Analysis of Concussion Metrics of Real-World Concussive and Non-Injurious Elbow and Shoulder to Head Collisions in Ice Hockey

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Acknowledgment

I must admit, I was a little naïve when I decided to pursue my education and become a PhD candidate. Like all previous academic steps, it seemed like it was the natural decision to make once I had my Master's diploma in hand. I only had a meagre understanding of the academic and emotional challenges I would face during what I thought was going to take four years. This dissertation actually ended up taking six years to complete. This may seem like a long time to write a single dissertation; and looking back, I feel like I could have finished the work in two years. But doing so would have deprived me of the valuable lessons I have learned about myself. I find the title itself, "Doctor of Philosophy", quite fitting as the process involves countless cycles of exploration, inquiry, meditation, enlightenment, doubt, confusion, uncertainty, and perseverance. While I have spent a little too much time confused and uncertain of what I was doing and why I was doing it, I managed to find the motivation to persevere and successfully complete what very well may my biggest achievement in my academic career.

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Abstract

Concussions occur at an unacceptable rate in the sport of ice hockey. Efforts are made to improve its prevention by modifying protective equipment and implementing rules of conduct; yet the effectiveness of these methods remains unknown as there is a lack of evidence pointing to a mechanical metric able to adequately predict concussion. The purpose of this thesis was to identify metrics which best characterize concussion following ice hockey collisions and provide values reflecting concussion risk. The first study reported effective mass of shoulder checks, extended elbow strikes, and tucked-in elbow strikes using 15 competitive ice hockey players as subjects. The results were used to guide the impact mass and compliance of laboratory reconstructions of real-world ice hockey collisions done in the second study. Analysis of these reconstruction showed that concussions following shoulder and elbow to head collisions in ice hockey occurred at low peak linear and angular accelerations and that impulse duration played a large role in the mechanism of injury. The results also indicated that concussion risk estimations are specific to the mechanism of loading. A 50% likelihood of concussion following a shoulder check to the head was established for peak angular accelerations of 9.2, 6.9, 4.6, and 2.2 krad/s^2 for impulse durations of 15, 20, 25, and 30 ms, respectively. A 50% likelihood of concussion following an extended elbow to the head was established for peak linear accelerations of 23, 15, and 7 g for impulse durations of 15, 20, and 25 ms, respectively. Finally, the third study reported brain tissue stress and strain comparable to the ones obtained

reconstructing concussive impacts in American football, rugby, and Australian rules football despite having lower peak linear and angular acceleration values. This thesis has provided a new sport concussion data set acquired using a methodology guided by the biomechanics of ice hockey player volunteer testing, has identified metrics which can adequately predict concussion, and has established concussion risk levels. This information will be of use to helmet manufacturing companies, companies developing concussion detection sensors, and governing bodies in their efforts to eliminate concussion from the sport of ice hockey.

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Chapter One

INTRODUCTION

The National Collegiate Athletic Association Injury Surveillance System published injury data collected from 1988 to 2004 from 15 collegiate sports [Hootman et al., 2007]. The report stated that concussion was the most reported injury in men's and women's ice hockey programs and identified the sport of ice hockey as having the highest incidence of concussion per athlete exposures. The results support previous epidemiological studies that reported that concussion accounted for 6% to 14% of injuries in ice hockey and had an incidence of 1.5 to 18.7 per 1 000 player-game hours [Covassin et al., 2003; Jørgensen and Schmidt-Olsen, 1986; Koh et al., 2003; Pelletier et al., 1993; Tegner and Lorentzon, 1996].

Concussion is defined as a rapid onset of neuronal impairment that may present in a wide range of domains including clinical symptoms, physical signs, behavioural changes, cognitive impairment and sleep disturbance. While symptoms typically resolve spontaneously over a short period of time, some cases may require a lengthy convalescence to fully recover [Binder et al., 1997; McCrory et al., 2009]. More alarmingly, it has been suggested that the early onset of dementia such as Alzheimer's disease may be associated with repetitive concussions [Guskiewicz et al., 2005].

Long-term neurodegenerative changes in a professional National Hockey League (NHL) player consistent with chronic traumatic encephalopathy (CTE) were observed post-mortem [Stern et al., 2011]. The disorder reflects a progressive neurological deterioration possibly associated with repetitive brain trauma; its early symptoms include memory and cognitive difficulties, depression, impulse control problems and behavioural changes and may later result in dementia [Stern et al., 2011]. While the severity of CTE seems to be proportional to the length of time spent playing a sport and usually appears years following an athlete's retirement [Stern et al., 2011], the early onset of CTE has been diagnosed following an autopsy of an 18 year old high-school football player [CSTE, 2009].

It is important the note that the causality between a history of multiple sports concussion and neuropathological disorder such as CTE has not been established. Current findings were derived from small samples of convenience and the tau protein aggregates present in normal aging were overlooked [Karantzoulis and Randolph, 2013]. Nevertheless, it cannot be denied that participation in contact sports can result in various injuries to the brain. It is hoped that the growing body of research identifying brain trauma risk associated with participation in contact sports encourages a discussion between coaches, athletes, physicians and parents about the importance of concussion prevention and management. As stated by Dr. Cantu, "it is morally and ethically wrong to allow our children to voluntarily suffer this kind of brain trauma without taking the simple educational steps needed to protect them" [CSTE, 2009].

Statement of the problem

Designing and optimizing protective equipment such as helmets require an accurate understanding of injury mechanics and the risk associated with the environment in which they are used. While the gross mechanism is known, the head's dynamic response as well as the brain strains and stresses generated by an impact to the head in ice hockey have yet to be determined. Identifying the brain injury metrics that best predict concussion would allow for a proper assessment of concussion risk and the ability of behavioural changes and protective equipment to reduce it.

Body checking, defined as a defensive skill designed to separate a player from the puck using the hip or body as a point of contact, is the primary cause of concussion in ice hockey [Emery and Meeuwisse, 2006; Gerberich et al., 1987; Hutchison, 2011; Hutchison and Comper, 2013]. While players are taught to body check without making contact to the opponent's head, 68% of concussions occurring in the National Hockey League between 2006 and 2009 were attributed to body-to-head impacts [Hutchison, 2011]. The analysis also revealed that the shoulder (68%) and elbow (23%) were the body parts that came most often into contact with an opponent's head [Hutchison, 2011].

Many brain tissue deformation metrics have been associated with neuronal impairment; yet their ability to predict concussion following a shoulder or elbow to head collision has not been established [Bain and Meaney, 2000; Cater et al., 2006; Elkin and Morrison, 2007; Galbraith et al., 1993; LaPlaca et al., 1997; Mao and Yang, 2010; Morrison et al., 2003; Stålhammar 1975b; Stålhammar and Olsson, 1975; Yu et al., 2009]. The analysis of head impacts occurring in elite

sports can be achieved using high quality video recordings of real-world elite sports [Fréchède and McIntosh, 2009; McIntosh et al., 2000; Pellman et al., 2003]. Video analysis provides information concerning the impact velocity, location and orientation as well as player identification and equipment worn. These parameters can then be reconstructed in a laboratory to quantify the event and establish a link between brain injury metrics and concussion. The quality of the result is dependent of the reconstruction parameters; thus, it is necessary to establish a valid and reliable method to determine impact parameters prior to testing.

Impact velocity, location and orientation can be obtained directly from the video. Velocity is typically measured using the dimension of a visible player's helmet or limb to scale the frame [Fréchède and McIntosh, 2009; McIntosh et al., 2000; Newman et al., 1999]. The location of the head contact and the orientation of the impacting object can be determined qualitatively using camera close-ups, by comparing the post-impact kinematics of the reconstruction to the real-event, or by pictures of superficial trauma such as bruising [Fréchède and McIntosh, 2009; McIntosh et al., 2000; Newman et al., 1999]. Conversely, the mass used by the striking player, referred to as effective impact mass, is difficult to define as it cannot be calculated using video analysis. Previous studies have used the estimated mass of the limb that came into contact with the head or the mass of the anthropomorphic surrogates used for testing to approximate effective impact mass [Fréchède and McIntosh, 2009; Newman et al., 1999]. Both methods may underestimate the impact mass used by the striking athlete as they do not account for the athlete's ability to apply force during a collision. Striking technique was reported to increase the

effective impact mass in boxing and football and could also influence effective impact mass in an ice hockey body check [Viano et al., 2007; Walilko et al., 2005]. Considering that a variation of 2 kg was shown to change head peak linear and angular acceleration by up to 12% and 6%, respectively [Karton, 2012], accurately defining the impact mass of a body-to-head collision in ice hockey would increase the validity of laboratory reconstruction.

Purpose

Thus, the purpose of this thesis was to identify the metrics which best characterize concussion following ice hockey collisions and provide values reflecting concussive and non-injurious events. This was achieved by:

1. Calculating the effective impact mass of three common striking techniques: extended elbow strike, tucked-in elbow strike, and shoulder check.
2. Reconstructing real-world concussive and non-injurious elbow and shoulder to head collisions in ice hockey in a laboratory.
3. Performing a Finite Element Simulation of the dynamic response of the reconstructed real-world concussive and non-injurious elbow and shoulder to head collisions in ice hockey.

Chapter Two

LITERATURE REVIEW

The Human Brain

The human brain is a complex organ in charge of storing, retrieving and processing information. It can be divided structurally and functionally in three parts: the cerebrum, the brain stem, and the cerebellum. Although the different regions of the brain are attributed specific functions, they work in unity to achieve appropriate responses to different stimuli.

The Cerebrum

The cerebrum is the largest part of the brain and is composed of two hemispheres separated by a deep midline cleft named longitudinal cerebral fissure. A thick and fibrous membrane, called the falx cerebri, descends vertically in the fissure to provide stability and reduce lateral motion of the brain [Margulies et al., 1990]. Deeper within the cerebrum lies the corpus callosum and the anterior commissure. These structures are composed of white matter tracts and allow for learning and memory to be shared between both hemispheres. Deep within each cerebral hemisphere are other white matter formations that connect the neurons within each hemisphere (associational fibers) and connect the cortex to the rest of the central nervous system (subcortical fibers).

The superficial layer of the cerebrum is composed of gray matter and is known as the cerebral cortex. It is organized in a series of convolutions named gyri and folds named sulci or fissures, depending on their size. The main fissures are the lateral fissure, the central fissure, and the parieto-occipital fissure (Fig. 2.1).

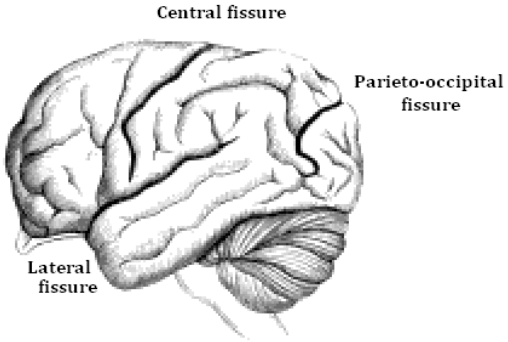


Figure 2.1. The three cerebral fissures used to define the cerebral lobes.

These fissures divide the cerebral hemispheres in four lobes, each with its own predominant functions (Fig. 2.2). These regions are responsible for motor, sensory, auditory and visual integration. The frontal lobe is mostly connected to emotional and intellectual thinking; the temporal lobe is responsible for hearing, balance, language recognition; the parietal lobe is associated with sensation; and the occipital lobe is responsible for vision.

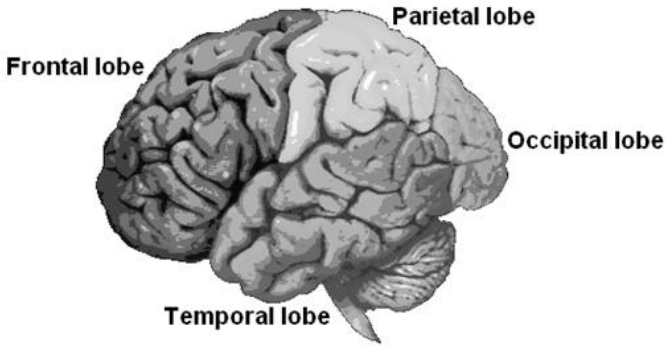


Figure 2.2. The four lobes forming the cerebral cortex.

The frontal lobe is the most anterior region of the brain and is located under the forehead. The frontal lobe initiates voluntary actions (e.g. looking toward an object of interest) and controls learned motor skills (e.g. writing); complex intellectual processes (e.g. concentration); and emotional response. The frontal lobe also influences one's perception of one's relation to the environment; judgment about what occurs in daily activities; and memory for habits and motor activities [McAllister et al., 1999]. Injuries to the frontal lobe may result in loss of flexibility of thinking; impairment of recent memory; inability to focus on tasks; changes in mood and social behavior; and difficulty with problem solving.

The parietal lobe is located at the top of the brain and is separated from the frontal lobe by the central sulcus. The postcentral gyrus can be found just posterior to the central sulcus and is the area which interprets sensory stimuli from cutaneous and muscular receptors throughout the body. The parietal lobe stores spatial memories that allow people to orient themselves in space and maintain a sense of direction; influences mathematical skills and language

comprehension; integrates different senses to understand a single concept; and interprets shapes and textures of objects as they are handled. Mild brain injuries affecting the parietal lobe may elicit difficulties with eye and hand coordination; drawing objects; reading; focus visual attention; as well as a disorientation of environment space.

The temporal lobe is located below the parietal lobe behind the frontal lobe, and is separated from both by the lateral sulcus. The temporal lobe houses the auditory centers responsible for hearing and the interpretation of auditory and visual events. It also plays the primary role in the acquisition, sequencing, storing and retrieving of recent and long-term memories. Injuries to this area can result in hearing impairment; difficulty in recognizing faces; difficulty in understanding spoken words; irritable behaviour; short-term memory loss; and interference with long-term memory retrieving.

The occipital lobe is located at the most posterior area of the cerebrum. The lobe lies immediately above the cerebellum and is not distinctly separated from the temporal and parietal lobes. The principal functions of the occipital lobe are visual processing; interpretation; visual association (i.e. correlating visual images with previous visual experiences); and other sensory stimuli. Injury to the occipital lobe following a direct blow to the posterior aspect of the head may result in vision deficiencies such as hallucinations; inaccurately seeing objects; and inability of recognizing the movement of an object.

The Brain Stem

The brain stem lies between the spinal cord and the diencephalon and is composed of the medulla oblongata, the pons and the midbrain. The three sections are divided based on their functionality rather than their anatomy. The brain stem is mostly composed of white matter nerve fibers connecting the cerebral and cerebellum hemispheres. It is responsible for basic functions such as breathing, swallowing, and level of consciousness. Vertigo, nausea, and vomiting associated with other neurological symptoms are almost always indicative of brain stem lesions at the lower pons or upper medulla oblongata while alterations in the level of consciousness may indicate lesions in the upper brain stem.

The Cerebellum

The cerebellum lies directly behind the cerebrum. It has two hemispheres divided by a structure called the vermis. Its outside layer is composed of gray matter while the inner layer is made of white matter. It is directly connected to the brain stem, spinal cord, and cerebrum and is the unconscious centre for motor movement coordination allowing for voluntary movement to occur smoothly and accurately. Lesions to the cerebellum may cause tremors and lurches, movements are no longer dampened and may oscillate. Lesions may also impair the ability to adjust to sudden displacements, decreasing stability of posture and gait. Lesions to the lateral aspect of the cerebellum may impair executive functions, such as abstract reasoning, planning and visual spatial organization, and alter a person's behaviour by disinhibiting inappropriate actions.

Definition of Concussion

Concussion has been defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces [McCrory et al., 2009]. Concussion manifests itself in a range of clinical symptoms, physical signs, behavioural changes, cognitive impairment and sleep disturbance. [Aubry et al., 2002; Chen et al., 2004; Denny-Brown and Russell, 1941; Johnston et al., 2001; Lovell, 2009; Ptito et al., 2007]. Detection is difficult because the neuropathological changes do not appear as abnormalities on standard neuroimaging (e.g., CT and MRI scans) [Aubry et al., 2002; Bazarian et al., 2006; McAllister et al., 1999]. While most individuals see their symptoms resolve spontaneously over a short period of time, some require a lengthy convalescence and may never fully recover [Binder et al., 1997; Guskiewicz et al., 2005; McCrory et al., 2009; McKee et al., 2009].

Incidence

The National Collegiate Athletic Association Injury Surveillance System (NCAA ISS) released injury data collected from 1988 to 2004 in 15 NCAA sports. Incidence rates for concussion in collegiate athletes were reported to range from 5% to 18%, with ice hockey accounting for the highest rate [Hootman et al., 2007]. This is comparable to previous epidemiological reports that found that 3% to 24% of high school and collegiate student-athletes reported being diagnosed with concussion in all sports [Cernich et al., 2007; Covassin et al., 2003; Erlanger et al., 1999; Gessel et al., 2007; Hootman et al., 2007; Powell & Barber-Foss, 1999; Schulz et al., 2004].

Concussion affects 5% to 20% of athletes playing ice hockey each season [Benson et al., 2011; Covassin et al., 2003; Goodman et al., 2001; Jørgensen and Schmidt-Olsen, 1986; Tegner and Lorentzon, 1996]. Accounting for the length of games and number of games per season, which varies per league and age group, the incidence of concussion in ice hockey has been reported to be between 17.6 and 21.5 per 1 000 player-game hours [Echlin et al., 2010; Roberts et al., 1999]. This is higher than the rate of 1.0 to 6.5 concussion per 1 000 player-game hours reported in other studies using data collected between 1979 and 2002 [Jørgensen and Schmidt-Olsen, 1986; Pelletier et al., 1993; Tegner and Lorentzon, 1996; Wennberg and Tator, 2003]. The methodology used to collect the data can partially explain the increase in concussion risk. The studies reporting lower rates used retrospective questionnaires completed by athletes and trainers while the two studies reporting higher rates had trained medical staff attending each game. Being more attentive to signs that could indicate a concussion may have influenced the reporting rate.

Interestingly, a research study that asked athletes to report concussion symptoms while omitting the word “concussion” found that 47% of American football players had most likely suffered from a concussion during the previous season [Langburt et al., 2001]. This was supported by studies conducted in Canada which reported that 70% of Canadian Interuniversity Sports football players and 63% of Canadian Interuniversity Sports soccer players claimed having suffered from symptoms related to concussion during a single competitive season despite only 17% of football players and 12% of soccer players acknowledging having

experienced a concussion [Delaney et al., 2002]. This clearly indicates that athletes may be at a higher risk of experiencing a concussion than once thought.

Signs and Symptoms

Concussed athletes present a wide variety of signs and symptoms following a violent collision involving head contact. Three immediate on-field markers of sport-related concussion are loss of consciousness (LOC), post-traumatic amnesia (PTA) and confusion. Sport-related LOC can be described as a state similar to sleep; however, arousal by external stimuli is not possible. Post-traumatic amnesia refers to a disruption in continuous memory function and can affect information which occurred before the injury (retrograde amnesia) or after the injury (anterograde amnesia). Confusion denotes the inability to think with customary speed, clarity, and coherence and is marked by some degree of inattentiveness and disorientation.

Athletes suffering from concussion may also be subjected to post-concussion syndrome (PCS) which presents itself with a variety of physical, cognitive, behavioral and emotional symptoms. The nature and severity of these signs and symptoms may be attributed to localised damage to the underlying brain tissue [Bailes & Cantu, 2001; Collins & Hawn, 2002; Kontos et al., 2004; McGee, 2010]. Loss of consciousness may be the result of disturbances to the upper brainstem [Ommaya, 1990; Ropper and Samuels, 2009; Viano et al., 2005b]; memory disturbances are more likely to occur following trauma to the right or left temporal lobe [Janowsky et al., 1989]; and confusion may reflect the loss of integrated activity of all of the

associative regions of the cortex [Ropper and Samuels, 2009]. Although certain PCS symptoms such as headache and anxiety have not been associated to lesions to a specific location in the brain, others, such as blurred vision and hearing deficit are attributed to a particular cerebral lobe (occipital and temporal lobes, respectively) [Ropper and Samuels, 2009].

The presence of headache, dizziness, blurred vision, LOC and nausea have been noted in prospective studies of concussion [Johnston et al., 2001]. Unfortunately, establishing relationships between symptoms and PCS duration can be difficult considering that PCS persistence is influenced by substance abuse, increasing age, low educational achievement, low level job skills, pre-morbid personality traits and a neuropsychiatric history [De Kruijk et al., 2002; Kibby & Long, 1997]. In addition, other non-neurological injuries occurring at the time of the brain injury such as litigation and the availability of compensation can prolong PCS and lead to a worse functional outcome while good family support can positively influence outcome [Olver, 2005].

Injury mechanism

Concussion is typically caused by a direct blow to the head or by impulse loading, which refers to the motion of the head following an impact to the body [Ommaya and Gennarelli, 1974]. Momentum and energy are transferred to the head, creating forces which modify its motion and deform the brain tissue.

Upon contact, the skull and brain move both as a unit and relative to each other [Bayly et al., 2005; Gosh et al., 1970; Gurdjian, 1978; Gurdjian et al., 1968; Hodgson et al., 1966; Nusholtz et al., 1984; Pudenz and Shelden, 1946; Stalnaker et al., 1977]. The difference in response between the brain and the skull causes an increase in pressure at the impact site as the skull presses against the brain and a decrease in pressure at the opposite side as the brain lags behind the rapidly moving skull [Gurdjian and Gurdjian, 1975; Thomas et al., 1967]. Moreover, the brain has a tendency of rotating and creating shear strain within its tissue even though the head may not have been accelerated angularly [Bayly et al., 2005].

The stretching of the neuronal tissue causes its structure to change and forces the opening of ion channels. This creates a sudden increase of extracellular potassium (K^+) which in turn triggers an excessive release of excitatory amino acids such as glutamate [Katayama et al., 1990]. Usually, any excess in extracellular K^+ would be reabsorbed by surrounding glial cells; however, the high amounts of excitatory amino acids activate the opening of ligand-gated ion channels which increase the outflow K^+ and influx of calcium (Ca^{2+}). Thus, following a blow to the head, the brain tissue undergoes a large excitatory process followed by an abrupt wave of neuronal deactivation. This is known as *spreading depression* and it was suggested that this phenomenon may be responsible for loss of consciousness, memory loss and cognitive alterations associated with concussion [Giza and Hovda, 2001]. The brain enters a hypermetabolic state as its K^+ and sodium (Na^+) pumps are activated. Unfortunately, the increased demand in glucose and ATP cannot be met because cerebral blood flow is reduced by the intra-cranial pressure, and the excessive accumulation of intracellular Ca^{2+} impaired

mitochondrial function [Giza and Hovda, 2001; Hovda et al., 1999]. Electrolyte homeostasis is usually restored within minutes but the brain may remain in a depressed state for several days [Katayama, 1990].

Injury metrics

Measuring the head's response to insult and establishing tolerance limits for the neuronal tissues composing the human head are two of the primary objectives set by impact biomechanics researchers [Zhang et al., 2001]. Reaching these goals can improve the evaluation of risk associated with specific situations and help devise new strategies to reduce the severity and incidence of concussion.

As mentioned above, mechanically induced stretching of the neuronal tissue creates a state of depression which partially explains the transient nature of concussion. Direct measurement of brain tissue deformation would be ideal to predict the occurrence of concussion; yet the methods to do so are invasive and cannot be used with human subjects. Thus, most of the research is concentrated on identifying relatively easy to measure injury metrics which can evaluate concussion risk. It is understood that kinematic parameters cannot predict the diverse injury mechanisms of concussion; however, they are currently used to assess the safety of equipment and play structures based on the assumption that a decrease in one parameter results in a decrease in all other parameters.

Most of the research conducted in the late 20th century used animal models to study concussion. These models offered the possibility to test above injury thresholds and allowed for a direct analysis of concussion and severe traumatic brain trauma [Genarelli et al., 1971; Gennarelli et al., 1972; Ommaya et al., 1966; Pudenz and Shelden, 1946]. These animal models relied on the assumption that human traumatic brain injuries could be replicated in non-humans. Unfortunately, the use of anesthetics, differences in brain size, inherent tolerance and skull characteristics reduces the reliability of data extrapolation. Recently, reconstructions of real-world concussive incidents occurring in professional sports have showed to be a promising source of data [Fréchède and McIntosh, 2009; McIntosh et al., 2000; Patton et al., 2012; Pellman et al., 2003; Walilko et al., 2005; Withnall et al., 2005]. Using anthropomorphic dummies or computer simulations, researchers have been able to replicate these events to assess concussion risk in American football, Australian rules football and rugby.

