., Bishop, P.J., & Wells, R.P. (1988). Three dimensional analysis of linear and angular accelerations of the head experienced in boxing. In: Proceedings of the IRCOBI Conference on the Biomechanics of Impact, Gurdjian, E.S., Roberts, V.L., and Thomas, L.M. (1966). Tolerance curves of acceleration and intracranial pressure and protective index in experimental head injury. *Journal of Trauma*, 6(5), 600-604.

Sosin, D.M., Sniezek, J.E., & Thurman, D.J. (1996) Incidence of Mild and Moderate Brain Injury in the United States, 1991. *Brain Injury*, 10(1): 47-54.

Stalhammar, D. (1975a). Experimental brain damage from fluid pressures due to impact acceleration – 1. Design of experimental procedure. *Acta. Neurol. Scand.*, 52 7-26.

Stalhammar, D. (1975b). Experimental brain damage from fluid pressures due to impact acceleration – 2. Pathophysiological observations. *Acta. Neurol. Scand.*, 52, 27-37.

Stalhammar, D. and Olsson, Y. (1975). Experimental brain damage from fluid pressures due to impact acceleration – 3. Morphological observations. *Acta. Neurol. Scand.*, 52, 38-55.

Stalnaker, R., Melvin, J., Nusholtz, G., Alem, N., and Benson, J. (1977). Head impact response. In Proc. of the 21st Stapp Car Crash Conf., SAE paper no. 770921.

Stapp, J.P. (1970). Voluntary human tolerance levels. In *Impact Injury and Crash Protection*, ed. E.S. Gurdjian, W. Lange, L.M. Patrick and L.M. Thomas, pp. 308-349. Charles C Thomas, Springfield, IL.

Suh, W., Yang, W., and McElhaney, J.H. (1972). Rarefaction of liquids in a spherical shell due to local radial loads with application to brain damage. *Journal of Biomechanics*, 5, 181-189.

Thibault, L.E. & Gennarelli, T.A. (1985). Biomechanics of diffuse brain injuries. Proc. 29th Stapp car crash conference, SAE paper No. 856022.

Thibault, L.E. (1993). Brain injury from the macro to the micro level and back again: what have we learned to date?. In: Proceedings of the IRCOBI Conference, Eindhoven, The Netherlands, 3-25.

Thomas, L.M., Roberts, V.L. and Gurdjian, E.S. (1966) Experimental intracranial pressure gradients in the human skull. *Journal of Neurology, Neurosurgery & Psychiatry* 29(5): 404-411.

Thomas, L.M., Roberts, V.L., and Gurdjian, E.S. (1967). Impact-induced pressure gradients along three orthogonal axes in the human skull. *Journal of Neurosurgery*, 26(3), 316-321.

Thurman, D. J., Branche, C. M. & Snizek, J. E. (1998). The epidemiology of sports-related traumatic brain injuries in the United States: recent developments. *Journal of Head Trauma Rehabilitation*, vol. 13, no. 2, pp. 1-8.

Trosseille, X., Tarrière, C., Lavaste, F., Guillon, F., and Domont, A. (1992). Development of a F.E.M. of the human head according to a specific test protocol. Proceedings of the 36th Stapp Car Crash Conference, Seattle, Washington, USA, SAE 922527.

Unterharnscheidt, F.J. & Sellier K. (1966). Mechanics and pathomorphology of non-penetrating brain injuries. Reprinted from *Excerpta Medica international congress series*, No. 110, pp. 93-103.

Unterharnscheidt, F.J., and Higgins, L.S. (1969) Traumatic lesions of brain and spinal cord due to nondeforming angular acceleration of the head. *Texas Reports on Biology and Medicine* 27(1): 127-166.

Unterharnscheidt, F.J. (1971). Translational versus rotational acceleration-animal experiments with measured input, SAE International, Paper Number: 710880, DOI: 10.4271/710880.

van den Bosch, H. 2006. Crash helmet testing and design specifications, PhD thesis, Library Eindhoven University of Technology, Sept.

