Evaluation of Impact Attenuation of Facial Protectors in Ice Hockey Helmets

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Abstract

The purpose of this study was to investigate whether ice hockey facial protectors can decrease overall head acceleration during blunt impacts as well as to identify if attenuation differences exist between visors and cages. Commercial models of three cages and three visors were assessed. Blunt impacts were simulated permitting the measurement of peak accelerations (PA) within the surrogate head form. Results indicated that indeed face protectors in combination with helmets substantially reduced PA during blunt impacts within threshold safety limits (below 300 g's). In general, cages showed lower PA than visors (p=0.004). Differences between models were also observed during repeated impacts and impact site (p=0.0001, p=0.007). In conclusion, this study demonstrates that facial protectors function beyond their role in preventing facial injuries, complementing the role of the helmet in attenuating head deceleration during impact. Consequently, the utilization of facial protectors may reduce the severity and incidence of mTBI.

Résumé

L'objectif de cette étude fut de déterminer si les protecteurs faciales pouvaient réduire l'accélération aux cours d'impacts au visage, ainsi que déterminer s'il existe une différence entre la cage et la visière. Six (6) modèles de protecteurs faciales (3 cages et 3 visières) furent évalués. Les simulations furent produites sur une tête NOCSAE médium relié par armature à un monorail. L'accélération maximale lors de l'impact (PA) fut mesurée pour les différents types d'impacts. Les impacts facials ont démontré que le port d'un protecteur facial combiné avec un casque de hockey réduisait d'avantage le PA que le port de seulement le casque de hockey. De plus, les cages ont des valeurs PA plus faibles que les visières (p=0.004). Des différences furent observées pour le nombre d'impacts répétés et pour le site d'impact. En conclusion, cette étude démontre que les protecteurs facials font plus que simplement protéger le visage; ils complémentent le rôle du casque de hockey en atténuant les forces d'impacts. Par ce fait, l'utilisation de protecteurs facials peut réduire la sévérité ainsi que l'incidence de commotions cérébrales.

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I. INTRODUCTION

The beginnings of North American ice hockey have been traced back to 1783, when British troops used natural arena of frozen ponds and rivers to ease the bleakness of winter of the colonized country (8). Today, ice hockey is the most popular team sport played on ice. It is one of the world's fastest sports; players on skates are capable of going high speeds on natural or artificial (indoor) ice surfaces. Ice hockey offers a setting that is peculiar to injuries: players move on sharp skates at speeds of up to 30 mph on a solid ice surface that is confined by rigid boards, players handle novel technological sticks made of wood, carbon graphite, or aluminium, and propel a piece of vulcanized rubber at speeds of up to 100 mph (18). Hence, it is recognized among the most dangerous of contact & collision sports. The purpose of this review is (1) to establish the prevalence of head injuries, specifically concussion, in ice hockey and to determine the causes and mechanisms of concussion injury; (2) to elaborate on the clinical effects of concussion; (3) to discuss the existing measures of prevention, and (4) to analyse the role of facial protectors in ice hockey.

II. PREVALENCE OF INJURIES

The risk of injury is an integral part of the game. An ice hockey injury refers to a traumatic injury event occurring during a practice or game that causes the player to miss the remaining of the practice or game or more, and/or requires the player to consult a health professional (16, 33). The National Collegiate Athletic Association (NCAA) have reported that game injury rates have increased almost from 0.7 injuries/game in 1986 to 2.6 injuries/game in 2000. This is a fourfold increase over almost 15 years. Injuries are the result of player collision with another player or with the environment (side boards, goal posts, ice etc.). The increases in injury incidences may be attributed, in part, to the fact that over the past 20 years, there has been a 10 lbs increase in the average weight of NHL hockey players. Greater speeds and mass increase the momentum, the energy, and the force when collisions occur. Consequently, collisions can result in greater magnitudes of injuries (56).

Head and neck injuries have consistently ranked as one of the most commonly injured body sites, followed by the knee, shoulder, hip/thigh/groin region, and hands/fingers (3). Marchie et al. reported in a study, on the role of body checking, that each season 10-12% of minor league hockey players 9-17 years old report a head injury, most commonly a concussion (35). Additional studies report head injuries to represent up to 20% of all injuries (14, 18, 21, 25). These injury types include cerebral concussions, lacerations, contusions and fractures. An average of 30% of all hockey-related head injuries have been reported to be cerebral concussions. The prevalence of concussions in contact sports in general has reached a substantial level. Recent studies demonstrate that there are more than 300,000 new cases of concussion which occur annually in contact sports (7, 44). Moreover, it is estimated that 3.9 to 7.7% of high school and college athletes, respectively, sustain concussions each year, in all sports (24). Since 1986, media reports of national hockey league injuries show stable concussion rates for 10 seasons, followed by a tripling of the reported rate over two seasons in

the late 1990's, and then stability once again at a new, higher rate for the next three seasons observed during the study period. Concussions have been reported to account between 2 and 14% of all hockey-related injuries and 15 and 30% of all hockey related head injuries (21). Variations in reported values are mainly due to differences in study designs and different research groups. The dramatic increase is partly due to the increased recognition and attention given to concussion with defined diagnostic criteria. Irrespective of the greater attention to mTBI symptoms, clearly mTBI represents a substantial health concern for participants.

There are many factors which influence the reported incidence of injury in ice hockey: such as, age, sex and region. It may be argued these three factors influence the attitude and type of play which numerous studies have shown direct correlation with the number of injuries. The incidence of injuries increases with age in parallel with level of play. The most vulnerable group in competitive ice hockey are youths in their early teens. The age groups with the highest incidence of injuries are adolescents (12-17 years old) and young adults (18–24 years old), probably because training for competition is most intense and contact checking is introduced at these age groups (30). Serious injuries start at the Pee Wee level; for example, in a 2 year prospective study of the British Columbia Junior Hockey league, Goodman et al. report the mean age of a first concussion as 15 years old (21). Benson et al. showed in a cohort study conducted during the 1997-1998 Canadian inter-university athletics union hockey season, a total of 79 concussions were reported, with 67% occurring during the regular season (7). Over all, annual rates of concussion range between 7% - 10%. Yet, retrospective histories show an evidence of underreporting of concussion during studies. Thus, as much as 20% of elite ice hockey players will sustain a concussion in a season (21, 53). In recreational leagues, the incidence of concussion tends to decrease as age increases. This is probably because play becomes less competitive and more technical for older age

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groups. At both the professional and recreational levels, proper management of concussive injuries is warranted for long term health and well-being.

The prevalence of injuries in the adolescents group can also be explained by the lack of experience and skills of the sport which results in riskier behaviours. Throughout the years, an experience athlete will have developed reflexes which will help him protect himself. Under experience players may not be able to carry out skills as well as professional players they see on television which in turn puts them at risk for potential injuries.

The prevalence of ice hockey injuries is different for male and female players. Female players account for less than 10% of all ice hockey players in the United States (30). Injury rates in women leagues are similar to those in men's leagues where body checking is prohibited. However, when body checking is permitted, women injury rates are much less than men injury rates. According to a cohort study by Dryden et al. on female ice hockey players of the greater Edmonton area in Alberta, Canada, the injury rate for women's recreational hockey is 0.49 injuries/player during the season. Head and neck injuries account for 15.2% of the injuries with concussion representing 5.6% of all injuries (16). This is on the lower range of men hockey statistics. Furthermore, the women league players sustained less severe injuries than male league players as estimated as a function of time lost from hockey play. In a retrospective analysis study on ice hockey head and neck injuries presenting to emergency departments, almost 90% of injuries were to male individuals. This implies that mostly male will have severe injuries requiring medical assistance. Women leagues show many potential reasons for a lower incidence of injuries: absence of intentional body checking, mandatory full facial protection, differences in the nature of the game associated with body mass, speed and impact force, gender differences in behaviours, and gender specific mechanical differences. Women hockey players are competitive and play aggressively, but do not show intimidation, dominance and strength as compared to men

hockey players (at least in this stage of development of female competitive hockey). In addition, there appears to be a greater respect of fair play in women's ice hockey. As a result, the incidence of injuries are lower for women leagues compared to men leagues and generally, the injuries are less severe than men injuries.

The incidence of ice hockey injuries is also a function of the region where hockey is played. Ice hockey is popular in North America, but also in northern and eastern European countries. In many European leagues, it has been compulsory to wear a helmet since the 1960s. During this period, in Canada and the United States, wearing a helmet was mandatory only for non-adults. It was one decade later that all amateur players were required to wear standard certified helmets. Moreover, helmets were not required in the professional National Hockey League (NHL) until 1981 (8). In a prospective epidemiological study on Finnish ice hockey injuries, results show that less than 5% of the injuries are concussions. Similarly, in a 4 year study on elite Swedish ice hockey players, it was showed that 6% of all injuries are concussion. In addition, on average, players return to training and game player after a week. In comparison to North American hockey injuries, the severity of European injuries is much lower. Likewise, catastrophic ice hockey injuries are less frequent in Finland than North America (30). This appears to be due to larger rink sizes and less aggressive play behaviour. An inverse correlation between the ice size and collision rates during game play has been documented (56). In large ice rinks, there are fewer collisions. The greater space on the ice decreases the chances collision between players, be it volitionally or accidentally.

Ice hockey head injuries are caused by collisions. The main cause of concussion is the head striking the boards and/or the end glass and collision with the ice. Moreover, they can also be caused by player-player contact. They can be the result from direct elbow contact to the jaw or another part of the head (21). Unsurprisingly, the risk of injury is greater during game conditions as compared to practice conditions. According to NCAA's Injury

Surveillance System for the academic years 2002-2003 and 2003 – 2004, approximately 80% of concussions occur during games. During games, players play with increased intensity and forcible body contact. In practices, there is respect for team mates. The high intensity of the sport found in games results in frequent collisions between players and forceful impacts with the side boards, goal posts, other players, pucks and hockey sticks. Furthermore, the high skating speeds amplify the impact forces. Given that the players play with increasing force and speed, there is greater frequency of collision and greater force at which the players' heads are struck. The position of the player appears to be an important factor regarding concussion susceptibility. Goodman et al. show that almost 60% of concussions are experienced by forwards, where as defences and goal tenders experience approximately 30% and 5% respectively (22). Forwards are the most vulnerable players, especially because of checks from behind. Players are focused on the puck and lose sight angles of the opposite team. Unsuspicious of the coming check, the players have relaxed neck muscles resulting in slower reaction rates to protect themselves. This combined with the increased energy at which they are hit increases the risk and severity of injury.

III. CLINICAL REVIEW OF CONCUSSION

The linguistic origin of the word concussion derives from the Latin word, *concussus,* which means to shake violently (10). In the 1960's, the Congress of Neurological Surgeons defined concussion as a clinical syndrome characterized by immediate transient impairment of neural function such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to mechanical forces (44). After extensive research over the following decades, the original definition was revised. Sport concussion refers to a complex pathophysiological process affecting the brain induced by traumatic biomechanical forces (2, 37). It is the most common type of brain injury, and therefore, it is often referred to as mild traumatic brain injury (mTBI). There is typically a rapid onset of short-lived impairment of neurological

function that resolves spontaneously. Many symptoms can appear following the impact; head ache, dizziness, nausea, confusion and visual disturbances are among the most common ones. In general, cerebral concussion is considered as a non specific diagnosis for an event which results in a wide variety of signs and symptoms, whether they are general symptoms, cranial nerve symptoms, memory problems, cognitive problems, somatic complaints or a loss of consciousness (LOC). A concussion can result from one or more of the following: (a) a direct blow to the head, face, neck, or elsewhere on the body with an impulsive force transmitted to the head/brain, (b) rapid onset of short lived impairment of neurological function that resolves spontaneously, (c) a functional disturbance rather than a structural injury, (d) resolution of clinical and cognitive symptoms that typically follows a sequential course, and (e) grossly normal structural neuroimaging scans (37). The pathophysiological changes include neuronal changes, chemical changes, neuroelectrical imabalances and microstructural damages.

Sport therapists can recognize concussion in athletes when there is a sudden or brief impairment of consciousness or LOC. It resembles a state of generalized seizure activity that may result in tissue deforming collisions with the internal wall of the skull similar to the agitation the brain experiences during other types of closed head injuries (31). Mental status alteration is revealed by the array of self-reported symptoms, LOC, post-traumatic amnesia or

Table 1 – Signs & Symptoms of Concussion

Immediate	Delayed
Confusion	Irritability
Amnesia	Headaches
Headache	Depression
Loss of consciousness	Sleep disturbances, including insomnia or difficulty waking
Ringing in the ears (tinnitus)	Fatigue
Drowsiness	Poor concentration
Nausea	Trouble with memory
Vomiting	Getting lost or becoming easily confused
Unequal pupil size	Increased sensitivity to sounds, lights and distractions
Convulsions	Loss of sense of taste or smell
Unusual eye movements	Difficulty with gait or in coordinating use of limbs
Slurred speech	

retrograde amnesia. Table 1 shows the potential signs and symptoms of concussion. Positive signs and symptoms imply the presence of signs or symptoms that are not present in normal behaviour. Negative signs and symptoms refer to the absence of normal behaviour, signs or symptoms. As reported by Killam et al., neuropsychological evaluative testings have shown that concussed athletes show significant lower immediate memory and delayed memory scores (31). Moreover, processing speeds are inversely correlated with the severity of head injury (grade 1, 2 or 3). Lastly, learning disabilities can result from multiple concussions. Killam et al., identified that rest from sport participation buffers athletes from the deleterious effects of contact play; therefore, it appears that memory recovery is possible with time (31).

