An Analysis of Catcher's Mask Performance to Attenuate Head Acceleration

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(ABSTRACT)

The goals of this study were to measure the ability of catcher's masks to attenuate head accelerations upon impact with a baseball, and to compare these head accelerations to established injury thresholds for concussions. Testing involved using a pneumatic cannon to shoot baseballs at an instrumented (3-2-2-2 accelerometer array) Hybrid III headform (a 50th percentile male head and neck) with and without a catcher's mask on the head. The ball speed was controlled from approximately 26.8 – 35.8 m/s (60 – 80 mph) and regulation NCAA baseballs were used.

Peak linear resultant acceleration was 140 – 180 g without a mask and 16 – 30 g with a mask over the range of balls speeds investigated. Peak angular resultant acceleration was 19500 – 25700 rad/sec^2 without a mask and 2250 – 3230 rad/sec^2 with a mask. The Head Injury Criterion was 93 – 181 without a mask and 3 – 13 with a mask and the Severity index was 110 – 210 without a mask and 3 – 15 with a mask. Catcher's masks reduced head acceleration metrics by approximately 85% when baseballs were impacted with just the headform. Head accelerations with a catcher's mask were substantially lower than contemporary injury thresholds, yet evidence indicates that baseball impacts to the mask still result in concussions.

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Chapter 1

Introduction and Background

This chapter contains an overview of concussions, including epidemiology, etiology, and pathophysiology. Basic anatomy and physiology of the brain and neurons are given along with an extensive literature review of relevant research.

The second chapter is an article that was submitted to the Clinical Journal of Sports Medicine and discusses the attenuation of head accelerations when impacted by a baseball.

1.1 The Matheny story

The spring of 2006 marked the end of a career for gold glove winning catcher, Mike Matheny. Matheny began his major league career with the Milwaukee Brewers in 1994. After four years, he was traded to Toronto where he spent one season. After another trade and four years with St. Louis he ended up with the San Francisco Giants in 2004 with a three year contract. Unfortunately, Matheny was not able to finish out this contract with the Giants. Halfway through his second season in San Francisco, Matheny was bombarded with six foul tips to his catcher's mask within a one week. One of these foul tips came off a pitch that was reported to be 100 mph. This impact left the catcher stunned and dazed. The team trainer immediately pulled him from the game for medical evaluation. The day following the 100 mph impact, he was placed on the 15-day disabled list so he could be examined by specialist at the University of Pittsburgh Medical Center. During the examination Matheny had an increased heart rate, fatigue, and cognitive

problems leading to the diagnoses of a concussion. At the time Matheny did not know the seriousness of the concussion or the implication this brain injury would have on his career. For the next several months, he struggled with post-concussion syndrome, and was unable to return [2]. On February 1, 2007 Mike Matheny officially announced his retirement from baseball.

It has been reported that 20 – 25% of the active major league catchers have experienced concussive symptoms [3]. In addition, baseball was reported to be among the top three sports during which head injuries occurred and were treated in emergency rooms [4]. Although these data were not limited to concussions (they also included intracranial injuries and extracranial injuries without concussions, and facial injuries), they illustrate the high occurrence of head injuries in baseball. Dick et al. (2007) accumulated and reviewed injury data on collegiate baseball for a 16 year period. An injury to the head/neck was reported in 9% of games and 6.6% of practices over this period. In addition, 10% of the injuries reported in baseball were due to a batted ball. Although this number includes all positions, 257 injuries occurred to the catcher from batted balls. It is important to note that this number excludes impacts to the catcher that did not result in an immediately apparent injury. Four fatalities occurred during this span - one of these resulting from a batted ball to the head and a coma [5]. These injuries are not limited to the professional and collegiate players. Collins (2008) reports that 12.3% of the injuries reviewed in high school baseball were to the head/neck, and 3.5% of these injuries were concussions. Of the injuries to the high school catchers over the two year study, 34.6% were the result of batted balls. In addition, 8% of the injuries by batted balls were concussions [6]. Again, it is important to note that the numbers reported in these

articles may under estimate that actual amount of concussions due to the difficulty of determining whether a concussion occurred when there was no loss of consciousness. While concussions are not as prevalent in baseball as during contact sports such as football or hockey, theses reports indicate that concussions are also a problem in baseball

1.2 Background on concussions

One widely-accepted definition of a concussion was proposed by the Committee on Head Injury Nomenclature of the Congress of Neurological Surgeons [7]. The committee defined a concussion (derived from the Latin verb *concutere* "to shake violently") as "a clinical syndrome characterized by immediate and transient post-traumatic impairment of neural function, such as alteration of consciousness, disturbance of vision, equilibrium, etc. due to brainstem involvement." The severity of a concussion has been divided into three grades based on the symptoms exhibited. The symptoms for a grade on concussion are transient confusion, no loss of consciousness, and concussion symptoms or cognitive abnormalities lasting less than 15 minutes. Grade one concussions are the most frequent, making up more than 50% of reported concussions, but is often the most difficult to determine because there is no loss of consciousness. Grade two is considered a moderate concussion and is defined as transient confusion, loss of consciousness lasting less than 5 minutes, and cognitive abnormalities that resolve in more than 15 minutes. Some athletes "blackout" for a matter of seconds from an impact to the head (and have no recollection of this) making it difficult for medical staff to determine the concussion a grade one or two. A grade three (severe concussion) is a loss of consciousness for more than 5 minutes [7].

1.3 Brief overview of brain and neuron anatomy

There are three main regions of the human brain: (1) the hindbrain, (2) the midbrain, and (3) the forebrain.