Van Gijn, J. (2009). Cerebral involvement in head injury. A study based on the examination of two hundred cases. By W. Ritchie Russell, MD. *Brain* 1932; 55: 549-603. *Brain* 132(3): 565-567.

Versace, J. (1971). A review of the severity index. Proc. 15th Stapp Car Crash Conference, Coronado, CA, pp. 771-796.

Viano, D.C. (1988) Biomechanics of head injury: toward a theory linking head dynamics motion, brain tissue deformation and neural trauma. Proc. 32nd Stapp Car Crash Conference, Atlanta GA, pp. 1-20.

- Viano, D., King, A., Melvin, J. & Weber, K. (1989). Injury biomechanics research: An essential element in prevention of trauma. *Journal of biomechanics*, 22(5), 403-417.
- Viano, D.C., Casson, I.R., Pellman, E.J., Zhang, L., King, A.I., & Yang, K.H. (2005). Concussion in professional football: Brain responses by finite element analysis: Part 9. *Neurosurgery*, 57(5), 891-915.
- Viano, D.C., Casson, I.R., Pellman, E.J., Bir, C.A., Zhang, L., Sherman, D.C. and Boitano, M.A. (2005). Concussion in professional football: comparison with boxing head impacts—part 10. *Neurosurgery*, Dec; 57(6): 1154-72.
- Victorian Football League Medical Officer's Association. (1986). A three-year survey of Victorian football league injuries, H. Seward and J. Patrick Melbourne, 11–19.
- Voo, L., Kumaresan, S., Pintar, F.A., Yoganandan, N., & Sances Jr., A. (1996). Finite-elements models of the human head. *Medical & Biological Engineering & Computing*, 34(5), 375-381.
- Walilko, T. J., Viano, D. C., & Bir, C. A. (2005). Biomechanics of the head for Olympic boxer punches to the face. *British Journal of Sports Medicine*, 39(10), 710-719.
- Walsh, E.S. & Hoshizaki, T.B. (2010). Sensitivity analysis of a Hybrid III head- and neckform to impact angle variations. In: 8th Conference of the International Sports Engineering Association, Vienna, Austria.
- Ward, C. and Thompson, R.B. (1975). The development of a detailed finite element brain model. *Proceedings of the 19th Stapp Car Crash Conference*, San Diego, California, USA, SAE 751163.
- Ward, C.C., and Nahum, A. (1979). Correlation between brain injury and intracranial pressure in experimental head impacts. *Proceedings of the international Conf. on Biomechanics of Trauma*, IRCOBI, Gotenborg, Sweden.
- Ward, C.C., Chan, M., and Nahum, A.M. (1980). Intracranial pressure – a brain injury criterion. *Proc. 24th Stapp Car Crash Conference*, Troy, MI, pp. 347-360.
- Ward, C.C., & Nagendra, G.K. (1985). Mathematical models: Animal and human models. In *The Biomechanics of Trauma*, ed. A.M. Nahum and J. Melvin, pp.77-100. Appleton-Century-Croft, Norwalk, CT.
- Wennberg, R.A. & Tator C.H. (2008). Concussion incidence and time lost from play in the NHL during the past ten years, *Canadian Journal of Neurological Science*; 35, 647-651.
- Willinger, R., Kopp, C., and Cesari, D. (1992a). New concept of contrecoup lesion mechanism; modal analysis of a finite element model of the head. In *Proc. of the 1992 International IRCOBI Conf. on the Biomechanics of Impacts*, pp. 283-297.

Willinger, R., Ryan, G., McLean, A., and Kopp, C. (1992b). Mechanisms of brain injury related to mathematical modelling and epidemiological data. In Proc. of the 1992 International IRCOBI Conf. on the Biomechanics of Impacts, pp. 179-192.

Willinger, R., Taleb, L., Viguier, P., and Kopp, C. (1994). Rotation translation duality in head trauma? perceptive and objective evidence. In Proc. of the 1994 International IRCOBI Conf. on the Biomechanics of Impacts, pp. 63-76.

Willinger R, Ryan GA, McLean AJ, et al. (1994). Mechanisms of brain injury related to mathematical modelling and epidemiological data. *Accident Analysis and Prevention*, 26:767.