On field evaluation of a concussion is difficult. Following a forced impact with possible concussion, the initial decision a therapist must make is whether the athlete can or cannot return to play. There is no consensus among experts and practitioners concerning concussion grading scales, and standardized method of concussion assessment (46). The three traditional grading scales, used commonly in sports medicine, include the Colorado Medical Society System, Cantu Grading System, and the American Academy of Neurology (AAN) Guidelines. However, none of the scales have been universally accepted or followed with much consistency by the sports medicine community (22). The major issue with the grading scales is that they are based upon a subjective evaluation of the athletic trainer or physician at the moment of the incident. Moreover, the evaluation of the concussion will change depending on the scale the athletic therapist uses. The Colorado Scale system and the AAN guidelines report the state of mind of the athlete with loss of consciousness (LOC) identifying a severe concussion. The Cantu System determines severity evaluation solely based on LOC and post trauma amnesia (PTA). Loss of consciousness and amnesia seem to be the basic symptoms on which the scales attribute the severity of the cerebral concussion; however, according to the committee on Head Injury Nomenclature of the Congress of Neurological

Surgeons, neither is required for an injury to be diagnosed as concussion. Furthermore, a number of authors have documented no association between brief LOC and abnormalities on neuropsychological testing at 48 hours post concussion (22, 23, 24). Furthermore, studies involving high school and collegiate athletes with concussion revealed no association between (a) LOC and duration of symptoms or (b) LOC and neuropsychological and balance testing at 3, 24, 48, 72 and 96 hours post injury. This is indicative that athletes who experience LOC perform similarly to athletes without LOC on the same injury event. This indicates that LOC may not necessarily be a reliable predictor of concussion severity. Therefore, the grading scales available do not offer the best method to quantify severity of concussion appropriately.

There is plenty of controversy in regards of concussion severity evaluation. According to recognized grading scales, as shown in Table 2, LOC is associated with most severe concussions. Indeed, in a study on professional football players of the NFL, LOC occurred in 9.3% of reported concussion cases and involved more lost days than for players with concussion without LOC (46). However, as it was referred to previously, Guskiewicz et al. reported that athletes experiencing LOC performed similarly to athletes without LOC on post-

Table 2 – Criteria for Determining Concussion Severity

| | Severity Classification | | |
System	Grade 1	Grade 2	Grade 3
Cantu	No LOC, PTA <30	LOC <5 min or PTA >30 min	LOC>5min or PTA >24h
Colorado	Confusion, no amnesia, no LOC	Confusion and amnesia, no LOC	LOC
AAN	Brief confusion, no LOC, mental status clear <15min	Brief confusion, no LOC, mental status not clear in 15 min	any LOC, brief or prolonged

injury neuropsychological testing (24). Therefore, LOC may be a result of early neurological disturbances but may not be indicative of prolonged neuropsychological abnormalities following concussion. Grades of concussion severity should be associated with greater prevalence of symptoms and long duration of mental state impairment, and not on LOC at the

time of the event. Thus, factors initially associated with prolonged return to play include the number of symptoms, any general symptom, any memory or cognitive problem, and any somatic complaint. For instance, Asplund et al. have shown that persistent head aches and persistent concentration difficulties for more than 3 hours are significantly correlated with a prolonged return to play (>7days). Likewise, retrograde and post traumatic amnesia have been linked to prolonged return to play (1). Consequently, the therapist or physician must receive adequate training in dealing with concussion in order to understand that LOC itself is not enough to make the treating staff more concerned regarding the potential seriousness of the injury. Epidemiological studies on concussion measured severity of the injury as a result of time lost from practices and games. This further suggests that the grading scales should be revised. There is no consistent report of the evaluation according to the grading scales and time lost from play.

In the second part of their epidemiological study on concussion in professional football, Pellman et al. report the location and direction of helmet impact resulting in concussion. Analyzing the resulting symptoms from the various types of head impact could help determine the most dangerous impact types and possibly provide future directions for safety emphasis and research. This orientation could provide a link between symptoms and the type of impact to the head, for which at the moment none exists. For example, if impact to the back of the helmet shell produces concussion with a greatest number of symptoms, thus greater severity, regulation reinforcement can be implemented on certain types of plays in order to avoid these severe injuries.

Another difficulty in determining whether an athlete can or cannot return to play is that symptoms may not necessarily arise spontaneously after the impact to the head. In certain individuals, symptoms may appear 15-30 minutes post impact, and in rare cases, symptoms may appear after 24 hours. In contrast, symptoms may resolve immediately or may last an

indefinite amount of time. Consequently, no two concussions are identical and resulting symptoms can be very different. The variety on resulting symptoms is independent of the force of the blow to the head, the degree of metabolic dysfunction, the tissue damage and duration of time to recover, the number of previous concussions, and the time between injuries. Therefore, there is no definite treatment that can be applied in response to concussion. The Concussion in Sport Committee recommended that combined measures of recovery should be used to assess injury severity, and hence, individually guide return-to-play decisions (2, 37). This includes assessment of self-reported symptoms from the athlete, reference to all traditional grading scales and follow-up evaluations post injury.

During on field evaluation, the presence of positive and negative symptoms is an important marker for concussion severity and concussion outcome. The symptoms are the initial tools the therapist has in order to make a decision. According to the First International Symposium on Concussion in sport in Vienna, 2001, when a player shows a symptom or sign of a concussion, the player should not be allowed to return to play in the current game or practice; the player should be monitored for any deterioration of his condition and return to

Table 3 – Stepwise Process of Return to Play after Concussion

STEP	Directions
1	NO ACTIVITY/COMPLETE REST; once asymptomatic, Proceed to level 2
2	Light Aerobic exercise (Walking, stationary cycling); If asymptomatic, proceed to level 3
3	Sport-Specific Training (but not regular practice) If asymptomatic, proceed to level 4
4	Non-Contact Sport-Specific Training Drills; If asymptomatic, proceed to level 5
5	Full-contact training after medical clearance;
6	Game play/Competition Return

play should follow a supervised 6 step process of Return to Play After Concussion (2). Table 3 shows the gradual process that should be followed during the return-to-play transition. A player should never return to play while symptomatic. Premature return to play may place an athlete at an increased risk of repeat injury, possibly resulting in cumulative damage, or even, catastrophic outcome (34).

There exist important factors that the therapist must consider in addition to symptoms. These factors include the play environment, level of play and the age of the athlete. Increases in these factors correspond to increase the risk of concussion. Although, return to play should be granted only if the player is asymptomatic at rest and during physical exertion, return to play management decisions are easily influenced by an athlete's desire to return to competition or a coach's will to win. Play environment such as impact energy, location and frequency are important to consider in relation to the incidence of mTBI. The incidence of concussion increases with the level of play. It is related to the increased force and frequency at which the players play and are struck. The age of the athlete is another important factor influencing the decision of allowing the athlete return to play or not. The young developing brain is more at risk for injury than any mature developed adult brain. Therefore, younger athletes are more vulnerable to injury. Consequently, younger high school athletes recover more slowly than older collegiate athletes to equivalently matched concussions. Thus, high school athletes should have a larger return to play period.

Neuropsychological and postural balance testing are the available tools to test neurological damage resulting from concussion on individual athletes. Neuropsychological tests measure domains such as attention, memory, and speed of information processing. Posturography allows for the identification of individual neurological system (vestibular, visual, and somatosensory) contributions to postural control. In a prospective epidemiological study on intercollegiate athletes participating in football, soccer, basketball and cheerleading,

Peterson et al. demonstrated that concussed athletes show a significant decrease in information processing speed and composite balance compared to uninjured control subjects (47). The vestibular system appears most disrupted by concussion, yet returns to pre-injury baseline levels within 3 days post-injury (47). It is surprising that no significant differences were observed at other cognitive domains; all domains are susceptible to be affected by concussion. In animal models, repeated mTBI may lead to permanent learning disabilities and other neurological and psychiatric problems (34). Peterson et al. should pursue their analysis on a larger population in order to obtain significant measures.

There exist two main types of head injuries: focal head injuries and diffuse head injuries. Focal head injuries involve trauma localised to a specific region of the head and usually result from a direct blow to the head. 66% of head injury deaths are from focal head injuries. Diffuse head injuries involve a widespread or global neural disruption of the brain which may include structural damage with the loss of brain cells (10). Concussions are an example of diffuse head injuries. Following concussive head injury, living brain cells may exist in a state especially vulnerable to minor changes in cerebral blood flow, increases in intracranial pressure, anoxia, and most importantly, repeated concussion. After an initial concussion event, the chance for a second concussion is found to increase 4-6 times over the chances of an initial cerebral concussion. It is thought that the increased vulnerability of the brain to repeated impact is due to the acute metabolic and ionic changes, the cerebral blood flow-glucose metabolism uncoupling, intracellular calcium influx and accumulations, mitochondrial dysfunction and delayed glucose hypometabolism, reductions in magnesium, neurotransmitter alterations, and diffuse axonal injury caused by the first concussive injury. Repeated impacts, even below the accepted threshold, can lead to cumulative brain damage similar to that of one concussion and can lead to persistent symptoms or neuropsychological deficits that result in social dysfunction, loss of productivity and excessive healthcare costs.

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There is evidence that support the idea of specific developmental periods that the young brain may be more vulnerable to injury. During this period, diffuse mechanical injury can have lasting effects on the complex sequence of neurochemical and anatomical events occurring during normal development. Consequently, developmental brain injury can lead to impaired plasticity (20). Repeated concussions can result in permanent neurological disability due to the relevant cumulative effects.

Concussion evaluation is limited because damage cannot be measured neuroanatomically or physiologically, therefore, we cannot unambiguously quantify measure the extent of the injury or the severity of the metabolic dysfunction. Moreover, it cannot be measured precisely when it has cleared. A player who returns to play before the succession of acute post concussive symptoms is at risk of repeated injury. The potential for a second concussion lays neurochemically, as well as, behaviourally. A player who experiences dizziness, attention deficits, and slowed reaction times is at greater risk of a second injury because balance, the ability to attend and anticipate play development, and the ability to react appropriately are also necessary to avoid potentially dangerous body contact (8).

Catastrophic sport injuries are rare, tragic events. Second Impact Syndrome (SIS) occurs when an athlete who sustained a head injury, either a concussion or worse injury, such as a cerebral contusion, sustains a second head injury before symptoms associated with the first have cleared (2). The resulting ischemic condition and oedema of the first injury increase the brain's vulnerability to a second concussion. For similar reasons, an individual with a history of concussions remains at greater risk for repeated injury than an athlete with no history. The second blow may be remarkably minor, perhaps involving a blow to the chest, side or back that merely snaps the athlete's head and imparts accelerative forces to the brain. Pathophysiologically, there is a loss of autoregulation of the brain's blood supply leading to vascular engorgement within the cranium, consequently, increasing intracranial pressure and

herniation either of the medial surface (uncus) of the temporal lobe, or lobes below the tentorium, or the cerebellar tonsils through the foreamen magnum (20). Immediate medical attention is required in order to prevent fatal outcomes. The length of time the brain is vulnerable after a concussion remains unknown (34). Moreover, repeated minor head injuries may cause irreversible cumulative brain damage.

Extensive research on traumatic head injuries has only been done in the last twenty years. Therefore, there is limited understanding of the mechanism, sequelae and long term effects of concussions. Due to ethical issues, the only conclusions that can be drawn come from experimental animal models. Microscopic examination of brain tissue from primates exposed to experimental models of concussion revealed axonal shear strain on autopsy (4). Such shear strain is not detected in patients with mild head injuries using gross neuroimaging techniques, Magnetic Resonance Imaging (MRIs) or Computed Tomography Imaging (CT scans). Neuronal cellular injury causes disturbances of the membrane potential, thereby disrupting normal neuronal activity. Severe axonal shear strain, resulting in apoptosis, can cause permanent disabilities because Central Nervous System (CNS) neurones do not have the ability to regenerate. Laurer et al. have shown in experimental studies on rabbits and rats, that repetitive mTBI results in increased vascular permeability, behavioural dysfunction, and cytoskeletal abnormalities (34). Increased vascular permeability is indicative of a partial breakdown of the blood-brain-barrier; behavioural dysfunction and cytoskeletal abnormalities indicate a functional change of the injured neurones. This exposes the CNS to immunoglobulins, and thus, increases the risk of an immune reaction at the brain which are further complications of concussion. Interestingly, animal models show that systemic blood pressures are not affected by single or repeated injuries. Therefore, disturbances are limited to the CNS.