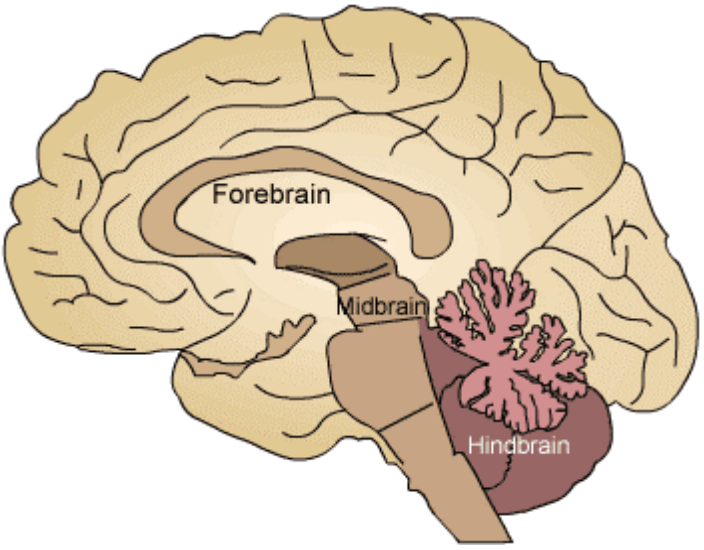

Figure 1. Main structures of the human brain [8].

The hindbrain consists of the brainstem, which is composed of the medullar oblongata and the Pons, and the cerebellum. The medullar oblongata is responsible for controlling respiration, regulating heartbeat, and adjusting cardiac flow [9]. The Pons relays sensory information between the cerebellum and the cerebrum and assists the medullar oblongata in regulating respiration. The "coordinator of body movements" or the cerebellum contains as many neurons as all of the rest of the brain combined and is responsible for controlling most muscles, neuromuscular facilitation, movement and timing resulting in a smooth, coordinated body movement. The midbrain consists of the reticular formation, substantia nigra, and ventral tegmental area. The reticular formation collect sensory input (sight, sound, smell, taste, and touch) from other portions of the brain and passes it on to

motor neurons (reflexes). It is also involved in sleep, arousal, and vomiting. The substantia nigra controls fine motor movement (i.e. the eye). The forebrain is consists of the thalamus, hypothalamus, and cerebrum. The function of the thalamus is to regulate states of sleep and alertness, arousal, and relay sensory information to various parts of the cerebrum. The hypothalamus is the control center of the autonomic nervous system (ANS). The ANS is responsible for homeostasis; therefore, the hypothalamus regulates body temperature and pH levels [9]. The cerebrum is divided into two halves, called hemispheres. The left side of the cerebrum controls the right side of the body while the right side of the cerebrum controls the left side of the body. The outer most sheet of neural tissue in the cerebrum is the cerebral cortex and is a major site of neurological disturbances in a concussion. Four lobes makeup the cerebral cortex, (1) the frontal lobe: responsible for reasoning, planning, parts of speech and movement, emotions, and deduction. (2) The parietal lobe: primary function is responding to stimuli from touch, pressure, pain, and temperature. (3) The occipital lobe is solely linked to vision and (4) the temporal lobe which perceives and recognizes auditory stimuli and memories [9].

Neurons, which are electrically excitable cells that relay electrical signals, are the functional units of the nervous system. The typical neuron contains dendrites, soma (cell body), axon, and axon terminal. Neurons may or may not have myelin sheaths which are used to speed up the rate of the signal (Figure 3). The brain consists of both myelinated neurons (white matter) and nonmyelinated neurons (gray matter). The dendrites are branched projections from the soma that receive information from neighboring neurons and transmit the signals to the cell body. The axon is a long conduit that for an electrical signal. This signal travels down the axon to the axon terminals causing the release of

neurotransmitters from the axon terminals into the synapse. The neurotransmitters than activate dendrites of neighboring neurons or other cells in the body such as a muscle cell [9].

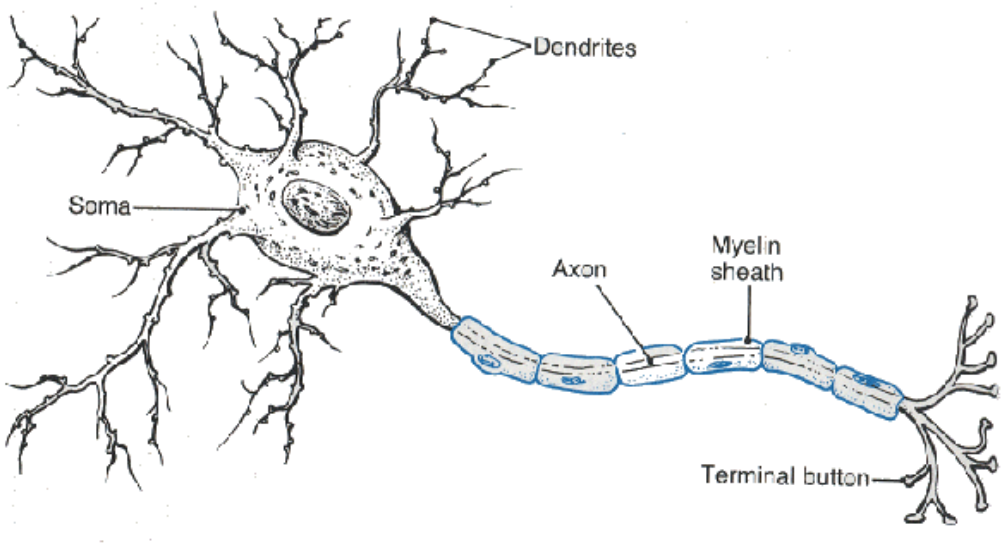

Figure 2. Typical neuron of the Central Nervous System [10].

1.4 Etiology of a concussion

Numerous studies have been completed in an attempt to better understand concussion mechanisms, but the specific etiology of a concussion remains unclear. Several mechanisms have been theorized but remain controversial because of the nature of the testing (cadavers, animal testing, etc.,) and the difficulties in measuring the motion of the brain. Zhang et al. (2001) completed a review of several theories of concussion mechanisms. One theory is that the shear strains that are generated by rotational acceleration cause a concussion. Another theory is that the relative displacement between the brain and skull produces coup and/or contrecoup cavitations (or the forming

of bubbles in low pressure areas). The brain is slightly less dense than the surrounding cerebral fluid and is suspended. Therefore, the cavitations that are formed cause the brain to move in the opposite direction of the cerebral fluid. This motion causes the brain to contact the skull and produce brain injury. A coup injury describes the brain traveling towards the site of impact while contrecoup injury describes the brain traveling away from the site of the impact due to the displacement of cerebral fluid [11]. Another theorized mechanism for concussions is that the pressure gradients caused by impact loading results in shear stress, distortion, or mass movement of the brainstem. This mechanism is mostly dependent upon translational or linear acceleration, and rotational acceleration is of minimal significance.