While concussion prediction may be improved by using numerical models, it is important to note that results are dependent on the material properties chosen to represent brain tissues. Furthermore, the size of the modelled head has an influence on intracranial stresses associated with injury [Kleiven and von Holst, 2002]. The physical properties are usually taken from the literature, which are often assumed, inaccurate or incomplete [Panjabi, 1979]. Properties taken from in-vivo tests on animal tissues are scaled, limiting its reliability, it is also unknown how the properties obtained using in-vitro tests on human samples are altered by their cadaveric state [Gilchrist, 2003].While constitutive properties are a limitation to all existing FE models, the systemic response can be analysed to draw global conclusions. The concussion metrics most

commonly used and sufficiently correlated to concussion to be used by impact biomechanics researchers are presented below.

Linear acceleration

Peak resultant linear acceleration is the most common parameter used to predict the occurrence of brain injury. It is calculated using the following equation:

$$a = \frac{\Delta v}{t}$$

where a is linear acceleration of the head, generally reported in g (9.807 m/s^2), Δv is the change of velocity of the head (m/s), and t is the time duration over which the acceleration takes place (s). Linear acceleration can easily be acquired using accelerometers located either in an anthropometric headform [Padgaonkar et al., 1975], fastened to a cadaveric skull [Yoganandan, et al., 2006], inserted in a volunteer's mouth [Higgins et al., 2007], or attached to an athlete's helmet [Beckwith et al., 2007; Duma et al., 2005; Gwin et al., 2006].

Studies using mongrel dogs and primates identified 90 g as being a threshold for concussion [Gurdjian et al., 1964; Ono et al., 1980]. Interestingly, it was determined that a head peak resultant linear acceleration of 90 g would only cause concussion if the duration of the impulse was equal to or higher than 9 ms [Ono et al., 1980]. Shorter impacts, with duration of 2 ms, required a peak linear acceleration of 220 g to result in a concussion. The threshold proposed in these early studies are difficult to extrapolate to humans due to the use of anaesthetics, differences in brain size, inherent tolerance, and skull characteristics of the tested animals. Furthermore, concussion was at the time defined as a short period of unconsciousness followed

by minor confusion. Since the animals were sedated prior to impact, concussion was assessed using physical signs such as loss of corneal reflex, fluctuation in blood pressure, apnea and bradycardia. It is now known that loss of consciousness is a relatively uncommon sign of concussion and is not an adequate proxy for concussion [Aubry et al., 2002; Collins et al, 2003; Guskiewicz et al., 2005; Guskiewicz et al., 2000].

Peak linear acceleration thresholds for sports concussion have been proposed following impact reconstructions of 27 collisions and four falls occurring in the National Football League (NFL) [Pellman et al., 2003]. Twenty-two of the struck players involved in head-to-head contact and three players who fell and struck their head on the turf were diagnosed with a concussion by a team physician. The remaining player who fell on his head, the five struck players, and all 27 striking players did not report a head injury. While concussion was assessed by team physicians and the clinical reports were verified by team physicians, the researchers did not report the criteria used to determine a concussion. In this study, the anthropomorphic test device representing the struck player was guided in free-fall from a height sufficient to achieve the same impact velocity as that determined from video analysis. A second helmeted head/neck assembly attached to a freely suspended Hybrid III torso was used to mimic the striking player. The results indicated that linear acceleration had a strong positive correlation with concussion ($r = 0.72$, $p < 0.001$), which was slightly higher than angular acceleration ($r = 0.68$, $p < 0.001$). The peak head acceleration for concussed players was 98 ± 28 g, which was significantly greater than the 60 ± 24 g for uninjured struck players ($p < 0.005$). Risk curves for peak linear acceleration were established using logistic regression analysis [Zhang et al., 2004].

The 25%, 50% and 80% probability of sustaining a concussion were estimated to be 66 g, 82 g and 106 g, respectively. While impact velocity, orientation, and location were obtained using video analysis, assumptions were made for impact mass and compliance. Effective mass was assumed to be comprised of the mass of the dummies and the active force generated by both athletes was assumed to be negligible. Furthermore, it was assumed that compliance of the collision was accurately recreated using two Hybrid III dummies equipped with American football protective equipment. The accuracy of these assumptions is unknown because no data currently exists identifying human effective mass and compliance in football head-to-head collisions.

Mathematical Dynamic Models (MADYMO) were used to recreate 27 collisions which occurred in rugby and Australian rules football [Fréchède and McIntosh, 2009]. Results indicated that concussion was associated with a linear acceleration of 103 g and an angular acceleration of 8 020 rad/s^2. Nonparametric tests revealed that peak linear acceleration, head impact power, and head injury criterion were significantly different between impacts which resulted in a diagnosed concussion without loss of consciousness (86 ± 14 g) and impacts which resulted in a diagnosed concussion with a loss of consciousness lasting more than one minute (123 ± 20 g). Mass and inertias of each body segment were calculated based on the known anthropometry of the athletes and closing velocities were estimated using the videos. The modeled body segments used mass and compliance found in the literature; however, validation was done using human cadavers in motor vehicle accident scenarios and it is not known how

accurate the model can recreate athletic collisions (lower velocities, higher compliance) [de Lang et al., 2006].

A higher linear acceleration threshold for concussion was suggested by Funk et al. [2007] following the analysis of 27 000 head impacts from Virginia Tech football players captured during the 2003-2006 seasons. Despite collecting such a high number of impacts, only four concussive impacts were captured. The mean peak acceleration associated with concussion was 137 ± 47 g, which is similar to the data reported by Pellman et al. [2003]. A regression analysis of the data identified a 10% risk of concussion for peak linear acceleration of 165 g, which is dramatically higher than what was proposed by Zhang et al. [2004]. The difference may be in part due to their respective data set; Funk et al. [2007] utilized a large data set of 27 000 exposures, of which only four resulted in a physician-diagnosed concussion (2.97 concussions per 1000 athletic exposures), while Zhang et al. [2004] mostly selected injurious impacts. Furthermore, Funk et al. [2007] collected live data during the games using a Head Impact Telemetry system (HITs). While the usefulness of such a tool is undeniable, its validation has been severely critiqued [Jadischke et al., 2014]. The system was validated using a Hybrid III headform for low velocity impacts but not the high velocities which occur often in a game [Gwin et al., 2006]. It cannot be assumed that the system is able to accurately measure head acceleration when impacted at high velocity because the helmet may move independently from the head in such conditions. Second, the system was only validated for impacts through the centre of gravity of the head and does not consider non-centric impacts [Gwin et al., 2006]. Third, the validation was performed using a medium helmet which created an unrealistic

coupling with the Hybrid III headform [Jadischke et al., 2014]. This reduces the motion of the helmet during the impact underestimating error in the process. Finally, acceleration was collected at 1000 Hz and filtered using a 400 Hz low pass filter, both of which are below the 10 000 Hz collection rate and 1650 Hz low pass filter recommended by the industry [SAE, 1995]. Using such a low collection rate and filter to capture acceleration during direct impacts to a helmeted head prevent researchers from obtaining true peak values.

Average linear acceleration

Peak linear acceleration is highly correlated to skull fractures but only mildly to concussion indicating it may not accurately define the response of a viscoelastic structure like the brain [Gadd, 1966; Newman, 1980]. While mechanical failure of an elastic material can be predicted using a single value, the same cannot be accomplished for viscoelastic materials such as the brain which are dependent of the rate at which the stress is applied [Bandak, 1995].

One method to account for the brain's rate dependence is to integrate the area under the acceleration-time pulse. In other words, impacts causing 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 more commonly used description of the relationship between acceleration and time is the Wayne State University Cerebral Concussion Tolerance Curve (WSTC) (Fig. 2.3). The curve defines the limit between fatal and non-fatal head responses to impacts and it illustrates the brain's ability to withstand high accelerations for short time durations and its vulnerability to longer impulses.

It is apparent that the curve does not provide information in regards to concussion; yet the same relationship was observed by Ono et al. [1980] as mentioned previously.

The original curve was developed by Gurdjian and colleagues [Gurdjian et al., 1964; Gurdjian et al., 1953; Gurdjian et al., 1955; Gurdjian et al., 1966]. Tolerance for short duration impacts (2-6 ms) was determined from linear skull fracture data in human cadaveric heads [Gurdjian et al., 1964]. Tolerance for medium duration impacts (6-20 ms) was derived from analyzing the concussive effects in anesthetized dogs subjected to varying pressure pulses applied directly to the dural sac as well as head acceleration-pressure data obtained from a series of embalmed human cadaver drop tests [Gurdjian et al., 1955]. The curve was later extended for longer duration impacts (above 20 ms) using sub-injurious data obtained with the help of military volunteers [Stapp, 1970]. It is important to note that "sub-injurious" meant no apparent trauma and does not mean that the participants did not experience concussion symptoms.

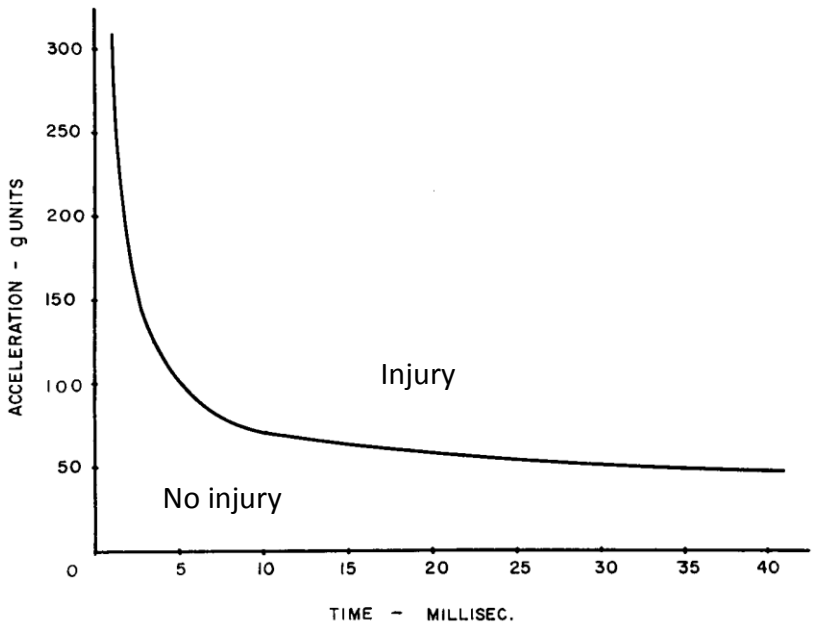


Figure 2.3. Wayne State University Concussion Tolerance Curve [Zhang, 2001]

The WSTC was critiqued for a number of reasons and is considered to be an inaccurate representation of the relationship between head motion and head trauma. The protocol used to obtain cadaver head accelerations was based on the assumption that the skull was non-deformable, thus the accelerometers were positioned at the back of the head [Newman, 1980]. Furthermore, the membranes surrounding the lobes of the brain were removed to allow all parts of the cranial cavity to communicate during cadaver testing [Prasad et al., 1985]. While this was judged necessary to allow for the measurement of intracranial pressure gradients, such a procedure likely had an effect on the response of the brain within the cranium [Bayly et al., 2005; Margulies et al., 1990; Pudenz and Shelden, 1946]. Newman [1980] also observed that some data points were not properly plotted and others were ignored. Furthermore, it is unclear whether the "effective acceleration" obtained from the military study [Stapp, 1970] referred to the change in velocity of the sled or the volunteers' head [McElhaney, 1976].The use of the WSTC as a criterion is limited because it is largely based on the acceleration response of skulls to front impacts and was never validated for living human beings [Newman, 1980; Prasad et al., 1985]. Nevertheless, the creation of the curve had a positive effect on impact biomechanics by demonstrating that a relationship between injury and impulse duration existed.

A curve similar to the WSTC was developed by the Japan Automobile Research Institute (JARI) (Fig. 2.4). The curve, named Japanese head tolerance curve (JHTC) was developed using experimentations on primates and human cadaveric skulls [Ono et al., 1980]. The data were scaled by comparing fracture tolerance between the primate and human skulls. It was stated

that a peak resultant linear acceleration of 200 g and 150 g could be tolerated for a period lasting less than 2 ms and 3 ms, respectively. The data used by JARI were considered better quality than what was used at Wayne State because multiple impact conditions were used and the injuries were categorized [Ono, 1998]. This curve was similar to the WSTC and supports the hypothesis that brain trauma risk is linked to a combination of peak acceleration and impulse duration. A comparison with modern data sets of sports concussion shows that while the methodologies used to obtain both curves was limited; the curves are in accordance with concussion thresholds obtained using reconstructions of American Football concussive impacts [Pellman et al., 2003].

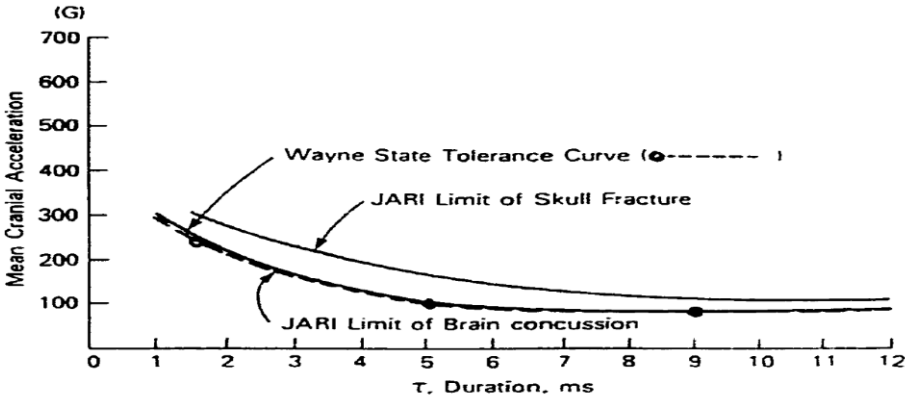


Figure 2.4. A comparison between the WSTC and the JARI tolerance curve [Zhang, 2001]

Gadd Severity Index

The Gadd severity index (GSI) was developed to do what peak linear acceleration could not: compare impacts with different acceleration-time curves [Gadd, 1966]. The GSI was obtained by plotting the data points of the WSTC as well as volunteer data obtained by the NASA

[Eiband, 1959] on a log-log plot. This study reported that subjects strapped onto an accelerating seat could tolerate a uniform acceleration of 45 g for 0.044 s without any loss of consciousness or confusion. Tolerance was observed to decrease to 40 g for a pulse duration of 0.1 s and 25 g for a pulse duration of 0.2 s.

The process reduced the curve to a straight line whose equation was the following:

$$at^{0.4} = 15.83$$

where a is linear acceleration (g), t is duration (s), and *15.83* is the tolerance limit. For ease of use, the tolerance level was set at 1000, which changed the equation to:

$$a^{2.5}t = 1000$$

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

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

where $a\ dt$ is linear acceleration (g) expressed as a function of time. Tolerance thresholds for GSI were proposed using the NFL concussion data mentioned above and were 24, 291 and 559 for a 5%, 50% and 95% risk of mild TBI, respectively [Newman et al., 2002].

While integrating the acceleration pulse may better predict the onset of concussion, the equation used for GSI may not be the best fit. The response was assumed to be linear despite a curved line offering a better representation of the threshold identified by the WSTC [Versace, 1971]. The acceleration data used to create the equation was an average of the effective acceleration of the head, which is quite different from the NASA data, where the deceleration

of the seat was recorded in lieu of the head's response. It is unknown how accurately GSI can predict concussion in direct head impacts as they do not typically result in a uniform acceleration impulse. Finally, the equation does not impose time limits, meaning that indirect impacts, which typically have longer pulses, will yield unrealistically high GSI values [Zhang et al., 2001].

Head injury criterion

The Head injury criterion is a different extrapolation of the WSTC and was developed in response to Versace's [1971] critique of the GSI. The equation is the following:

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

where t and t_0 (referred to as the HIC interval) are the initial and final times (in seconds) during which HIC attains a maximum value. The tolerance value was originally maintained at 1000. It was observed 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 was incorporated into the criterion by the international standards organization [Prasad and Mertz, 1985].

Although a HIC threshold for life threatening injuries was originally set at 1000, it was a non-validated arbitrary value. The threshold was finally tested several years later, when Prasad and Mertz [1985] associated a HIC_{15} of 1400 with a 50% probability of sustaining a life threatening injury while a value of 1000 was associated with a risk of 16%. Table 2.2 shows HIC_{15} thresholds for concussion that were estimated to be between 150 and 400 [Funk et al., 2007; Marjoux et al., 2008; Newman et al., 2002; Prasad and Mertz, 1985; Versace, 1971; Zhang et al., 2004].

Head injury criterion, like GSI, was criticised because of the limitations of the WSTC. Moreover, it was demonstrated that for impacts lasting less than 5 ms, HIC would underestimate the severity of an injury and was not a good injury predictor [Ward et al., 1980]. Finally, a later analysis of injury data revealed that HIC was an unreliable criterion for helmet testing as it was not strongly correlated with either skull fractures or brain vascular injury [Newman, 1975].

Angular acceleration

Holbourn [1943; 1945] first proposed angular acceleration as a possible injury metric using the mechanical properties of brain tissue to establish a proportional relation between angular acceleration and injury severity. Knowing that brain tissue has a high tolerance to compression (bulk modulus) and a poor ability to resist tension (Young's modulus), he suggested that shear stress was the true cause of injury and that rotational forces, not linear, were responsible for concussion and haemorrhage. Many studies were conducted in the 70s and 80s to determine the role of angular acceleration in concussion using monkeys [Adams et al., 1981; Adams et al., 1983; Gennarelli and Thibault, 1982;Gennarelli et al., 1971; Gennarelli et al., 1972; Gennarelli et al., 1981; Gennarelli et al., 1982; Unterharnscheidt and Higgins, 1969]. The results described the predominant role of angular acceleration in the mechanism of concussion, diffuse axonal injury and subdural hematoma. This led to the theory that angular acceleration alone was capable of causing sufficient shear stress to cause any type of brain injury, if applied properly.

Angular acceleration is more complicated to measure than linear acceleration which could explain why it hasn't been incorporated in safety standards. It can be obtained by using

differential displacement of an array of linear accelerations. The most popular array was developed by Padgaonkar, Krieger, and King [1975] and requires nine uni-axial accelerometers positioned in an orthogonal arrangement (Fig 2.5). The method used the following equations to obtain angular acceleration:

$$\alpha_X = \frac{(A_{Zside} - A_{Zcentre})}{2\rho_{side}} - \frac{(A_{Ytop} - A_{Ycenter})}{2\rho_{top}}$$

$$\alpha_Y = \frac{(A_{Xtop} - A_{Xcentre})}{2\rho_{top}} - \frac{(A_{Zfront} - A_{Zcentre})}{2\rho_{front}}$$

$$\alpha_Z = \frac{(A_{Yfront} - A_{Ycentre})}{2\rho_{front}} - \frac{(A_{Xside} - A_{Xcentre})}{2\rho_{side}}$$

where α is angular acceleration (rad/s^2) about the three axes (X, Y, and Z), A is linear acceleration (g) along the three axes (X, Y, and Z), and ρ is the distance between the centre accelerometer block and the side, front, or top accelerometer blocks. Other techniques exist, such as a two-dimensional in-line method; however, Padgaonkar, Krieger, and King's method is accurate, robust and most commonly used in research [Newman et al., 2005].

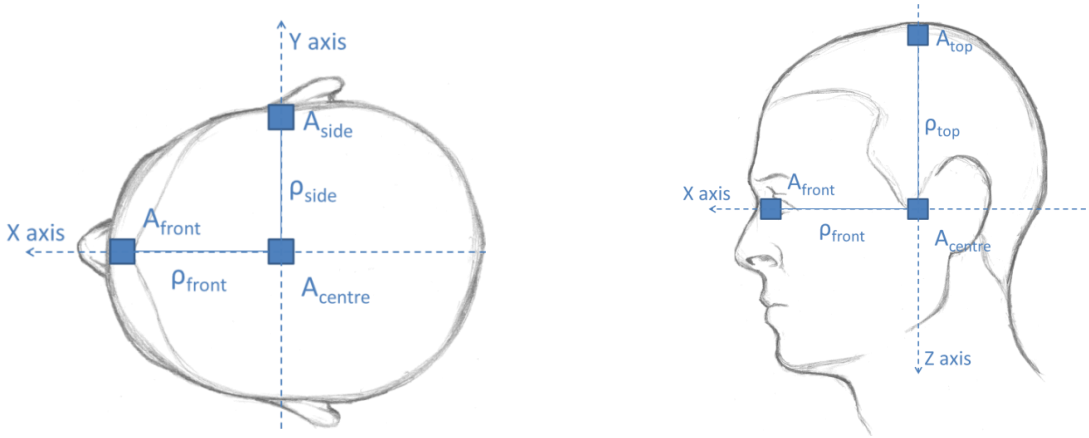


Figure 2.5. Location of accelerometers in a 3-2-2-2 array

Brain tolerance to angular acceleration was first established by producing cerebral concussion in monkeys by direct impact to the occipital area of the head and by whiplash induced by an impact to the base of a mobile chair carrying the monkey [Ommaya et al., 1967; Ommaya and Hirsch, 1971]. In these studies, concussion was described as loss of coordinated response to external stimuli, apnea, bradycardia, loss of corneal reflexes, loss of voluntary movement, and pupillary dilatation. By scaling the animal data to the adult human, it was determined that humans may suffer concussion when exposed to a peak angular acceleration of 1 800 rad/s^2 and was almost certain to suffer concussion when exposed to a peak angular acceleration of 7 500 rad/s^2.

Human concussion tolerance extrapolated from animal data is in agreement with concussion risk curves proposed by Zhang, Yang, and King [2004] who fitted regression curves to NFL reconstruction data of concussive events. A 25%, 50% and 80% probability of sustaining a concussion were estimated to be associated with peak angular acceleration magnitudes of 4 600 rad/s^2, 5 900 rad/s^2 and 7 900 rad/s^2, respectively. Once again, Funk et al. [2007] proposed a higher tolerance threshold based on their data obtained with the HITs; a magnitude of 9 000 rad/s^2 was associated with a 10% concussion risk. Similar to linear acceleration, the accuracy of the HITs to collect angular acceleration has been contested. While claims were made on the system's ability to capture angular acceleration, testing was done at low level impacts and through the centre of gravity of the headform and cannot be extrapolated to the high energy collisions seen in sports [Gwin et al., 2006; Jadischke et al., 2014].

Interestingly, peak angular acceleration well above the aforementioned thresholds has been reported in boxing [Pincemaille et al., 1989; Stojsih et al., 2008]. Ten male volunteer boxers equipped with accelerometer-outfitted helmets sparred for a total of 15 rounds. Despite recorded impacts exceeding 3 500 rad/s^2 with the highest 13 600 rad/s^2, no concussion was reported [Pincemaille et al., 1989]. A more recent study involving 27 male boxers and 28 female boxers reported similar results. Each boxer sparred for four rounds of two minutes and completed a neurocognitive assessment prior to and directly subsequent to the data collection. The average angular acceleration recorded was 2 571 ± 1 852 rad/s^2 and 2 533 ± 1 524 rad/s^2 for male and female fighters, respectively. The highest recorded angular acceleration was 17 156 rad/s^2 and 13 113 rad/s^2 for male and female fighters, respectively. Despite experiencing such high magnitudes repeatedly over one sparring session, no concussions were diagnosed (no decrease in the ImPACT neurocognitive test) [Stojsih et al., 2008]. In both studies, angular acceleration was collected using accelerometers inserted in the pugilists' helmets. While both protocols were validated prior to testing, it is possible that the accelerometers captured the motion of the helmet thus increasing the magnitude of the recorded accelerations and give the illusion of a higher tolerance to angular acceleration.

Stress and strain

Holbourn [1943] was the first to suggest that shear stress, which causes two segments of a structure to displace in the opposite direction (Fig 2.6), as the most probable cause of brain trauma. The hypothesis was founded on the brain's bulk and shear moduli. A material's bulk modulus is a measure of its resistance to uniform compression while shear modulus is a

measure of its resistance to deformation (shear strain). The brain possesses a high bulk modulus and a low shear modulus making it much more likely to change in shape than change in size. Therefore, deformation of the brain as whole applies a certain amount of shear stress to the neuronal network which may cause it to tear. The hypothesis was supported by many studies which directly observed brain tissue deformation following an impact and it is now widely accepted that the it may be the cause for brain injury [Al-Bsharat et al., 1999; Bayly et al., 2005; Gosh et al., 1970; Hardy, et al., 1997; Hardy et al., 2001; Hodgson et al., 1966; Troseille et al., 1992].

Brain tissue deformation can be quantified using strain or stress. Strain is measured by comparing the length of the deformed brain tissue to its original length. Its mathematical form is:

$$\varepsilon = \frac{\Delta L}{L_0}$$

where ε is strain, ΔL is change in length (m) and L_0 is the original length (m). The unit of strain is m/m and is therefore reported as a percentage.

Stress is a measure of the force used to deform the brain tissue. Unidirectional stress is obtained with the following equation:

$$\sigma = \frac{F}{A}$$

where σ is stress, F is the applied force (N) and A is the unit of area stress is applied to (m^2). The unit of stress is the pascal (Pa). Tensile stress pulls apart the brain tissue, compressive stress pushes it towards each other, and shear stress skews it (Fig. 2.6).