Multiple vectors of acceleration and deceleration in response to forces applied to the brain likely account for the greatest histokinetic changes, or axonal injuries, in mild head injury (4). MTBI is associated with post-traumatic oedema formation and changes in cerebral blood flow and metabolic rates. In addition, there are ion dishomeostasis and metabolic alterations which persist for days following concussive TBI, without creating overt morphological damage, and may represent the pathological basis for an increased vulnerability. The younger developing brain is at a higher risk of injury. Consequently, the brain acquires an increased vulnerability to a second mild traumatic injury for at least 24 hours after an initial episode of a mild TBI. Repeated injuries present potential for permanent damage and a possible risk of developing neurodegenerative disease later in life even if initial injuries are not severe enough to result in long-lasting disability or impairment.

Preventive measures, as explained above, remain the best solid ground on which therapist can rely to guarantee a normal and safe lifestyle for athletes. As a result, athletes have often been forced to retire from their sport because of prolonged post-concussion syndrome due to several repeated injuries.

There is limited information in regards of the biomechanical causes of traumatic injuries. Biasca (2002) has postulated three different possible mechanisms responsible for causing mTBI in ice hockey: (a) a direct blow to the head resulting in translational and rotational forces; (b) a direct blow to the face or jaw, also, resulting in translational and rotational forces; and (c) a blow directed to the chin resulting in transverse forces transmitted through the lower jaw, through the temporomandibular joint at the base of the skull, to the brain (8). Focal head injuries are caused by the force of contact and the head acceleration of direct blunt trauma. Wearing helmets certified to safety standards can reduce the severity of these injuries. A concussion is the mildest form of a diffuse brain injury. Diffuse head injuries are caused by the inertial effect of the mechanical blow to the head which can result in a

progressive widespread but heterogenous shearing of axons resulting in a global disruption of neurological function. It is hypothesized that the proportion and total number of axons damaged, as well as their anatomical location, could be correlated to the severity and reversibility of the clinical syndromes and the neurological deficits. Moreover, in cases with higher impact forces there will be relative movement of the brain and the skull in response to the impact, causing a contre-coup effect.

IV. PREVENTION MEASURES

No area of sports medicine involves more clinical uncertainty and controversy than the management of concussion. Indeed, no individuals will be affected by concussion exactly in the same manner. Each individual will have their own characteristic of self-reported symptoms to varying degrees and have their own neuropsychological disturbances. Likewise, the resolution of the concussive symptoms will be different for each individual. Consequently, for the health of the athlete, it is better to prevent the syndrome than to attempt to treat it.

Professional diagnosis and treatment of concussion are expensive and there is no guaranteed full recovery (MRI scans for brain damage, neuropsychological testing for memory and cognition evaluations etc). Athletes may have to miss several weeks of training and games in order to potentially fully recover from concussion. Injuries result in physical pain and loss of pleasure to play the sport. Consequently, as the incidence of concussion in sports is increasing preventive strategies are necessary to avoid the foreseeable risks and to attempt to control that which is inherent to the sport.

The main preventive measures taken are the standardization of protective equipment and regulation reinforcement of illegal play. In sports like football, the National Operating Committee on Standards for Athletic Equipment (NOCSAE) standards have proposed better helmet designs. The helmet shell and padding function to distribute the load of impact,

increasing the surface area on which impact is absorbed and lowering the risks of more serious brain injuries and cranial fractures. The cushioning effect of helmets increases the distance of deceleration and reduces the forces associated with concussion injuries. Since the implementation of revised NOCSAE standards for football helmets in 1978, for college players, and 1980, for high school players, there has been a 51% reduction in fatal head injuries, a 35% reduction in concussion, and a 65% reduction in cranial fractures observed over the first five years (44). For concussions in ice hockey, players should use properly fitted and maintained helmets. This increases the quality of protection of the helmet. The single chin strap of ice hockey helmets appears to allow the helmet to ride back on the head when a force is directed to the frontal region, exposing the chin. This is a typical force that occurs in collisions (33). Thus, modification of the single chin strap to prevent the helmet from riding back is recommended.

The use of facial protectors combined with the helmet would prevent several facial and concussion injuries. Northern American league regulations require players less than 18 years old players to wear full facial protectors, whereas, players more than 18 years old have the option to wear or not to wear a facial protector. Economic studies have shown that if every hockey player were given a hockey face protector for free, society would still make a profit in medical expenses avoided by use of a protective device (14).

For concussion in ice hockey, it is proposed that existing preventive measures include the use of mouth guards. Primarily, mouth guards are effective at reducing jaw fractures and dental injuries. In addition, it has been hypothesized that the mouth guard can serve as a shock absorber for impacts originating at the chin and the mandible area which could be transmitted to the brain. Particular attention has been given to mouth guard use in sports and there is evidence that mouth guard use reduces concussion severity. Results form Benson et al. show that 14 players from the half face shield cohort who did not wear mouth guards at the

time of concussion missed more than twice as many practices and games per injury than the 23 players who sustained concussion while wearing a half shield and mouth guard at the time of injury (7). Indeed, properly fitted mouth guards, particularly during a linear impact that involve the mandible, increase the time and distance of deceleration, and likely, offer cerebral protection. Thus, depending on the type of impact to the head, the mouth guard may help absorb impact forces to the head, and reduce concussion severity.

In consequence of effective protection, players tend to adopt riskier behaviour. Players have the feeling of being invincible; therefore, they tend to take more risks and be more aggressive. When tackling, the striking player generally hits the other player on the facemask. Facemask impacts can be severe when the struck player is unaware of the closing angle. Striking players receive impact on the top portions of the helmet where there is most protection. Although, the facemask, chin strap, brow, and side padding all contribute to the head loading, 76% of facemask impacts are below the head's center of gravity. As a result of being surprised by the tackle, there is not sufficient time for motor reflexes to prepare and protect from the fall. Moreover, some striking players use spearing technique which, banned years ago to decrease the risk of severe cervical spine injury

Unfortunately, equipment has led to risk compensation. Protective equipment creates the false perception of invincibility. Players frequently overestimate the protection provided by the required protective gear and demonstrate riskier behaviour (14). Equipment creates the false belief of invincibility of his own protection and of the protection of other players. Consequently, the player may play more aggressively in regards of the other players, increasing the risk of injuring them. As well, the player may put himself in vulnerable positions increasing the risk of his own injuries.

As well, strict enforcement of rules and stiffer penalties for illegal play will influence the way players will play hockey. If officials show consistent enforcement of existing game-

play rules, then players will respect rules and show fair play attitudes. In order to reduce the risk of injury, enforcement of rules penalizing actions where a clear intent to injure another player is demonstrated is necessary, especially when the injured player is in a vulnerable position. Such dangerous plays include elbowing at the jaw or head, charging, boarding, checking from behind, high stick to the head etc.

Intentional fist, stick and illegal body contact are a hockey fact of life (38). Players persist to carry out such plays even though they risk to be penalized for them and referees have been accused of leniency in regards of penalizing players for illegal plays. Indeed, foul play allowed by referees could be one of the most important reasons for injury, especially at the junior and high school levels. Rule enforcement can lead to increased safety without jeopardizing the nature of the game. Banned behaviours such as high sticking and slashing are among the most common types of illegal plays causing injury, and they should not be tolerated. Many concussions result from checking from behind or from cross checking. Hitting from behind can induce head first collisions with the boards or the ice. The danger in such actions is that checked players are not expecting to be hit. A player struck in the head by an opponent's body is in a vulnerable position with the head usually lowered and body bent at the waist. In addition, the forces are large and are concentrated towards the head, resulting in serious consequences including permanent brain or spinal injuries (26). As a result, the thrust of impact does not allow sufficient time for proper reflex reactions to fall safely. Thus, the risk of injury is highly present.

Checking from behind, cross checking and checks to the head are all illegal plays. However, checking from behind and cross checking tend to be treated less severely. There should be the enforcement of the no-checking-from-behind rule. Maintaining an environment with consistent reprimand for illegal actions is the responsibility of the officials. Consistent reprimands will render players less likely to perform such illegal plays. If a player repeats

illegal plays, then reprimands of bigger consequences should be granted. In women ice hockey leagues, no intentional body contact is permitted. Their injury rates are significantly lower than in men leagues. Accordingly, as a result of strict rule enforcement, a decrease in injury severity and injury rates could be expected.

Body checking is an important skill that allows players to take control of the puck, creates scoring opportunities and helps with defensive positioning and coverage, making it valuable to overall team play. However, body checking is often used as physical and mental intimidation to gain control of the game. It is the most common cause of trauma in hockey and it accounts for 86% of all injuries among players 9-15 years old (25). Illegal body checks cause up to 8% of the reported injuries in the same age group. Thus, a significant percentage of injuries could be avoided if regulations were properly enforced and encouraged to be respected by the referees, the coaches and the parents. Concussions are also most often caused by body checking. Therefore, body checking in minor hockey leagues prolongs the risk exposure of the child to concussion. Moreover, as mentioned before, the young developing brain is thought by some researchers to be more susceptible for concussion, therefore, proper surveillance of regulations is necessary in the minor hockey leagues. Players should not be introduced to body checking until they can make a mature, informed choice regarding the issue, thereby reducing the potential risk for injuries. Furthermore, regulation reinforcement can shift the game away from violent behaviour towards a game focused on skill.

The rules of ice hockey strategically permit a player to impede an opponent who has possession of the puck. Proper checks are legal actions in ice hockey. It was noted that 51% of concussion cases were caused by what was considered legal body checking, most often to the head (26). Head checking is referred to as an incident where a player is struck above the shoulders with the body, shoulder, elbow, knee, or stick of an opposing player. Likewise, penalties (major or minor) were called only in 45% of concussion cases (26). This

demonstrates the inherent risk of injury in ice hockey and rule changes are required to reinstate safety for all players.

The experience of the referee is essential when ambiguous situations arise. First, referees must be able to identify a player's clear intentions during play; referees must be able to distinguish between accidental and voluntary actions to hurt the opposing team players. Vitality and intensity should be allowed in ice hockey but the intent to hurt players can not be tolerated. Secondly, referees can not alter the way they police the game in function of the protective equipment worn by the athletes. Just as players may demonstrate riskier behaviour with belief that their protective equipment renders them invincible, referees have become more lenient as to how they police the game. Injuries occur when rules are not enforced and illegal plays are permitted.

Conditioning the neck musculature has been shown to improve the control of the head during surprised impacts leading to falls, especially in cases where the head follows a whip lash movement. Furthermore, coaches should universally train players how to properly give and receive checks. Enhanced coaching techniques will prepare players to the competitive conditions of the game.

Supplemental prevention measures include the replacement of the seamless glass around the ice rink with traditional clear plexiglass. The seamless glass offers better sight for the fans; however, it is more rigid than traditional plexiglass. Thus, collisions with seamless glass produce harder impact forces to the players' heads. Secondly, there should be a yearly adjustment or replacement of used equipment. This applies especially to pee wee and Bantam leagues where parents can not necessarily afford for new equipment every year for their children.

Improved medical care for on field recognition and treatment of injuries would prevent unnecessary complications of injuries. Immediate attention can be given to each player

following any blow to the head. Evaluation of the condition by qualified unbiased personnel will reduce any possible risk exposure. A certified athletic therapist or sport medicine specialist should be present at every practice and game of each team. In addition, programs similar to the NHL concussion study program should be effective in all competitive leagues of all age groups. There should be baseline neuropsychological testing with repeat testing performed 24-48 hours post concussion with further follow-up at five to seven days or until symptoms resolve. This will provide regularized supervision of the incidence of concussion and will insure that the player returns to play when he is ready.

The development of protective equipment requires a sound understanding of the injury mechanism, location and type. Likewise, it is important to be able to predict the tissue's response to stress, fatigue, displacement and velocity. Most importantly, equipment should neither be a source of new injuries nor compromise the nature of the game.

When hockey was played out doors on the large ice sheets with limited boundary materials, injuries were not a great concern. As the game moved to the enclosed indoor arena, play became more interactive between players and the environment. Players can reach high speeds of approximately 30mph when skating, and 15mph when gliding. Players make contact with numerous hard surfaces and objects: boards, glass, ice, goal posts, and other players. These cause tremendous acceleration and deceleration forces to the head. Unsurprisingly, the rate of injury increases as the size and the speed of the players increases.

As a result of the prevalence of catastrophic head injuries, various prevention measures have been taken in order to reduce or eliminate the injury rates. In 1965, the Canadian Amateur Hockey Association (CAHA) made helmet wearing mandatory for all non-adults. In 1975, all CAHA players were required to wear helmets certified with specific Canadian Standard Association (CSA) criteria. Example of CSA standardized tests include: the impact attenuation drop test, the faceguard penetration resistance test and the faceguard

projectile test. The introduction of rigorous standards for ice hockey helmets combined with increased wearing rates has been responsible for the reduction in fatal and serious head injury. Since 1979, helmets have been mandatory in all hockey leagues in North America including the professional National Hockey League (26).