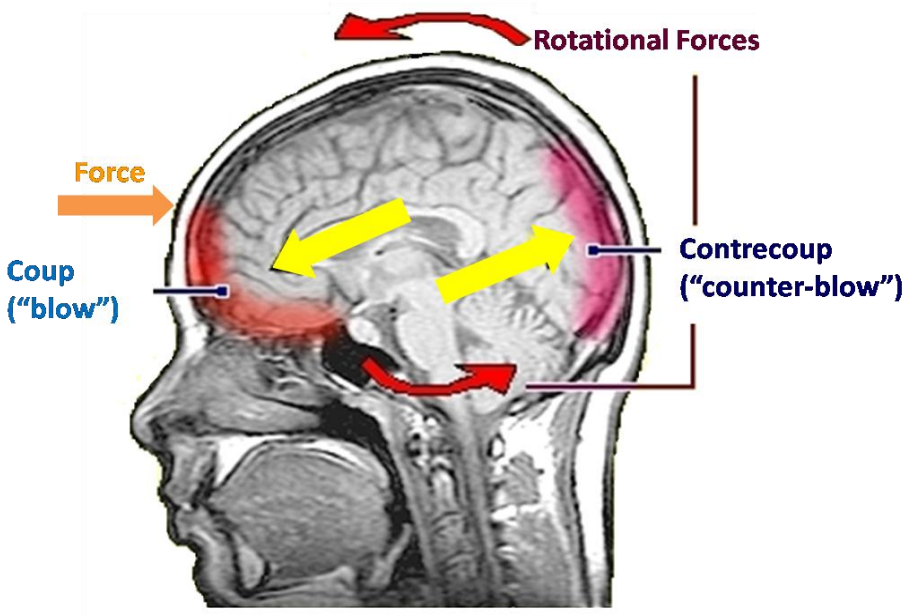

Figure 3. Coup and contrecoup injuries [12].

The common thread of these theories is that brain deformation or strain is the principal cause of brain injury. However, it remains unclear as to whether linear or rotational accelerations are more significant in concussions and other brain injuries. Linear accelerations are thought to result in a pressure gradient, resulting in localized (area of impact) effects, cerebral contusions and intracerebal hematomas [13]. In contrast, rotational acceleration is thought to generate shear and tensile strains. In a series of work by Gennarelli et al. (1986), it was concluded that angular acceleration was more injurious to the brain than linear acceleration in the production of concussive injuries [14]. In addition, the researchers claimed that every type of head injury can be caused by angular accelerations.

1.5 Pathophysiology of a concussion

The underlying mechanisms to brain injury are at the microscopic levels. The underlying mechanisms to brain injury are at the microscopic levels. One theory states that concussions are caused when the neurons are stretched and torn disrupting the transduction of electrical signals. The other theory states that concussions are caused by a rapid metabolism of the neuron cells followed by energy depletion. The unavailability of energy is believed to cause post-concussion symptoms.

One theory, outlined by Gennarelli (1986), is called the strain theory. A direct blow to the head or neck results in the acceleration of the head from rest. This head acceleration results in skull and brain accelerations. Rotational and linear accelerations lead to stress, and this stress leads to deformation/strains in the brain. From the centripetal distribution theory, which indicates that the maximum strain occurs at the

surface of the brain and smaller strains are located further inward, the severity of the injury is greater at the outer layers of the cerebral cortex. These strains can result in the stretching and tearing of neuron cells. This in turn alters or damages the synaptic distances, causing disruptions in signal transduction [14].

Another theory is that the impact disrupts the neuron's ability to produce adenosine triphosphate (ATP). Following injury to the brain, random release of neurotransmitters and unrestrained ionic fluxes can occur in the neuron cells, causing unintended depolarization of these cells. In order to restore the membrane potential, active transport mechanisms (i.e. the sodium-potassium pump) work vigorously to return the membrane potential to the resting state. These pumps are active transport mechanisms, and therefore require ATP. To meet the demands, the body increases glucose metabolism and rapidly depletes cerebral blood flow of glucose. In an attempt to lower the metabolic demand, a to the body decreases cerebral blood flow. The increase in ATP consumption is followed by a rapid decrease in the availability of ATP, resulting in an energy crisis. It is hypothesized that this energy crisis is the cause for post-concussion symptoms [15]. As a person recovers, the cells re-establish chemical balances and restore energy levels. The more frequently neurons are injured, the more likely recovery will take longer and the recovery of function maybe incomplete.

1.6 Symptoms of a concussion

Concussion symptoms are grouped into physical, cognitive, and behavioral symptoms and can occur at the onset of the injury or after a period of time. Physical symptoms of a concussion can include headache, dizziness, lightheadedness, and nausea.

Numbness, postural instability, fatigue, blurred vision, and ringing of the ears are also common symptoms. Any sensitivity to physical stimuli such as light and noise is also considered a concussive symptom. Cognitive symptoms of a concussion can include confusion, attention or concentration problems, amnesia, and difficulty organizing words. Behavioral symptoms can include restlessness, acting upon impulse, easily upset and angered, depression, anxiety and nervousness. Other notable symptoms can include bradycardia, hypertension, respiratory irregularities, and sleep apnea [7].

It is common for headache, disorientation, and nausea to occur immediately after the concussion, but other symptoms can appear several days to weeks after the injury. This is referred to as post concussion syndrome (PCS). It is suggested that PCS is present from the time of injury, but more symptoms become present as time goes on due to the delayed pathophysiologic reaction of the brain cells, meaning that the function of the cells can change during PCS [16].

The areas of the brain that are affected in a concussion can be determined by examining the nature of the symptoms. For example, hypertension, apnea, vomiting, and respiration irregularities can be traced back to the lower Pons and medulla. Damage to the cerebral cortex can result in posttraumatic amnesia, attentiveness, and mental concentration.