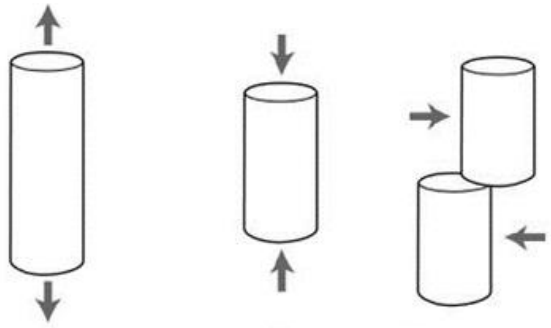


Figure 2.6. Illustration of tensile stress (left), compressive stress (centre), and shear stress (right)

The mechanical (structural) and physiological (functional) tolerance levels of materials are characterised using the relationship between stress and strain [Goldsmith and Plunkett, 2004]. For example, an artery subject to tensile stress greater than its tensile strength will tear (mechanical failure), causing intracranial haematoma [Goldsmith and Plunkett, 2004]. Physiological failures occur at lower levels and may temporarily disturb normal activity [Galbraith et al., 1993]. It is unclear whether concussion is the result of mechanical or physiological failure of the neuronal tissue or a combination of both.

The behaviour of tissue under stress is in part dependent of its composition, which determines whether the atoms composing it bind to resist the stress or reorient to deform [Bandak, 1995]. Elastic behaviour designates the property of a material to return to its original

shape following the unloading of a stress, like the human skull [Goldsmith and Plunkett, 2004]. The brain, on the other hand, is a viscoelastic tissue meaning that energy is dissipated during its deformation and not completely returned when the tissue regains its initial shape [Goldsmith and Plunkett, 2004]. It is also rate dependant meaning that the amount of energy absorbed prior to tissue failure is influenced by the rate of loading [Galbraith et al., 1993; LaPlaca et al, 1997; Singh et al., 2009; Viano et al., 1989].

Von Mises stress

Von Mises stress is a combination of the stresses along the three orthogonal axes (x, y, and z) and was reported to be a better representation of the stresses experienced by brain tissue [Horgan and Gilchrist, 2003; Horgan and Gilchrist, 2004; Kleiven, 2007; Willinger and Baumgartner, 2003; Zhang et al., 2004]. High von Mises stress localized in the upper brainstem and thalamus regions and shear stress in the upper brainstem were identified as better concussion predictors than HIC, GSI, and acceleration [Zhang et al., 2004].

Von Mises stress is calculated using the following equation:

$$2\sigma_v^2 = (\sigma_1 - \sigma_2)^2 + (\sigma_2 - \sigma_3)^2 + (\sigma_1 - \sigma_3)^2$$

where $\sigma_v$is the von Mises stress and σ_1, $\sigma_2$and $\sigma_3$are principal stresses, along the three axes. This measure was created to determine if a structure was near its yielding point by combining all six degrees of freedom into one metric for tension.

Von Mises stress thresholds were first proposed following FE analyses of the NFL cases reconstructed by Pellman et al. [2003]. Zhang et al. [2004] selected 12 of the events which

resulted in 9 concussions and used an FE model developed at Wayne State University validated against cadaveric pressure [Nahum et al., 1977] and brain displacement [Troseille et al., 1992]. A 25%, 50% and 80% concussion risk were estimated to be associated with peak shear stress magnitudes of 6.0 kPa, 7.8 kPa, and 10.0 kPa, respectively. A similar threshold, 8.4 kPa for a 50% risk, was proposed by Kleiven [2007] who used the FE model developed at the Royal Institute of Technology in Stockholm to analyse a larger data set (58 cases of which 29 were concussions). The data reported by Zhang, Yang, and King [2004] revealed that the highest stress was present in the upper brainstem and thalamus regions while shear stress in the upper brainstem was the best predictor of concussion. Kleiven [2007] on the other hand found that the highest von Mises stress was present in the midbrain and brainstem while von Mises stress in the corpus callosum was identified as the best predictor of concussion. Considering that the input was practically identical (NFL impacts), the different injury patterns between both studies are likely due to differences in models characteristics.

Von Mises stress has also been linked to concussion following the reconstruction of 64 real-world impacts [Willinger and Baumgartner, 2003]. Reconstructed motorcycle and American football accidents were performed by impacting a headform while accidents involving pedestrians were reconstructed using an analytical model. Shear stress was calculated using a FE model developed at the Strasbourg Louis Pasteur University validated against cadaveric intracranial pressure [Nahum et al., 1977], brain displacement [Troseille et al., 1992] and skull fracture [Yoganandan et al., 1995]. The analysis showed that von Mises stress was a good indicator for neurological lesions and identified 18 kPa for a 50% risk of moderate lesions

[Willinger and Baumgartner, 2003]. The term “moderate” was not defined; nevertheless, knowing that 22 cases were taken from the NFL studies [Pellman et al., 2003], it was inferred that concussion falls in that category.

Marjoux et al. [2008] analysed the same data set and published much higher thresholds for moderate neurological lesions. Going through the same process as Willinger and Baumgartner [2003], they found that von Mises stress values of 20 kPa, 27 kPa and 35 kPa were associated with a 5, 50 and 95% risk of moderate neurological injury, respectively. The difference may be explained by the classification of injury severity. While Willinger and Baumgartner [2003] did not provide the reader with a definition, Marjoux et al. [2008] labelled injuries as moderate when loss of consciousness lasted at least 30 minutes and less than 24h, excluding all but the most severe concussions.

Maximum principal strain

The effect of strain on neuronal conductivity was measured by Galbraith et al. [1993]. By using giant squid axons, the authors were able to study the physiological response associated with an applied load without the interference of surrounding structures or physiological interactions. Giant squid axons are easy to manipulate due to their size, absence of myelination which provided uniform properties and the fibres remain functional for hours following their excision from the body. It was observed that following an elongation of 0.12 in 14 milliseconds the axon was unable to elicit an action potential response when stimulated. This loss of functionality was only transient and the action potential reappeared after a three minutes period of rest. Axons subjected to strains above 0.20 were found to never fully regain their

resting potential while axons subjected to strains above 0.25 failed structurally. While the changes observed cannot be extrapolated to the entire nervous system, the data is strong evidence that deformation can cause electrical dysfunction in neurons.

Axonal trauma was also studied using guinea pig optic nerve to determine the magnitude of strain necessary to cause functional and structural failure [Bain and Meaney, 2000]. Functional impairment was assessed by measuring changes in electrical potentials; failure was identified when the peaks were located two standard deviations outside what was considered normal. Structural failure was assessed by light microscopy three days post-injury and was reported if axonal swellings or retraction balls in any section of the nerve were present. Functional thresholds were determined to be 0.13, 0.18 and 0.28 for a conservative, liberal and optimal threshold, respectively. Structural thresholds were determined to be 0.14, 0.21 and 0.34 for a conservative, liberal and optimal threshold, respectively. A conservative threshold was defined as one where no false negatives are identified (sensitivity of 1), a liberal threshold was defined as one where no false positives are identified (specificity of 1) and an optimal threshold was defined as one where the sum of sensitivity and specificity is maximised. While the results of this study were limited to the guinea pig optic nerve, the authors believe that mechanical behaviour for brain tissue may not vary significantly from species to species and these strain-based thresholds may be applied to the human brain. Nevertheless, the authors recognise that the proposed axonal thresholds may be influenced by the white matter's microstructural arrangement.

The effect of low level strains was studied *in vivo* using organotypic brain slice cultures [Yu et al., 2009]. For the procedure, the hippocampus of a rat pup was removed and thinly cut. The slices were transferred to silicone membrane complex and maintained in a cell-culture incubator for 16 days. The tissue was then stretched over a hollow indenter to create an equi-biaxial strain. While previous research reported a correlation between tissue cell death and strains above 0.10, the relationship between tissue deformation and tissue electrical function was unknown [Cater et al., 2006; Elkin and Morrison, 2007]. Six days after strains of 0.05 and 0.10 were applied, the hippocampal slices exhibited lower maximal evoked responses and required a higher stimulus to evoke a half maximal response. Thus, it was concluded that strains of 0.05 and 0.10 were sufficient to impair neuronal function.

Maximum principal strain represents the highest strain value along the three principal axes (x, y, and z) and is commonly used in head injury biomechanics to describe tissue deformation obtained with an isotropic brain finite element model [Horgan and Gilchrist, 2003; Horgan and Gilchrist, 2004; Kleiven, 2002; Willinger and Baumgartner, 2003; Zhou et al., 1995]. Maximum principal strain thresholds were proposed following FE analyses of the reconstructed NFL cases [Kleiven, 2007; Zhang et al., 2004]. Concussion risk levels were proposed by Zhang, Yang, and King [2004] using the NFL research data. The results identified the upper brainstem and thalamus as the regions subjected to the highest strain and a 25%, 50% and 80% probability of concussion was associated with strain values of 0.14, 0.19, and 0.24, respectively. An analysis performed on the same dataset but with a different FE brain model found similar results

[Kleiven, 2007]. A 50% probability of sustaining a concussion was associated with a maximum principal strain of 0.21 in the corpus callosum and 0.26 in the grey matter.

Strain rate

The effect of strain rate on neuronal conductivity was measured by Galbraith, Thibault, and Matteson [1993] using giant squid axon. For a similar elongation of 0.19, the membrane potential of axons exposed to a low strain rate (0.284 mm/s) returned to its resting level, while the membrane potential of axons exposed to a high strain rate (275 mm/s) had a brief period of hyperpolarization followed by a slow depolarization and required 30 min to recover its resting potential. Thus, it was suggested that strain-induced trauma was reduced at low strain rates by viscous dissipation. At higher strain rates, the influence of the viscous component was reduced and the axons reacted more like elastic materials.

The effect of loading rate was also studied by LaPlaca, Lee, and Thibault [1997] who measured the free calcium concentration ($[Ca^{2+}]_i$) and lactate dehydrogenase (LDH) released by human neuron-like cultured cells to assess cell damage. Following an exposure to fluid shear stress at rates ranging from 1.8 to 7.3 s^{-1}, the data showed that an increase in loading rate resulted in larger deformation and elevated peak $[Ca^{2+}]_i$, increased average $[Ca^{2+}]_i$, indicating an increase in membrane permeability, and immediate release of LDH, indicating cell damage.

Brain tissue damage was observed to be minimal when subjected to a strain of 0.10 at a rate of 5, 10, 20 and 50 s^{-1} [Morrison et al., 2003]. This indicates that strain rate may not have an influence on concussion when the magnitude of strain is low. Strain rate was shown to have an effect on brain tissue damage at a strain of 0.20, with the higher rates generating higher tissue

damage. Furthermore, brain tissue subjected to strains of 0.35 and 0.50 at a rate of 10 s^{-1} recovered within the first four days. This was not the case for tissue which was subjected to identical strains applied at a rate of 50 s^{-1} which generated lasting damage and cell death. This study was performed on organotypic hippocampal slice cultures and was designed to be an exploratory study with no statistical significance reported.

Following FE analyses of the NFL reconstruction data, the average strain rate was reported as 84 s^{-1} for injury cases and 38 s^{-1} for non-injury cases, respectively [King et al. 2003]. Mid-late strain rate (between 60 and 80 s^{-1}), referring to peaks occurring later in the analysis, in the midbrain and fornix showed the strongest correlations with the diagnosis of concussion [Viano et al., 2006], The thresholds for a 25%, 50% and 75% probability of concussion were associated to strain rate values of 46, 60, and 80 s^{-1}, respectively [King et al. 2003].

Summary

In summary, concussion is a complex pathophysiological process which typically resolves in a few days [McCrory et al., 2009]. Athletes diagnosed with concussion experience a range of signs and symptoms which may be a manifestation of a depressed state caused by a metabolic imbalance caused by a mechanical stretch of the neuronal tissue.

Upon contact, momentum and energy are transferred to a struck athlete's head causing it to change its motion. Due to inertia, the brain and the skull do not move as a unit, which creates an increase in pressure at the impact site and imparts stress to the soft neuronal tissue

[Gurdjian and Gurdjian, 1975; Thomas et al., 1967]. Stretching of the neuronal tissue creates a sudden increase of extracellular K^+ that in turn triggers an excessive release of excitatory amino acids such as glutamate and an influx of Ca^{2+} [Katayama et al., 1990]. The brain enters a hypermetabolic state as it attempts to reabsorb the K^+; unfortunately, the increased demand in glucose and ATP cannot be met because cerebral blood flow is reduced by the accumulation of intracellular Ca^{2+} which impairs mitochondrial functioning [Giza and Hovda, 2001; Hovda et al., 1999]. Electrolyte homeostasis is usually restored within minutes but the brain may remain in a depressed state for several days [Katayama et al., 1990]. It was suggested that this phenomenon may be responsible for loss of consciousness, memory loss and cognitive alterations associated with concussion [Giza and Hovda, 2001].

Neuronal impairment has been observed experimentally. The inability of eliciting an action potential was reported following axonal elongations between 0.05 and 0.2 while elongations above 0.25 resulted in structural failure [Bain and Meaney, 2000; Cater et al., 2006; Elkin and Morrison, 2007; Galbraith et al., 1993]. The rate at which the neuronal tissue deformed was also reported to influence brain function impairment. A high strain rate was observed to increase membrane permeability to Ca^{2+} and lengthen recovery time [Galbraith et al., 1993; LaPlaca et al., 1997]. The effects of strain rate were reported to have little effect when the neuronal tissue was stretched by 0.10 [Morrison et al., 2003]. While important, correlating axonal trauma to strain and strain rate does not provide insight in the level of stress necessary to create the signs and symptoms that are typical of concussion. Moreover, they do not provide

information concerning the mechanism of injury which is critical for successful prevention and treatment.

The emergence of sophisticated brain models provides an opportunity to study the mechanical metrics associated with common events which are known cause concussion. Recently, researchers began to study the relationship between mechanical metrics and concussion using sports as a source of data. Concussive events captured on camera have been reconstructed in laboratory using anthropomorphic test dummies [Pellman et al., 2003]. Thresholds for concussion were proposed for peak resultant linear acceleration, peak resultant angular acceleration, maximum principal strain, strain rate, and von Mises stress [Kleiven, 2007; Marjoux et al., 2008; Willinger and Baumgartner, 2003; Zhang et al., 2004]. Unfortunately, these studies all used the same data set that is specific to American football collisions and have yet to be replicated. While impact velocity, orientation, and location were obtained using video analysis, effective mass was assumed to be comprised of the mass of the dummies and any active muscle force generated by the both athletes was disregarded. Furthermore, it was assumed that compliance of the collision was accurately recreated using two 50^{th} percentile Hybrid III dummies equipped with American football protective equipment.

It is hypothesized that a similar data set could be obtained using elite ice hockey as a source of concussive events. The sport lends itself well to a biomechanical analysis as it is played on a small surface (in comparison to American football or Australian rules football) marked with lines and circles, the motion of the players is captured by multiple cameras allowing for many angles, the players wear standardized equipment and medical experts are often on-site to

diagnose concussion immediately. Effective impact mass and compliance could be estimated by using human test subjects. This has been done in boxing and soccer and has provided researchers with valuable data concerning punching and elbowing biomechanics [Walilko et al., 2005; Withnall et al., 2005]. This additional biomechanical information would provide a more accurate reconstruction methodology which could provide the necessary input for finite element brain models.

Chapter Three

First Study

Defining the Effective Impact Mass of Elbow and Shoulder Strikes in Ice Hockey

Introduction

Ice hockey was identified as the collegiate sport having the highest incidence of concussion per athlete exposure [Hootman et al., 2007]. Concussion in ice hockey accounts for 6 to 14% of all sport-related injuries and has an incidence of 1.5 to 18.7 per 1 000 player-game hours [Covassin et al., 2003; Jørgensen and Schmidt-Olsen, 1986; Koh et al., 2003; Pelletier et al., 1993; Tegner and Lorentzon, 1996]. While symptoms typically resolve spontaneously over a short period of time, some cases require a lengthy period of time to fully recover [Binder et al., 1997; McCrory et al., 2009] and may lead to the early onset of dementia [Guskiewicz et al., 2005] and chronic traumatic encephalopathy (CTE) [Stern et al., 2011].

Competitive ice hockey players are typically struck to the head a couple of times during a game [Brainard et al., 2009]; yet most complete the season without being diagnosed with a concussion [Echlin et al., 2010; Emery and Meeuwisse, 2006; Koh, Cassidy, and Watkinson, 2003; Wennberg and Tator, 2003]. This suggests that players, coaches and trainers are poor at diagnosing concussion but also that not all head impacts result in equal brain stress and strain.

Reconstruction of real life events can be used to investigate the relationship between the mechanical parameters of the impact and concussion risk [Fréchède and McIntosh, 2009; Newman et al., 1999; Zhang et al., 2004]. Such variables as impact velocity, location, orientation mass, and compliance of the striking bodies have all been shown to affect the response of the head and brain and should be replicated as accurately as possible when doing a reconstruction [Fréchède and McIntosh, 2009; Karton, 2012; Oeur, 2012; Walsh et al., 2011; Zhang et al., 2001].

The effective striking mass of a player coming into contact with another player's head has typically been approximated as being the mass of the body part coming into contact with the head without any additional muscular or inertial forces applied by the striking athlete [Fréchède and McIntosh, 2009; Newman et al., 1999]. Hockey players are taught to anticipate a collision and position themselves by flexing their knees, leaning forward, keeping their feet shoulder-width apart, and driving their legs through the collision. Striking technique has been reported to increase effective impact mass in boxing and football and it is expected to also influence effective striking mass in ice hockey body checking [Viano and Pellman, 2005; Viano et al., 2005; Viano et al., 2007; Walilko, Viano, and Bir, 2005]. Considering that an increase in mass was reported to increase head acceleration [Karton, 2012]; defining the effective mass of a head collision in ice hockey is important to ensure the validity of the reconstruction. Upper extremities are the most common body parts to contact the head in collisions resulting in concussion [Hutchison, 2011]; thus, the purpose of this study was to document the effective impact mass of elbow and shoulder strikes to the head.

Methods

The effective impact mass of elbow-to-head and shoulder-to-head collisions was calculated in laboratory using the change in velocity of the striking player and a suspended Hybrid III headform. Figure 3.1 illustrates the three common striking techniques analyzed in this study which were: 1) *extended elbow*, defined as contact with an elbow extended away from the body; 2) t*ucked-in elbow*, defined as contact with an elbow located close to the torso; and 3) *shoulder check*, defined as contact with the shoulder. Any obvious attempt to strike an opponent to the head is severely penalized in ice hockey; most elbow strikes are the result of accidental, or careless, contact. The test subjects were thus instructed to not actively strike the headform but rather to skate into it while bracing their bodies to withstand the impact for the elbow conditions. The instructions were different for the shoulder condition as it was designed to replicate an intentional body check; thus, the athletes were told to skate forcefully and align themselves to maximize the force of the impact.

Figure 3.1. Typical ice hockey body-to-head collisions. The three techniques shown are extended elbow (left), tucked-in elbow (centre), and shoulder check (right)

Experimental procedure

Fifteen test subjects were instructed to strike a suspended 50th percentile Hybrid III headform at least three times for each of the three techniques. Video clips of the desired technique were shown prior to testing to ensure they understood what was required from them. The tests were performed on rollerblades and consisted of a quick acceleration followed by a glide phase to prepare for impact with the headform (Fig. 3.2). Test subjects were allowed to warm up and stretch before beginning as well as take practice runs to ensure that they were able to comfortably strike the headform. They were equipped with the same elbow, and shoulder protective gear.

Figure 3.2. Illustration of the apparatus used to suspend the Hybrid III headform

The temporal region of the headform was targeted to remove the influence of headform geometry [Foreman, 2010]. A rectangular target (h: 3.4 cm; w: 5.0 cm) placed in-line with the headform's centre of gravity. The accuracy of the strikes was assessed by reviewing the high speed video. Impacts above or below the target were clearly visible while impacts to the left or right of the target were identifiable due the headform's resulting Z-axis (longitudinal) rotation. Impacts that generated angular accelerations above 2.0 krad/s^2 were not recorded and participants were asked to repeat the trial.

A 50th percentile Hybrid III headform (FTSS, Plymouth, MI) was used to represent the struck player's head. It had a mass of 4.54 kg and was engineered to respond similarly to a human head under impact [Foster et al., 1974; Hubbard and McLeod, 1974; Mertz, 1985]. The headform was suspended and free to move post-impact. The height was adjustable allowing the test subjects to strike the headform in a manner that was comfortable and natural. The mass of the body below the neck was not added in this study to facilitate the calculation of the effective impact mass of the striking player using conservation of momentum. . It was not intended to replicate the complex kinematics of a collision between two players in movement. The protocol was reviewed and approved by the University of Ottawa Office of Research Ethics and Integrity.

Volunteer test subject

Fifteen male volunteers with experience playing competitive ice hockey participated in the study. The average age was 27.8 ± 7.4 years (range: 18 to 44 years), the average height was

1.79 ± 0.07 m (range: 1.65 to 1.91 m), and the average mass was 88.5 ± 9.5 kg (range: 69.4 to 104.3 kg). A description of each subject is given in table 3.1.

TABLE 3.1
Description of participating competitive ice hockey players

ID	Age (years)	Experience (league)	Height (m)	Mass (kg)
S01	24	Minor hockey	1.80	85.3
S02	19	Collegiate	1.80	69.4
S03	19	Junior hockey	1.83	84.8
S04	30	Collegiate	1.80	104.3
S05	24	Collegiate	1.91	95.3
S06	29	Junior hockey	1.78	99.8
S07	29	Professional	1.77	88.9
S08	25	Junior hockey	1.91	98.4
S09	30	Collegiate	1.75	81.6
S10	42	Minor hockey	1.83	89.4
S11	28	Junior hockey	1.75	92.5
S12	27	Collegiate	1.65	77.6
S13	29	Minor hockey	1.68	78.1
S14	44	Minor hockey	1.85	95.3
S15	18	Junior hockey	1.75	86.2

Instrumentation

Nine single-axis Endevco (San Juan Capistrano, CA) 7264C-2000 accelerometers were mounted inside the headform to capture its three dimensional movement. They had a measurement range of ± 500 g and were calibrated by Humanetics (Plymouth, MI). They were positioned in an orthogonal arrangement following a 3-2-2-2 array [Padgaonkar et al., 1975]. Head acceleration was collected at a frequency of 20 kHz and filtered using a 300 Hz low-pass filter in accordance with the SAE J211 protocol. The accelerometer signals were passed through a TDAS Pro Lab system (DTS, Calabasas CA) before being processed by TDAS software.

Impacts were captured with a calibrated PCI-512 Fastcam high-speed camera (Photron USA Inc., San Diego, CA) collecting at 100 fps. A faster frame rate creates minimal displacement between frames that inhibits velocity measurements. The camera was located perpendicular to the line of action creating a sagittal view of the impact. The camera's position, orientation, zoom, and field of vision was fixed during the whole data collection (Fig. 3.3).

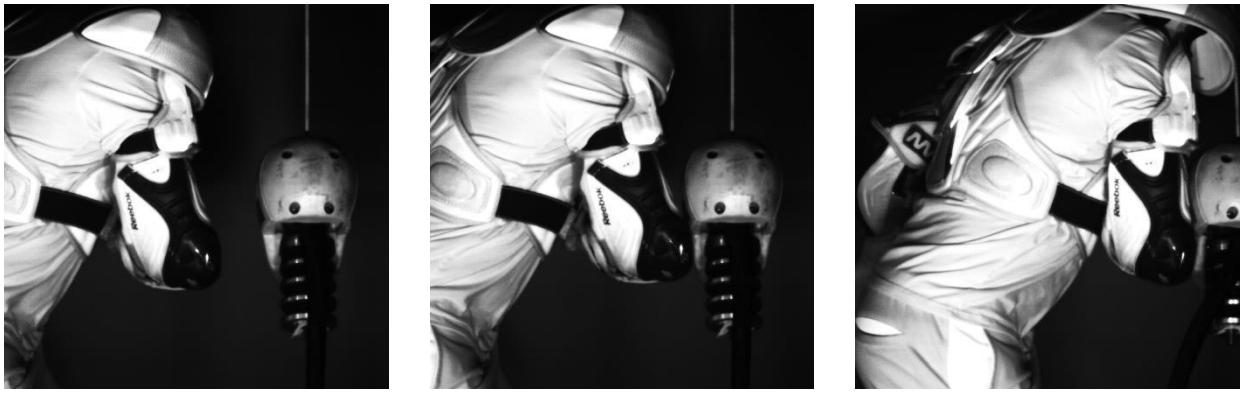


Figure 3.3. Sagittal view of a test subject performing a tucked-in strike

Velocity measurement

Velocity of the elbow or shoulder prior to the impact was captured using Photron motion analysis software (Photron USA Inc., San Diego, CA). The camera was set at a rate of 100 fps, thus time between each frame was known 0.01 s. Image digitization was done using the width of the jaw of the headform (0.136 m) as a reference. Velocity was calculated by dividing the average distance travelled in the last ten frames prior to contact by 0.1 s.