The helmet's function is primarily to attenuate the energy of impact and to distribute the load force over a greater area. First, hard helmet shells permit the spread of impact forces during collisions, thereby reducing the potential for skull fractures. Secondly, liners inside increase the distance of shock absorption provided by the helmet. Several factors determine the performance of the helmet, including the liner foam, the liner thickness and density, and the shell stiffness. During impact, the liner is compliant and distorts as it is absorbing energy. After impact, the material is resilient, and thus, returns to its original form. Liners must reduce head acceleration to below relevant tolerance levels by allowing forces to be transmitted over thicker distances. Overall, the helmet reduces the magnitude of the forces applied to the head, thereby reducing stress and strain at the skull and brain (5). Moreover, ice hockey helmets are categorized as multiple-impact helmets and are designed to withstand a number of impacts without substantial performance degradation.

A helmet can function to reduce the risk of injury only if it can reduce the head impact force and the head acceleration to below relevant tolerance levels, and especially, under sport specific impact scenarios. Helmets must be worn properly for optimal protection. Improperly worn helmets are worn tipped back over the forehead or with the chin strap loosely fastened. If the helmet is placed further back on the head, the protective effect of the padding is minimized over the forehead area. The work of the helmet is limited by the context of the fall causing the head to collide with another player, the boards, or the ice. For example, when a player falls and hits his head on the ice, the distance travelled is greater than when it collides with the boards. The increased distance may result in increased acceleration upon impact due

to the pull of the body. The liner system in a helmet can only adequately decelerate the head upon impact. It is the role of the neck to decelerate the torso.

The helmet in ice hockey is effective in reducing fatal injuries but no helmet can completely protect the brain from injury (35). The mandatory use of ice hockey helmets fitted with facial protectors coincided with an increase in the occurrences of spinal cord trauma: injuries almost exclusively due to flexion/hyperflexion of the neck. It was believed that the added mass of a helmet and face shield to a player's head places the player in a position which is potentially vulnerable for cervical spine trauma. For instance, in chest (front) impacts, the neck muscles contract which directs shearing forces in the posterior direction. In rear impacts (impacts from behind), the neck is compressed causing the shear forces to be directed anteriorly. In a study on Hybrid III anthropomorphic test dummies simulating chest and rear impacts to the trunk of a hockey player, Smith et al. show that the addition of a helmet combined with a facial protector increases angular displacement. However, the increased angular displacement did not cause significant angular accelerations worthy of causing cervical spine trauma (28). Consequently, head and neck risk management strategies should include the mandatory use of full facial protection.

Similarly, a loose chin strap allows the normally proper fitted helmet and facial protector unit to shift from its proper position during impact with an opposing player, thereby eliminating its protective effect. The chin piece of the full face shield also helps hold the helmet in place during impact, thereby limiting forces created at the mandible to be transmitted to the brain and maintaining maximum player protection from brain injury. Facial protectors protect the face from contact with dangerous obstacles in the environment. Flying pucks and high sticks can easily hit the face causing bruises, laceration as well as fractures (dental, mandible or nasal). In addition, it is hypothesized that the facial protectors can help to

disperse and absorb impact forces that would otherwise be transmitted to the brain from a direct blow to an exposed jaw.

V. FACIAL PROTECTORS

In ice hockey, there exist two main types of facial protectors: the half shield visor and the full shield cage. A half shield, such as a visor, is a clear plastic visor that is attached to a helmet and extends approximately down to the tip of a player's nose. A full face shield, such as a cage, extends down to the bottom of a player's chin and covers the entire face. The Canadian Academy of Sport Medicine has produced position statement to promote prevention of eye and facial injuries by advocating the use of facial protection in ice hockey (9). As it became mandatory to wear full facial protection for players 18 years old or less, there has been hybrid products which have evolved in order to incorporate the advantages of both the cage and visor, and omit their respective inconveniences. The use of facial protectors in the professional league has raised plenty of controversy. Many argue that cages obstruct the field of view which impedes play. On the other hand, clear visors tend to accumulate fog which also obstructs the field of view. Consequently, research on the role of facial protectors is required to show evidence of efficient protection and minimal obstruction to play.

Current ice hockey research has documented a reduced risk of head and facial injuries with helmet use and facial protection. The helmet-face mask combination has fulfilled its role admirably, reducing the incidence of facial injuries by 70%. By 1988, not one eye injury had been reported for a player wearing a Canadian Standards Association (CSA) certified face protector (40). Facial protectors achieve perfectly their primary intended role: that to protect from fast moving pucks and high flying sticks. Laprade et al. report a lower incidence of facial lacerations with the mandatory use of face masks (34).

A trend of increasing catastrophic hockey-related injuries to the cervical spine had led to speculations regarding the use of the full face shield increasing players' risk of sustaining a neck injury, possibly due to biomechanical alterations. It was proposed that the added mass and area of a helmet and full face shield may increase the rotational acceleration of a player's head following impact, producing greater neuronal shearing forces, increased concussion severity and increased vulnerability for spine trauma. Smith et al. studied the effect of a hockey helmet and face shield on the head and neck during inertial loading using chest and rear impacts on a HYBRID III anthropomorphic test dummy. The results showed that the addition of a helmet and face shield to the head caused an increased angular displacement of the head's center of mass (51). However, it would not appear that the magnitudes involved are sufficient, by themselves, to be the cause of the increased cervical spine trauma observed. As well, angular accelerations were not substantially different between the two conditions. Consequently, the addition of a helmet and face shield does not, in and of itself, predispose the player to the risk of cervical injury.

Later, Benson et al. attempted to determine the risk of sustaining head or neck injury among intercollegiate players wearing full face shields compared with those wearing half shields (6). The prospective cohort study was conducted during the 1997-1998 Canadian Inter-University Athletics Union hockey season. There was no significant difference in risk of sustaining a concussion, neck or other injury for athletes wearing half shields than those players wearing full face shields. Wearing full face shields reduced the risk of facial lacerations and dental injuries by more than two and almost 10 fold, respectively. Based on this epidemiological study, there is no evidence to support speculation the full face shield use increases player's risk of sustaining a neck injury or concussion

In another epidemiological study, Stuart et al. reported no neck injury to players wearing full or partial protection. Thus, full and partial facial protection significantly reduces

eye and facial injuries without increasing neck injuries. In addition, it was found that junior A players with full facial protection were half as likely to sustain head or facial injury compared to players with partial facial protection. Those players wearing no facial protection were injured at a rate more than twice that of players wearing partial protection and almost seven times higher than the injury rate to players wearing full facial protection. Therefore, full face guards were found to provide almost a five fold reduction of eye injury incidences and reduction rate of concussion from 12.2 to 2.9 concussions per 1000 players hours compared with no faceguards (53).

In a later study, Benson et al. attempted to identify specific risk factors associated with increased concussion severity among ice hockey players wearing half shields compared to players wearing full face shields. Results revealed that the overall risk of sustaining a concussion was not significantly different for players wearing half shields compared with those who wore full facial protection at the time of injury (7). The total incidence of concussion for both groups was similar; 41 and 38 for the half shield and full shield groups, respectively. However, players who wore half face shields at the time of injury missed significantly more playing time (practices and games) per injury than players who wore full facial protection. Whether it was the first concussion or recurrent concussion, whether they played defence or offence position and independent of their experience level, half shield group players lost greater amount of time than full shield group players. Players who wore half face shields lost on average 3.29 sessions per concussion, whereas players who wore full face shields lost half the number of sessions, approximately 1.70 sessions per concussion (7). Thus, the use of half face shields compared with full face shields was a specific risk factor for increased concussion severity as measured by time lost from play. This research implies that a full face shield helps to disperse and absorb impact forces more adequately than half face shields.

Pellman et al. has conducted a series of research projects aimed at defining the biomechanics of concussion impacts in professional football. First, an epidemiological study between 1996 and 2001 was done to report the incidence of concussion in the NFL. Second, they have analysed videotapes made from several broadcast cameras during NFL games with significant head impacts and concussions. They analysed game clips from two video cameras positioned near the two ends of the field. The two cameras permitted cinematographic analysis of three-dimensional impact velocity, orientation, and helmet kinematics and permitted to determine the speed at which the players were moving, relative to each other, before colliding. This method, however, limited the number of possible concussions to be analysed. Sufficient lines markings were necessary in both camera views to determine the kinematics. Thus, concussions occurring near or in the end zones could not be reliably analysed and were therefore omitted from analysis. There were 182 concussions recorded on videotape. 61% of the cases were helmet-helmet impacts and 16% were helmet-shoulder impacts, indicating that nearly 80% of concussions are the result of impact with another player. Concussions resulting from impact with the ground represented approximately 16%.

Thirdly, they have reconstructed the event under laboratory conditions to analyse the head biomechanical responses related to concussion. Reconstructions were used with Hybrid III male anthropometrical test devices, similar to those used by Smith et al. Head acceleration was determined from three orthogonal accelerometers measured in the dummy's head. The primary response of the head is the resultant translational acceleration of the head center of gravity, the point selected for motion tracking. There were 31 events reconstructed in the laboratory because of limitations experienced with end zone regions. There were 25 struck players who experienced concussions from helmet impacts and 6 who were struck but not injured. The majority of the impacts for the struck player were either on the face mask, or on the helmet shell close to the side of the facemask. If the concussion resulted from a helmet-

ground impact, then the impact was on the back of the helmet shell. There was higher correlation between concussion and helmet-helmet impacts compared to concussion and than helmet-ground impacts. In contrast, impacts of striking players were all on the top of the helmet shell. Interestingly, in each of the impacts the striking player generally did not suffer from concussion, whereas the struck generally did. The strike was oblique on the facemask or facemask attachment to the helmet, usually below the head center of gravity, or on the side of the helmet, above the head center of gravity. The head acceleration of injured struck players averaged 98 ± 28 g, with 15 ms duration, whereas, uninjured players experienced head accelerations averaging 60 ±24 g, where g represents acceleration due to gravity ($9.8m/s^2$). These impact accelerations are well below the accepted threshold values by the Wayne State University Concussion Tolerance curve. This curve expresses the relationship between acceleration level and the impulse duration with respect to head injury. The curve was first developed from cadaver head drops onto flat steel plate resulting in cranial fractures and impulses of less than 6 ms. Pellman et al. added to the curve with their NFL data showing that concussions result from smaller peak acceleration but with longer lasting impulse of approximately 15 ms.

Why is it only the struck player which will have a concussion? Pellman et al.'s biomechanical analyses show that the struck player experiences a greater change in head velocity and a greater head acceleration resulting in a greater impact velocity. This can be explained by the role of each player at the time of impact. The struck player is on the offensive team and his attention is given to the ball, if he is a receiver, or to his team mate receivers, if he is the quarterback. Striking players spot an offensive player and must block him from bringing the ball to the end zone. Striking players voluntarily collide with the opposing players, therefore, they will adopt a position in such a manner that they remain protected. Conversely, struck players lose sight of the striking player, and therefore cannot

anticipate where and when they will be struck. Consequently, the momentum is transferred to the struck player during the collision resulting in increased head velocity and acceleration, and a reduction in head velocity and loss of momentum for the striking player. Therefore, the same collision is more severe for struck players than for striking players.

Though facemask impacts have the highest average impact velocity, they have the lowest change in velocity, thus, lowest head acceleration. They are direct impacts which occur spontaneously where reflex latencies are too long to initiate protective behaviour. Moreover, if unaware of the impending impact, players' reflex latencies do not permit appropriately alignment of their body during the fall, creating torque, rotational or twisting forces. This further complicates the computation of the sum of forces brought to bear on the brain. Ground impacts to the back of the helmet involve the lowest impact velocity but the highest change in velocity, thereby, highest head acceleration. In ground impacts, there is a greater time lapse by which the player uses reflexes while falling backward, so the impact is higher on the helmet, because of the angle of fall and shoulder pads prevented a lower level of impact.

Even under conditions in which there is no overt impact, trauma to the brain is possible. Trauma can result from a rapid change in the head's velocity, either acceleration or deceleration (4). A significant force, in the absence of direct impact to the head, can have a detrimental effect on brain tissue similar to any physical impact. A smaller stopping distance will result in a larger resulting force on the brain. A coup injury is when the brain strikes against the inner skull in the direction it was initially traveling. In a contre-coup injury, the brain rebounds from the direction of the deceleration and strike the inner lining of the skull in the opposite direction. Rotational forces increase the areas in which the brain strikes the inner skull. Therefore, even when there is no direct impact to the head, the brain can undergo severe damage depending on the forces that are transferred to the head.

VI. Conclusion

Studies by Benson, Laprade, Smith and Stuart research groups have demonstrated epidemiological evidence of the protective effect of facial protectors from concussion in ice hockey. Nonetheless, there is no research which has been conducted to identify the biomechanical properties of protection associated with the use of the different types of facial protectors. The following research project uses novel methods to identify impact attenuation responses of facial protectors when combined with ice hockey helmets.