1.7 Recovery and return to play

Many times, high school, collegiate, and professional athletes return to play before they have fully recovered from the concussion. This can leave an athlete susceptible to another and far more serious brain injury. Due to the physiologic cascade

and flux in the cellular metabolism that occurs in the brain post-trauma, the brain is the most vulnerable to further injury for days to weeks after the incident. To account for the increased cellular metabolism following a concussion, there is a decrease in cerebral blood flow, lowering metabolic demand. A second impact at this point in the physiologic cascade could result in brain cell death and is referred to as second impact syndrome (SIS) [15].

Determining when an individual can return to play following a concussion is an extremely difficult decision for a sports medicine physician. Variations in recovery time are based on individual factors and may not be the same from patient to patient. Factors that have been researched include gender, age, and injury history. Broshek (2005) reported that female athletes had more post-concussion symptoms compared to males [17]. Currently, the mechanisms for the difference in patient response to concussion based on gender are still not understood. Recent studies have hypothesized that younger individuals may have prolonged diffuse cerebral swelling after a concussion [18]. Another theory is that younger athlete's brains are up to 60 times more sensitive to glutamate [19]. Glutamate is the most abundant excitatory neurotransmitter in the body. In brain injuries such as concussions, glutamate accumulates outside of neuron cells (because glutamate transporters may work in reverse). This increase in extracellular glutamate causes calcium ions to enter glutamate transporters, resulting in neuronal damage and possibly cell death. This is also known as excitotoxicity [20]. Although younger athletes recovered more slowly from older athletes, there was no significant difference in the outcome of the recovery [21]. Brain injury history is an important individual determinant for return to play. Evidence is continually being gathered on the

cumulative effects of multiple hits and concussions. Studies have shown there are neuropsychological impairment and neurologic abnormalities in boxers [22, 23]. Other studies have shown neurocognitive discrepancies in football players and long-term consequences in soccer players from repetitive blows to the head [24]. Currently, there is no precursor to determine how many concussions an athlete can sustain before retirement from sport [20].

During the last three decades, twenty different concussion management guidelines have been published. The problem with these guidelines prior to the conference in Vienna, Austria in 2001, is that they could not adequately assure proper management of all concussions in athletes because they failed to implement post-injury neuropsychological testing [20]. It was decided at this conference that neuropsychological testing should be a “cornerstone” of concussion management. The management guidelines that were produced at this conference recommended that neuropsychological testing be used whenever possible and used to develop a return to play protocol (Table 1). Neuropsychological testing provides a baseline post-injury analysis of cognitive function that is likely affected by concussive injury. An example of a computer-based model for neuropsychological testing is Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT). ImPACT consists of seven test modules to assess cognitive domains such as reaction time, verbal memory, visual memory, processing speed and impulse control [20].

Table 1: Vienna Concussion Conference: Return to Play Recommendations [25].

1. **Removal from contest following and signs / symptoms of concussion.**
2. **No return to play in current game**
3. **Medical evaluation following injury**
    a. Rule out more serious intracranial pathology
    b. Neuropsychological Testing considered "cornerstone" or proper post-injury assessment
4. **Stepwise return to play**
    a. No activity at rest until asymptomatic
    b. Light aerobic exercise
    c. Sport-specific training
    d. Non-contact drills
    e. Full-contact drills
    f. Game play

****** Each step (a-f) is designed to occur 24 hours after the previous.

If an athlete is unable to complete a step to return to play (a-f) without post-traumatic symptoms returning, they shall drop to a previous level and repeat the process. University of Pittsburgh Medical Center Sports Concussion Program states that an athlete should not return to play with abnormal neuropsychological test results and/or symptomatic until these are resolved [20].

1.8 Head injury tolerance and criteria

Zhang et al. (2001) defined tolerance level as "the magnitude of loading of the body which produces a specific type of injury at a specified injury severity level" and injury criterion as "a physical parameter which correlates well with injury severity of the body part in question" [25]. Together, injury criterion and tolerance level have been used to quantify risk of injury in various scenarios such as a concussion and have been integral in improving protective equipment. As previously mentioned, linear and rotational head

acceleration are parameters used to quantify stresses and strain of the brain. To relate these accelerations to concussive injuries head injury criteria and tolerance levels were developed using cadavers and animal models. These allowed researchers to take the measured accelerations and determine if a concussive injury occurred by simply comparing their numbers to injury thresholds [25].

The first experiments to investigate brain injury took place in the 1940s at Wayne State University. Researchers investigated the effects of linear skull fracture and pressure as injury mechanisms on the brain by experimenting with cadavers and anesthetized animals. The product of this research was the Wayne State Tolerance Curve (WSTC). The WSTC (Figure 1) shows the effective head acceleration against the impact duration and is based on the assumption that linear skull fracture (breaks in the bone that are transverse to the thickness of the bone) occurs for 80% of all concussive impacts. Head accelerations exceeding the WSTC have a strong possibility of resulting in a concussion [25].

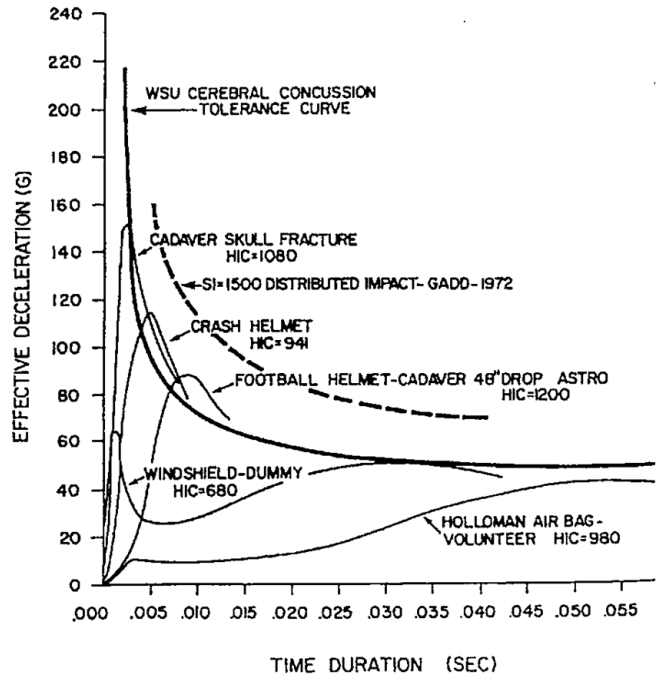

Figure 4. Wayne State Tolerance Curve [25].