Willinger, R., Taled, L. & Kopp, C. (1995). Modal and temporal analysis of head mathematical models, *Journal of Neurotrauma*, 12, 743-754.

Willinger, R., Taled, L., and Pradoura, P. (1995). Head biomechanics from finite element model to the physical model. *Proceedings of the 1995 IRCOBI Conference*.

Willinger R, Baumgartner D, Chinn B and Schuller E. (2001). New Dummy head prototype: development, validation and injury criteria, *International Journal of Crashworthiness*, 6(3):281-293.

Willinger, R., & Baumgartner, D. (2003). Human head tolerance limits to specific injury mechanisms. *International Journal of Crashworthiness*, 8(6), 605-617.

Willinger, R., and Baumgartner, D. (2003b). Numerical and physical modelling of the human head under impact – towards new injury criteria. *Int. J. Vehicle Design*, Vol 32, 94-115.

Wright, P., Stamatakis, E.A. & Tyler, L.K. (2012). Differentiating hemispheric contributions to syntax and semantics in patients with left-hemisphere lesions. *Journal of Neuroscience*, 32(24), 8149-8157.

Yang, Y., Raine, A., Colletti, P., Toga, A.W. & Narr, K.L. (2009). Abnormal temporal and prefrontal cortical gray matter thinning in psychopaths. *Molecular Psychiatry*, 14, 561–562; doi:10.1038/mp.2009.12

Yoganandan ,N., Zhang, J., Pintar, F.A., King Liu, Y. (2006). Lightweight low-profile nine-accelerometer package to obtain head angular accelerations in short-duration impacts. *Journal of Biomechanics*, 39(1), 1347-1354.

Yogandandan N, Li J, Zhang J, Pintar FA, Gennarelli TA. (2008). Influence of angular acceleration-deceleration pulse shapes on regional brain strains. *Journal of biomechanics*, 41, 2253-2262.

Zazryn, T.R., Finch, C.F. & McCrory, P. (2003). A 16 years study of injuries to professional boxers in the state of Victoria, Australia. *Br J Sports Med*; 37:321-24.

Zhang, L., King, H., and King, A. (2001a). Comparison of brain responses between frontal and lateral impacts by finite element modeling. *Journal of Neurotrauma*, 18(1), 21-30.

Zhang, L., Yang, K., Dwarampudi, R., Omori, K., Li, T., Chang, K., Hardy, W., Khalil, T., and King, A. (2001b). Recent advances in brain injury research: A new human head model development and validation. *Stapp Car Crash Journal*, 45, 369-393.

Zhang, L., Yang, K. H., & King, A. I. (2004). A proposed injury threshold for mild traumatic brain injury. *Journal of Biomechanical Engineering*, 126(2), 226-236.

Zhou, C., Khalil, T.B., King, A.I. (1995). A new model for comparing responses of the homogeneous and inhomogeneous human brain. In: *Proceedings of the 39th Stapp Car Crash Conference*, 121-136.

APPENDIX A

Examples of concussive injury cases from the four (4) specific impact events in this study

Collisions Impact Event

Case #13

Test ID: [NHL-34]

Date: March 27, 2014 + May 27, 2014

Event details

Mechanism: Collision

Striking athlete: [1.93 m; 10 kg]

Helmet: Reebok R7 + Oakley Straight Pro Visor

Outcome:

Reported concussion

3 Previous Concussions

Recovery time: End of season / Resumed play at start of the following season

Video Analysis

Impacting surface: Shoulder

Impact velocity: 10.17 m/s

Impact location: L1 - E

Impact orientation: Rotation: 51 degrees

Elevation (Impact):NA

Extra:

Velocity calculations

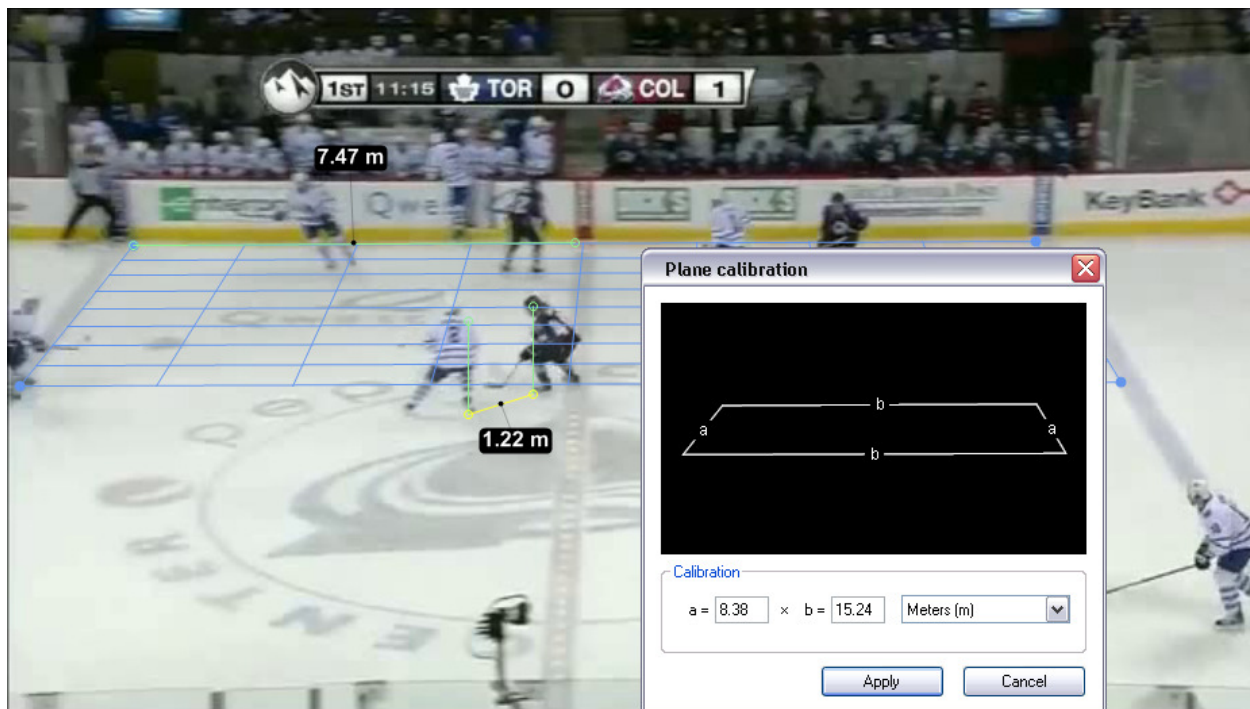


Fig 1 Location of players 3 frames prior to impact

Frame rate: 25 fps

Frames: 3

Reference distance: length: 15.24 m

width: 8.38 m

Time to impact: 0.12 s

Distance travelled: 1.22 m

Velocity: 10.17 m/s

Impact location

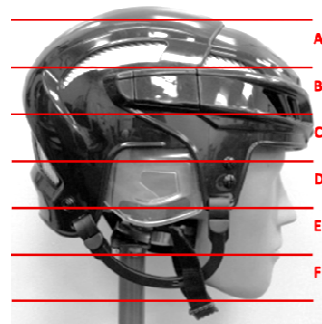
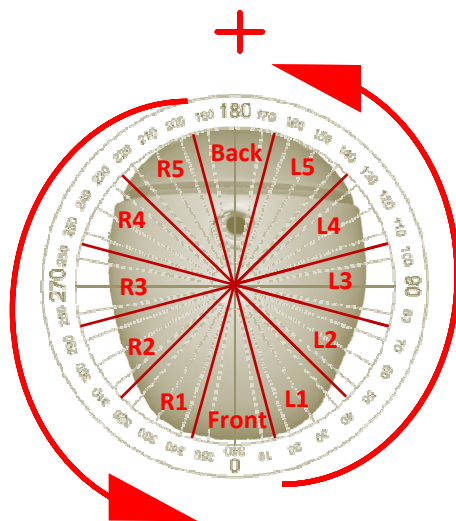