Evaluation of Impact Attenuation of Facial Protectors in Ice Hockey Helmets

Introduction

Ice hockey involves hard contacts, collisions and fast-moving pucks. Hence, the risk of injury is an integral part of the game. In leagues permitting body checking, previous epidemiological studies have shown that head and neck injuries have consistently ranked as one of the most commonly injured body sites, followed by the knee, shoulder, hip/thigh/groin, and hands/fingers (Azuelos et al., 2004). More specific studies on head injuries in hockey reported that concussions (or mild traumatic brain injuries, mTBI) account between 2 and 14% of all hockey-related injuries and 15 and 30% of all hockey-related head injuries (Goodman, 2001). More recently, Flik and colleagues (2005) reported that head injuries in ice hockey represent up to 20% of all trauma. Hence, concussions form a substantial and persistent problem in ice hockey that has acute and long term consequences to the health of the players.

To address this concern, the wearing of helmets (covering the cranial skull of a player's head) is obligatory in most competitive, contact ice hockey leagues. Helmets are designed to diminish the magnitude of impact forces during collisions by both distributing the contact load over a wider area of the cranium and by means of an energy absorbing liner system. Manufactured helmets must pass safety standard guidelines such as ASTM International F1045-04, NOCSAE DOC 001-04m05 standards for certification or both. Typically, these standardized tests involve controlled impact simulations of surrogate head forms with helmets to assess impact attenuation gains (as quantified by reduced peak head form acceleration). In most contact sports, the intervention of helmets has reduced the severity of head injuries; however, the incidence of mTBI remains prevalent (Pellman et al., 2003).

Recently, the potential to reduce mTBI incidence by virtue of wearing facial protectors in conjunction with helmets has been proposed (Biasca et al., 2002). Some prospective cohort studies have been conducted to investigate this possibility. For instance, Benson et al. (1999) monitored the number head or neck injury sustained among intercollegiate players wearing full face shields compared with those wearing half face shields. They found no evidence to support speculation the full face shield use increases player's risk of sustaining a neck injury or concussion. In a later study, Benson et al. (2002) found similar results; however, independent of prior injury history, played position and experience, the half shield group players lost greater amount of time than full shield group players (average of 3.29 sessions per concussion versus 1.70, respectively). In a similar prospective study by Stuart and colleagues (2002), athletes were monitored from within competitive Junior A league where none, partial and full facial protection was allowed. In contrast to Benson's results, they observed that players wearing no facial protection were injured at a rate more than twice that of players wearing partial protection and almost seven times higher than the injury rate to players wearing full facial protection. Therefore, full face guards were found to provide almost a five fold reduction of eye injury incidences and reduction rate of concussion from 12.2 to 2.9 concussions per 1000 players hours compared with no facial protectors. Hence, these studies suggest that the use of facial protectors has a positive relation with decreasing, at least, the severity and, at most, the rate of head injuries.

The dynamics of impact at an incident vector towards the face in terms of net head acceleration is not well understood with respect to concussion events, let alone the intervening effects of facial protectors in combination with helmets. Thus, the purpose of this project is to determine: (1) if facial protectors can attenuate head acceleration within acceptable limits (i.e. below 300 g's) during blunt facial collisions, (2) if acceleration attenuation differs between cages and visor facial protectors conjoined with ice hockey helmets, and (3) if helmet liner

material (vinyl nitryl, VN, or expanded polypropylene, EPP) significantly modify the above responses.

Methods

Within this section, the testing materials (samples), instrumentation, protocol, as well as planned statistical data analysis will be presented.

Materials

Six models of commercial available ice hockey facial protectors were evaluated: three full-face shield (cages) and three half-face shields (visors). The cage models included the Bauer Nike FM8500, Itech FM 480 and the CCM RBE VIII. The visor models included the Bauer Nike FM1000, Itech HLC and the Oakley Aviator. Four samples of each model were tested for each condition as described below (see Protocol). During testing, all facial protectors were mounted on a common reference ice hockey helmet model (NBH8500). In addition to the above, two separate liners covering the inside of the ice hockey helmet were evaluated: vinyl nitryl (VN) or expanded polypropylene (EPP).

Instrumentation

A *Drop Rig Monorail Tester Guide Assembly* (ASTM F1045-04) was used to control the free drop height and direction of the head form mounted with the helmet and facial protector. The full facial headform used (NOCSAE DOC 001-04m05) was attached to the armature of the drop rig monorail guide. The headform's orientation was adjustable, allowing impacts to be delivered to any point on the helmet or the facial protector. Given that the ASTM F1045-04 standards reference head form lacks facial geometry (i.e. consists of only the cranium analog), the full facial NOCSAE head form was used. Headforms with approximate facial geometry were essential to assess the visors and cages, since the latter designed to function around the former. In addition, the NOCSAE headform has a high

degree of biofidelity, incorporating a visco-elastic rate dependant response to impact accelerations. The medium size headform (head circumference of 578 mm) was used during impact testing. The combination of the two standard's instrumentation provided the optimal configuration wherein consistent site and magnitude specific impacts could be produced repeatedly to a full faced head form. The combined mass of the assembly was 6.98 kg. Within the headform, a tri-axial accelerometer was mounted at its center of mass (CoM) to measure impact acceleration or deceleration. Acceleration measures were amplified and digitally collected (V4.6, Cadex Inc.).
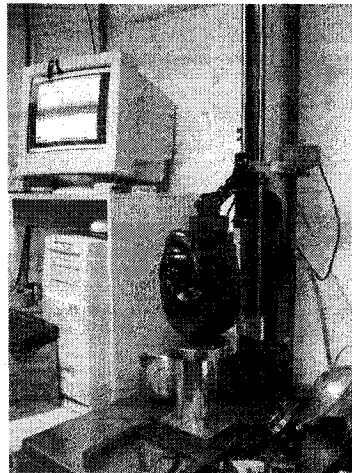

Figure 1 – Monorail Drop Rig tester; Head form, and Steel anvil

The impact surface for the dropped head form / helmet / cage or visor assembly was to a flat, circular steel surface anvil. This is a non-compliant landing surface. Unlike the ASTM standard, an intervening silicone pad (modular elastomer programmer, MEP), was not used. Pilot testing indicted that the inherent compliance of facial protectors permitted direct impact on to the steel anvil.

Test Protocol

As noted above, the testing protocol adopted was a hybrid of the ASTM International F1045-04 and NOCSAE DOC 001-04m05 standards. Facial protectors and helmets were positioned on the head form (attached to the armature and guide rail) then dropped 77 ± 1 cm in order to achieve a pre-impact kinetic energy of 45 ± 3 J. At the impact event, peak acceleration and the integral of acceleration-time (i.e. Gadd Severity Index, GSI) were recorded.

Testing was performed at ambient temperatures ($20 \pm 2°C$). Helmet positioning and tightness were checked before each impact test. The helmet's ear aperture were aligned concentric with the head form's index ear holes and the helmet's front rim was positioned properly along a pre-measured line on the head form forehead (5 cm above the basic plane). By re-orienting the head form on the drop rig's armature, each facial protector was impacted at four different sites including:

1. Crown (C) of helmet (intersection of the midsagittal and the coronal planes). The crown was used as a reference site to compare direct helmet to direct facial protector impacts.

2. Front (F) of facial protector (located at the intersection of the basic plane and the coronal plane).

3. 45° to Front Boss (FB45) of facial protector (located on the basic plane at the 45° angle from the coronal plane). Impact was done on the left cheek.

4. Jclip (JCL) of helmet (located on the basic plane at the approximate intersection of the basic plane and the midsagittal plane). The J clip is a structure to secure the facial protector to the side of the helmet. Impacts were done on the left ear side of the head form.
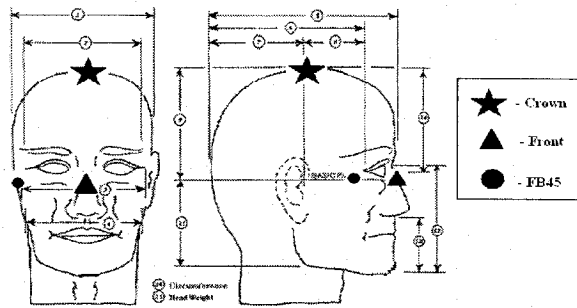
Figure 2 – Impact Location Sites. (note: JC1 was defined on the ice hockey helmet.)

Before impact, the head form was oriented to impact the designated site. Then, the helmet and the facial protector were mounted on the head form. Therefore, impacts were conducted with respect to the reference lines on the head form not with respect to helmet references. This permitted repetition of the design across all the facial protector models. Prior to each drop test, the helmet was adjusted on the head form. Each site was impacted three times with, approximately, 1 minute intervals. Both new helmets and facial protectors were used for each site specific test, so as to avoid potential prior impact damage from one site affecting the results of other sites later.

In addition, for each test the face of the headform was covered with white paste to identify potential contact between the face and anvil during impact. After impact, the extent of paste transferred either to the facial protector and / or the steel anvil were documented. The white paste permitted a qualitative description of the impact events. To further understand the nature of the collisions, front impacts were filmed using high speed video camera (Fastec Imaging, TroubleShooter) at 1000 frames/sec. The camera was positioned lateral and perpendicular to the monorail's drop plane. Subsequent analysis permitted the linear kinematics of headform and facial protector to be calculated. Specifically, the amount of deformation (linear compression) of the headform's facial structure (nose) was measured.

Statistical Analysis

From the dependent variables of peak acceleration (PA) the affect of independent variables (model, site, repeated impacts) were assessed with a standard reference helmet lined with VN foam, such as:

$$PA = Model_6 \quad x \quad Site_3 \quad x \quad Repeat\ impact_3$$

For two models, the effect of helmet liners (EPP, VN) was assessed separately, such as:

$$PA = Model_2 \quad x \quad Site_3 \quad x \quad Repeat\ impact_3\ x \quad Foam_2$$

Statistical analyses were preformed using Statistica (V.6, GLM & Post Hoc tests). Significant differences were evaluated at α level of 0.05. Given that the intent is not to endorse nor denigrate particular products, in the following results specific name brands will not be cited, instead, they will be referred to as cage A, B, C and visor A, B, C (in no particular order)

Results

Peak impact acceleration (PA) was lowest for VN lined ice hockey helmets and greatest for EPP lined ice hockey helmets as shown in Figure 3, ($p< 0.001$). PA for VN liners ranged from 75g to 110g whereas PA for EPP liners ranged from 90g to 130g. The wide range of PA was dependent on the repeated impact sequence ($p=0.028$). The general trend observed was for the PA to increase progressively from the first impact number to the second impact and to the third impact number. Significant differences between the first and the third impact number were calculated ($p=0.036$, Tukey HSD).
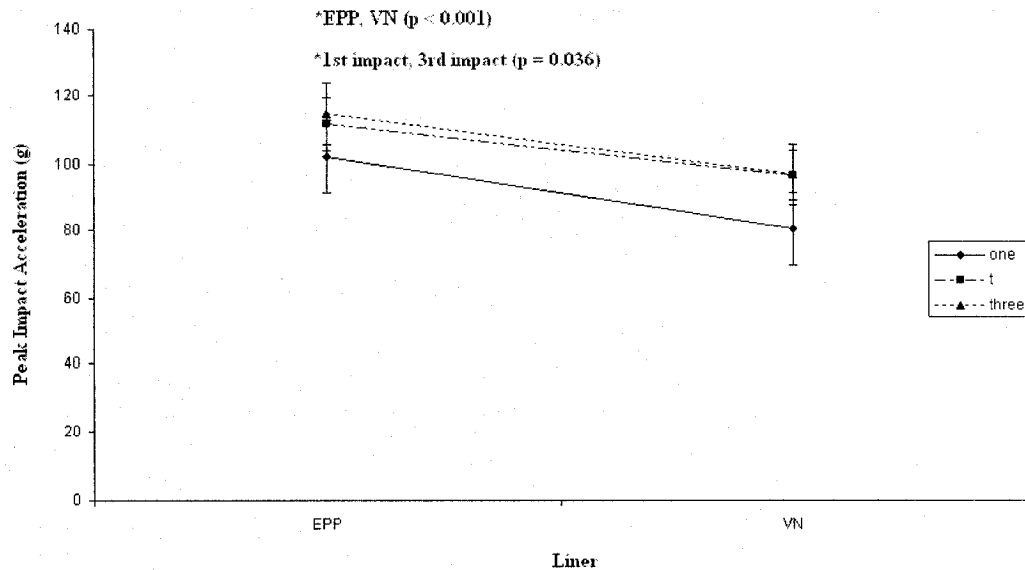
Figure 3 – Peak Acceleration (g) for Crown Impacts at third impact.