Analysis of pilot data revealed that the test subjects's elbow or shoulder coupled with the Hybrid III headform and moved as one following the collision (Fig 3.4). This was also observed by other researchers and confirmed that the complex mechanics of two bodies colliding could be simplified using inelastic collision mechanics [Budescu and Iacob, 2008; Walilko et al., 2005].

Thus, velocity of the limb coupled with the Hybrid III headform was calculated by integrating the full centre of gravity Y-axis acceleration curve to ensure that the compression phase and restitution phase had ended.

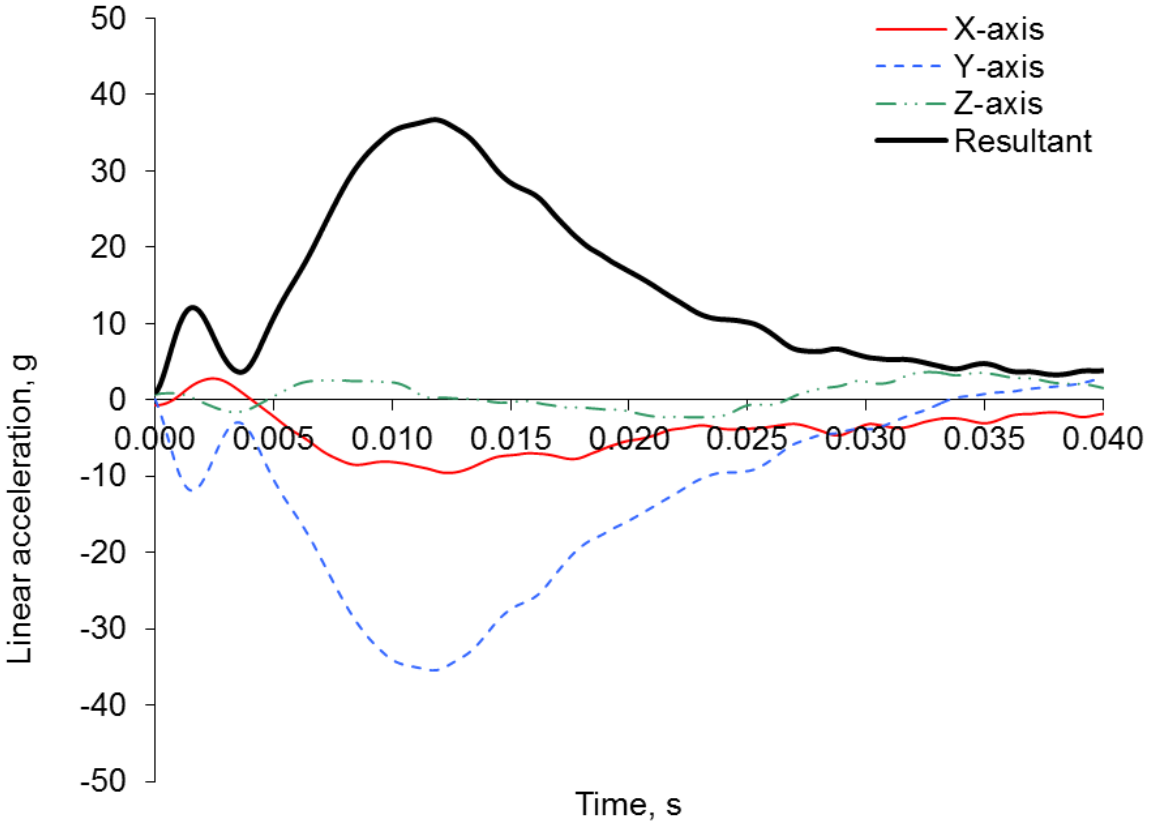


Fig. 3.4. Head linear acceleration history of a shoulder check. The image on the left is at time 0 s (contact), the image at the centre is at time 0.012 s (peak acceleration), and the image at the right is at time 0.030 s (acceleration back to baseline).

Effective mass calculation

During a collision, contact force, $F(t)$, is imparted to both bodies and can be integrated over a time period to obtain linear momentum, $p\ (t)$

$$p(t) = \int_{t_0}^{t} F(t)\, dt \quad [1]$$

Using Newton's second and third laws, the equation can be rewritten as

$-\Delta p = m_1 \Delta v_1$ and $\Delta p = m_2 \Delta v_2$ [2]

where, m_1 and m_2 are the effective impact masses, and Δv_1 and Δv_2 are the velocity variations of the striking and struck bodies, respectively. Therefore, the velocity of both players at the end of the collision can be written as

$$u_1 = v_1 - \frac{p}{m_1} \quad [3]$$

$$u_2 = v_2 + \frac{p}{m_2} \quad [4]$$

If the relative velocity between the two bodies, v_{rel}, is defined as

$$v_{rel} \equiv v_1 - v_2 \quad [5]$$

then

$$\Delta v_{rel} = \Delta v_1 - \Delta v_2 = -\frac{\Delta p}{m_1} - \frac{\Delta p}{m_2} = -\left(\frac{1}{m_1} + \frac{1}{m_2}\right)\Delta p = -\frac{\Delta p}{m_{1\cup 2}} \quad [6]$$

where $m_{1\cup 2} = \frac{m_1 m_2}{m_1 + m_2}$ and can be considered as the mass of the system (both players).

During the collision, the variation of the relative velocity is obtained by

$$v_{rel_f} = v_{rel_i} - \frac{p}{m_{1\cup 2}} \quad [7]$$

Considering that a collision between two players can be represented by an inelastic collision [Budescu and Iacob, 2008; Walilko et al., 2005] where both bodies are moving together, v_{rel_c} is equal to zero. Thus, equation [7] becomes

$$p = m_{1\cup 2} v_{rel_i} = \frac{m_1 m_2}{m_1 + m_2}(v_1 - v_2) \quad [8]$$

A substitution of equation [8] into equations [3] and [4] can be used to calculate the final velocity of the striking player, u_1, and the struck player, u_2, following a collision

$$u_1 = v_1 - \frac{p}{m_1} = v_1 - \frac{m_2}{m_1 + m_2}(v_1 - v_2) \quad [17]$$

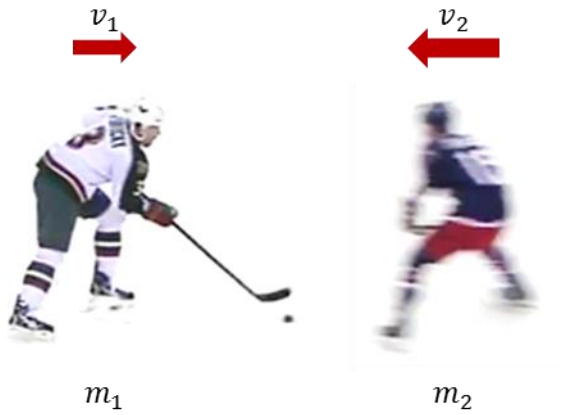
$$u_2 = v_2 + \frac{p}{m_2} = v_2 + \frac{m_1}{m_1 + m_2}(v_1 - v_2) \quad [18]$$

Effective mass of the striking player can be readily obtained by rearranging equation [17]

$$m_{striking} = \frac{-\, m_{head}\, v_{striking}}{u_{striking} - v_{striking}} - \, m_{head} \quad [19]$$

where $m_{striking}$ is the effective impact mass, m_{head} is the mass of the headform, $v_{striking}$ is the velocity of the striking body prior to the impact, and $u_{striking}$ is the velocity of the striking body following the impact as illustrated in figure 3.5.

a)



b)

c)

Figure 3.5 Typical impact sequence in ice hockey. Players come into contact at a certain velocity (a); the force of the impact creates an impulse which modifies their velocities (b); both athletes will move in the same direction for a short period of time until other forces come into play (c)

Prior to testing, the accuracy of the method was experimentally assessed. The experiment consisted of five impacts to a suspended Hybrid III headform using a 9.79 kg pendulum. Error between the mass of the pendulum and the calculated mass is reported in table 3.2.

TABLE 3.2
CALCULATED MASS AND VARIANCE OF A PENDULUM STRIKE TO A HYBRID III HEADFORM

Impact	Striking velocity (m/s)	Calculated effective impact mass (kg)	Percentage difference from actual mass (%)
1	4.25	9.65	-1.6
2	4.24	9.91	1.0
3	4.32	9.45	-3.7
4	4.15	10.16	3.5
5	4.15	9.89	0.9
Average		9.81 ± 0.27	2.2 ±1.3

Statistics

A one-way analysis of variance was conducted to evaluate differences in effective impact mass between the extended elbow, tucked-in elbow, and shoulder check conditions. Post-hoc comparisons using the Tukey HSD test were conducted when statistical significance, set at an alpha of 0.05, was found. Pearson correlations were performed to assess the relationship between effective impact mass, body mass, and velocity.

Results

A summary of the results is presented in table 3.3, more details can be found in tables 3.4 to 3.6. Linear acceleration histories used to calculate post-impact velocities are illustrated in figures 3.6 to 3.8.

TABLE 3.3
Effective impact mass of shoulder and elbow to head impacts in ice hockey

	Striking velocity (m/s)	Post-impact velocity (m/s)	Effective impact mass (kg)	Percentage of body mass (%)
Extended elbow	5.5 ± 0.9	2.6 ± 0.6	4.8 ± 1.7	5.4 ± 1.8
Tucked-in elbow	6.3 ± 1.8	2.2 ± 0.4	3.0 ± 0.8	3.4 ± 0.8
Shoulder check	6.1 ± 0.8	4.3 ± 0.7	12.9 ± 3.3	14.6 ±3.6

Mean effective impact mass of the extended elbow was 4.8 ± 1.7 kg. Accounting for body mass, which had a low correlation with striking mass ($r=0.437$, $p=0.003$), the tested ice hockey players used on average 5.4 ± 1.8% of their body mass when striking a static Hybrid III headform with an extended elbow. Impact velocity ranged from 3.6 to 7.7 m/s and its correlation with striking mass was not statistically significant ($r= -0.239$, $p=0.114$).

The effective impact mass of the tucked-in elbow was lower than the extended elbow ($p<0.001$) with a mean of 3.0 ± 0.8 kg. Accounting for body mass, which once again had a low correlation with striking mass ($r=0.369$, $p=0.015$), the tested ice hockey players used on average 3.4 ± 0.8% of their body mass when striking a static Hybrid III headform with a tucked-in elbow. Impact velocity ranged from 4.1 to 9.5 m/s and its correlation with striking mass was not statistically significant ($r= -0.207$, $p=0.183$).

The effective impact mass of the shoulder check was highest ($p<0.001$) with a mean of 12.9 ± 3.3 kg. Accounting for body mass, which had a low correlation with striking mass ($r=0.266$, $p=0.089$), the tested ice hockey players used on average 14.6 ± 3.6% of their body mass when striking a static Hybrid III headform with a their shoulder. Impact velocity ranged from 4.8 to 7.5 m/s and its correlation with striking mass was not statistically significant ($r=0.229$, $p=0.145$).

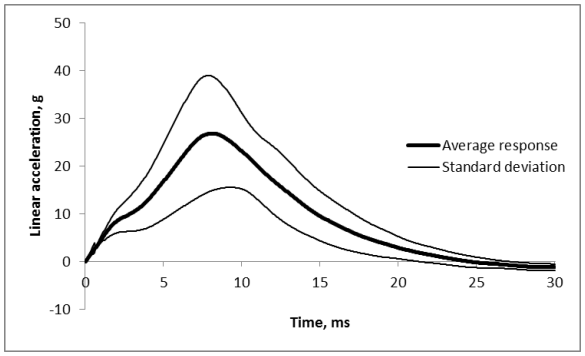


Fig. 3.6. Linear acceleration history (mean and standard deviation) of an extended elbow impact to a Hybrid III headform

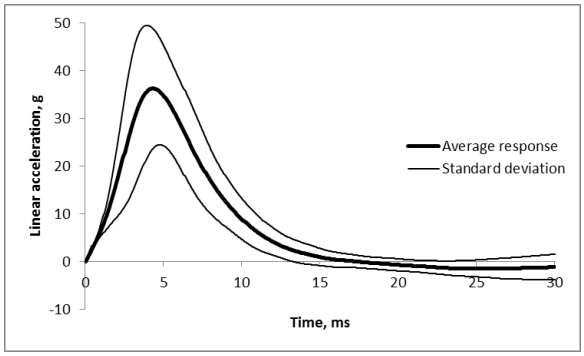


Fig. 3.7. Linear acceleration history (mean and standard deviation) of a tucked-in elbow impact to a Hybrid III headform

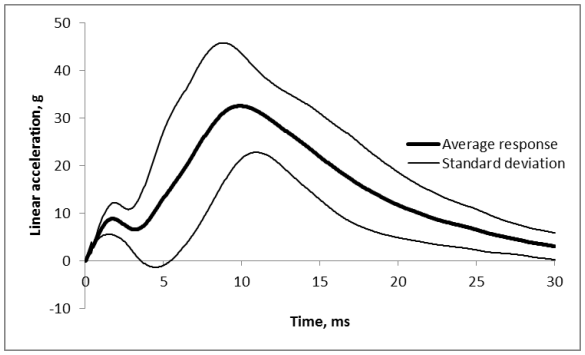


Fig. 3.8. Linear acceleration history (mean and standard deviation) of a shoulder check to a Hybrid III headform

TABLE 3.4

Effective impact mass of an extended elbow strike to the head in ice hockey

ID	Striking velocity (m/s)	Post-impact velocity (m/s)	Effective impact mass (kg)	Percentage of body mass (%)
S01	5.9 ± 0.5	3.2 ± 0.3	5.6 ± 0.5	6.6 ± 0.6
S02	6.1 ± 0.4	2.5 ± 0.2	3.4 ± 0.5	4.9 ± 0.7
S03	5.1 ± 0.8	2.0 ± 0.1	3.3 ± 0.4	3.9 ± 0.5
S04	4.6 ± 0.3	2.5 ± 0.1	5.4 ± 0.4	5.2 ± 0.4
S05	5.2 ± 0.7	2.6 ± 0.2	5.2 ± 0.4	5.5 ± 0.5
S06	4.7 ± 0.3	2.2 ± 0.2	4.2 ± 0.4	4.2 ± 0.4
S07	5.1 ± 0.2	3.2 ± 0.01	8.1 ± 0.7	9.1 ± 0.7
S08	6.1 ± 1.0	2.7 ± 0.01	4.7 ± 0.3	4.8 ± 0.3
S09	7.2 ± 0.4	3.5 ± 0.3	4.6 ± 0.3	5.6 ± 0.4
S10	4.5 ± 0.1	2.7 ± 0.1	7.3 ± 0.6	8.2 ± 0.7
S11	6.1 ± 0.2	2.7 ± 0.4	3.9 ± 0.8	4.2 ± 0.9
S12	5.5 ± 0.3	2.5 ± 0.1	4.0 ± 0.3	5.2 ± 0.3
S13	5.5 ± 0.5	1.9 ± 0.7	2.7 ± 1.2	3.5 ± 1.5
S14	6.7 ± 0.6	3.6 ± 0.6	5.8 ± 1.3	6.1 ± 1.4
S15	4.0 ± 0.4	2.1 ± 0.1	5.5 ± 0.9	6.3 ± 1.1
Average	5.5 ± 0.9	2.6 ± 0.6	4.8 ± 1.7	5.4 ±1.8

TABLE 3.5

Effective impact mass of a tucked-in elbow strike to the head in ice hockey

ID	Striking velocity (m/s)	Post-impact velocity (m/s)	Effective impact mass (kg)	Percentage of body mass (%)
S01	6.1 ± 0.4	2.2 ± 0.1	2.8 ± 0.5	3.3 ± 0.6
S02	8.8 ± 0.7	2.5 ± 0.2	2.0 ± 0.04	2.9 ± 0.1
S03	6.6 ± 0.4	2.3 ± 0.3	2.6 ± 0.3	3.1 ± 0.3
S04	5.4 ± 0.1	2.1 ± 0.1	3.1 ± 0.2	2.9 ± 0.2
S05	6.5 ± 0.2	2.4 ± 0.3	3.0 ± 0.4	3.1 ± 0.4
S06	4.4 ± 0.3	1.8 ± 0.2	3.8 ± 0.3	3.8 ± 0.3
S07	4.9 ± 0.1	2.1 ± 0.01	3.7 ± 0.2	4.1 ± 0.2
S08	6.6 ± 0.5	2.7 ± 0.1	3.4 ± 0.1	3.4 ± 0.1
S09	8.3 ± 0.3	2.6 ± 0.3	2.2 ± 0.3	2.7 ± 0.3
S10	5.7 ± 0.4	1.7 ± 0.2	2.0 ± 0.2	2.3 ± 0.2
S11	6.8 ± 0.05	2.3 ± 0.3	2.6 ± 0.5	2.8 ± 0.5
S12	5.8 ± 0.4	1.8 ± 0.1	2.1 ± 0.01	2.8 ± 0.02
S13	5.8 ± 0.4	1.7 ± 0.1	2.1 ± 0.2	2.7 ± 0.2
S14	6.2 ± 0.2	3.0 ± 0.1	4.5 ± 0.3	4.7 ± 0.3
S15	5.5 ± 1.0	2.2 ± 0.2	3.5 ± 0.6	4.0 ± 0.7
Average	6.3 ± 1.8	2.2 ± 0.4	2.8 ± 0.7	3.2 ± 0.6

TABLE 3.6

Effective impact mass of a shoulder check to the head in ice hockey

ID	Striking velocity (m/s)	Post-impact velocity (m/s)	Effective impact mass (kg)	Percentage of body mass (%)
S01	-	-	-	-
S02	6.8 ± 0.4	4.8 ± 0.3	11.9 ± 1.4	17.2 ± 2.1
S03	5.2 ± 0.4	4.0 ± 0.2	15.7 ± 1.7	18.6 ± 2.1
S04	6.4 ± 0.3	4.6 ± 0.4	13.2 ± 3.3	12.6 ± 3.1
S05	6.3 ± 1.1	4.7 ± 0.7	14.7 ± 1.4	15.4 ± 1.5
S06	6.6 ± 0.2	4.5 ± 0.3	19.0 ± 4.3	19.0 ± 4.3
S07	7.1 ± 0.3	5.2 ± 0.2	13.9 ± 0.6	15.6 ± 0.7
S08	7.0 ± 0.4	4.8 ± 0.01	11.4 ± 2.2	11.6 ± 2.2
S09	6.4 ± 0.4	4.8 ± 0.3	14.4 ± 2.6	17.6 ± 3.2
S10	6.9 ± 0.3	4.2 ± 0.4	13.6 ± 3.1	15.2 ± 3.5
S11	6.4 ± 0.6	4.7 ± 0.3	13.4 ± 2.1	14.4 ± 2.3
S12	5.0 ± 0.1	3.4 ± 0.3	10.5 ± 2.0	13.5 ± 2.6
S13	5.7 ± 0.1	3.9 ± 0.2	11.0 ± 2.2	14.1 ± 2.8
S14	5.5 ± 0.6	3.7 ± 0.4	10.7 ± 1.2	11.2 ± 1.3
S15	5.0 ± 0.3	3.0 ± 0.2	7.3 ± 0.8	8.5 ± 0.9
Average	6.1 ± 0.8	4.3 ± 0.7	14.2 ± 3.0	15.9 ± 3.3

Discussion

This study was designed to measure the effective impact mass of elbow-to-head and shoulder-to-head impacts in ice hockey. Fifteen hockey players struck an anthropomorphic headform with their extended elbow, tucked-in elbow and shoulder for a total of 135 impacts. The results indicated that impacts delivered with the shoulder involved the highest amount of mass with an average near 15% of a player's body mass while impacts performed with an elbow were between 3% and 5%, depending on its alignment with the striking player's body. The increase in effective impact mass suggests that the players were better at bracing themselves when their elbow was extended then when their arm was aligned with their torso. This was unexpected as it was hypothesized that the arm would couple with the trunk during the tucked-in elbow impact thus incorporating mass in the impact. While the arm did bind with the player`s torso, it happened long after the 20 ms taken to impart acceleration to the headform. The observed difference between both elbow strike techniques is important because an increase in mass results in an increase in headform acceleration for a striking mass below 10 kg, [Karton, 2013]. It is difficult to compare the results to anthropometric data because the mass of the segment is reported and the relative contribution of each is unknown. Comparisons with other studies should be done cautiously due to differences in technique and equipment used; nevertheless, the masses used in the elbow strikes were comparable to the calculated mass of a straight punch to the jaw (2.9 ± 2.0 kg) [Walilko et al., 2005] while the mass of the shoulder strike was comparable to the suggested mass of a head-to-head collision in football (14.0 kg) [Viano and Pellman, 2005].

Limitations

The results presented in this study are specific to the size and composition of the population tested. Fifteen participants were recruited for this study, which is higher than what has previously been done in impact biomechanics research. Typically, 4 to 23 participants are recruited per study and further divided in groups or weight classes [Schwartz et al., 1986; Smith et al., 2000; Viano et al., 2005; Walilko, Viano, and Bir, 2005; Whiting et al., 1988; Withnall et al., 2005]. Despite the relatively low number of participants, the standard deviations associated to the three techniques are small indicating that the mean is not likely to change dramatically. Most of the ice hockey players who participated in this study did not play at a professional level. While each of them played competitive hockey at some point of their life, it is possible that they do not have the strength and power of professional athletes of the same age. While professional ice hockey players may be more skilled at using these techniques in a game situation, it is reasonable to hypothesize that they have similar striking mass when hitting a static Hybrid III headform in laboratory.

The data were collected in a controlled laboratory setting that did not replicate an actual ice hockey game. Factors such as fatigue may modify a player's technique and thus change the effective mass of an elbow or shoulder strike [Gabbett, 2008]. Furthermore, the impacts were delivered to a static headform, which does not replicate the rapid movements and maneuvers of the opponent. It is possible that the striking masses calculated in this study are different from those that are seen in a game; however, the calculated values remain a good representation and offer important information for impact reconstructions.

Conclusion

The purpose of this study was to document the effective impact mass of three common body-to-head collisions in ice hockey. Mean effective impact mass for impacts with an extended elbow, a tucked-in elbow, and a shoulder were 4.8, 3.0, and 12.9 kg, respectively. The data collected in this study can be used to guide the striking mass during laboratory reconstructions of collisions in ice hockey and thus better understand the injury mechanism of head injury.

Chapter Four

SECOND STUDY

HEAD DYNAMIC RESPONSE ANALYSIS OF REAL-WORLD CONCUSSIVE AND NON-INJURIOUS ELBOW AND SHOULDER TO HEAD COLLISIONS IN ICE HOCKEY

Introduction

Despite being the most common head injury, concussion remains poorly understood and the relationship between impact mechanics and the onset of the injury remains unknown. A number of research projects involving the biomechanics of head injury have been conducted over the last century yet only a few studies have been conducted using volunteer subjects [Bayly et al., 2005; Eiband, 1959]. For ethical reasons, impacts have to remain well under injury levels, providing little data concerning concussion mechanisms. While cadaveric heads have been used to study skull deformation and fracture [Gurdjian et al., 1950], impacting cadaveric heads does not reveal information concerning concussion mechanism as it cannot identify neural tissue trauma on a microscopic level. Animal models offer the possibility to test above injury thresholds and allow for a direct analysis of brain injury [Gennarelli et al., 1971; Gennarelli et al., 1972; Ommaya et al., 1966; Pudenz and Shelden, 1946]. Unfortunately, the

use of anaesthetics, differences in brain size, inherent tolerance and skull characteristics limit the benefits of data extrapolation.

A recent increase in the availability of data concerning real-world sports impacts resulting in diagnosed concussion has given researchers the opportunity to establish a direct link between impact mechanics and concussion [Fréchède and McIntosh, 2009; Kleiven, 2007; Pellman et al., 2003; Withnall et al., 2005]. Ice hockey is one of the contact sports that lend itself well to biomechanical analyses. The game is played on a small surface (in comparison to American football or Australian rules football) marked with lines and circles, the motion of the players is captured by multiple cameras allowing for many angles, the players wear standardized equipment and medical experts are often on-site to diagnose concussion. Thus, the purpose of this study was to characterize head dynamic response of concussive and non-injurious ice hockey elbow and shoulder to head strikes by reconstructing real-world collisions in laboratory.

Methods

Professional hockey games are played on an ice surface that adheres to the dimensions and specifications of their respective leagues. The ice rinks are 61.0 m in length and 25.9 m in width and covered in markings (lines and circles), which can be used to identify location and distances (Fig. 4.1). Cameras located around the playing surface sometimes offer an overhead as well as an eye-level view of a collision between two players. Distant views can be used to calculate impact velocity while close-ups can be used to determine impact location, direction and striking technique.