Fig 3 Reference coordinates

Impact location: L1-E

Impact orientation

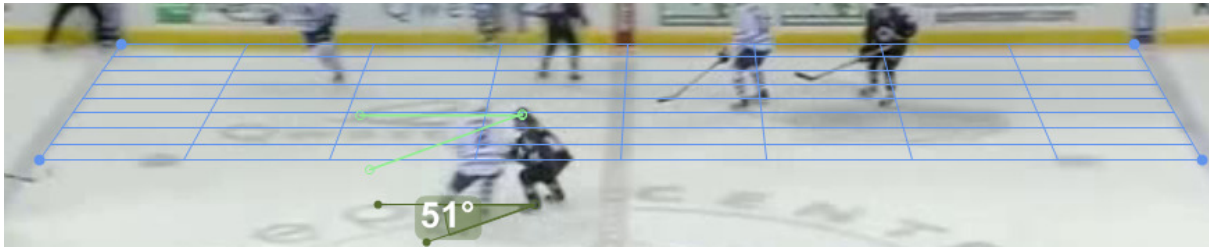


Fig 4 Impact orientation



Fig 5 Impact elevation

Impact orientation:

Rotation: 51 degrees

Elevation (Head): None

Elevation (Impact): None



Helmeted Fall Impact Event

Case #3

Test ID: [NHL54]

Date: April 30, 2014

Event details

Mechanism: Fall

Helmet: Bauer 4500 [VN]

Outcome:

Reported concussion

0 Previous Concussions

Recovery time: 6 games / 17 days

Video Analysis

Impacting surface: Ice

Impact velocity: 5.2 m/s

Impact location: R5 - B

Extra:

Velocity calculations

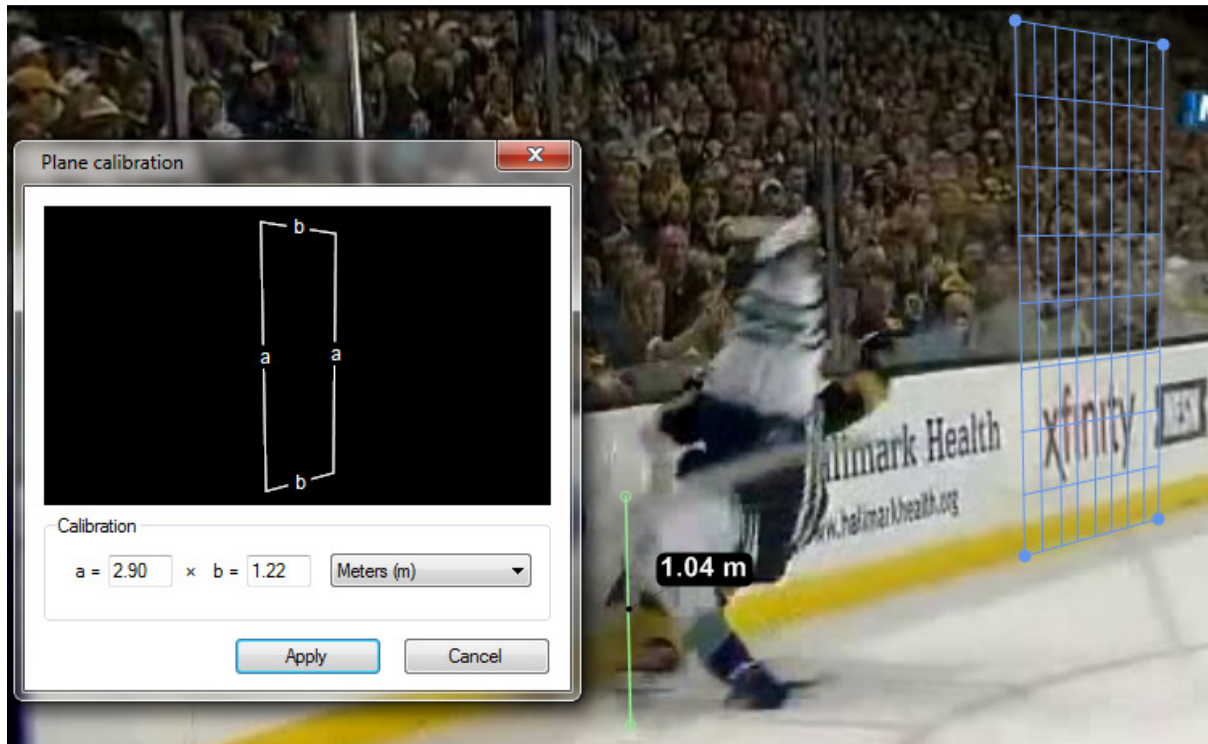


Fig 1 Location of players 5 frames prior to impact

Frame rate:	25 fps
Frames:	5
Reference distance:	length: 2.9 m width: 1.22 m
Time to impact:	0.2 s
Distance travelled:	1.04 m
Velocity:	5.2 m/s

Impact location

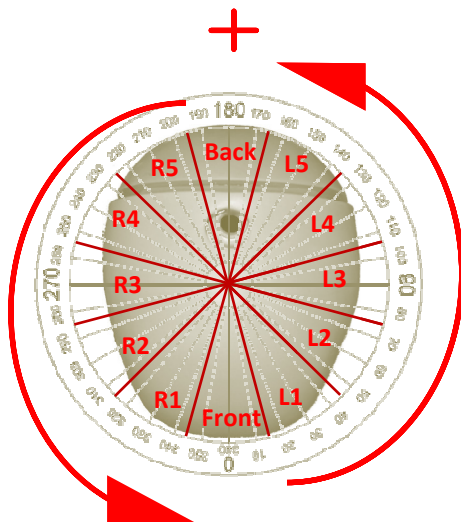
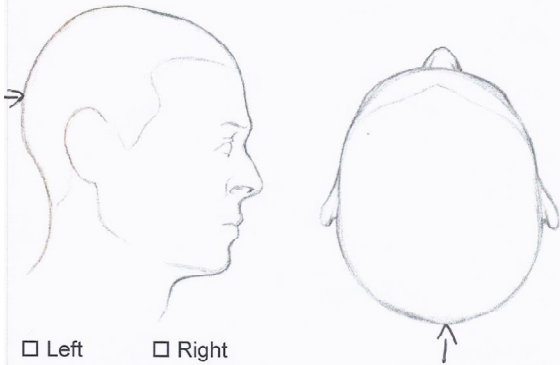


Fig 3 Reference coordinates

Impact location: R5-B

Unhelmeted Fall Impact Event

Case # 10

Neurotraumatic Injury Report	
Patient Information	
Age: <u>37</u>	Gender: ☐ Male
Weight: _____ kg	☒ Female
Height: _____ cm	Ethnicity: _____
Previous Head Injury: ☐ No ☐ Yes – Number _____	
Pre-existing Medical Conditions	
<u>Transcatheter ischemic attacks</u>	
Diagnosis and General Description	
Injury Diagnosis: <u>Concussion</u>	
Incident Description: <u>slipped on ice while walking, went up in the air and landed on occiput</u>	
Primary Impact Location	
Please indicate the location of primary impact.	
	
☐ Left	☐ Right
Documentation	
Physician: _____	<u>6.7 m/s</u>
Hospital: _____	<u>3.8 m/s</u>
Date: (YYYY/MM/DD)	
Glasgow Coma Scale	
Eyes: ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ Closed	
Verbal: ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ Tube	
Motor: ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6	
Loss of Consciousness	
☒ No ☐ Yes – Duration _____ minutes	
Symptoms of Confusion	
☐ No ☐ Yes – Duration _____ minutes	
Fall ☐ N/A	
Drop: <u>height</u> cm Initial Contact: ☒ Head ☐ Other	
Impact Surface:	
☐ Sand ☐ Grass ☐ Gravel ☒ Concrete	
☐ Ice ☐ Steel ☐ Other: _____	
Collision ☒ N/A	
Object: _____	
Weight: _____ kg Speed: ☐ Slow	
Material: _____ ☐ Moderate	
☐ Fast	
Mode:	
☐ Walk ☐ Run ☐ Bike ☐ Skate	
☐ Ski ☐ Car ☐ Other: _____	
Helmet ☒ N/A	
Make: _____ Model: _____	
Size: _____	
Available for Analysis: ☐ No ☐ Yes	
Supporting Documents (Please attach)	
Injury Picture: ☐ No ☐ Yes	
Incident Video: ☐ No ☐ Yes	
MRI: ☐ No ☐ Yes	
CT: ☐ No ☐ Yes	
DTI: ☐ No ☐ Yes	