Several injury criteria have arisen from the WSTC, but the two most commonly used today are the Gadd Severity Index, commonly referred to as the Severity Index (SI), and the Head Injury Criterion (HIC). The SI was proposed by C.W. Gadd in 1966 [26]. Gadd (1966) plotted the WTSC on a logarithmic scale and determined that between 4 – 50 msec the WTSC could be approximated by a line with a slope of -2.5. He then used the slope of the line as a gain and proposed SI:

$$SI = \int_0^T a^{2.5} dt \qquad \textbf{[1]}$$

where a is the instantaneous linear resultant acceleration and the integral is calculated over the entire acceleration curve. Gadd (1966) proposed a SI of 1000 as a threshold for injury. A problem with SI is its tendency to have impractically high values for indirect impacts (impacts that glance off the headform) [25]. Today, the National Operating

Committee on Standards for Athletic Equipment (NOCSAE), whose goal is to commission research on and, where feasible, establish standards for athletic equipment. NOCASE has instituted safety criteria for protective helmets stating that the SI shall not exceed 1200 for the drop test (headform and helmet is dropped from a preselected height) nor the projectile test (impact test with a baseball traveling at 60 mph), and that no structural changes to the masks (permanent material deformation or failure) are permitted [27].

A more widely used injury criterion is the Head Injury Criterion (HIC). The HIC is conceptually similar to SI with the difference between the two being the consideration of the time over which acceleration occurs:

$$HIC = (t_2 - t_1)\left[\frac{1}{(t_2 - t_1)}\int_{t_1}^{t_2} a(t)dt\right]^{2.5} \quad \textbf{[2]}$$

where *a(t)* is the resultant head acceleration measured at the center of mass, t_1 and t_2 are the initial and final times over which the integral is calculated (t_1 and t_2 vary between 0 and 15 msec in a HIC_{15} calculation). Zhang et al. (2001) has reported a HIC value of 1000 as a brain injury threshold. Both HIC and SI only use linear resultant accelerations and do not incorporate angular acceleration [25].

Thresholds for angular acceleration of the head are not well known and thresholds using this parameter are primarily based off of cadaver tests and human volunteers (Table 2). The values shown from primate tests were calculated using a scaling factor and are somewhat controversial because of the inaccuracy associated when comparing animal test results to humans. The differences in animal and cadaver test results have demonstrated the need for a combined rotational and linear acceleration threshold [25].

Table 2: Proposed tolerance limits in terms of angular acceleration and velocity [25]

References	Type of surrogate	Proposed tolerance limits
Ommaya, 1967	Primate	7500 rad/sec^2
Ommaya et al., 1971	Primate	1800 rad/sec^2 and 60-70 rad/sec
Lowehielm, 1975	Cadaver	4500 rad/sec^2 and 50-70 rad/sec
Advani et al., 1975	Mathematical model	2000-3000 rad/sec^2
Ewing, 1975	Volunteers	1700 rad/sec^2 and 32 rad/sec
Pincemaile et al., 1988	Volunteer boxers	16000 rad/sec^2 and 25 rad/sec 13600 rad/sec^2 and 48 rad/sec
Margulies et al., 1990	Primate tests and physical models	16000 rad/sec^2 and 46.5 rad/sec

There is some controversy using the above injury metrics. The SI was generated from data based on severe concussion or diffuse brain injury and is appropriate when evaluating life-threatening brain injuries. SI is calculated over the entire interval and therefore a long, but low acceleration impact could produce the same SI value as a short, but high acceleration impact. Each impact could have drastically different affects that cannot be discerned from an SI value [28]. For example, Gennarelli and Thibault [29] suggested that acute subdural haematoma can could occur from an impact of 200g acceleration over a 3.5 ms time duration, while Ommaya [30] stated that diffuse axonal injury (commonly associated with a concussion) occur from distrubuted loading with a time duration greater than 10 ms. Lastly, SI can only assess head injury from impacts located at the forehead [28].

HIC is intended to correct some of the discrepancies disscused with SI, but HIC has some of its own. For example, Prasad and Mertz [31] conducted a study that looked at skull fracutre and brain injury data to calculate HIC thresholds. They determined that a HIC of 1000 is 16% risk of a life-threatening brain injury. In actuallality, the probability

of a life-threatening injury can be given for HIC value of 500 and 2500 [28]. Furthermore, HIC and SI thresholds do not incorporate rotational accelerations, which have been shown to cause brain injury [32]. Failing to report angular accelerations could under estimate brain injury potential [28]. Despite inaccuracies associated with both SI and HIC, many researchers use both injury criterion to assess risk of potential brain injury. Current research is investigating rotational accelerations in an attempt to combine linear and rotational accelerations into a injury criterion.

1.9 Concussions in sports

Most of the sports-related concussion research has been focused on football, soccer, hockey, and boxing due to the high prevalence of head impacts. As a result of this high prevalence, these sports provide the ability to record head accelerations in living human subjects and later relate theses accelerations to injuries. For example, Pellman et al. (2003) used NFL game videos of selected collisions between players to determine the speed and direction of the players involved. This information was then used to recreate the collisions in a laboratory with test dummies to estimate head accelerations.. Over a five year period, 1996 – 2001, 186 impacts were recorded and used for the analysis. Of these impacts, 31 were reconstructed in the lab. Using the translational acceleration from the trials, the SI and HIC values were calculated. Risk of injury was determined using the concussive impacts and the resulting SI and HIC values. The reconstructed data suggested that nominal injury values for the NFL impacts were peak linear acceleration of 79 g, SI of 300, HIC of 250, and peak rotational acceleration of 5757 rad/s^2 [33].

These reconstructed impacts included 23 definite cases of concussion within the 31 reconstructed impacts.