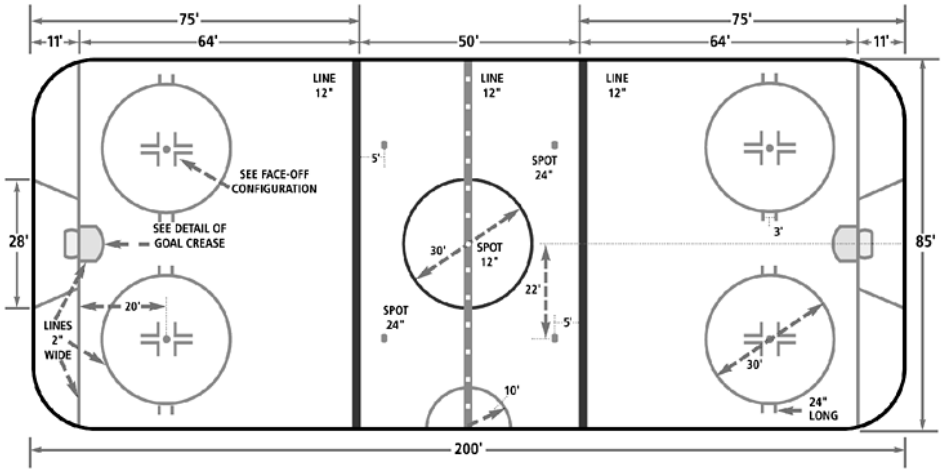


Figure 4.1. Standard North American ice rink dimensions in feet (imperial unit)

To accurately reconstruct a concussive event, the following parameters have to be determined: 1) velocity, 2) direction, 3) location, 4) impact mass, and 5) compliance. Impact velocity, orientation, and location were directly obtained using video analysis while impact effective mass and compliance were selected to replicate the mean response of elbow-to-head or shoulder-to-head strikes performed by competitive ice hockey players [Study One].

Case selection

A thorough search of injury reports of North American professional leagues' 2011-2012 season identified 93 collisions that resulted in a diagnosed concussion, of which 37 had video recordings available on the Internet. Fifteen events met the following inclusion criteria and were included in the study: 1) the event was a single impact to the head; 2) the head was the initial point of contact; 3) the striking player's elbow or shoulder was the initial point of contact; 4) a sufficient number of lines and circles were visible in the plan of view of the camera to

determine plan perspective using Kinovea 0.8.20 (open source, kinovea.org); 5) a close-up view of the impact was available.

A search for video recordings of professional hockey players struck to the head by an opponent but did not report a concussion nor miss the following scheduled game was performed on the Internet. The terms “head”, “hit”, and “ice hockey”, were combined with the Boolean operator “AND”. Fourteen events met the inclusion criteria listed in the previous paragraph and were included in this study. A description of the 27 cases that were reconstructed is presented in table 4.1. The outcomes were categorized as *concussion* if the struck player was diagnosed with a concussion by a physician or *no injury* if the struck player did not report a concussion and did not miss the subsequent scheduled game.

TABLE 4.1
Player anthropometrics and outcome of 27 ice hockey collisions

Case	Outcome	Mechanism	Struck player		Striking player	
			height	mass	height	mass
1	Concussion	Shoulder check	1.83 m	91 kg	1.85 m	108 kg
2	Concussion	Shoulder check	1.85 m	93 kg	1.88 m	97 kg
3	Concussion	Shoulder check	1.80 m	91 kg	1.98 m	111 kg
4	Concussion	Shoulder check	1.80 m	94 kg	1.85 m	77 kg
5	Concussion	Shoulder check	1.80 m	86 kg	1.83 m	87 kg
6	Concussion	Shoulder check	1.80 m	91 kg	1.78 m	93 kg
7	Concussion	Shoulder check	1.83 m	91 kg	1.88 m	89 kg
8	Concussion	Extended elbow	1.85 m	97 kg	1.88 m	97 kg
9	Concussion	Extended elbow	1.75 m	84 kg	1.83 m	86 kg
10	Concussion	Extended elbow	1.91 m	91 kg	1.83 m	86 kg
11	Concussion	Extended elbow	1.88 m	88 kg	1.91 m	96 kg
12	Concussion	Extended elbow	1.75 m	83 kg	1.85 m	102 kg
13	Concussion	Tucked-in elbow	1.80 m	91 kg	1.88 m	100 kg
14	Concussion	Tucked-in elbow	1.88 m	100 kg	1.91 m	98 kg
15	No injury	Shoulder check	1.85 m	86 kg	1.93 m	99 kg
16	No injury	Shoulder check	1.98 m	106 kg	1.85 m	93 kg
17	No injury	Shoulder check	1.88 m	95 kg	1.91 m	98 kg
18	No injury	Shoulder check	1.85 m	91 kg	1.93 m	102 kg
19	No injury	Shoulder check	1.91 m	93 kg	1.80 m	90 kg
20	No injury	Shoulder check	1.85 m	94 kg	1.98 m	111 kg
21	No injury	Shoulder check	1.91 m	98 kg	1.96 m	106 kg
22	No injury	Shoulder check	1.85 m	89 kg	1.91 m	98 kg
23	No injury	Shoulder check	1.85 m	102 kg	1.83 m	89 kg
24	No injury	Extended elbow	1.88 m	96 kg	1.78 m	79 kg
25	No injury	Extended elbow	1.85 m	95 kg	1.85 m	89 kg
26	No injury	Extended elbow	1.85 m	97 kg	1.80 m	93 kg
27	No injury	Tucked-in elbow	1.85 m	83 kg	1.85 m	86 kg

Laboratory reconstruction

The 27 selected events were reconstructed by striking a male 50th percentile Hybrid III headform instrumented with nine single-axis Endevco accelerometers positioned in an orthogonal arrangement following a 3–2–2–2 array to collect its three-dimensional dynamic response to the impact [Padgaonkar et al., 1975]. The Hybrid III headform was supported by a sliding table weighing 12.8 kg installed on rails, allowing it to slide backwards with little resistance following the impact. The table was designed to allow for complete control over impact location. It could be adjusted with five degrees of freedom, including fore-aft (x), lateral (y), and up-down (z) translation, as well as fore-aft (y) and axial (z) rotation of the neck base. The adjustments were lockable and remain fixed throughout testing. The headform was equipped with an ice hockey helmet and /or a visor with similar characteristics to the one worn by the struck player (Fig. 4.2). The headform was struck three times at three velocities (calculated velocity, ± 5%) for a total of nine impacts.

Figure 4.2. Frontal view of the helmeted Hybrid III headform used to represent the struck player.

Shoulder impacts were reconstructed using a tubular mobile impact arm that had a length of 1.28 m, diameter of 0.05 m, and total mass of 14.7 kg. It was held horizontally by two cylinders containing rows of bearings to keep friction low. The impact arm was accelerated with a piston and released prior to impact to eliminate the driving force of the compressed air. The striking surface consisted of a nylon disc (diameter 13.2 mm) covered with a 142 mm thick layer of vinyl nitrile 602 foam and a Reebok 11k shoulder pad (Fig. 4.3). This striker was found to generate a linear acceleration peak and duration similar to what was produced by ice hockey players for impacts to the temporal region of a suspended Hybrid III headform at low and high velocity (Fig. 4.4) [Study One].

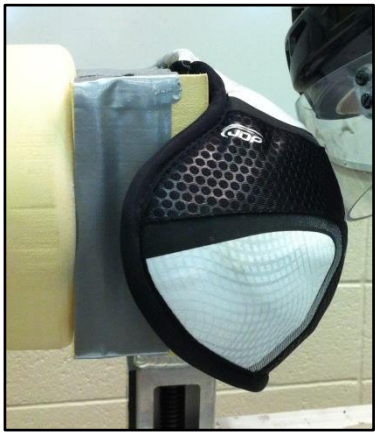


Figure 4.3. Shoulder striker used in this study to mimic mass and compliance of a shoulder check in ice hockey.

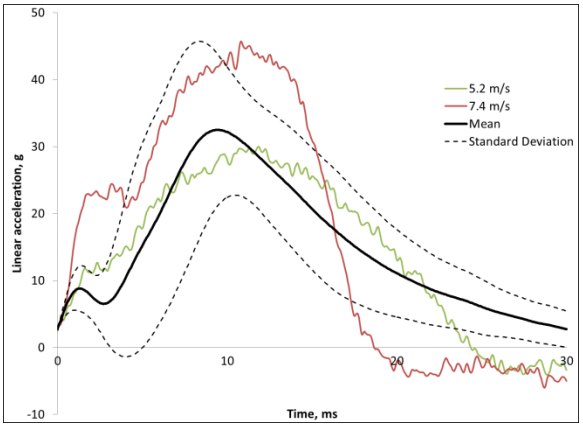


Figure 4.4. Linear acceleration history comparison between the shoulder striker and the mean response (± 1 standard deviation) of 15 test subjects for a shoulder check in ice hockey [Study One].

Extended elbow impacts were reconstructed using a hollow metal frame pendulum. The striking surface consisted of an aluminum L-shaped frame covered with a 25.4 mm thick layer of vinyl nitrile 602 foam, a 14.5 mm thick layer of vinyl nitrile R338V foam, and a Reebok 11k

elbow pad (Fig. 4.5). Foam type and thickness were selected to simulate similar peak and duration of the mean linear acceleration produced by ice hockey players for impacts to the temporal region of a suspended Hybrid III headform in laboratory recreations (Fig. 4. 6) [Study One]. The total mass of the pendulum was 6.2 kg.

Figure 4.5. Elbow striker used in this study to mimic mass and compliance of an extended elbow strike in ice hockey.

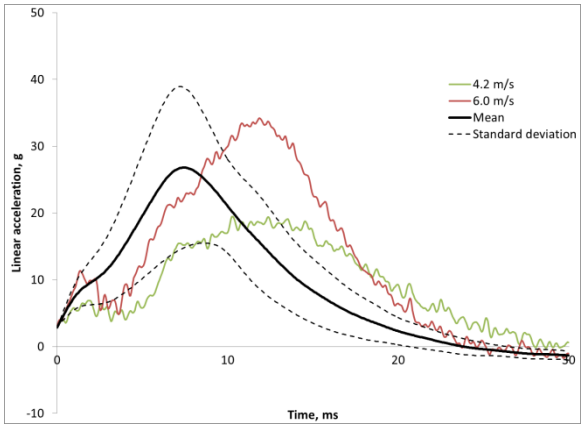


Figure 4.6. Linear acceleration history comparison between the elbow striker and the mean response (± 1 standard deviation) of 15 test subjects for an extended elbow strike in ice hockey [Study One].

Tucked-in elbow impacts were reconstructed using an aluminum L-shaped frame covered with a 14.5 mm thick layer of vinyl nitrile R338V foam and a Reebok 11k elbow pad (Fig. 7). The total mass of the pendulum was 2.9 kg. Foam type and thickness were selected to simulate similar peak and duration of the mean linear acceleration produced by ice hockey players for impacts to the temporal region of a suspended Hybrid III headform in laboratory recreations (Fig. 4. 8) [Study One].

The pendulums were suspended from the ceiling using four aviation cables. Velocity was reached by raising the pendulum at the appropriate height. The pendulum was released by switching off a magnet to ensure consistent releases.

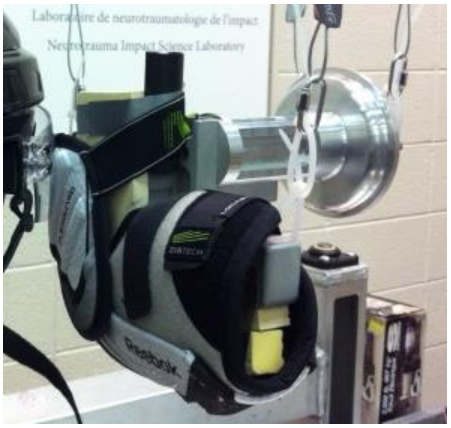


Figure 4.7. Elbow striker used in this study to mimic mass and compliance of a tucked-in elbow strike in ice hockey.

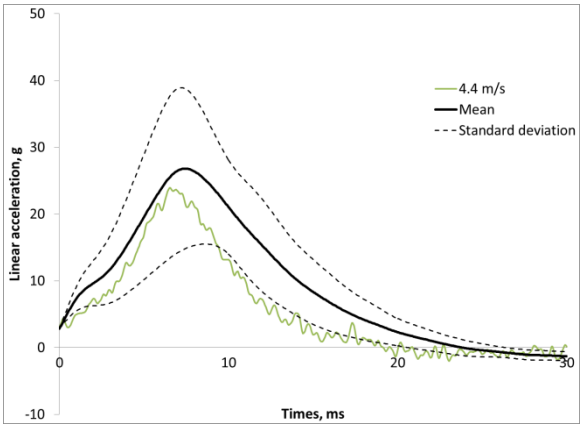


Figure 4.8. Linear acceleration history comparison between the elbow striker and the mean response (± 1 standard deviation) of 15 test subjects for a tucked-in elbow strike in ice hockey [Study One].

Impact parameters

Each case was reconstructed using five impact parameters (Table 4.2). Impact velocity was calculated by measuring the distance separating the struck player's head from the striking player's elbow, or shoulder, five frames prior to the impact. While both players were in movement; relative velocity was used in the reconstruction as a simplification. It has been demonstrated that impacting a static headform using the calculated relative velocity as opposed to impacting a moving headform creates different head linear and angular accelerations; however the method was explorative and too inconsistent for this study [Kendall et al., 2013]. The lines and circles visible in the plane of the camera view were used to draw a

perspective grid (Kinovea 0.8.20) and used as a scaling reference to convert pixels to metres (Fig. 4.9). The video footage of elite games played in North America followed the National Television System Committee (NTSC) encoding, which consists of 29.97 frames per second. Thus, time between each frame was known (0.0334 s) and used to obtain time duration (0.167 s). Error associated with the conversion of pixels to meters was assessed by performing the scaling process for three different events and measuring a third known distance. The influence of the error was accounted for by adding and substracting 5% to the measured velocity.

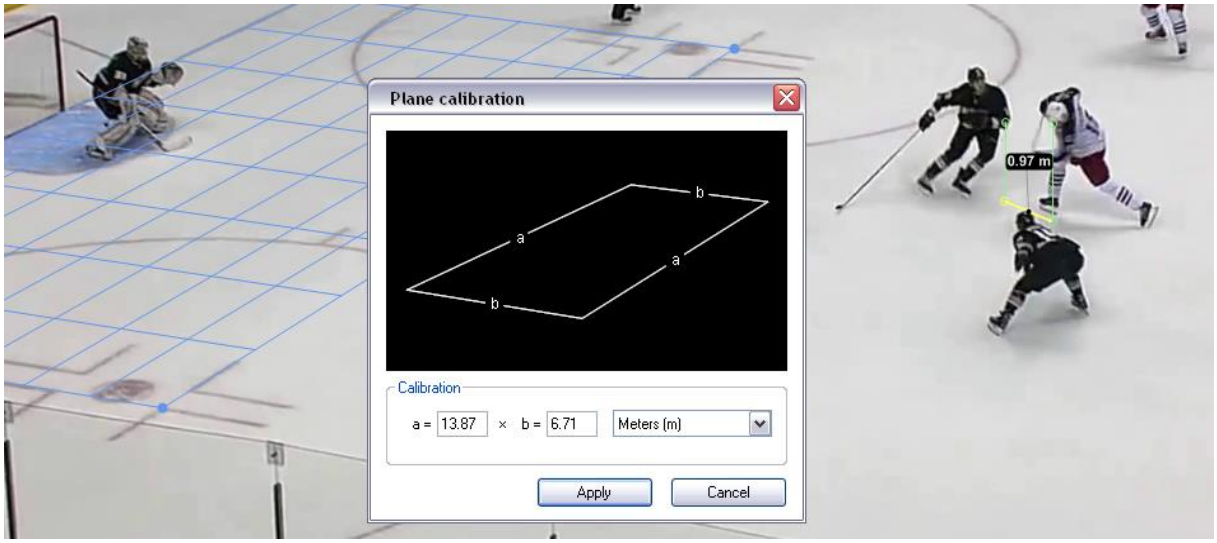


Figure 4.9. Example of a perspective grid calibration in ice hockey

Impact location was determined using a close-up view of the collision. Location was labelled using the reference system illustrated in Fig. 4.10. The head was divided in 12 sectors of 30° in the transverse plane, with eyes forward being 0°, and six levels of elevation in the sagittal plane. This reference system created rectangular regions of at most 5 cm in width, where the head circumference is the longest, and 4.51 cm in height. The level of precision used in this study was comparable to other studies reporting impact location [Crisco et al., 2010; Fréchède

and McIntosh, 2009; Hutchison, 2011; Mihalik et al., 2008; Pellman et al., 2003]. Impact orientation was determined using the position and displacement of the striking player in relation to the struck player and the orientation of the struck player's head. It was estimated using increments of 15°. Pilot testing was performed prior to this study to establish error levels for variations in angle and location. This was done using the pneumatic linear impactor used for shoulder checks; however the striking surface was changed to a hemispherical nylon striker containing a 3.6 mm thick layer of vinyl nitrile foam layer for consistency. The estimated level of error for a variation of 15° was 2.2% for both peak linear acceleration and angular acceleration. The estimated level of error for variation of one sector (R2 to R3) was 3.3% and 16.0% for peak linear acceleration and peak angular acceleration, respectively [Walsh et al., 2012].

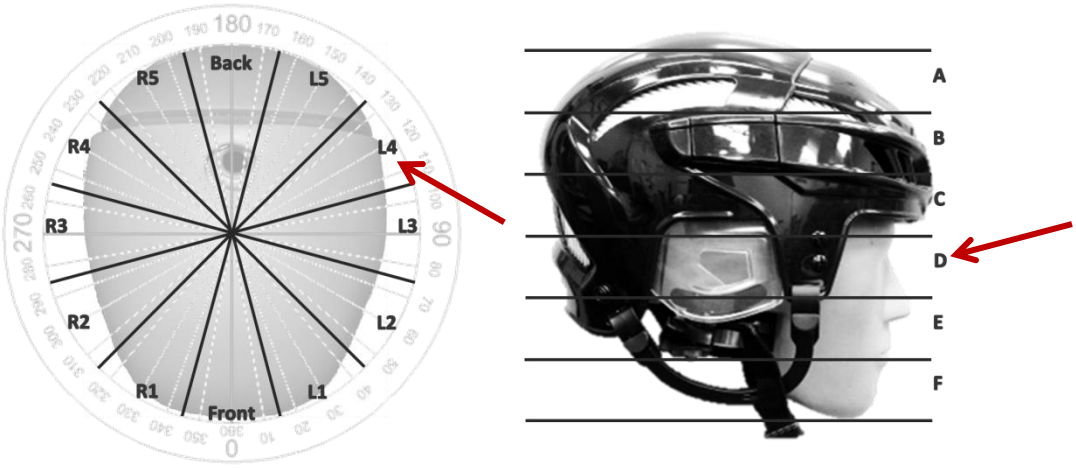


Figure 4.10. Top and side view of a head illustrating the 12 sectors and six levels used in the reconstruction protocol. The arrows demonstrate orientation for case 11: Z-axis rotation of 60° and Y-axis rotation of -15°.

Impact mass and compliance were predetermined using data reported in a previous study, which identified the effective mass of shoulder-to-head and elbow-to-head collisions in ice hockey [Study One]. Due to the low correlation between effective impact mass and the players' body mass, impact mass was 14.7 kg, 6.2 kg, and 2.9 kg for the collisions made with a shoulder, an extended elbow, or a tucked-in elbow, respectively. The strikers used in this study werer designed to generate similar linear acceleration peaks and durations to the ones generated by volunteer ice hockey players [Study One].

TABLE 4.2

Impact parameters for 27 reconstructions of ice hockey collisions

Case	Outcome	Striker		Velocity	Location		Head rotation	
		type	Mass		sector	level	Y-axis	Z-axis
1	Concussion	Shoulder	14.7 kg	9.0 m/s	L1	E	-30°	45°
2	Concussion	Shoulder	14.7 kg	6.6 m/s	R1	D	15°	240°
3	Concussion	Shoulder	14.7 kg	5.0 m/s	R2	F	0°	105°
4	Concussion	Shoulder	14.7 kg	6.3 m/s	L2	F	0°	300°
5	Concussion	Shoulder	14.7 kg	5.5 m/s	R1	E	0°	285°
6	Concussion	Shoulder	14.7 kg	8.7 m/s	R2	F	0°	255°
7	Concussion	Shoulder	14.7 kg	6.5 m/s	Front	D	0°	285°
8	Concussion	Extended elbow	6.2 kg	4.8 m/s	L1	F	0°	75°
9	Concussion	Extended elbow	6.2 kg	4.4 m/s	L1	E	0°	120°
10	Concussion	Extended elbow	6.2 kg	4.1 m/s	R1	F	0°	240°
11	Concussion	Extended elbow	6.2 kg	5.9 m/s	L3	D	-15°	60°
12	Concussion	Extended elbow	6.2 kg	6.0 m/s	L3	D	0°	90°
13	Concussion	Tucked-in elbow	2.9 kg	5.8 m/s	R4	D	-30°	285°
14	Concussion	Tucked-in elbow	2.9 kg	5.9 m/s	L2	E	0°	75°
15	No injury	Shoulder	14.7 kg	5.0 m/s	L2	B	0°	105°
16	No injury	Shoulder	14.7 kg	4.8 m/s	R4	C	0°	270°
17	No injury	Shoulder	14.7 kg	4.8 m/s	R2	D	0°	60°
18	No injury	Shoulder	14.7 kg	6.6 m/s	R1	F	0°	15°
19	No injury	Shoulder	14.7 kg	5.9 m/s	R2	D	0°	315°
20	No injury	Shoulder	14.7 kg	6.7 m/s	Front	E	0°	0°
21	No injury	Shoulder	14.7 kg	7.3 m/s	R1	E	0°	315°
22	No injury	Shoulder	14.7 kg	8.6 m/s	L1	C	-15°	60°
23	No injury	Shoulder	14.7 kg	8.4 m/s	L4	C	0°	90°
24	No injury	Extended elbow	6.2 kg	4.4 m/s	L4	E	-15°	90°
25	No injury	Extended elbow	6.2 kg	4.3 m/s	R5	B	-30°	300°
26	No injury	Extended elbow	6.2 kg	3.2 m/s	R1	F	0°	270°
27	No injury	Tucked-in elbow	2.9 kg	5.2 m/s	R1	D	0°	270°

Collection system

Impact velocity for reconstructed shoulder impacts was measured using an electronic time gate (width 0.2525 m) and recorded by computer using VI Logger (National Instruments, Austin TX). Impact velocity for reconstructed elbow impacts was measured using a High Speed Imaging PCI-512 Fastcam running at 1 kHz and Photron Motion Tools computer software (Photron, San Diego CA). The nine accelerometers mounted in the Hybrid III headform were single-axis Endevco 7264C-2KTZ-2-300 accelerometers (Endevco, San Juan Capistrano CA). Headform acceleration was sampled at 20 kHz and filtered using a 1600 Hz low-pass filter in accordance with the SAE J211 protocol. The accelerometer signals passed through a TDAS Pro Lab system (DTS, Calabasas CA) before being processed by TDAS software. Data collection was triggered when any of the accelerometers located at the centre of gravity of the headform reach a 3 g threshold.

Statistics

Independent-samples t-tests were conducted to compare linear and angular acceleration peak and duration in *no injury*, and *concussion* conditions; statistical significance was set at an alpha of 0.05. Binomial logistic regressions were performed to evaluate the effects of linear acceleration (peak and duration) and angular acceleration (peak and duration) on the likelihood of sustaining a concussion.

Results

Twenty-seven elite ice hockey collisions were reconstructed in this study. Fourteen collisions resulted in a diagnosed concussion while 13 resulted in no reported injury. Sixteen players were struck to the head by a shoulder (7 concussions and 9 no injuries), eight were struck by an extended elbow (5 concussions and 3 no injuries), and three were struck by a tucked-in elbow (2 concussions and 1 no injury) (Table 4.1). The point of contact was most often to the face (74%), L2 to R2, which was covered by a visor but not protected by the helmet. All concussive and most non-injurious impacts (85%) were the result of impacts coming from the side, Z-axis rotation between 45° and 135° or 235° and 315°.Most impacts (74%) had no elevation, Y-axis rotation between -15° and 15° (Table 4.2).