Figure 4 shows PA for cage and visor facial protectors as a function of the helmet liner and impact site. The graph shows results for the third impact. A significant main effect for facial protector was found (p=0.004). PA were substantially lower for cages versus visors. As well, a significant main effect for impact site was found (p=0.007). Significant differences between the Front site and both the FB45 and JCL sites (p=0.029 and 0.0056, respectively, Tukey HSD), were observed. There were no significant differences between the FB45 and JCL sites. For facial protectors, there were no significant main effect differences observed between EPP and VN liners (p=0.391). No significant interactions between factors were observed.
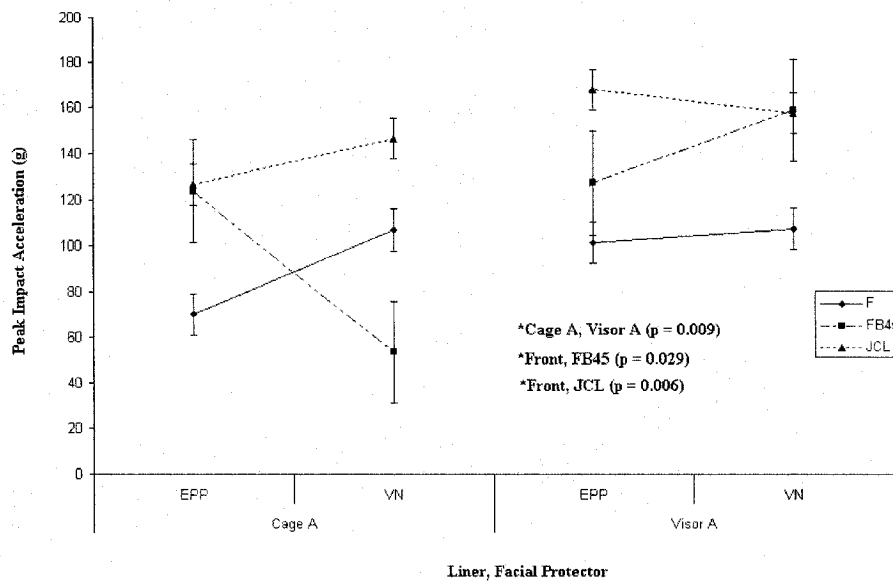
40

Figure 4 – Peak Acceleration as a function of Helmet Liner and Impact site for third Impact.

PA observed for each facial protector model as a function of repeated impact at front impact site are shown in figure 5. PA observed for each facial protector model as a function of impact site for the third impact are shown in figure 6. A significant main effect for facial protector model, impact site and impact number are found ($p < 0.0001$). In general, PA was lowest for cages as opposed to visors. There were no significant differences between the three cage models, however, Visor C was showed lower PA than Visor A and Visor B ($p=0.000715$ & 0.000813, respectively). In general, for impact site, Front impacts had lowest PA and JCL impacts had the greatest PA with values for the FB45 site being intermediate. The Front impact differed significantly from the FB45 and JCL site ($p=0.01602$ & 0.000022, respectively). As well, FB45 and JCL were significantly different. ($p=0.000022$). For repeated impact, PA was lowest for the first impact number and greatest for the third impact number with values for the second impact being intermediate. The first impact was significantly different from both the second and third impact number ($p=0.000044$ & 0.000022, respectively). The second and third impact were also significantly different from each other

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(p=0.02099). Specific differences between brand models and sites were observed; for instance, Cage A and Cage C were significantly different (p=0.0128) at the third impact. As well, Visor C was significantly different from Visor A and Visor B (p=0.000058 & p=0.019, respectively) at impact site FB45. Cage A and Cage C were significantly different (p=0.013) at the Front impact site.
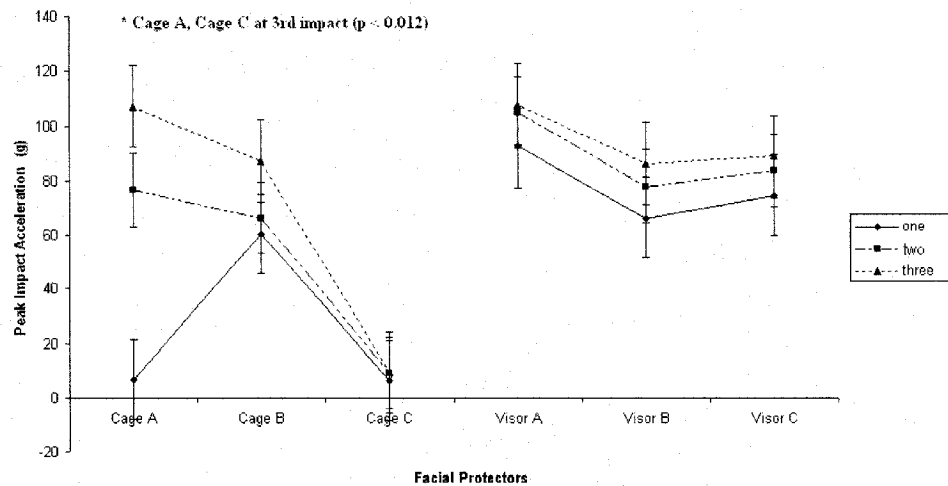

Figure 5 – Peak Acceleration (g) as a function of Impact number and Front impact site.

Figure 7 shows the distance of nose compression for the 6 facial protector models for the third impact at the front site. In general, nose compression was greater in visors than cages (p<0.001) but was dependent on the model. The main effect of impact repetition was dependent on the facial protector type. For cages, the impact repetition significantly affected the extent of nose compression (p<0.001), where as for visors, there was no significant differences in nose compression for each impact repetition (p=0.307). There were significant differences across each model. Cage B was significantly less than Cage A and Cage C (p=0.041 and p<0.001, respectively) and Visor C was significantly lower than Visor A (p=0.027).
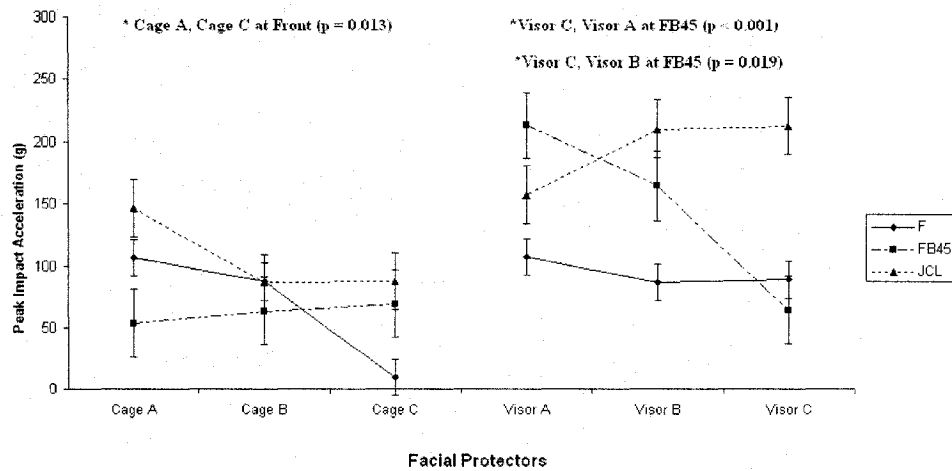
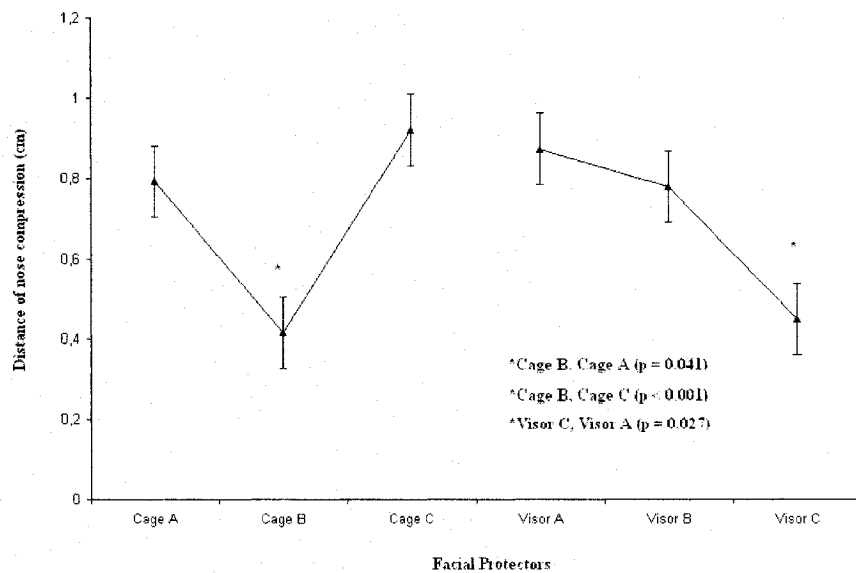Figure 6 – Peak Acceleration (g) as a function of Impact site for third impact.

Figure 7 – Distance of nose compression for Facial Protectors during 3rd impacts at Front site.

Discussion

This study demonstrated that facial protectors can serve to attenuate impact accelerations within acceptable tolerance criteria (i.e. 300g) (2) as applied to helmet shells and liners. Hence, it is appropriate to conclude that the use of facial protectors in combination

with helmets can reduce the risk of mTBI. Table 1 shows the comparison of peak impact acceleration of the NOCSAE head form under three conditions for front impacts. Impacts of the headform alone were as high as 400g and above the threshold value causing concussion.

Table 1 – Comparison of Peak Impact Acceleration for Front Impacts.

	Peak Acceleration (g)
Head Form Alone	380 - 420 g
Helmet with no FaceMask	100 - 130 g
Helmet with Face Mask	10 - 100 g

Worth noting was the observation that the use of an ice hockey helmet decreased PA by up to 4-fold during Front impacts. Though initially unexpected, the helmet shell covering the forehead was observed to hit the outer portions of the steel anvil concurrently with facial contact; thereby reducing the PA for impacts directed along the basic plane. The use of a facial protector combined with the ice hockey helmet showed greater reductions in PA. Indeed, facial protectors exhibited comparable PA attenuation as ice hockey helmet shells and liners. Consequently, facial protectors fulfill their primary function in providing protection to the face, while simultaneously reducing the PA of direct impact of forces from facial vector incidence. This is important to consider in future designs of facial protectors; special concern must be given to the mechanical function that they can accomplish. For example, should facial protectors prevent facial contact?

The type of helmet liners has been shown in previous studies to alter shell impact response: not so with facial protectors. Figure 3 shows that at Crown impacts, on the helmet shell, there were significant differences in the PA between EPP and VN lined helmets.

However, for impact on the facial protector (Front, FB45 or JCL), no significant difference was observed. It was initially thought that the different rigidity of the two foams tested may change the stability of the facial protectors anchoring to the shell; in turn altering the latter's impact response. It appears that for facial impacts, the function of the liner is replaced by the flexibility of the facial protector and the distance present between it and the surface of the head form. Although, there is impact on the forehead of the helmet, the liner does not alter the response of visors or cages. Thus, given that there is no liner on the facial protector compressing to accomplish load absorption, the facial protector must be designed in consequence of the head protection purpose.

The results collected in this study validated our second hypothesis. Overall, cages perform better than visors. There are several factors which can explain such result. Primarily, cages provides firm chin support, whereas, a visor's lower margin are not supported and easily collapse by buckling inward and downward. The chin support provides a greater distribution of the forces of impact to the portions of the facial protector above and below the basic plane; whereas in the visor, the forces are distributed only above the basic plane. The greater distribution of the forces can allow a greater absorption of energy by the facial protector. This entails that less energy is being transmitted to the CoM of the head, and thereby, reduces the PA. Secondly, cages and visors exhibited two different behaviours upon impact. The cage was permanently deformed at the area of impact including the bulging of the wires and flattening of the cage. Visors offered insufficient resistance to the forces of impact, collapsing but then elastically rebounding to their original geometric configuration. Thus, the permanent deformations seen in cages will have absorbed part of the force of impact, thereby reducing the PA transmitted to the CoM of the headform. The foam liner of the chin cup would also contribute to load dampening. Lastly, the design of the cage and the visor differ completely. The cage forms a hemisphere covering the entire face, whereas the visor forms a

hemi-cylinder covering only the upper portion of the face. Consequently, at impact, the forces are distributed differently for both the cage and the visor. The cage shape permits the radial distribution of the forces. The visor shape only allows forces to be distributed medially-laterally parallel to the basic plane. .

Main Factors

A major factor in this research project was the effect of impact repetition. The general trend observed was that the first impact had lower PA than the second impact, which in turn, had lower PA than the third impact ($RI_1 < RI_2 < RI_3$). Thus, with impact repetition, we observe a progressive increase in the PA. We describe the increase in PA to correspond to a decrease in shock absorption capacity of the facial protector. Nonetheless, all PA at each impact repetition are below the officially recognized criterion value for mTBI (300g). Thus, the results obtained emphasize the enhanced head protection provided by facial protectors.

The repeated impact was present for all cases independent of facial protector model, impact site, or liner type. This is probably a result of the damage to the product caused by the impact, and with repeated impact, the damage increases. Thus, with increased impact number, there is a progressive increase in the PA at impact. This corresponds with qualitative damage observed after testing, as described further below.