This study had two main limitations. First, the head accelerations were indirectly measured (calculated by reviewing video feeds of the impacts). Second, these accelerations were dependent on video reconstruction and dummy impacts. Duma et al. (2005) reported measurements of head accelerations upon impacts from 38 players during one season of collegiate football. The study used the Head Impact Telemetry System which records head accelerations during gameplay and wirelessly transmitss these measurements to a computer for later analysis [34]. Each helmet was configured with six spring-mounted accelerometers integrated into the helmet. Peak linear head acceleration, peak rotational head acceleration, SI, HIC, and impact location were calculated. For the 2003 season, 3312 valid head impacts were recorded. The average peak head acceleration was 32g ± 25g, with 89 % of the impacts having a peak head acceleration under 60g compared to an average of 98g (for concussive impacts) reported by Pellman et al. (2003) Averages for SI, and HIC were 36 ± 91, and 26 ± 64 respectively. Average peak rotational accleration was 905 rad/s^2 ± 1075 rad/s^2 about the x-axis and 2020 rad/s^2 ± 2042 rad/s^2 about the y-axis. Five concussions were recorded during this season, one of which involved a peak head acceleration of 81 g which fell in the optimal injury risk range reported by Pellman et al. [34]. Several results from this study contradicted nominal tolerance for concussion values reported by Pellman et al. and King et al. [35, 36]. A total of 25 hits surpassed the nominal tolerance values for SI, HIC, and peak head acceleration without any reports of concussions. Authors of this study suggest this could

be a result of athletes not realizing they have suffered a concussion. More data collection is needed for conclusions to be made on published injury thresholds.

In 2000, Naunheim et al. compared the acclerations that occurred in three high school sports including football, hockey, and soccer[37]. The purpose of these studies was to quantify head accelerations that athletes are exposed to in order to quantify concussions by determining injury thresholds. This in turn could lead to improved helmet design and sports regulations to help prevent concussions. For football and hockey, players were fitted with high school regulation helmets that had a triaxial accelerometer. In the hockey helmet, it was located at the vertex of the helmet. In the football helmet it was integrated in the padding. To measure the head acceleration of soccer players while heading a ball, participants were fitted with a headpiece that contained three triaxial accelerometers. Head accelerations for the football and hockey players were collected during game play and practices. For soccer, headers were simulated in a laboratory. Only impacts with peak head accelerations greater than 10g were recorded. Results showed that that the average peak acceleration was 29.2g ± 1.1g for football (for 158 recorded impacts), 35.0g ± 1.7g for hockey (for 161 recorded impacts), and 54.7g ± 4.1g for soccer (25 trials while heading a soccer ball with an average velocity of 39.3 miles per hour). No concussions were recorded in this study [37].

1.9.1 Concussions in baseball

The majority of the research on concussions in baseball has focused on ball impacts to the unprotected head, which is a fairly common head injury scenario [5, 6]. In 1994, Heald and Pass investigated the effects of hardness and liveliness of baseballs and

softballs on the risks of head injuries. Hardness is defined by the magnitude of force needed to compress a baseball a quarter inch, and liveliness is defined as the coefficient of restitution of the ball. Baseballs of different hardness and liveliness were propelled at humanoid head models, rigid head forms (Hybrid III), and cadaver heads. Head accelerations were used to determine HIC and SI values over a range of ball speeds. The risk of injury was determined using the Prasad-Mertz risk curves which relates SI values to risk factors for head injury. The Prasad-Mertz risk curves presents the relationship between head injury probability and HIC_{15} values. All impacts were located at the temple area of the head models, head forms, and cadavers [38]. The conclusions from this research were that (1) the humanoid head form and SI test are reasonable for assessing risk of head injury, (2) using slightly softer baseballs can reduce the incidence of head injury, and (3) increasing the hardness of the ball will increase the risk of head injury [38].

Another study investigated the differences between the head accelerations measured with three different headforms [39]. A baseball pitching machine propelled baseballs at speeds of 75 – 85 mph at the forehead and temple area of each headform. Measured head acclerations were compared for each headform and no statistical difference between mean values of the peak acceleration for the three headforms were found for forehead impacts. There was statistical differences between the mean values of peak accelerations the three headforms were found for side impacts. Melvin [39] attributed the differences between headforms to construction (curvature, materials, etc.) differences.

Jones and Mohan [40] conducted frontal (forehead) and side (temple) baseball impacts and measured head accelerations to calculate SI and HIC values using an anthropometric test dummy. Ball speeds ranged from 90 – 100 mph and resulted in average HIC values of 1025 ± 631 and peak head accelerations of 268g ± 55g for side impacts. Tests resulted in average HIC values of 1035 ± 20 and peak head acceleartions of 359g ± 9g for frontal impacts [40].

While these studies provide estimates of head accelerations upon impact with a baseball, there are no published studies to our knowledge that have investigated the effectiveness of catcher's masks at attenuating these accelerations. A group of students from Kettering Univerisity compared the effectiveness of two different types of catchers masks, as reported in a news article on the university webpage [41]. The students used a modified skeet shooter to catapult baseballs at an anthropometric dummy at speeds 90 – 105 mph. For frontal impacts, the ball struck the cage of the masks. For side impacts, the ball stuck the cage for the one-piece mask and a the shell of the skull cap for the two-piece mask. The article reported that for the frontal impacts, the peak "G-force" was 2.763 and 9.814 for the two-piece and one-piece mask, respectively. For the side impacts, the one-piece mask record a G-force value of 13.57 while the two-piece mask had a G-force of 32.02. There conclusion was that the one-piece mask protected better during frontal impacts, while the two-piece mask protected better during side impacts (which they declared represented a batter's back-swing) [41].

1.10 History of catcher's masks

In 1876, Harvard catcher Alexander Tyng was having difficulty catching a new pitch called the curve ball. The difficulty of determing the baseball's trajectory lead to foul tips, wild pitches, and the overall inability of the catcher to catch the pitch. Tyng's face, like many other catchers of the time, were being badly beaten by foul tips and wild pitches. At this time, the only protection available for catchers was a mouthguard, and this only prevented catchers from losing teeth. In order to prevent further injury, fellow teammate and coach, Fred Thayer, developed the "bird cage" (Figure 5). The bird cage was a fencing mask that was modified by a local tinsmith to provide better vision and increased padding for the chin and forehead. In the spring of 1877, Tyne became the first catcher to wear a catcher's mask. A year later, Spalding began selling the patented catcher's mask. The popularity of the catcher's mask was not widely accepted until the end of the decade and became an essential piece of equipment that would influence how the game was played [1].