Headform acceleration resulting from the 27 reconstructions was analyzed and summarized in table 4.3. An extended list of the results can be found in table 4.7. Mean peak linear acceleration was 27 ± 12 g (range: 10-58 g) for the concussion cases and 25 ± 11 g (range: 5-52 g) for the no injury cases. Mean impulse duration was 23 ± 6 ms (range: 10-33 ms) for the concussion cases and 19 ± 5 ms (range: 11-28 ms) for the no injury cases. Collisions that resulted in concussion had a similar peak linear acceleration to collisions that resulted in no injury ($t(229) = 1.519$, $p = 0.130$) but a higher impulse duration ($t(229) = 4.651$, $p < 0.001$). Peak angular acceleration was 3.0 ± 1.1 krad/s^2 (range: 1.4-6.6 krad/s^2) for the concussion cases and 2.8 ± 1.1 krad/s^2 (range: 1.1-5.9 krad/s^2) for the no injury cases. Angular acceleration curve duration was 28 ± 5 ms (range: 15-38 ms) for the concussion cases and was 23 ± 4 ms (range: 16-31 ms) for the no injury cases. Collisions that resulted in concussion had a similar peak

angular acceleration to collisions that resulted in no injury ($t(229) = 1.768$, $p = 0.078$) but a higher impulse duration ($t(229) = 6.626$, $p < 0.001$).

While peak linear and angular accelerations were similar between *concussion* and *no injury* conditions when the data were pooled regardless of striking technique, statistical differences were found when the mechanisms were analyzed separately. Shoulder checks that resulted in concussion had similar peak linear acceleration ($t(133) = 0.45$, $p = 0.964$) but higher peak angular acceleration ($t(133) = 2.229$, $p = 0.027$) than shoulder checks that resulted in no injury. Impacts made with an extended elbow resulting in concussion had higher peak linear acceleration ($t(67) = 4.4254$, $p < 0.001$) and peak angular acceleration ($t(67) = 3.221$, $p = 0.002$) than impacts that resulted in no injury. Finally, impacts made with a tucked-in elbow resulting in concussion had higher peak linear acceleration ($t(25) = 8.875$, $p < 0.001$) and peak angular acceleration ($t(25) = 4.824$, $p < 0.001$) than impacts that resulted in no injury.

TABLE 4.3

Mean headform acceleration response for 27 reconstructions of ice hockey collisions

Mechanism	Outcome	n	Linear acceleration		Angular acceleration	
			peak	duration	peak	duration
Shoulder check	Concussion	7	28 ± 7 g	25 ± 5 ms	3.6 ± 1.1 krad/s^2	30 ± 4 ms
	No injury	9	28 ± 9 g	19 ± 5 ms	3.1 ± 1.1 krad/s^2	24 ± 3 ms
Extended elbow	Concussion	5	17 ± 6 g	25 ± 2 ms	2.2 ± 0.5 krad/s^2	27 ± 2 ms
	No injury	3	12 ± 3 g	22 ± 1 ms	1.8 ± 0.5 krad/s^2	21 ± 4 ms
Tucked-in elbow	Concussion	2	48 ± 5 g	11 ± 1 ms	3. 2 ± 0.6 krad/s^2	18 ± 1 ms
	No injury	1	30 ± 5 g	14 ± 1 ms	2.0 ± 0.4 krad/s^2	20 ± 1 ms
All data	Concussion	14	27 ± 12 g	23 ± 6 ms	3.0 ± 1.1 krad/s^2	27 ± 5 ms
	No injury	13	25 ± 11 g	19 ± 5 ms	2.8 ± 1.1 krad/s^2	23 ± 4 ms

*framed values were significantly different ($p < 0.05$)

A binary logistic regression analysis was performed to evaluate the effects of linear acceleration (peak and duration) and angular acceleration (peak and duration) on the likelihood of sustaining a concussion (Tables 4.4 to 4.6). The test indicated that linear acceleration model was did not adequately predict concussion following a shoulder check to the head (Hosmer and Lemeshow $\chi^2 < 0.05$). Conversely, the angular acceleration model was found to reliably predict concussion (H-L $\chi^2 = 0.965$). The model explained 85.4% (Nagelkerke R^2) of the variance in concussion risk and correctly classified 92.6% of the shoulder check cases. A 50% likelihood of concussion was established for a peak angular acceleration of 9.2, 6.9, 4.6, and 2.2 krad/s^2 for impulse durations of 15, 20, 25, and 30 ms, respectively.

The linear acceleration model was found to reliably predict concussion following an extended elbow strike to the head (H-L $\chi^2 = 0.082$). The model explained 31.1% of the variance in concussion risk and

correctly classified 65.2% of the extended elbow cases. A 50% likelihood of concussion was established fora peak linear acceleration of 23, 15, and 7 g for impulse durations of 15, 20, and 25 ms, respectively. Peak linear acceleration explained 24.1% of the variance in concussion risk and correctly classified 66.7% of the extended elbow cases. This was not the case for the angular acceleration model, which did not adequately predict concussion (H-L χ^2 = 0.048).

While the linear acceleration model was found to reliably predict concussion following a tucked-in elbow to the head (H-L χ^2 = 0.999), the Wald criterion demonstrated that neither peak acceleration (p = 0.999) nor impulse duration (p = 0.997) made a significant contribution to prediction. The same was true for the angular acceleration model, which was also found to reliably predict concussion (H-L χ^2 = 0.999), but neither peak acceleration (p = 0.102) nor impulse duration (p = 0.154) made a significant contribution to prediction.

TABLE 4.4
Logistic Regression Predicting Concussion following shoulder checks to the head

Predictor variables	B	Wald χ^2 Statistic	p
Linear acceleration model			
Peak acceleration, g	0.074	6.522	0.011
Impulse duration, ms	0.262	30.549	< 0.001
Constant	-8.082		
Hosmer and Lemeshow χ^2 = 21.646 p = 0.006			
Pseudo R^2= 0.373			
% correct classification = 69.6			
Angular acceleration model			
Peak acceleration, krad/s^2	2.876	20.743	< 0.001
Impulse duration, ms	1.339	19.968	< 0.001
Constant	-46.558		
Hosmer and Lemeshow χ^2 = 2.434 p = 0.965			
Pseudo R^2= 0.854			
% correct classification = 92.6			

TABLE 4.5

Logistic Regression Predicting Concussion following extended elbow strikes to the head

Predictor variables	B	Wald χ^2 Statistic	p
Linear acceleration model			
Peak acceleration, g	0.143	3.876	0.049
Impulse duration, ms	0.228	3.771	0.052
Constant	-6.755		
Hosmer and Lemeshow χ^2 = 13.986 p = 0.082			
Pseudo R^2= 0.311			
% correct classification = 65.2			
Angular acceleration model			
Peak acceleration, krad/s^2	2.8998	7.473	0.006
Impulse duration, ms	0.469	14.102	< 0.001
Constant	-17.67		
Hosmer and Lemeshow χ^2 = 15.639 p = 0.048			
Pseudo R^2= 0.603			
% correct classification = 81.2			

TABLE 4.6

Logistic Regression Predicting Concussion following tucked-in elbow strikes to the head

Predictor variables	B	Wald χ^2 Statistic	p
Linear acceleration model			
Peak acceleration, g	0.333	0	0.999
Impulse duration, ms	-33.122	0	0.997
Constant	400.356		
Hosmer and Lemeshow χ^2 = 0.001 p = 0.999			
Pseudo R^2= 1.00			
% correct classification = 100			
Angular acceleration model			
Peak acceleration, krad/s^2	18.195	2.668	0.102
Impulse duration, ms	-4.069	2.031	0.154
Constant	30.951		
Hosmer and Lemeshow χ^2 = 0.259 p = 0.999			
Pseudo R^2= 0.882			
% correct classification = 92.6			

TABLE 4.7

Headform acceleration response for 27 reconstructions of ice hockey collisions

Case	Outcome	Mechanism	Linear acceleration		Angular acceleration	
			Peak	duration	peak	duration
1	Concussion	Shoulder check	36 ± 3 g	20 ± 2 ms	4.6 ± 0.5 krad/s^2	27 ± 1 ms
2	Concussion	Shoulder check	35 ± 3 g	26 ± 2 ms	2.7 ± 0.3 krad/s^2	29 ± 2 ms
3	Concussion	Shoulder check	16 ± 1 g	32 ± 1 ms	2.5 ± 0.2 krad/s^2	37 ± 1 ms
4	Concussion	Shoulder check	23 ± 3 g	26 ± 2 ms	3.3 ± 0.3 krad/s^2	32 ± 2 ms
5	Concussion	Shoulder check	25 ± 3 g	29 ± 3 ms	2.5 ± 0.4 krad/s^2	33 ± 3 ms
6	Concussion	Shoulder check	32 ± 3 g	18 ± 2 ms	5.4 ± 0.8 krad/s^2	25 ± 1 ms
7	Concussion	Shoulder check	26 ± 2 g	25 ± 2 ms	3.8 ± 0.3 krad/s^2	33 ± 2 ms
8	Concussion	Extended elbow	14 ± 2 g	24 ± 1 ms	2.5 ± 0.3 krad/s^2	26 ± 1 ms
9	Concussion	Extended elbow	12 ± 1 g	26 ± 2 ms	1.6 ± 0.1 krad/s^2	28 ± 1 ms
10	Concussion	Extended elbow	11 ± 1 g	23 ± 1 ms	2.4 ± 0.2 krad/s^2	29 ± 2 ms
11	Concussion	Extended elbow	24 ± 3 g	26 ± 1 ms	2.4 ± 0.3 krad/s^2	25 ± 2 ms
12	Concussion	Extended elbow	24 ± 3 g	26 ± 2 ms	2.2 ± 0.5 krad/s^2	30 ± 2 ms
13	Concussion	Tucked-in elbow	46 ± 3 g	11 ± 1 ms	2.7 ± 0.1 krad/s^2	19 ± 1 ms
14	Concussion	Tucked-in elbow	51 ± 5 g	10 ± 1 ms	3.7 ± 0.6 krad/s^2	17 ± 1 ms
15	No injury	Shoulder check	278 ± 1 g	26 ± 1 ms	2.1 ± 0.1 krad/s^2	27.1 ± 1 ms
16	No injury	Shoulder check	24 ± 1 g	27 ± 1 ms	2.3 ± 0.1 krad/s^2	26.7 ± 1 ms
17	No injury	Shoulder check	25 ± 1 g	27 ± 1 ms	1.8 ± 0.2 krad/s^2	29.8 ± 1 ms
18	No injury	Shoulder check	15 ± 1 g	18 ± 2 ms	3.8 ± 0.3 krad/s^2	26.3 ± 3 ms
19	No injury	Shoulder check	23 ± 2 g	19 ± 4 ms	2.2 ± 0.2 krad/s^2	25.4 ± 1 ms
20	No injury	Shoulder check	26 ± 3 g	16 ± 1 ms	3.4 ± 0.5 krad/s^2	24.3 ± 1 ms
21	No injury	Shoulder check	26 ± 1 g	14 ± 2 ms	3.7 ± 0.3 krad/s^2	22.0 ± 1 ms
22	No injury	Shoulder check	48 ± 4 g	16 ± 0.4 ms	3.1 ± 0.3 krad/s^2	21.1 ± 1 ms
23	No injury	Shoulder check	37 ± 2 g	15 ± 1 ms	5.1 ± 0.6 krad/s^2	19.2 ± 0.4 ms
24	No injury	Extended elbow	13 ± 2 g	26 ± 1 ms	1.8 ± 0.2 krad/s^2	28.0 ± 1 ms
25	No injury	Extended elbow	14 ± 1 g	22 ± 1 ms	2.3 ± 0.2 krad/s^2	18.0 ± 1 ms

26	No injury	Extended elbow	7 ± 1 g	16 ± 1 ms	1.2 ± 0.1 krad/s^2	16.8 ± 1 ms
27	No injury	Tucked-in elbow	30 ± 5 g	14 ± 1 ms	2.0 ± 0.4 krad/s^2	19.9 ± 1 ms

Discussion

This study was designed to characterize head dynamic response of concussive and non-injurious ice hockey elbow and shoulder to head strikes by reconstructing real-world collisions in laboratory. Video analysis of the 27 cases revealed that the most common point of contact was the face which was unprotected by the helmet. Furthermore, most concussions were the result of impacts coming from the side while non-injurious impacts had a large proportion of impacts coming from the front. This indicates that of the injured struck players may have been caught off guard placing them at a disadvantage. The data showed that impacts resulting in concussion had higher peak linear acceleration and impulse duration than impacts resulting in no injury. The same relationship was observed for peak angular acceleration and impulse duration.

Collisions resulting in concussion had a mean peak linear acceleration of 24.7 ± 12 g and a mean peak angular acceleration of 3.0 ± 1.1 krad/s^2; both are lower than the values associated with sports concussion reported in the literature. Thresholds for concussion have been proposed following reconstructions of 27 collisions and four falls occurring in the National Football League (NFL) [Pellman et al., 2003]. Twenty one players struck to the head by another athlete and three players who fell and struck their head on the turf were diagnosed with a concussion by their team physician. Peak head acceleration was higher for the concussed players (98 ± 28 g and 6.4 ± 1.8 krad/s^2) than the uninjured striking players (60 ± 24 g and 5.0 ±

1.1 krad/s^2). Likewise, concussion was associated with a peak linear acceleration of 103 ± 30 g and an angular acceleration of 8.0 ± 3.5 krad/s^2 following numerical reconstructions of 27 rugby and Australian rules football collisions [Fréchède and McIntosh, 2009].

The estimated tolerable peak linear acceleration levels for a 50% risk of concussion following an extended elbow strike to the head were 23, 15, and 7 g for impulse durations of 15, 20, and 25 ms, respectively. The estimated tolerable peak angular acceleration levels for a 50% risk of concussion following a shoulder check to the head were 9.2, 6.9, 4.6, and 2.2 krad/s^2 for impulse durations of 15, 20, 25, and 30 ms, respectively. The linear levels are different than the ones found in the literature. A 25, 50 and 80% probability of sustaining a concussion were associated to 66, 82 and 106 g for linear acceleration and 4.6, 5.9, and 7.9 krad/s^2, for angular acceleration [Zhang et al., 2004]. Differences in concussion risk may be a result of case selection, impact parameters inherent to each sport, and the methodology used.

The outcome of the rugby and Australian rules Football collisions was categorized using loss of consciousness (LOC) [Fréchède and McIntosh, 2009]. Nine of the cases resulted in the struck player being diagnosed with a concussion but no LOC (Grade 1); nine resulted in a concussion with LOC lasting less than one minute (Grade 2); and nine resulted in a concussion with LOC lasting more than one minute (Grade 3). Considering that collisions resulting in LOC lasting more than one minute were found to have statistically higher peak linear accelerations than collisions resulting in no LOC, it would be more accurate to compare head accelerations of collisions resulting in a Grade 1 concussion (86 ± 14 g and 7.2 ± 3.5 krad/s^2) to the ones obtained in this study. Furthermore, the absence of a “no-injury” condition makes it difficult to

establish whether the head acceleration values were representative of concussive collisions or a reflection of the methodology used. While the selection of concussive American football impacts was similar to this current study and relied on physician diagnosis, the selection of non-injurious collisions differed [Pellman et al., 2003]. Of the 33 cases which did not result in a diagnosed concussion, 27 were striking players. This is important because impact mass, location, and orientation differ between striking and struck players and comparisons should be made for each impact mechanism [Viano and Pellman, 2005; Viano et al., 2007]. Furthermore, not selecting more impacts where the struck player was not injured may have biased the results towards high velocity open field collisions.

Inherent differences between both sports can further explain the lower values found in this study. Ice hockey is played with skates on an icy surface and is known to be a fast paced game; yet, mean impact velocity was 5.9 ± 1.5 m/s which is lower than the mean velocity of Australian rules football (7 ± 3 m/s) and American football (9.3 ± 1.9 m/s) collisions [McIntosh et al., 2000; Pellman et al., 2003]. This may be a reflection of the role of body checking in ice hockey; its purpose being to separate a player from the puck. This differs from rugby, Australian rules football, or American Football tackles which are meant to stop an opponent's forward progression. Moreover, ice hockey players are discouraged to accelerate over a long distance to maximize impact velocity and may be given a penalty for charging. Impact compliance also differs, with ice hockey players being protected by a helmet, visor, elbow and shoulder pads. This equipment reduces peak linear acceleration but increases the duration of the impulse. In this study, mean linear and angular acceleration impulse durations were 21 ± 6 ms and 26 ± 5

ms, which are higher than head-to-head collisions reported for American Football (linear acceleration: 15 ms, angular acceleration: not reported) [Pellman et al., 2003]. This is important because the results from this study indicate that longer impulse durations increase concussion risk; this is consistent with severe head injury risk research [Gurdjian et al., 1953; Gurdjian et al., 1966; Gurdjian et al., 1955; Ono et al., 1980].

Third, rugby and Australian rules football collisions were simulated numerically by positioning MADYMO models to reproduce the relative motion of each player before and after the impact [Fréchède and McIntosh, 2009]. While MADYMO's human model has had several of its parts validated against blunt impact (shoulder, pelvis) this has not been done for the head. Thus, direct comparison between both studies should not be performed in absolute terms but rather as relative change of injury risk related impact parameters [Forero Rueda and Gilchrist, 2009]. The methodology used to reconstruct American football collisions was similar to this study with the exception of impact mass and compliance. Striking mass was recreated by dropping a Hybrid III headform attached to a 25 kg mass onto a suspended torso-headform Hybrid III assembly meant to represent the struck player. The effective mass of a striking player was not calculated prior to the study. Furthermore, compliance was replicated by equipping both headforms with helmets.

The results reported in this study are similar to what was reported in three studies investigating real-time head acceleration occurring during youth and collegiate hockey games [Gwin et al., 2009; Mihalik et al., 2008; Reed et al., 2010]. This was done using an in-helmet telemetry system consisting of six single-axis accelerometers. Mean linear accelerations were

reported to be below 25g, which is consistent with the low values obtained in this study. Furthermore, the data are in agreement with a study conducted by the National Aeronautics and Space Administration [Eiband, 1959]. That study reported that subjects strapped onto an accelerating seat could tolerate a uniform acceleration of 45 g for 0.044 s without any loss of consciousness or confusion. Tolerance was observed to decrease to 40 g for a pulse duration of 0.1 s and 25 g for a pulse duration of 0.2 s.

Limitations

The data presented in this study are specific to shoulder and elbow strikes to the head in elite ice hockey. There are other mechanisms of injury that have their own specific impact parameters and head response. For example, elite players struck to the helmet with a puck shot at 35 m/s may experience head accelerations of 165 g and 15.4 krad/s^2 [Rousseau, Hoshizaki, and Gilchrist, In press]. Impact mass and compliance of a striking body has an influence on head acceleration and injury risk should be assessed for each mechanism separately. Adapting the protocol used in this study to assess concussion risk for puck impacts, falls into boards, and falls on the ice would complement this study's findings and offer a more global assessment of concussion risk associated with ice hockey.

Despite finding statistical differences between the concussion and the no injury group, the method used to select no injury cases may have created a bias towards high energy impacts. To be included in this study, multiple views of a collision had to be available, meaning that only high energy collisions were selected. Considering that ice hockey players are reported to be hit to their head on average three times per game, it is likely that most non-concussive collisions

generate lower head accelerations than what was reported in this study [Brainard et al., 2012]. Unfortunately, those impacts are not spectacular and video footage of the event is difficult to find. Furthermore, ice hockey players are not systematically assessed for concussion by physicians following big collisions and the injury may be unreported. Thus it possible that the collisions that were included in the no-injury condition may actually be concussive impacts.

The 50th percentile adult male Hybrid III head and neck used in this study were designed and validated for indirect head impacts following car crash [Deng, 1989; Mertz, 1985]. Although widely used, there is an inherent limitation when using rigid physical models to represent human head response. The headform is made of steel and can only approximate the dynamic properties and impact response of a real human head [Hubbard and McLeod, 1974; Seeman, Muzy, and Lustick, 1986]. Moreover, the Hybrid III neckform is made of stiff materials and validated against inertial loading rather than direct impact [Deng, 1989]. Nevertheless, the Hybrid III dummy has been used to assess head injury risk in sports [Pellman et al., 2003; Walilko et al., 2005; Withnall et al., 2005] and is widely used in the industry of sports protection making it a reasonable surrogate for physical reconstructions.

Conclusions

The purpose of this study was to describe the relationship between concussion and head acceleration using data obtained from reconstructions of 27 shoulder-to-head and elbow-to-head collisions in elite ice hockey. In 74% of the cases, the point of contact was the face, which

was covered by a visor but not protected by the helmet. Furthermore, in all concussive and 85% of the non-injurious collisions, the striking player came from the side. Mean peak linear and angular accelerations were similar for the group of 14 concussion cases and the group of 13 no injury cases, yet mean linear and angular acceleration impulse duration was higher for the concussion group. This is interesting because one popular method to increase a helmet's ability to reduce peak linear acceleration is to increase its compliance by modifying foam type, size, and stiffness. Increasing the compliance of the helmet also increases impulse duration, however, which was shown to increase concussion risk. The data presented in this study is specific to shoulder-to-head and elbow-to-head collisions and reconstructions of other injury mechanisms is required to adequately assess concussion risk associated with ice hockey.

Chapter Five

THIRD STUDY

FINITE ELEMENT ANALYSIS OF CEREBRUM STRAIN AND STRESS OF REAL-WORLD CONCUSSIVE AND NON-INJURIOUS ELBOW AND SHOULDER TO HEAD COLLISIONS IN ELITE ICE HOCKEY

Introduction

Competitive ice hockey players typically get hit to the head several times during a game [Brainard et al., 2009] yet few of those impacts result in diagnosed concussions [Echlin et al., 2010; Emery and Meeuwisse, 2006; Koh et al., 2003, Wennberg and Tator, 2003]. Concussion risk can be estimated experimentally by reconstructing real life head impacts under laboratory conditions. This approach has been used to analyze collisions and falls occurring in professional American football, Australian rules football, and rugby [Fréchède and McIntosh, 2009; McIntosh et al., 2000, Pellman et al., 2003].

Impact reconstructions of 27 collisions and four falls occurring in the National Football League (NFL) were analyzed using finite element models [Kleiven, 2007, Zhang et al., 2004]. Twenty-five of the players involved in these events were diagnosed with a concussion by team physicians: 21 of the struck players involved in head to head contact and four players who fell and struck their head on the turf. None of the striking players reported a head injury. Twelve of

these collisions, of which nine resulted in the struck player suffering a concussion, were selected and analyzed using an FE model developed at Wayne State University [Zhang et al., 2004]. Concussion risk for 25%, 50% and 80% were estimated to be associated with peak strain of 0.14, 0.19, and 0.24, respectively and peak shear stress of 6.0 kPa, 7.8 kPa, and 10.0 kPa in the brainstem.

Similar thresholds were proposed by Kleiven [2007] who used an FE model developed at the Royal Institute of Technology (KTH) in Stockholm to analyze a larger sample of the same data set (58 cases of which 29 were concussions). A concussion risk of 50% was associated with a strain of 0.21 in the corpus callosum or 0.26 in the grey matter; and a von Mises stress (VMS) of 8.4 kPa in the corpus callosum. Strain in the grey matter was reported to be the best predictor for concussion. Considering that the data used in both studies were taken from the same source, the different injury risks may be a reflection of differences in model characteristics rather than variability in the human population. The KTH head model was also used to analyze brain tissue deformation of 27 concussive impacts and 13 non injurious impacts occurring in Australian rules football and rugby [Patton et al., 2013]. A concussion risk of 50% was associated with a strain of 0.13 in the thalamus, 0.14 in the brainstem, 0.26 in the white matter, or 0.27 in the grey matter. Strain in the thalamus was reported to be the second best predictor for concussion, behind angular acceleration in the coronal plane.

Mean peak linear and angular acceleration for concussive cases was 98 ± 28 g and 7.4 ± 2.9 krad/s^2, respectively for the NFL cases and 103 ± 30 g and 8.0 ± 3.5 krad/s^2, respectively for the rugby and Australian rules football [Fréchède and McIntosh, 2009; Zhang et al., 2004]. These

values are considerably higher than the mean peak accelerations obtained by reconstructing 27 elite ice hockey shoulder-to-head and elbow-to-head collisions [Study Two]. The mean peak linear and angular acceleration for the 13 ice hockey collisions resulting in concussion measured in this study was 27 ± 12 g and 3.0 ± 1.1 krad/s^2, respectively. These impacts did, however, have linear and angular impulse durations of 23 ± 6 ms and 28 ± 5 ms, which are higher than the 15 ms impulses generated by the NFL head-to-head impacts [Pellman et al., 2003]. Thus, concussion risks reported in the aforementioned studies may be specific to collision sports like rugby, Australian rules rugby, and American football and may not be adequate to evaluate concussion risk of a contact sport like ice hockey. The purpose of this study was to characterize brain tissue strain and stress of concussive and non-injurious ice hockey elbow and shoulder to head strikes by performing a finite element analysis of the dynamic response obtained following reconstructions of real-world impacts.