Analysis of the results obtained by impact site do not corroborate with our initial hypothesis. We expected that the front impact site would have lower PA than FB45 which in turn would have lower PA than JCL (F < FB45 < JCL). This hypothesis was based on the shape of the facial protector with regards to the face. At the front site, there is greater distance between the facial protector and the face than at the FB45 site. At the JCL site, the facial protector slides along the helmet, thus, the distance is nil. Given that the distance progressively decreases as the impact site shifts laterally, we expected a progressive increase in PA across the circular impact shift. This did not turn out to be the general principal across

each model. Each facial protector model had different trends in regards of the PA for impact site. However, significant differences among model were very rare. Post-hoc analysis showed significant differences between Cage A and Cage C at the front site; between Visor A and Visor C, and Visor B and Visor C at the FB45 site.

Qualitative Observations

During front impacts, the cages were progressively further deformed as a function of repeated impacts. Typically, immediately after the first impact, the cage did not return to its original spherical shape. Damage was restricted to the area of impact; that is, the wires were distorted and bended and the area of impact was flattened. The impact deformation was different for all three cages although the impact site was the same. The damage to cage A's wire grid was located typically at the second and third rows, and the fourth, fifth and sixth columns. Damage to cage B was at the third and fourth rows, and the fourth, fifth and sixth columns. Damage to cage C was at the second and third rows, and fourth, fifth and sixth columns. It appears that the different geometric structures of each cage provided different impact attenuation effects.

Conversely, with the visors, we observed no permanent deformation comparable to the flattening seen in cages; instead visors flexed with impact and returned to their original form after impact (as noted from visual digital recording at front impacts). Some fractures or breakage were observed; for instance, in some sample of visor A, we observed the fracture of the plastic support in the upper portions of the visor area designed for aeration.

The use of superficial paste applied over the headform's face permitted the post-hoc identification of whether contact occurred between face and the facial protector or anvil during impact, as well as showing the specific facial contact points. For cages, we observed that the mark of the paste from the nose of the headform was increasing in surface area when

it came in contact with the steel anvil through the cage. For visors, we observed a similar increase in surface area of the paste mark on the inside of the visor with repeated impacts. Moreover, we observed a slippage of the nose-visor contact point at impact. Impact is intended on the nose but there is sliding of the face mask during impact against the steel anvil; it is this slippage that is observed on the inside of the visor (this was confirmed qualitatively from inspection of digital video records). In addition, for visors, there is paste trace from the mouth of the head form on the steel anvil for all repeated trials. The paste clearly demonstrates a difference in impact attenuation between cages and visors. The chin support from the cage prevents any slippage motion of the helmet and facial protector. As well, the chin support prevents any contact of the mouth on the steel anvil. Consequently, the cage facial protector provides better facial protection at the nose and at the mouth.

For FB45 impacts, cages showed similar deformation patterns as observed in front impacts. There was bending of the wires and flattening of the cage as a whole. In contrast to front impacts, the damage was not restricted to the area of impact. The flattening on the right side (impact side) of the cage caused a bulging or outward buckling of the wires of the cage on the left side (opposite impact site). Cage A showed damage along the second, third and fourth row, and the second and third column. Cage B showed majority of the damage at the second and third row, and the third and fourth column. We observed most flattening at these quadrants, but deformation in surrounding areas of these quadrants was also apparent. Paste mark observations demonstrate that the cheek did not contact the side of the cage during impact. Presumably, the chin support prevents the cheek from headform slippage.

For visors, fissures within the plastic were observed on the sides of the visor.. For all visors, it is the region of the second ventilation hole which is mostly affected. Thus, unlike the cages, the visors returned to their original shape and are damaged at other areas than the area of impact. Paste mark observations indicated that the contact of the cheek with visor

progressively increased with impact repetition. On the inside of the visor, the surface area of the paste mark gradually increased. Moreover, at third impact, there was paste from the side jaw on the steel anvil. Thus, unlike the cages, the visor did not prevent the chin from contacting the steel anvil.

JCL impacts did not cause as much deformation to the cages and visors. Impact was partially absorbed by the side of the helmet shell, therefore, leaving less pressure on the facial protector. For both cages and visors, the impact was at the area of the brand sticker/logo. The stickers were scratched on the inner and outer portions of the facial protector. On the exterior, it was slippage on the steel anvil which caused scratches to the stickers. On the interior, the screws of the J clip scratched the logo of the facial protector. Cages have stickers only on the exterior portions; however, visor had the scratches observed both on a lateral displacement and vertical displacement forming a crucifix. Damage from impact is only observed in cages. There was slight deformation of the wires of the second row, and the first and second column.

Experimental setup allowed high speed video capture for all front impacts. The video from high speed filming contradicted any certainty obtained from Faceguard projectile tests. To obtain certification, facial protectors are submitted to faceguard projectile tests; for example, NOCSAE DOC (ND) 021-98m05a. The facial protector must prevent any contact with the nose in order to pass the test and obtain certification. In these tests, pucks are thrown at the facial protector at 28 m/sec. Considering the weight of the puck, this represents impacts of 65 J which is greater than the drop impacts of this study. High speed filming showed that the nose contacts the steel anvil during impact. This is very surprising given that the facial protector can prevent the facial protector-nose contact for protocols requiring higher energy impacts. Indeed, the projectile testing and drop testing are different. The projectile testing consists of a high velocity, low impact on a small surface area. The impact testing consists of

low velocity, high mass impact on a large surface area. Figure 7 demonstrates the distance of nose compression as a function of each facial protector model. Like for PA, the nose of the head form was less compressed with cages as compared with visors. This is probably due to the better support of the cage on the entire face than that of visors. For cages, at first impact, the nose does not contact the steel anvil, but with impact repetition, damage accumulates on the cage, and the nose progressively becomes compressed at the second and third impact. As shown in figure 7, at third impact, nose compression can be as high as 80 mm. For visors, nose compression is not dependent on the impact repetition. The nose is as compressed at the first impact as for the third impact. On average, the nose gets compressed by more than 1 cm. The nose compression observed corresponds with the PA observed. Thus, the forces of impact causing concussion can be depicted on facial observations. The mechanical explanations given above for PA can also explain the incidence of nose compression.

Conclusion

This study emphasizes the importance of facial protection in ice hockey and its implementation is necessary for the safety of athletes. Facial protectors reduce PA transmitted to the brain which could cause mTBI. Results have shown that cages are better constructed for the facial impact tests performed in this study. This mechanism for decreasing PA may in turn decrease the severity and / or incidence of mTBI as observed in the prospective studies of Benson et al. (2002) and Stuart et al. (2002) where effects of facial protectors has been correlated to mTBI severity.

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Appendix 1

Photographs of Facial Protector Models

Figure A1 – FM8500 – Nike Bauer, front view & side view

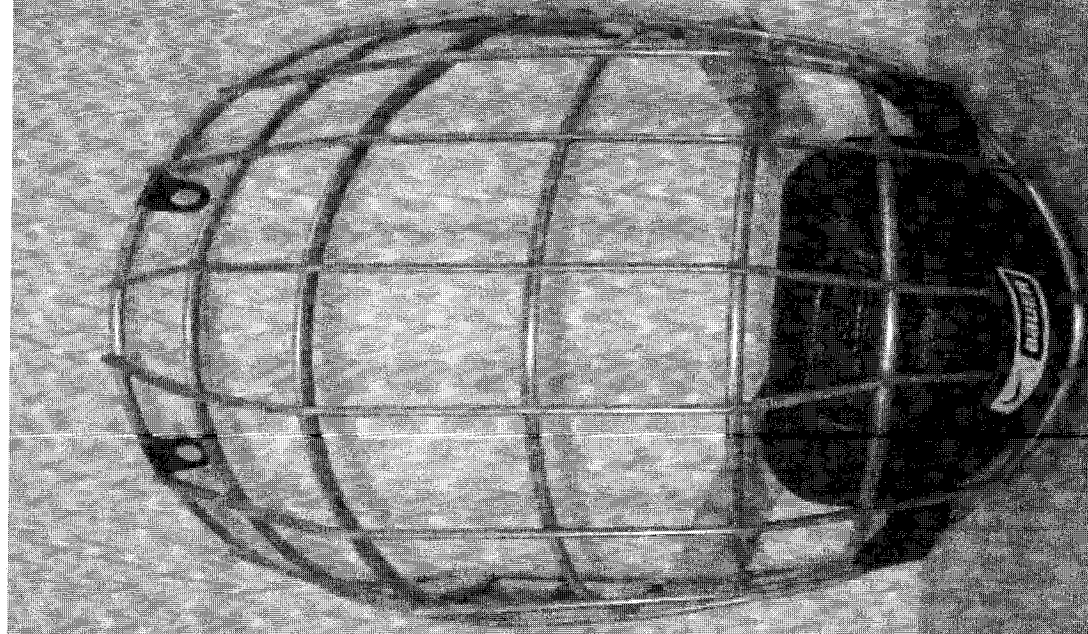Figure A2 – Itech FM480 – Itech, front view & side view

Figure A3 – CCM RBE III – CCM, front view & side view

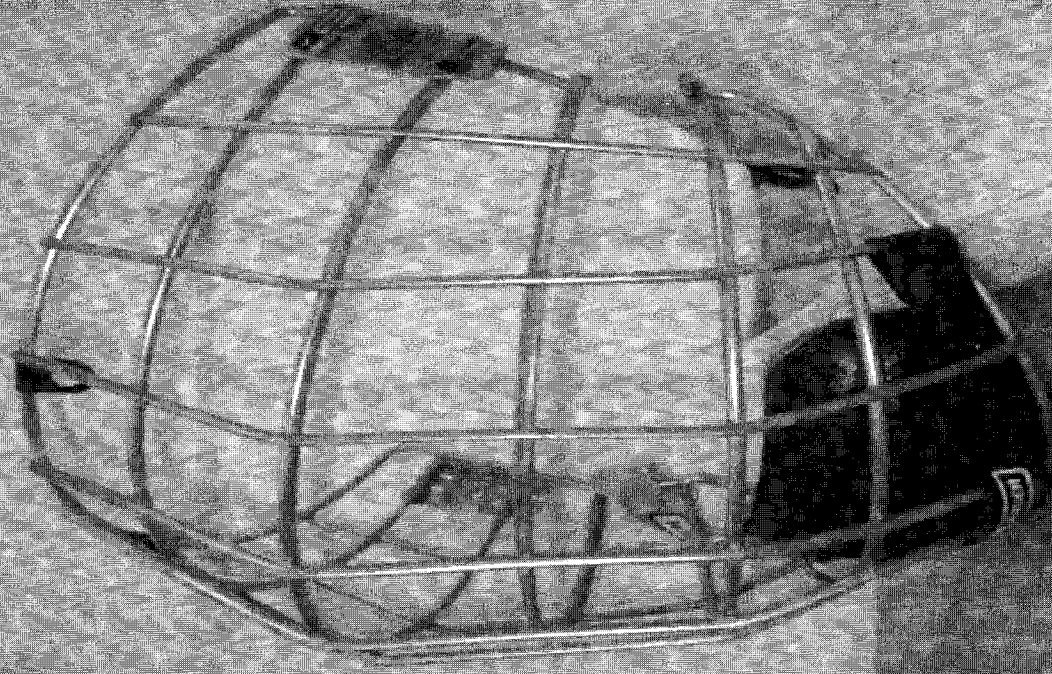Figure A4 – FM1000 – Nike Bauer, front view & side view