Punch Injury Impact Event

Case #14

Test ID: [MMA-0014]

Date: Aug. 30th 2014

Researcher: Marshall Kendall

Event details

Mechanism: Punch

Striking athlete: [1.78 m; 236 lbs]

Impacted athlete: [1.86 m; 236 lbs]

Outcome:

KO

Video Analysis

Impacting surface: Right fist

Impact velocity: 8.1 m/s

Impact location: L1 – F (tip of chin)

Impact orientation: Rotation: 20 degrees

Elevation (Impact): None

Extra:

Velocity calculations



Fig 1 velocity of 7.1 m/s distance of fist to head was then divided by the time for the fist to hit head (defined by the number of frames (4 frames = 0.08 s)).

Frame rate:	30 fps
Frames:	2
Reference distance:	length: 180 cm
Time to impact:	0.0666s
Distance travelled:	0.5394
Velocity:	8.1 m/s

Impact location

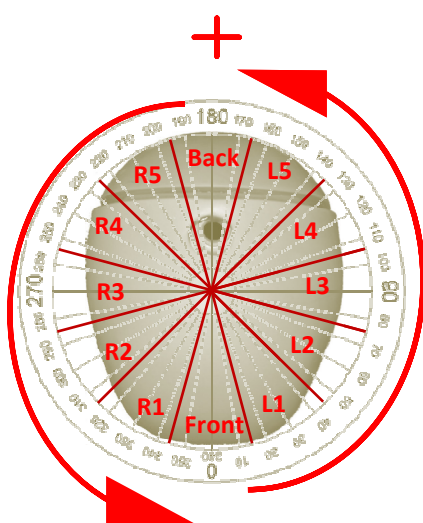


Fig 3 Reference coordinates

Impact location: L1-F (turn head to 290 degrees)