Figure 5. Fred Thayer's bird cage design from a modified fencing mask [1]

The evolution of the catcher's mask continued into the 20th century. For the first half of the century, the approach to protective equipment was a non-scientific, field-testing approach. The primary focus of catcher's mask design during this time was visibility and protection from physical damage i.e., broken bones, bruises, and lacerations. The "Open Vision" mask was the successor of the bird cage and debuted in 1910. A decade later, the first aluminum casting with horizontal crossbars was patented by umpire James E. Johnstone. During the next 50 years the metal cage evolved becoming a better shock-absorber and began to take on curved shapes to deflect the ball [1].

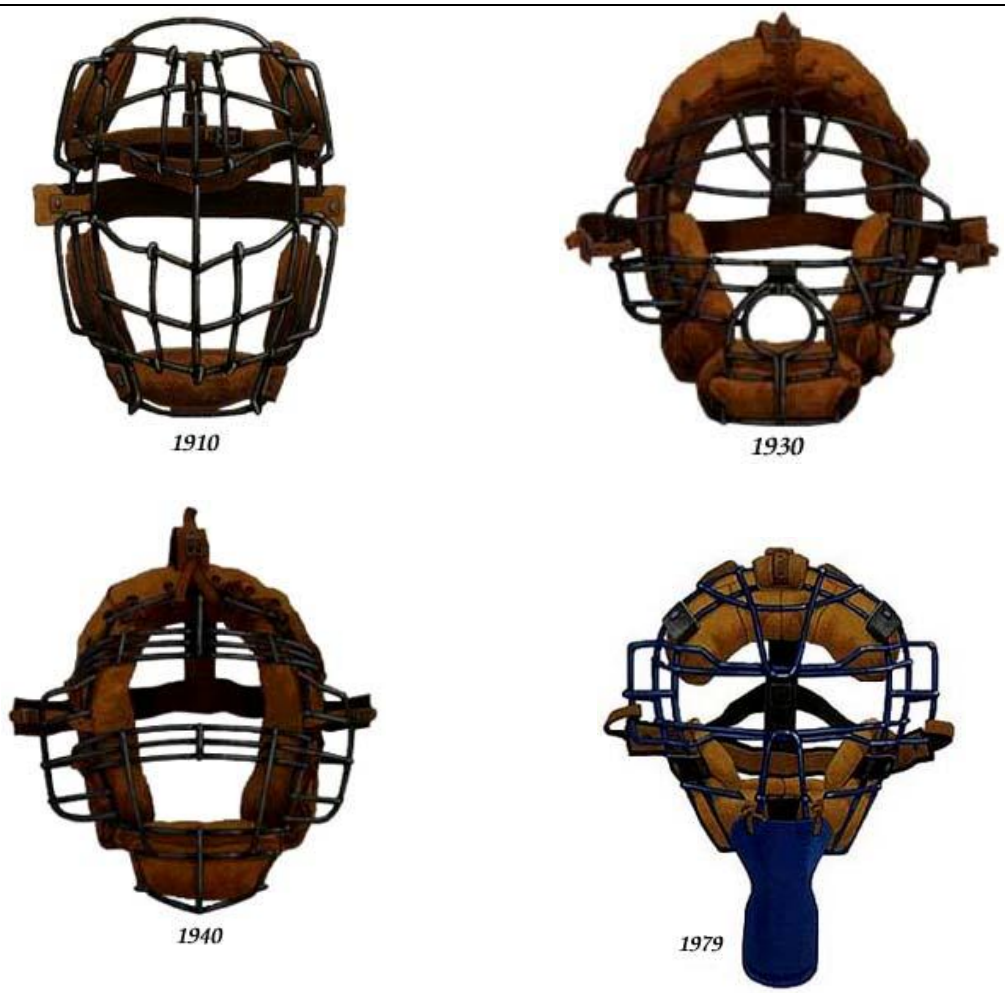

Figure 6. The evolution of the catcher's mask [1]

Today the primary masks worn by players are the traditonal two-piece mask (Figure 7) and the the one-piece, hockey-style mask first warn by Charlie O'Brien in 1997 (Figure 7). This hockey-style mask was designed to protect catchers from front, side, and rear impacts to the head [1].

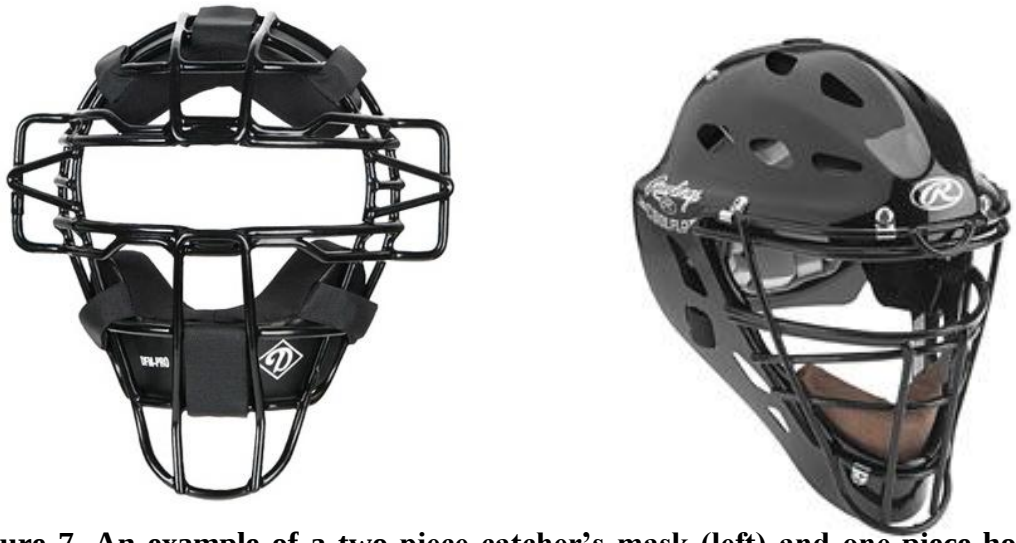

Figure 7. An example of a two-piece catcher's mask (left) and one-piece hockey-style mask (right) [1]

The liner located throughout the mask has also gone through mass evolution during the past century. For example, the forehead and chin liner of the bird cage was made from canine skin and its primary purpose was to help secure the mask to the head. Today, most manufacturers use energy-absorbing liners comprised of semirigid polyurethane foams or expanded polystyrene bead foams [1].