Figure A5 – Itech HCL – Itech, front view & side view

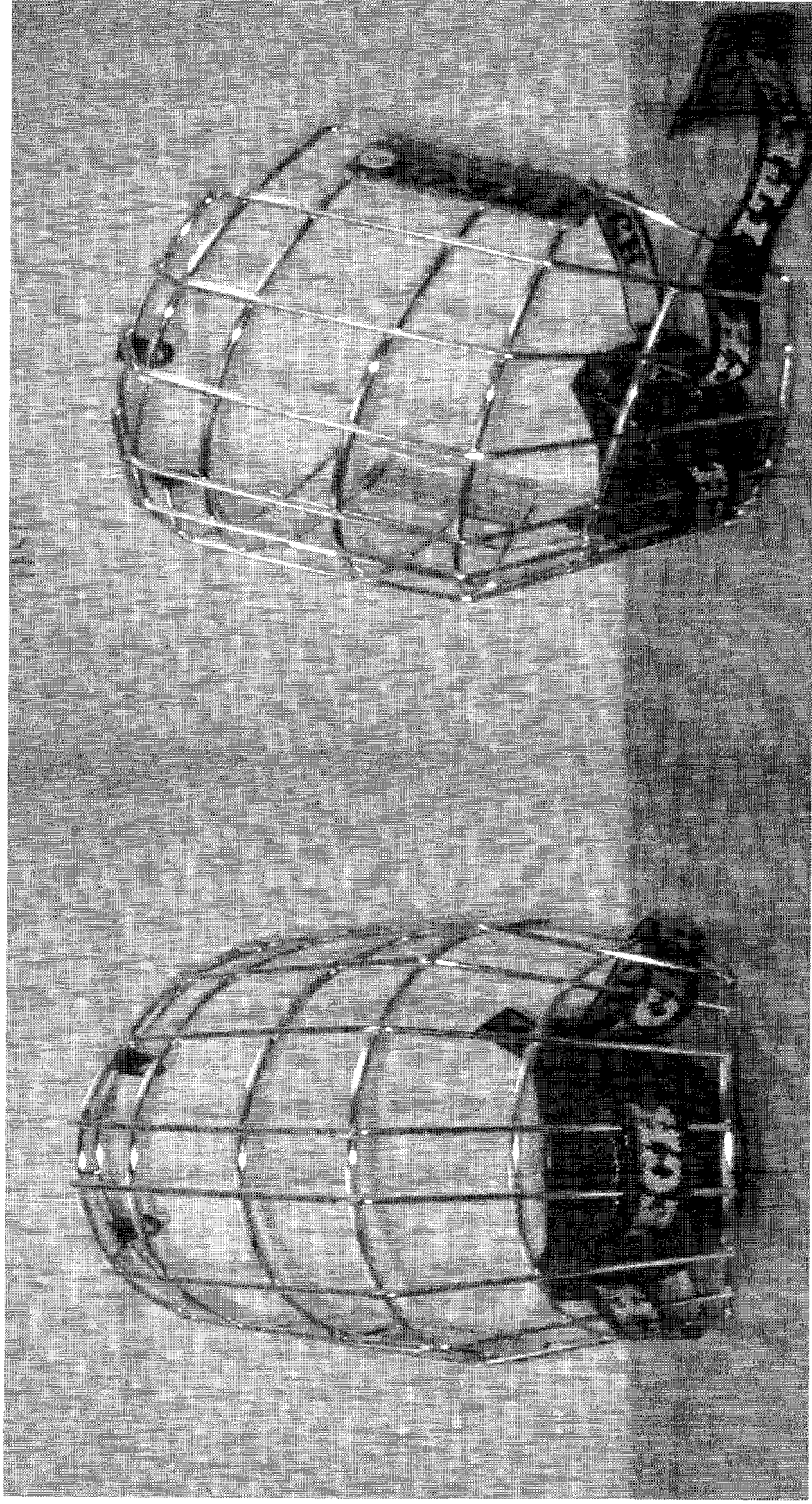Figure A6 – Oakley Aviator – Oakley, front view & side view
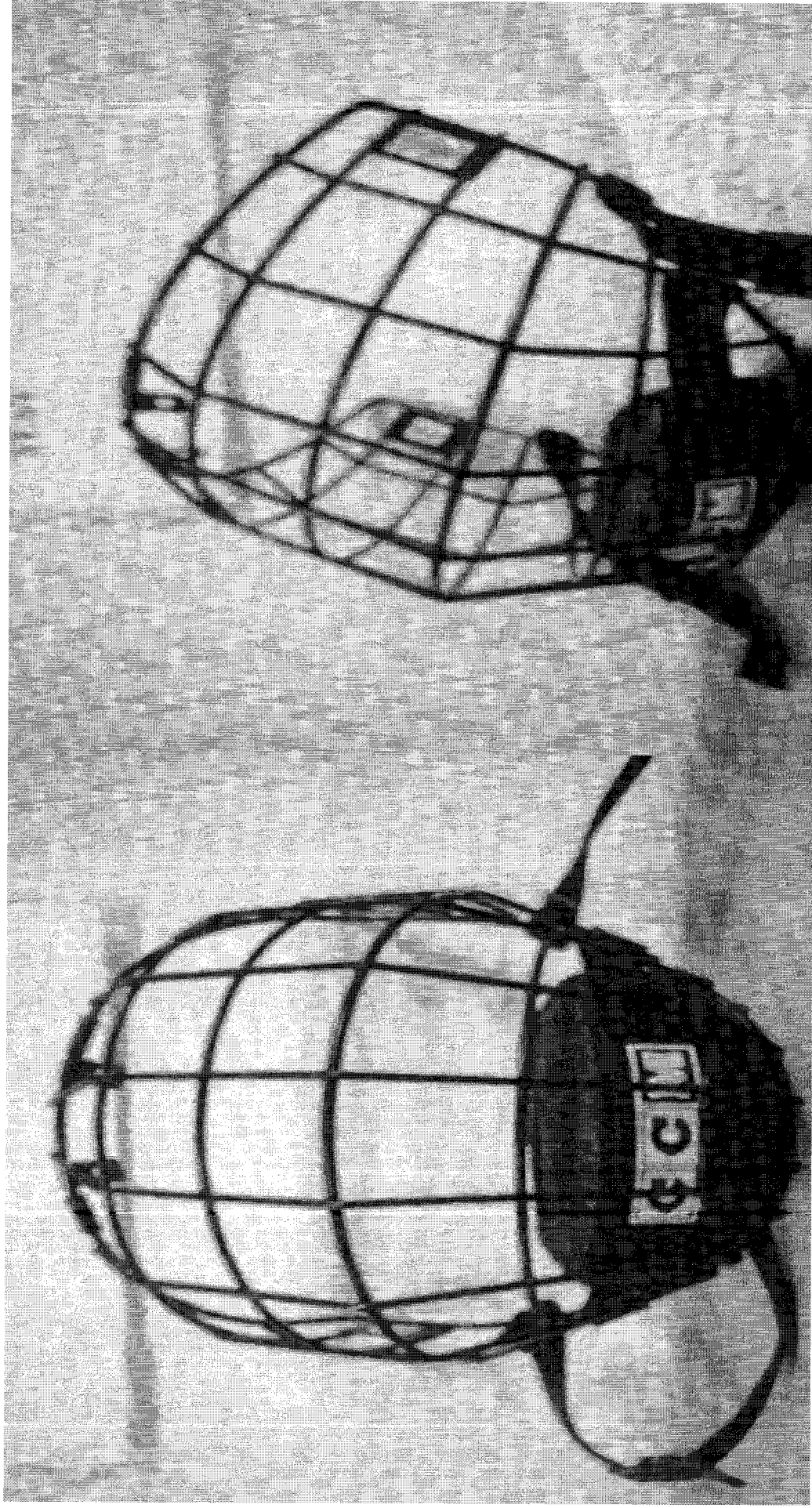
FM8500 – Side View

FM8500 – Front View

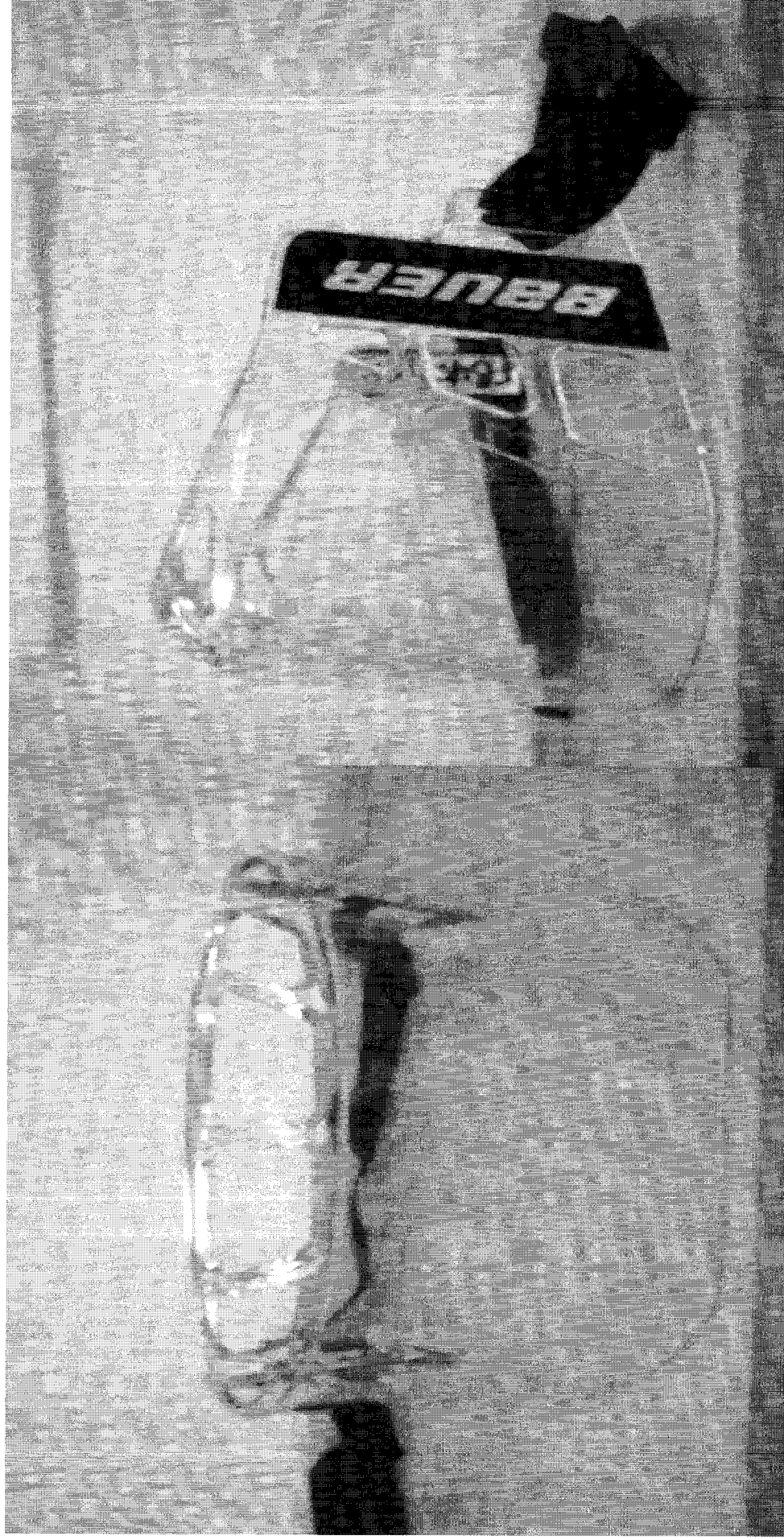Itech FM480 – Side View

Itech FM480 – Front View

CCM RBE VIII – Front View

CCM RBE VIII – Side View

FM1000 – Front View

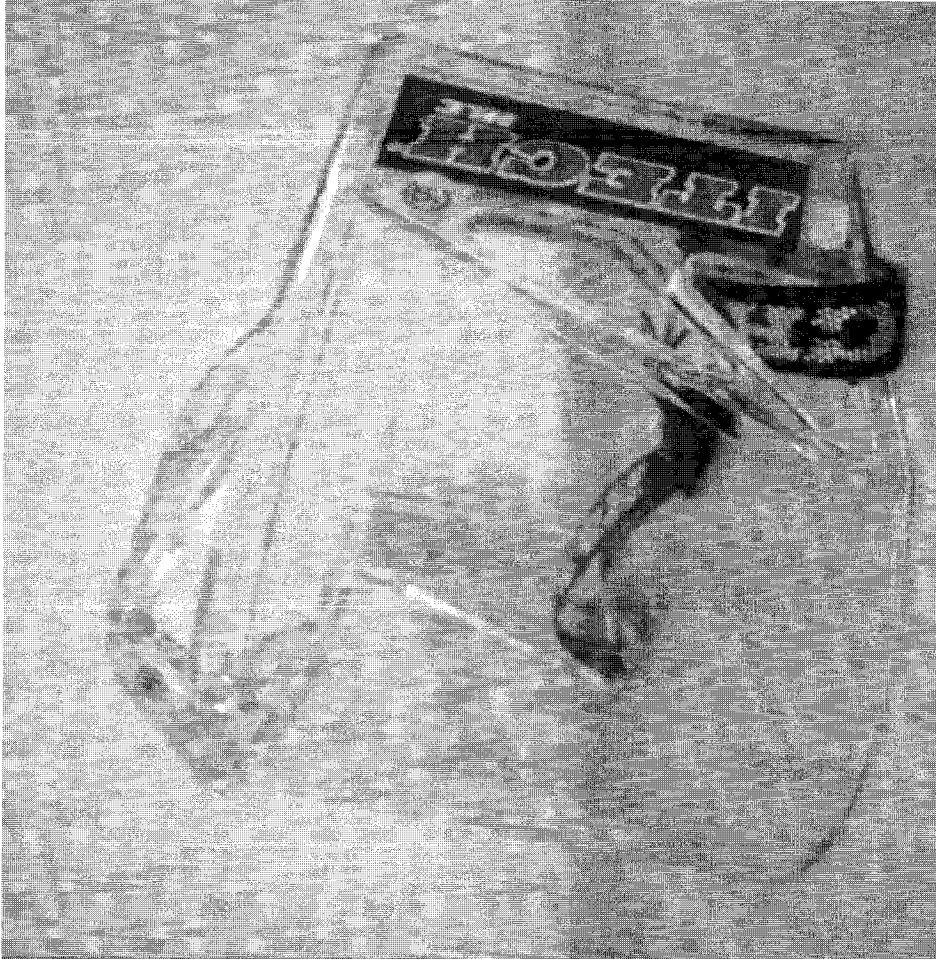FM1000 – Side View

Itech HCL – Side View

Itech HCL – Front View

Oakley Aviator – Side View

Oakley Aviator – Front View