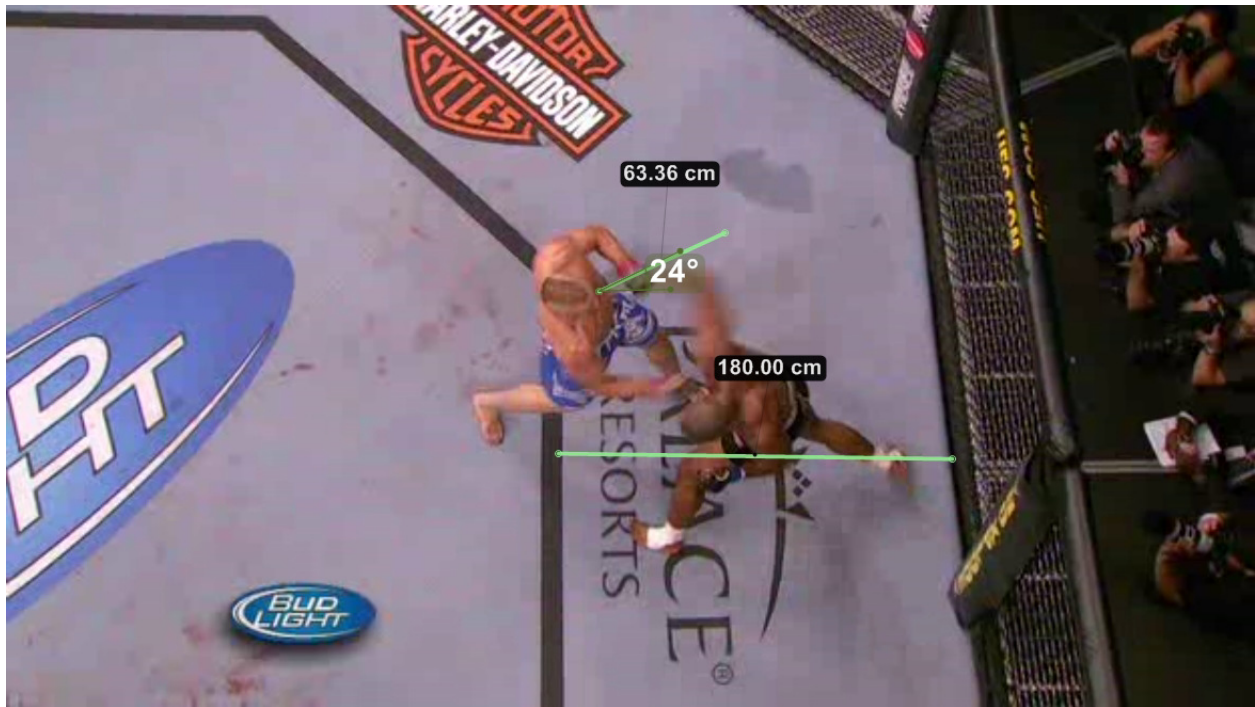


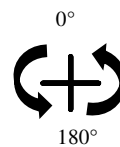
Fig 5 Impact orientation

Impact orientation:

Rotation: 290 degrees

Elevation (Head): None

Elevation (Impact): None



APPENDIX B

Unbiased neckform comparison to Hybrid III neckform

Table 1. Mean and standard deviation peak linear acceleration of a Hybrid III headform

Impact Site	Axis	Hybrid III		Unbiased		Unbiased 50	
FPE15	R	292.9	(11.2)	343.9	(20.2)	343.9	(31.0)
	X	-291.3	(11.4)	-341.1	(19.9)	-341.2	(27.0)
	Y	-11.6	(1.7)	-4.5	(14.8)	-1.6	(28.8)
	Z	-12.1	(41.5)	-17.9	(56.2)	14.7	(55.0)
FBPA	R	299.7	(62.7)	345.1	(20.8)	344.1	(8.0)
	X	-59.5	(15.2)	-61.3	(5.6)	-60.9	(3.4)
	Y	293.9	(60.9)	340.0	(20.3)	338.7	(7.8)
	Z	-17.5	(3.4)	-13.9	(0.8)	-15.1	(1.0)
SCG	R	336.0	(5.4)	336.5	(2.1)	346.8	(9.1)
	X	-13.4	(1.2)	-13.7	(3.3)	-20.5	(5.2)
	Y	335.5	(5.3)	335.1	(3.1)	345.0	(10.2)
	Z	-27.9	(0.2)	35.2	(10.5)	35.7	(10.8)
RBNA	R	233.7	(6.8) ¹	239.4	(1.7)	245.5	(3.5) ¹
	X	51.6	(1.9)	51.9	(5.1)	53.1	(7.3)
	Y	228.1	(7.0)	233.6	(0.9)	239.4	(2.3)
	Z	-22.6	(1.4)	-22.1	(1.6)	-22.2	(1.2)
RNA	R	218.7	(0.9)	239.7	(5.6)	215.0	(6.6)
	X	191.0	(2.9)	209.6	(2.6)	185.9	(20.1)
	Y	106.3	(3.3)	116.8	(6.8)	109.4	(13.0)
	Z	-38.2	(0.6)	-35.7	(1.7)	-30.4	(5.1)

¹ Significantly different at $p < 0.05$

Table 1. Mean and standard deviation peak angular acceleration of a Hybrid III headform

Impact Site	Hybrid III		Unbiased		Unbiased 50	
FPE15	20.6	(0.5)	20.3	(1.0)	20.0	(1.0)
FBPA	31.9	(8.0)	33.1	(2.6)	33.2	(1.5)
SCG	28.5	(0.3) ²	26.3	(0.8) ^{2,3}	27.7	(0.5) ³
RBNA	28.8	(0.5)	29.3	(0.3)	29.5	(1.3)
RNA	19.3	(0.5)	18.7	(0.9)	18.7	(1.1)

^{2 & 3} Significantly different at $p < 0.05$