Methods

A detailed stress and strain analysis of the cerebrum using the University College Dublin Brain Trauma Model (UCDBTM) was performed comparing 27 reconstructed real-world elbow and shoulder to head ice hockey collisions [Study Two]. Fourteen of the collisions resulted in the struck player being diagnosed with a concussion while no injuries were reported following the remaining 13 collisions (Table 5.1). The events were reconstructed in laboratory by striking a Hybrid III headform with an elbow or a shoulder surrogate. Resulting headform accelerations were applied to the centre of gravity of the brain model to calculate peak maximum principal

strain (MPS), peak strain rate (specific to the element which was identified as having the highest MPS), and peak von Mises stress (VMS) in the cerebrum.

Laboratory reconstruction

A helmeted Hybrid III headform representing the struck athlete was struck three times at three velocities to create a corridor (calculated velocity, ± 5%) for a total of nine impacts. Shoulder impacts were reconstructed using a tubular mobile impact arm which had a length of 1.28 m, diameter of 0.05 m, and total mass of 14.7 kg. The striking surface consisted of a nylon disc (diameter 13.2 mm) covered with a 142 mm thick layer of vinyl nitrile 602 foam and a Reebok 11k shoulder pad (Fig. 5.1). Extended elbow impacts were reconstructed using a hollow metal frame pendulum. The striking surface consisted of an aluminum L-shaped frame covered with a 25.4 mm thick layer of vinyl nitrile 602 foam; a 14.5 mm thick layer of vinyl nitrile R338V foam; and a Reebok 11k elbow pad (Fig. 5.1). The total weight of the pendulum was 6.2 kg. Tucked-in elbow impacts were reconstructed using an aluminum L-shaped frame covered with a 14.5 mm thick layer of vinyl nitrile R338V foam and a Reebok 11k elbow pad (Fig. 5.1). The total weight of the pendulum was 2.9 kg. A more detailed description of the apparatus and impact parameters can be found in Study Two. Headform acceleration was measured using nine single-axis Endevco accelerometers positioned in an orthogonal arrangement following a 3–2–2–2 array [Padgaonkar et al., 1975]. Sample linear and angular accelerations are presented in figures 5.2 to 5.4.

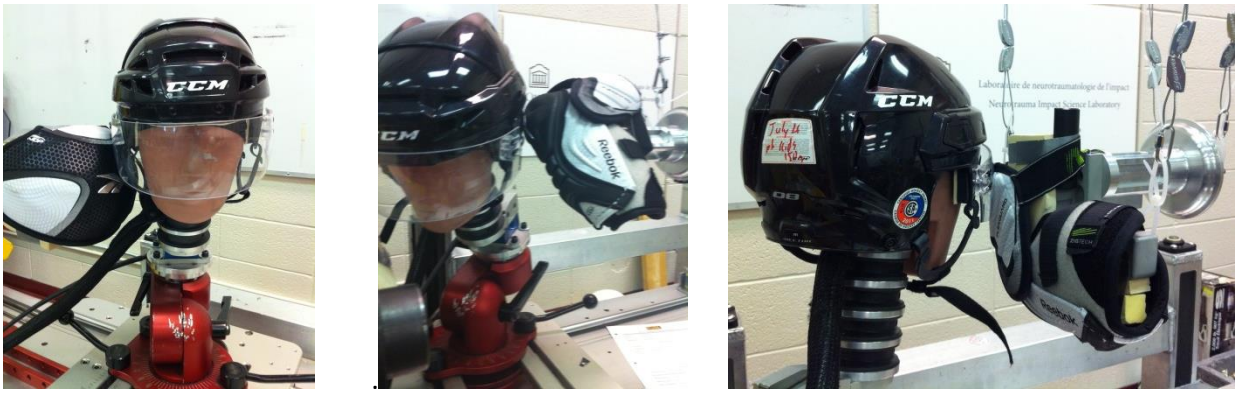


Figure 5.1. Examples of the experimental setup for physical reconstructions of shoulder check (left), extended elbow strikes (centre), and tucked-in elbow strikes (right).

TABLE 5.1

Mean headform acceleration response for 27 reconstructions of ice hockey collisions

Mechanism	Outcome	n	Linear acceleration		Angular acceleration	
			peak	duration	peak	duration
Shoulder check	Concussion	7	28 ± 7 g	25 ± 5 ms	3.6 ± 1.1 krad/s^2	30 ± 4 ms
	No injury	9	28 ± 9 g	19 ± 5 ms	3.1 ± 1.1 krad/s^2	24 ± 3 ms
Extended elbow	Concussion	5	17 ± 6 g	25 ± 2 ms	2.2 ± 0.5 krad/s^2	27 ± 2 ms
	No injury	3	12 ± 3 g	22 ± 1 ms	1.8 ± 0.5 krad/s^2	21 ± 4 ms
Tucked-in elbow	Concussion	2	48 ± 5 g	11 ± 1 ms	3. 2 ± 0.6 krad/s^2	18 ± 1 ms
	No injury	1	30 ± 5 g	14 ± 1 ms	2.0 ± 0.4 krad/s^2	20 ± 1 ms
All data	Concussion	14	27 ± 12 g	23 ± 6 ms	3.0 ± 1.1 krad/s^2	27 ± 5 ms
	No injury	13	25 ± 11 g	19 ± 5 ms	2.8 ± 1.1 krad/s^2	23 ± 4 ms

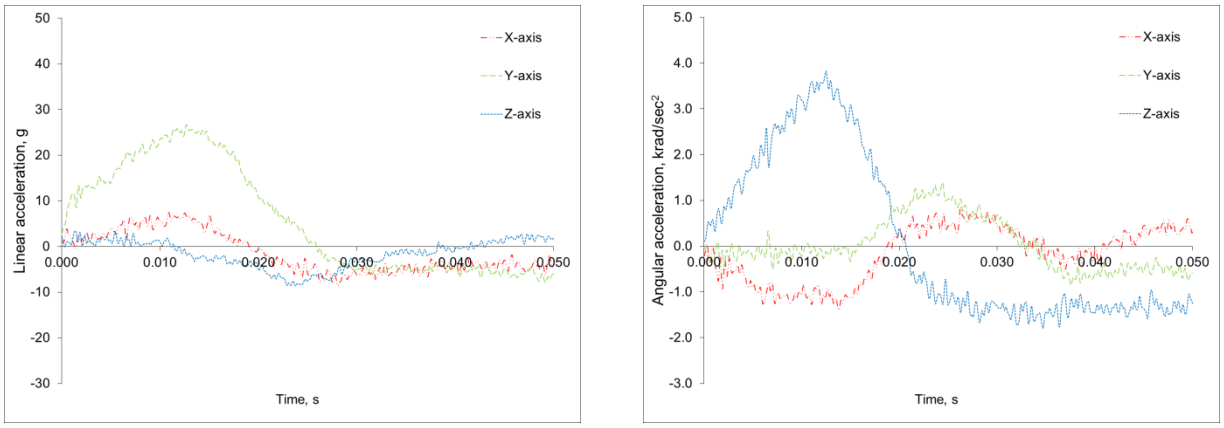


Figure 5.2. Sample linear and angular acceleration impulse for a reconstruction of a shoulder impact (Case 7 – concussive shoulder check)

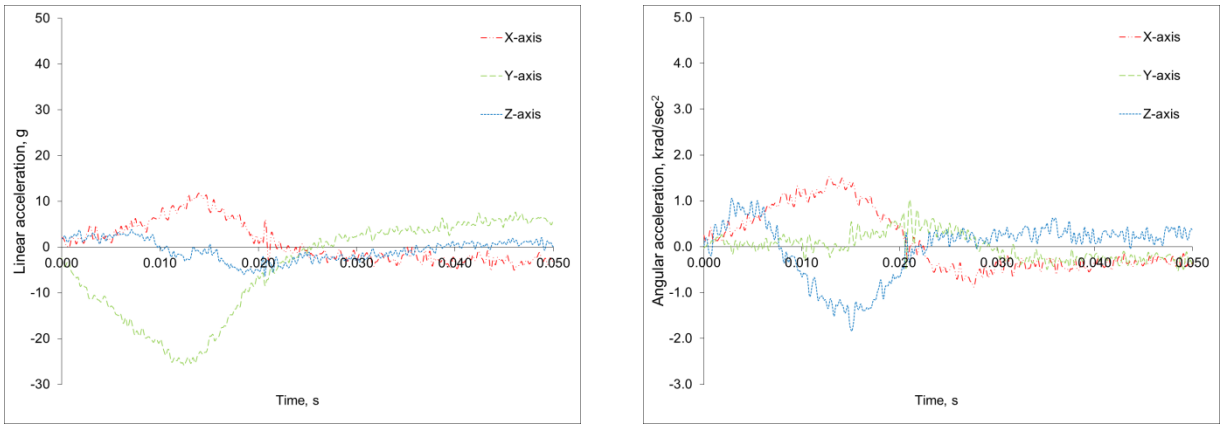


Figure 5.3. Sample linear and angular acceleration impulse for a reconstruction of an extended elbow impact (Case 11 – concussive extended elbow strike)

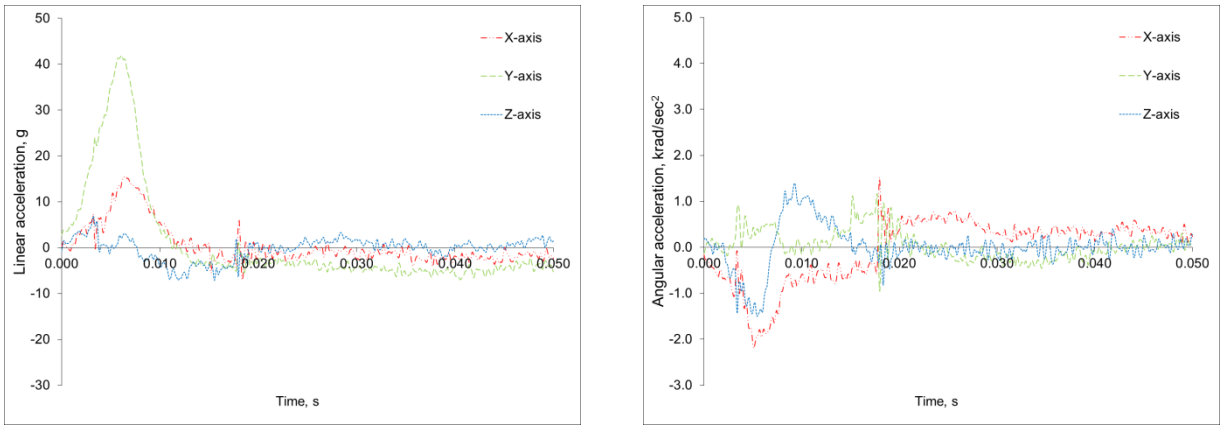


Figure 5.4. Sample linear and angular acceleration impulse for a reconstruction of a tucked-in elbow impact (Case 13 – concussive tucked-in elbow strike)

Finite Element Model

The resulting linear and angular accelerations were used as input for the University College Dublin Brain Trauma Model [Horgan and Gilchrist, 2003, Horgan and Gilchrist, 2004]. The model included the scalp, three-layered skull (cortical and trabecular bone), dura, cerebrospinal fluid (CSF), pia, flax, tentorium, cerebral hemispheres, cerebellum and brain stem. To improve brain deformation distribution, separate representations of the grey, white, and ventricular matter were implemented. Geometry was determined using Computer Tomography (CT) data available through the US National Library of Medicine Visible Human Database and was not meant to represent a 50^{th} percentile male head.

Various constitutive models have been used to describe the mechanical behaviour of brain tissue, including using an integral model in combination with hyperelasticity. In this present analysis, the neural tissue was simply characterized as viscoelastic in shear with a deviatoric stress rate (i.e., not the same in every direction) dependent on the shear relaxation modulus, while the compressive behaviour was considered as elastic. The CSF layer had a 1.3 mm depth and was modeled using solid elements with a low shear modulus and a high bulk modulus so that it would behave like a fluid. The boundary condition used at the brain/skull/CSF interfaces was a sliding boundary condition [Miller et al., 1998, Doorly and Gilchrist, 2006]. The model was found to correlate well with experimental data for head injury using a force free boundary condition at the foramen magnum [Horgan, 2005]. The remaining parts of the model were taken from the literature [Horgan and Gilchrist, 2003, Horgan and Gilchrist, 2004]. Material properties are reported in tables 5.2 and 5.3. The model was validated against the pressure

response of Nahum's [1977] cadaveric impact test and the displacement response of Hardy's [2001] high-speed x-ray cadaver impact test.

TABLE 5.2

Material properties for the human head finite element

Material	Density (kg/m^3)	Young's modulus (MPa)	Poisson's ratio
Facial bone	2100	5 000	0.23
Scalp	1 000	16.7	0.42
Cortical bone	2 000	15 000	0.22
Trabecular bone	1 300	1 000	0.24
Dura	1 130	31.5	0.45
Pia	1 130	11.5	0.45
Falx and tentorium	1 130	31.5	0.45

TABLE 5.3

Material properties for the neuronal tissue

Material	Density (kg/m^3)	Shear modulus (kPa)		Decay constant (MPa)	Bulk modulus (GPa)
		G_0	G_∞		
Brain white matter	1 060	12.5	2.5	80	2.19
Brain grey matter	1 060	10	2	80	2.19
Brain stem	1 060	22.5	4.5	80	2.19
Cerebellum	1 060	10	2	80	2.19

Statistics

Independent-samples t-tests were conducted to compare linear and angular acceleration peaks and durations in *no injury*, and *concussion* conditions; statistical significance was set at an alpha of 0.05. Pearson's correlations were computed to assess the relationship between head

acceleration and brain tissue deformation metrics. Binomial logistic regressions were performed to evaluate the effects of maximum principal strain, strain rate, and von Mises stress on the likelihood of sustaining a concussion.

Results

Brain tissue deformation metrics resulting from 27 ice hockey shoulder-to-head and elbow-to-head reconstructions are summarized in Table 5.4 (Stress and strain for each cases are reported in table 5.9). Mean MPS was 0.24 ± 0.09 (range: 0.12-0.50) for the concussion cases and 0.22 ±0.07 (range: 0.08-0.40) for the no injury cases. Mean strain rate was 47 ± 21 s^{-1} (range: 17-98s^{-1}) for the concussion cases and 42 ± 17 s^{-1} (range: 20-99 s^{-1}) for the no injury cases. Mean VMS was 8.0 ± 3.4 kPa (range: 3.3-18.3 kPa) for the concussion cases and 7.2 ± 2.5 kPa (range: 3.6-14.0 kPa) for the no injury cases. Collisions that resulted in concussion had a similar peak MPS to collisions that resulted in no injury ($t(229) = 1.861$, $p = 0.063$) but a higher strain rate ($t(229) = 1.977$, $p=0.047$), and VMS ($t(229) = 2.007$, $p=0.042$).

Differences in brain deformation metrics were greater when each mechanism was analyzed separately. Shoulder checks that resulted in concussion had higher MPS ($t(133) = 4.074$, $p < 0.001$), strain rate ($t(133) = 3.651$, $p = 0.003$), and VMS ($t(133)$ 3..651, $p < 0.001$) than shoulder checks that resulted in no injury. Impacts made with an extended elbow resulting in concussion had higher MPS ($t(67) = 2.926$, $p = 0.005$) han impacts that resulted in no injury but similar strain rates ($t(67) = 1.550$, $p = 0.126$) and VMS ($t(67) = 1.782$, $p = 0.079$). Finally, impacts made

with a tucked-in elbow resulting in concussion had higher MPS ($t(25) = 3.250$, $p = 0.003$), strain rate ($t(25) = 3.964$, $p = 0.001$), and VMS ($t(25) = 2.719$, $p = 0.012$) than impacts that resulted in no injury.

TABLE 5.4

Mean cerebrum stress and strain metrics for 27 reconstructions of ice hockey collisions

Mechanism	Outcome	n	Maximum Principal strain	Strain rate	von Mises stress
Shoulder check	Concussion	7	0.30 ± 0.08	58 ± 21 s^{-1}	9.9 ± 3.8 kPa
	No injury	9	0.25 ±0.07	48 ± 18 s^{-1}	8.0 ± 2.5 kPa
Extended elbow	Concussion	5	0.18 ± 0.03	29 ± 7 s^{-1}	6.1 ± 1.3 kPa
	No injury	3	0.16 ±0.04	32 ± 10 s^{-1}	5.6 ± 1.1 kPa
Tucked-in elbow	Concussion	2	0.18 ± 0.04	49 ± 18 s^{-1}	6.0 ± 1.1 kPa
	No injury	1	0.15 ±0.02	30 ± 6 s^{-1}	5.1 ± 0.6 kPa
All data	Concussion	14	0.24 ± 0.09	47 ± 21 s^{-1}	8.0 ± 3.4 kPa
	No injury	13	0.22 ±0.07	42 ± 17 s^{-1}	7.2 ± 2.5 kPa

Peak linear acceleration of the headform was found to have a low positive correlation with MPS and strain rate, and little to no correlation with VMS. Peak angular acceleration was found to have a high positive correlation with strain rate, and a very high positive correlation with MPS, and VMS. The four brain tissue deformation metrics were all highly or very highly positively correlated (Table 5.5).

TABLE 5.5

Correlation between head acceleration and cerebrum stress and strain metrics (n=231)

	Peak linear acceleration	Peak angular acceleration	MPS	Strain rate	VMS
Peak linear acceleration		0.475*	0.345*	0.445*	0.278*
Peak angular acceleration			0.915*	0.854*	0.906*
MPS				0.844*	0.941*
Strain rate					0.816*
VMS					

*p<0.001 level.

A binary logistic regression analysis was performed to evaluate the effects of MPS, strain rate, and VMS on the likelihood of sustaining a concussion (Tables 5.6 to 5.8). The test indicated that the MPS model was found to reliably predict concussion following a shoulder check to the head (H-L χ^2 = 0.104). The model explained 14.6% (Nagelkerke R^2) of the variance in concussion risk and correctly classified 65.9% of the shoulder check cases. A 50% likelihood of concussion was established for a peak MPS of 0.30. The strain rate model was also found to reliably predict concussion (H-L χ^2 = 0.226). The model explained 8.2% of the variance in concussion risk and correctly classified 65.2% of the shoulder check cases. A 50% likelihood of concussion was established for a peak strain rate of 59 s^{-1}. The VMS model did not predict adequately predict concussion following a shoulder check to the head (H-L χ^2 < 0.001).

TABLE 5.6
Logistic regression predicting concussion following shoulder checks to the head

Predictor variables		B	Wald χ^2 Statistic	p
Strain model				
Peak maximum principal strain, %		9.296	13.447	< 0.001
Constant		-2.794		
Hosmer and Lemeshow χ^2 =	13.236 p = 0.104			
Pseudo R^2=	0.146			
% correct classification =	65.9			
Strain rate model				
Peak strain rate, s^{-1}		0.026	8.025	0.005
Constant		-1.61		
Hosmer and Lemeshow χ^2 =	9.392 p = 0.226			
Pseudo R^2=	0.082			
% correct classification =	65.2			
Stress model				
Peak von Mises stress, kPa				
Constant		0.198	11.256	0.001
		-1.979		
Hosmer and Lemeshow χ^2 =	34.791 p < 0.001			
Pseudo R^2=	0.12			
% correct classification =	70.4			

The MPS model was found to reliably predict concussion following an extended elbow strike to the head (H-L χ^2 = 0.307). The model explained 15.5% of the variance in concussion risk and correctly classified 68.1% of the extended elbow cases. A 50% likelihood of concussion was established for a peak MPS of 0.14. While the strain rate model was found to reliably predict concussion following an extended elbow strike to the head (H-L χ^2 = 0.915), the Wald criterion demonstrated that peak strain rate (p = 0.130) did not make a significant contribution to prediction. The same was true for the VMS model, which was also found to reliably predict concussion (H-L χ^2 = 0.649), but peak VMS (p = 0.083) did not make a significant contribution to prediction.

TABLE 5.7

Logistic regression predicting concussion following extended elbow strikes to the head

Predictor variables	B	Wald χ^2 Statistic	p
Strain model			
Peak maximum principal strain, %	22.785	6.867	0.009
Constant	-3.25		
Hosmer and Lemeshow χ^2 = 9.435 p = 0.307			
Pseudo R^2= 0.155			
% correct classification = 68.1			
Strain rate model			
Peak strain rate, s^{-1}	0.05	2.297	0.130
Constant	-0.881		
Hosmer and Lemeshow χ^2 = 2.65 p = 0.915			
Pseudo R^2= 0.05			
% correct classification = 63.8			
Stress model			
Peak von Mises stress, kPa	0.361	3.015	0.083
Constant	-1.49		
Hosmer and Lemeshow χ^2 = 5.980 p = 0.649			
Pseudo R^2= 0.061			
% correct classification = 63.8			

The MPS model was found to reliably predict concussion following a tucked-in elbow strike to the head (H-L χ^2 = 0.051). The model explained 33.8% of the variance in concussion risk and correctly classified 77.8% of the tucked-in elbow cases. A 50% likelihood of concussion was established for a peak MPS of 0.15. While the strain rate model was found to reliably predict concussion following an extended elbow strike to the head (H-L χ^2 = 0.390), the Wald criterion demonstrated that peak strain rate (p = 0.057) did not make a significant contribution to prediction. The same was true for the VMS model, which was also found to reliably predict concussion (H-L χ^2 = 0.236), but peak VMS (p = 0.062) did not make a significant contribution to prediction.

TABLE 5.8

Logistic regression predicting concussion following tucked-in elbow strikes to the head

Predictor variables	B	Wald χ^2 Statistic	p
<u>Strain model</u>			
Peak maximum principal strain, %	43.16	4.003	0.045
Constant	-6.299		
Hosmer and Lemeshow χ^2 = 12.53 p = 0.051			
Pseudo R^2= 0.338			
% correct classification = 77.8			
<u>Strain rate model</u>			
Peak strain rate, s^{-1}	0.192	3.62	0.057
Constant	-6.176		
Hosmer and Lemeshow χ^2 = 6.306 p = 0.390			
Pseudo R^2= 0.481			
% correct classification = 66.7			
<u>Stress model</u>			
Peak von Mises stress, kPa	1.105	3.470	0.062
Constant	-5.382		
Hosmer and Lemeshow χ^2 = 9.244 p = 0.236			
Pseudo R^2= 0.243			
% correct classification = 70.4			

TABLE 5.9

Cerebrum stress and strain metrics for 27 reconstructions of ice hockey collisions

Case	Outcome	Mechanism	Maximum Principal Strain	Strain rate	von Mises stress
1	Concussion	Shoulder check	0.364 ± 0.047	76 ± 7 s^{-1}	12.2 ± 3.0
2	Concussion	Shoulder check	0.270 ± 0.038	52 ± 17 s^{-1}	6.6 ± 0.3
3	Concussion	Shoulder check	0.184 ± 0.006	34 ± 3 s^{-1}	5.8 ± 0.6
4	Concussion	Shoulder check	0.289 ± 0.035	58 ± 17 s^{-1}	10.6 ± 1.8
5	Concussion	Shoulder check	0.234 ± 0.022	32 ± 6 s^{-1}	6.2 ± 1.0
6	Concussion	Shoulder check	0.432 ± 0.057	85 ± 20 s^{-1}	15.7 ± 2.1
7	Concussion	Shoulder check	0.321 ± 0.043	60 ± 9 s^{-1}	11.1 ± 0.9
8	Concussion	Extended elbow	0.200 ± 0.031	30 ± 6 s^{-1}	6.7 ± 1.0
9	Concussion	Extended elbow	0.151 ± 0.017	23 ± 4 s^{-1}	4.6 ± 0.6
10	Concussion	Extended elbow	0.182 ± 0.012	35 ± 9 s^{-1}	7.0 ± 0.7
11	Concussion	Extended elbow	0.195 ± 0.019	42 ± 11 s^{-1}	6.7 ± 0.8
12	Concussion	Extended elbow	0.183 ± 0.042	32 ± 8 s^{-1}	5.8 ± 1.6
13	Concussion	Tucked-in elbow	0.151 ± 0.008	34 ± 4 s^{-1}	5.0 ± 0.3
14	Concussion	Tucked-in elbow	0.219 ± 0.022	64 ± 13 s^{-1}	7.0 ± 0.6
15	No injury	Shoulder check	0.196 ± 0.014	40 ± 10	5.5 ± 0.4
16	No injury	Shoulder check	0.230 ± 0.003	37 ± 3	5.9 ± 0.6
17	No injury	Shoulder check	0.167 ± 0.010	25 ± 3	5.1 ± 0.7
18	No injury	Shoulder check	0.298 ± 0.017	53 ± 5	8.9 ± 0.7
19	No injury	Shoulder check	0.175 ± 0.017	29 ± 6	5.4 ± 0.7
20	No injury	Shoulder check	0.263 ± 0.042	60 ± 12	8.9 ± 1.4
21	No injury	Shoulder check	0.283 ± 0.016	57 ± 15	9.5 ± 0.3
22	No injury	Shoulder check	0.239 ± 0.022	42 ± 5	7.7 ± 0.6
23	No injury	Shoulder check	0.382 ± 0.015	75 ± 13	13.0 ± 0.8
24	No injury	Extended elbow	0.160 ± 0.010	29 ± 8	6.0 ± 0.9
25	No injury	Extended elbow	0.191 ± 0.015	30 ± 7	6.2 ± 0.5
26	No injury	Extended elbow	0.103 ± 0.014	27 ± 5	4.0 ± 0.4
27	No injury	Tucked-in elbow	0.148 ± 0.021	30 ± 6	5.1 ± 0.6

Discussion

This study was designed to characterize brain tissue strain and stress of concussive and non-injurious elbow and shoulder to head strikes in ice hockey. The results showed that collisions resulting in concussion had higher strain rates, and VMS than collisions resulting in no injury but similar MPS. The data also indicated that the mechanism of injury has an effect on brain tissue deformation metrics. Extended elbow strikes had similar results to tucked-in elbow strikes, but both generated lower VMS, MPS, and strain rate than shoulder checks. In addition, shoulder checks resulting in no injury had higher MPS, strain rate, and VMS than elbow impacts resulting in concussion. This would suggest that these brain tissue deformation metrics are able to identify a concussion but that prediction could be improved by refining the methodology (e.g., account for the brain's sensitivity to impact direction, compare stress and strain in different functional sites of the brain). Furthermore, it is interesting to note that while peak linear and angular acceleration associated with concussion were lower for ice hockey shoulder-to-head and elbow-to-head collisions when compared to head-to-head collisions in the NFL and head impacts in rugby or Australian rules football, brain deformation metrics were similar [Kleiven, 2007; Patton et al., 2013; Zhang et al., 2004]. This may be in part due to the differences in the mechanism of injury. While head-to-head collisions generated head acceleration curves lasting approximately 15 ms, shoulder checks and extended elbow impacts had impulse duration above 25 ms. This would suggest that impulse duration has an influence on brain tissue deformation and should be considered when investigating head injury risk using head acceleration.

Shoulder checks were reconstructed using a 14.7 kg, high compliance striking arm; extended elbow impacts were reconstructed using a 6.2 kg, medium compliance pendulum; and tucked-in elbows were reconstructed using a 2.9 kg, low compliance pendulum. Shoulder checks generated higher angular acceleration peaks and impulse durations than both elbow conditions. Considering the very high correlations between angular acceleration and brain trauma metrics, it is not surprising that shoulder checks also resulted in high MPS, strain rate, and VMS. This was not the case however for both types of elbow impacts which had different head acceleration peaks and impulse durations but similar MPS, strain by strain rate, and VMS. This suggests that an interaction between impact mass and compliance is likely and both parameters play an important role in the risk of concussion.

Shoulder-to-head and elbow-to-head collisions in elite ice hockey had a mean peak MPS of 0.24 ± 0.09 for concussive impacts and 0.22 ± 0.07 for non-injurious impacts. The estimated tolerable peak MPS levels were 0.30, 0.14, and 0.15 for a 50% risk of concussion following a shoulder check, extended elbow strike, or tucked-in elbow strike to the head, respectively. The values are consistent with data reported in previous reconstructions of sports collisions. A finite element analysis of NFL collisions reported a mean MPS of 0.3 for concussive cases and 0.2 for no injury cases [Zhang et al., 2004]. A mean MPS of 0.47 ± 0.20 was associated with rugby and Australian rules football collisions resulting in concussion and 0.24 ±0.12 for collisions resulting in no-injury [Patton et al., 2013]. The study reconstructed 27 concussive impacts, of which 14 were accompanied by loss of consciousness. A previous study reported that these 14 cases had higher head acceleration so it is possible that the inclusion of collisions resulting in loss of

consciousness also increased reported mean MPS [Fréchède and McIntosh, 2009]. The values reported in this study are also consistent with anatomical studies involving the effects of strain on neuronal conductivity. By using giant squid axons, Galbraith et al. [1993] were able to study the physiological response associated with an applied load without the interference of surrounding structures or physiological interactions. It was observed that following an elongation of 0.12 in 14 milliseconds the axon was unable to elicit an action potential response when stimulated. This loss of functionality was only transient and the action potential reappeared after a three min period of rest. Axons subjected to strains above 0.20 were found to never fully regain their resting potential while axons subjected to strains above 0.25 failed structurally. Axonal trauma was also studied using guinea pig optic nerve to determine the magnitude of strain necessary to cause functional and structural failure [Bain and Meaney, 2000]. Functional thresholds were determined to be 0.13, 0.18 and 0.28 for a conservative, liberal and optimal threshold, respectively. While the results of this study were limited to the guinea pig optic nerve, the authors believed that mechanical behavior for brain tissue may not vary significantly from species to species and these strain-based thresholds could be applied to the human brain.

Shoulder-to-head and elbow-to-head collisions in elite ice hockey had a mean peak strain rate of 47 ± 21 s^{-1} for concussive impacts and 42 ± 17 s^{-1} for non-injurious impacts. While significantly different, the difference between both concussive and non-injurious impacts is not as pronounced as what was reported for NFL helmet-to-helmet collisions [Kleiven, 2007]. The difference of approximately 25 s^{-1} between concussion and no injury impacts could be

attributed to the methodology used. Most of the athletes selected for the no injury condition were in a striking position meaning that the headform used and the impact location and orientation was different [Pellman et al., 2003b]. The estimated tolerable peak strain rate level was 59 s^{-1} for a 50% risk of concussion following a shoulder check. This is consistent with anatomical research that linked strain rate to neuronal loss in conductivity. Galbraith et al. [1993] found that for a similar elongation of 0.12, the membrane potential of squid giant axons exposed to a low strain rate returned immediately to their resting level, while the membrane potential of axons exposed to a high strain rate required 30 min to recover their resting potential. The effect of loading rate was also studied by Morrison et al. [2003] who reported that brain tissue damage increased when subjected to a strain of 0.20 at rates of 5, 10, 20 and 50 s^{-1}. Furthermore, brain tissue subjected to strains of 0.35 and 0.50 at a rate of 10 s^{-1} were able to recover within four days while tissue subjected to identical strains applied at a rate of 50 s^{-1} resulted in permanent loss of function or death.

Shoulder-to-head and elbow-to-head collisions in elite ice hockey had a mean peak VMS of 8.0 ± 3.4 kPa for concussive impacts and 7.2 ± 2.5 kPa for non-injurious impacts. This is in agreement with previous reconstructions of NFL game impacts which reported 8.4 kPa as being associated with a 50% risk of concussion [Kleiven, 2007]. Furthermore, it is lower than values associated with a risk of moderate neurological injuries published in other studies. Sixty-four real-world accidents were reconstructed using anthropomorphic dummies for motorcycle and American football accidents and an analytical model for motor vehicle accidents involving pedestrians [Willinger and Baumgartner, 2003]. Stress was calculated using a finite element

model developed at the Strasbourg Louis Pasteur University. The analysis showed that VMS was a good indicator for neurological lesions and identified 18 kPa for a 50% risk of moderate lesions. The term “moderate” was not defined; nevertheless, knowing that 22 cases were taken from the NFL studies, it is inferred that concussion falls in that category.

Pearson correlations identified a low correlation between linear acceleration and brain tissue deformation metrics. This is troublesome because peak linear acceleration currently is the criteria used to assess the performance of sports helmets. The poor relationship could in part explain why the rate of concussion has not diminished despite the introduction and improvement of protective headgear [Benson et al., 2011; Casson et al., 2010; Wennberg and Tator, 2003]. Considering the very high relationship between peak angular acceleration and brain tissue deformation metrics, the data presented in this study would support the inclusion of angular acceleration in test protocols.

Limitations

The data presented in this study are specific to the mechanism of injury in ice hockey, the reconstruction method, and the finite element model. The data pertain to shoulder-to-head and elbow-to-head collisions in elite ice hockey. Other mechanisms of injury, which have their own specific impact parameters and head response, may generate different results. For example, elite players struck to the helmet with a puck shot at 35 m/s may experience head accelerations of 165 g and 15.4 krad/s^2 [Rousseau, Hoshizaki, and Gilchrist, In press]. It is likely that the mass and compliance of a striking body has an influence on head acceleration and injury risk and a comparison between other impact mechanisms in ice hockey needs to be

conducted. The 50th percentile adult male Hybrid III head and neck used in this study were designed and validated for indirect head impacts following car crash [Deng, 1989; Mertz, 1985]. The headform is made of steel and can only approximate the dynamic properties and impact response of a real human head [Hubbard and McLeod, 1974; Seeman, Muzy, and Lustick, 1986]. Moreover, the Hybrid III neckform is made of stiff materials and validated against inertial loading rather than direct impact [Deng, 1989]. Nevertheless, the Hybrid III dummy has been used to assess head injury risk in sports [Pellman et al., 2003; Walilko et al., 2005; Withnall et al., 2005] and is widely used in the industry of sports protection making it the best surrogate for physical reconstructions.

Comparisons between the brain tissue metrics computed with the UCDBTM should not be directly compared to metrics obtained using a different FE model or animal data. The data are a direct result of the model's representation of the head and material composition which differs between models. Finite element modeling requires many assumptions, and comparison with experimental data is crucial for their validation [Ward and Nagendra, 1985]. Correct constitutive properties are a limitation to all existing FE models. Properties taken from in-vivo tests on animal tissues are scaled, jeopardizing its reliability while it is unknown how the properties obtained by in-vitro tests on human samples are altered by their cadaveric state [Gilchrist, 2003]. Correlation with experimental data is difficult due to the scarcety of experimental data. A model is considered validated if its response is reasonably correlated to the response measured experimentally. It is therefore not proper to assume that the model has been validated for a different situation or parameter [Ward and Nagendra, 1985]. Nevertheless,

the metrics computed in this study can be put into context using a wide range representing concussion risk.

Conclusions

The purpose of this study was to describe the relationship between concussion and brain tissue deformation metrics using data obtained from reconstructions of 27 shoulder-to-head and elbow-to-head collisions in elite ice hockey. When the mechanism of injury was used to separate the data, mean maximum principal strain, strain rate, and von Mises stress were significantly higher for a group of 14 concussion cases when compared to a group of 13 no injury cases. Furthermore, peak linear acceleration had a low correlation with the three brain tissue deformation metrics while angular acceleration had a very high correlation. This is interesting because the primary focus of helmet manufacturers is to reduce peak linear acceleration, possibly at the expense of angular acceleration. The data presented in this study are specific to shoulder-to-head and elbow-to-head collisions. The different head injury mechanisms present in ice hockey should also be reconstructed and analyzed to obtain a more accurate depiction of the relationship between head injury metrics and concussion.

Chapter Six

SYNOPSIS AND CONCLUSION

The research presented in this dissertation was divided in three studies. The first study calculated effective impact mass of elbow-to-head and shoulder-to-head strikes by recreating the conditions in laboratory using human test subjects. The second study examined the head dynamic response of concussive and non-injurious ice hockey elbow and shoulder to head strikes by reconstructing real-world collisions in laboratory. The third study investigated brain tissue strain and stress of concussive and non-injurious ice hockey elbow and shoulder to head strikes by performing a finite element analysis of the dynamic response obtained following reconstructions of real-world impacts.

In the first study, 15 competitive ice hockey players were recruited to recreate three common striking techniques in laboratory. The results indicated that impacts delivered with the shoulder involved the highest amount of mass with an average near 15% of a player's body mass while impacts performed with an elbow were between 3% and 5%, depending on its alignment with the striking player's body. When compared to past studies, effective mass of elbow strikes was found to be comparable to a straight punch to the jaw (2.9 ± 2.0 kg) [Walilko et al., 2005] while effective mass of the shoulder strike was comparable to the head-to-head collision in football (14.0 kg) [Viano and Pellman, 2005].

In the second study, 14 concussive and 13 non-injurious elite ice hockey collisions were reconstructed in laboratory using an anthropomorphic test dummy. The results showed that impacts resulting in concussion had higher peak linear acceleration and impulse duration than impacts resulting in no injury. The same relationship was observed for peak angular acceleration and impulse duration. A 50% likelihood of concussion following a shoulder check to the head was established for a peak angular acceleration of 4.0 krad/s^2. A 50% likelihood of concussion following an extended elbow strike to the head was established for a peak linear acceleration of 11 g and a peak angular acceleration of 1.7 krad/s^2. A 50% likelihood of concussion following a tucked-in elbow strike to the head was established for a peak angular acceleration 2.5 krad/s^2.

Collisions resulting in concussion had a mean peak linear acceleration and a mean peak angular acceleration below values associated with sports concussion reported in the literature [Fréchède and McIntosh, 2009; Pellman et al., 2003; Zhang et al., 2004]. Differences in head acceleration may be a result of case selection, impact parameters inherent to each sport, and the methodology used. Conversely, the results reported in this study are similar to data reported by studies investigating head acceleration during youth and collegiate hockey games using in-helmet telemetry system [Gwin et al., 2009; Mihalik et al., 2008; Reed et al., 2010]. Furthermore, the data are in agreement with sub-injurious testing of volunteers that revealed that an acceleration of 45 g could be withstood for 0.044 s without any loss of consciousness or confusion. Tolerance was observed to decrease to 40 g for a pulse duration of 0.1 s and 25 g for a pulse duration of 0.2 s [Eiband, 1959].

In the third study, concussion metrics for the 27 reconstructed ice hockey collisions were analyzed using the University College Dublin Brain Trauma Model. The results showed that collisions resulting in concussion had significantly higher strain rates, and VMS than non-injurious collisions but similar MPS. The data also indicated that the mechanism of loading have an effect on brain tissue deformation metrics. Extended elbow strikes had similar results to tucked-in elbow strikes, but both generated lower responses than shoulder checks. In addition, shoulder checks resulting in no injury had higher MPS, strain rate, and VMS than elbow impacts resulting in concussion. This would suggest that these brain tissue deformation metrics are able to identify a concussion but that prediction could be improved by refining the methodology Furthermore, it is interesting to note that while peak linear and angular acceleration associated with concussion were lower for ice hockey shoulder-to-head and elbow-to-head collisions when compared to head-to-head collisions in the NFL and head impacts in rugby or Australian rules football, brain deformation metrics were similar [Kleiven, 2007; Patton et al., 2013; Zhang et al., 2004]. This may be in part due to the differences in the mechanism of injury. While head-to-head collisions generated head acceleration curves lasting approximately 15 ms, shoulder checks and extended elbow impacts had impulse durations above 25 ms. Considering the very high correlations between angular acceleration and brain trauma metrics, it is not surprising that shoulder checks also resulted in high MPS, strain rate, strain by strain rate, and VMS. This was not the case however for both types of elbow impacts, which had different head acceleration peaks and impulse durations but similar MPS, strain by strain rate, and VMS. This suggests that an interaction between impact mass and compliance is likely and both parameters play an important role in the risk of concussion.

Conclusion

The purpose of this thesis was to identify the metrics which best characterize concussion following ice hockey collisions and provide values reflecting concussive and non-injurious events. The first study reported effective mass of shoulder checks, extended elbow strikes and tucked-in elbow strikes. The techniques were selected because they are the most common injury mechanism in body-to-head collisions in ice hockey [Hutchison, 2011]. The results of this study were used to guide effective impact mass and compliance for the laboratory reconstructions or real-world ice hockey collisions. The second study reported that concussions following shoulder and elbow to head strikes in ice hockey occurred at lower peak linear and angular accelerations than expected. Conversely, it was reported that linear and angular acceleration time histories played a large role in the mechanism of injury. The results also indicated that concussion risk estimations are specific to the mechanism of loading. A 50% likelihood of concussion following a shoulder check to the head was established for a peak angular acceleration of 9.2, 6.9, 4.6, and 2.2 krad/s^2 for impulse durations of 15, 20, 25, and 30 ms, respectively. A 50% likelihood of concussion following an extended elbow to the head was established for a peak linear acceleration of 23, 15, and 7 g for impulse durations of 15, 20, and 25 ms, respectively. Finally, the third study reported that peak Maximum Principal Strain, strain rate, and von Mises stress were comparable to the ones obtained reconstructing head-to-head collisions in the NFL and head impacts in rugby or Australian rules football despite having lower peak linear and angular acceleration values. The estimated tolerable peak MPS levels were 0.30, 0.14, and 0.15 for a 50% risk of concussion following a shoulder check, extended elbow strike, or tucked-

in elbow strike to the head, respectively. The estimated tolerable peak strain rate level was 59 s^{-1} for a 50% risk of concussion following a shoulder check.

This information is particularly important to helmet manufacturing companies because the development and improvement of protective equipment is currently driven by head acceleration. Ice hockey helmets are designed to reduce peak linear acceleration effectively reducing the risk of traumatic head injuries. Unfortunately, impulse duration and angular acceleration have been of lesser importance due to a lack of data linking them to concussion in ice hockey. This thesis supports the inclusion of linear acceleration impulse duration, peak angular acceleration, and angular acceleration impulse duration in safety standards as they were shown to be good predictors of concussion following shoulder checks and extended elbows to the head in ice hockey. Furthermore, it was shown that head impacts in ice hockey did not generate the same linear and angular acceleration histories as other contact sports such as American Football. Thus, the specific loading mechanisms present in ice hockey should be mimicked when considering creating new safety standard protocols.

This study may also be of interest to companies creating head motion sensors designed to predict concussion. The results indicated that peak linear or angular accelerations used alone are not adequate predictors. Incorporating impulse duration in the sensors' algorithms would greatly improve their ability to detect concussion accurately. The results also indicated that the characteristics of the acceleration impulses are specific to the mechanism of loading and using a single cut-off value to indicate a possibility of concussion is inappropriate. While it may be

difficult for the sensor to detect the nature of the impact, coaches, trainers, and physicians could be taught to recognize the different mechanisms of loading and act accordingly.

Finally, this thesis supports the modification of player behaviour on the ice. Most concussions were the result of strikes to the face which was unprotected by the helmet, which indicates that improvement made to the helmets may not increase concussion protection from body-to-body collisions. A change in the regulations (banning strikes to the head) would be a more efficient way to reduce the rate of concussion in ice hockey due to shoulder and head impacts to the head.

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APPENDIX I

Consent Form

Project title: **A biomechanical analysis of ice hockey collisions**

Name of Researcher: T. Blaine Hoshizaki, Ph.D., University of Ottawa; Neurotrauma Impact Science Laboratory, University of Ottawa, Canada

Invitation to Participate: I am invited to participate in the abovementioned research study conducted by Dr. Hoshizaki, Associate Professor and Vice-Dean at the University of Ottawa's School of Human Kinetics.

Purpose of the Study: The purpose of the study is to document the dynamic response of a head form following a shoulder, arm, and elbow hit to the head. The results will be used to validate a concussive event reconstruction protocol designed to detect relationships between impact mechanical parameters and concussion.

Participation: My participation will consist essentially of a 90 minute session during which I will be asked to complete a consent form and a physical activity readiness questionnaire (PAR-Q) to ensure I am physically capable of completing the required tasks of the study. I will be given a standardized CSA certified elbow, arm, and shoulder protective gear and required to wear my own personal CSA certified ice hockey helmet. Following a warm-up, I will be instructed to strike a Hybrid III head form five (5) times for each of the three (3) striking methods. Between each trial, I will be given three (3) minutes of recovery. Each of the trial will be recorded on video for research purposes.

Risks: My participation in this study may result in muscle pain or delayed onset of muscle soreness which can develop due to the demand of full muscle contractions to achieve desired striking force. I may also experience minor bruising if I fail to place and secure the protective gear or employ an improper hitting technique. I have received assurance from the researcher that every effort will be made to minimize those risks and that I can withdraw from the study at any time and/or refuse to answer any questions.

Benefits: My participation in this study will not benefit me directly. My participation will however help develop further understanding of how brain injuries occur, and how they may be better prevented in the future.

Confidentiality and anonymity: I have received assurance from the researcher that the information I will share will remain strictly confidential. I understand that the contents will be used only for research purposes and that my confidentiality will be protected. Anonymity will be protected and all data will be associated with an alpha-numeric code only. I understand that any published results will be presented with complete anonymity. Data will be pooled for analysis, and all identifying documents will be kept separate from data. I understand that any personal data (e.g., address, telephone number) will be kept in a separate file folder.

Conservation of data: My consent form and PAR-Q questionnaire will be stored in a locked cabinet located in Dr. Hoshizaki's locked and password protected laboratory.

Test results will be kept in a secure manner on a password secured computer, located in Dr. Hoshizaki's locked and password protected laboratory. This information will be kept for 5 years once the data have been published and will be destroyed at the end of this period.

Voluntary Participation: I am under no obligation to participate and if I choose to participate, I can withdraw from the study at any time and/or refuse to answer any questions, without suffering any negative consequences. If I choose to withdraw, all data gathered until the time of withdrawal will be destroyed and will not be used.

Criteria for Participation: I am a healthy male aged between 18 and 30 years, have played high level competitive ice hockey (Junior A or above) and am not experiencing pain or discomfort from an injury to the upper limbs or shoulders (Initials_____).

Acceptance: I, _____, agree to participate in the above research study conducted by Dr. Hoshizaki of the University of Ottawa's Neurotrauma Impact Science Laboratory.

If I have any questions about the study, I may contact the researcher.

IF I HAVE ANY QUESTIONS REGARDING THE ETHICAL CONDUCT OF THIS STUDY, I MAY CONTACT THE PROTOCOL OFFICER FOR ETHICS IN RESEARCH, UNIVERSITY OF OTTAWA, TABARET HALL, 550 CUMBERLAND STREET, ROOM 154, OTTAWA, ON K1N 6N5, (613) 562-5387 OR ETHICS@UOTTAWA.CA

There are two copies of the consent form, one of which is mine to keep.

Participant's signature: _____ Date: _____

Researcher's signature: _____ Date: _____

Consent to use photographic, video and audio recordings: I give my permission to Dr. Hoshizaki and the Neurotrauma Impact Science Laboratory to use, reproduce, publish, transmit, distribute, broadcast and display photographs, videos or audio recordings that contain my image or voice, without my name associated with them. In any current or future Neurotrauma Impact Science Laboratory material, publications, multimedia productions, video, displays, advertisements and on the laboratory's website, social media website and other current or future media, without further notice to me or without my approval of the finished photographs, videos or audio recordings.

Participant's signature: _____ Date: _____

APPENDIX II

University of Ottawa Office of Research and Integrity ethics approval

File Number: H06-12-23 **Date (mm/dd/yyyy):** 08/23/2012

Université d'Ottawa University of Ottawa
Bureau d'éthique et d'intégrité de la recherche Office of Research Ethics and Integrity

Ethics Approval Notice

Health Sciences and Science REB

Principal Investigator / Supervisor / Co-investigator(s) / Student(s)

First Name	Last Name	Affiliation	Role
Blaine	Hoshizaki	Health Sciences / Human Kinetics	Principal Investigator

File Number: H06-12-23

Type of Project: Professor

Title: A Biomechanical Analysis of Ice Hockey Collisions

Approval Date (mm/dd/yyyy)	Expiry Date (mm/dd/yyyy)	Approval Type
08/23/2012	08/22/2013	Ia

(Ia: Approval, Ib: Approval for initial stage only)

Special Conditions / Comments:
N/A

1

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http://www.recherche.uottawa.ca/deontologie/index.html

File Number: H06-12-23 **Date (mm/dd/yyyy):** 08/23/2012

Université d'Ottawa University of Ottawa
Bureau d'éthique et d'intégrité de la recherche Office of Research Ethics and Integrity

This is to confirm that the University of Ottawa Research Ethics Board identified above, which operates in accordance with the Tri-Council Policy Statement and other applicable laws and regulations in Ontario, has examined and approved the application for ethical approval for the above named research project as of the Ethics Approval Date indicated for the period above and subject to the conditions listed the section above entitled "Special Conditions / Comments".

During the course of the study the protocol may not be modified without prior written approval from the REB except when necessary to remove subjects from immediate endangerment or when the modification(s) pertain to only administrative or logistical components of the study (e.g. change of telephone number). Investigators must also promptly alert the REB of any changes which increase the risk to participant(s), any changes which considerably affect the conduct of the project, all unanticipated and harmful events that occur, and new information that may negatively affect the conduct of the project and safety of the participant(s). Modifications to the project, information/consent documentation, and/or recruitment documentation, should be submitted to this office for approval using the "Modification to research project" form available at:
http://www.research.uottawa.ca/ethics/forms.html

Please submit an annual status report to the Protocol Officer 4 weeks before the above-referenced expiry date to either close the file or request a renewal of ethics approval. This document can be found at:
http://www.research.uottawa.ca/ethics/forms.html

If you have any questions, please do not hesitate to contact the Ethics Office at extension 5387 or by e-mail at: ethics@uOttawa.ca.

Riana Marcotte
Protocol Officer for Ethics in Research
For Daniel Lagarec, Chair of the Sciences and Health Sciences REB

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