The National Operating Committee on Standards for Athletic Equipment (NOCSAE) formed in 1969 to fund research toward injury prevention and began to develop safety standards for athletic equipment and headgear. The first standard

NOCSAE published was the football helmet standard in mid 1970s. The majority of their efforts focused on football which proved to be the most fatal sport due to the high frequency of head impacts. By 1978, the National Collegiate Athletic Association and the National Federation of State High School Association mandated that all players wear helmets that meet NOCSAE test standards. Current standards for protective helmets are constantly being modified as results from research become available. According to NOCSAE document 024 – 03m05 each catcher's masks (helmet with faceguards) are tested using both a projectile and drop test. NOCSAE uses a standard humanoid headform instrumented with a tri-axial accelerometer to measure linear head acceleration. During the drop tests, the headform with the helmet is attached to an anvil that freefalls to an impact surface (the impact surface is made of polyurethane thermoplastic elastomer). The headform is oriented six different positions (defined in NOCSAE document 001 – 08m08b) to change the impact location for each test. During impact tests, the headform, with the helmet attached to a load bearing table, is subjected to baseball impacts propelled at speeds of 60 mph. The headform is oriented in three different positions, (defined in NOCSAE document 024-10m10). No contact to the ocular area (location in metal cage with no vertical or horizontal bars designed for optimal vision) is permitted during these tests. The helmets must pass the sanctioned requirements before they can be sold. These requirements state that no helmet can be structurally altered or damaged during the test and that the SI shall not exceed 1200. If the mask passes the current standards, a NOCSAE emblem is placed on the mask and can be sold on the market.

1.11 Study goals

The Matheny story and stories in the mass media indicate that concussions are a problem in baseball. This fact led to the question as to how well currently used catcher's masks help to prevent concussions. The lack of stories describing facial injuries in catchers suggests that these masks adequately prevent these injuries. However, they may not be optimally designed to help prevent concussions. As such, the study described in the next chapter was performed to measure the ability of catcher's masks to attenuate head accelerations upon impact with a baseball. In addition, we compared head accelerations upon impact with a baseball with established injury thresholds. The results from this work will provide a foundation of knowledge for future research aimed at understanding the cause of concussions, improving protective equipment, and ultimately reducing the occurrence of concussions in catchers and umpires due to facial impact with a baseball.

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Chapter 2

Analysis of the Ability of Catcher's Masks to Attenuate Head Accelerations upon Impact with a Baseball

2.1 Introduction

An estimated 1.6 – 3.8 million sports-related traumatic brain injuries occur each year [42]. Mild traumatic brain injuries (MTBI), a term that is used interchangeably with the term "concussion", occur in approximately 75% of all traumatic brain injuries [43]. The symptoms of sports-related MTBI can include, but are not limited to, nausea, headache, dizziness, mood changes, and memory dysfunction. In most cases of MTBI, the individual fully recovers [44-46]. However, up to 15% of MTBI cases result in debilitating, long-term effects on quality of life [45, 46]. Individuals with a history of concussions are up to four times more likely to sustain another concussion compared to individuals with no prior concussions [47, 48]. Multiple concussions can lead to greater risk for long-term consequences [4].

Although not as prevalent as during contact sports such as football or hockey, MTBIs can also occur during baseball. The National Electronic Injury Surveillance System reports baseball to be among the top three sports during which head injuries occurred and were treated in emergency rooms [4]. Although these data were not limited to concussions (they also included intracranial injuries and extracranial injuries without concussions, but not facial injuries alone), they illustrate the high occurrence of head injuries in baseball. In addition, stories in the mass media document numerous incidents of concussions in baseball. One scenario for a concussion in baseball is for a foul tip to

impact a catcher or umpire in the mask [5, 6]. Based upon this, it would seem prudent to investigate the effect that baseball impact has on the head, and the ability of catcher's masks to help prevent MTBIs. Such information could improve our understanding of these injuries, and potentially contribute to improvements in the effectiveness of these masks at preventing MTBIs.

To our knowledge, no information is available in the literature on the effectiveness of catcher's masks at attenuating head acceleration upon impact with a baseball (head acceleration is commonly used as a surrogate measure of risk of MTBI). The National Operating Committee on Standards for Athletic Equipment (NOCSAE) has established safety requirements for new catcher's masks as supplied by manufacturers [49]. Using both projectile testing and drop testing, these requirements state that the Severity Index (SI) shall not exceed 1200 and that no structural changes to the masks (permanent material deformation or failure) are permitted. Jones and Mohan [40] documented peak head accelerations and Head Injury Criterion (HIC) when impacting an anthropomorphic dummy with a baseball. Melvin [39] documented differences in peak head accelerations and HIC between frontal and side baseball impacts in three different anthropomorphic dummies. Heald and Pass [38] reported peak head acceleration, HIC and Severity Index (SI) using a variety of baseballs and softballs, and using two anthropomorphic dummies and cadavers. To our knowledge, the only study that investigated head accelerations with catcher's masks was performed at Kettering University (albeit unpublished in the peer-reviewed literature). Peak head accelerations measured with an anthropomorphic dummy head were compared between two types of catcher's masks for frontal and side impacts [41]. Although their results support

improved performance of one-piece hockey-style masks for side impacts and two-piece traditional masks for frontal impacts, the effect of wearing a catcher's mask on head accelerations were not reported.

Based upon the severe and long-term effects that MTBI can have on players, and the limited information available in the literature, the goal of this study was two-fold. The first goal was to measure the ability of catcher's masks to attenuate head accelerations upon impact with a baseball. Quantifying this ability will provide a foundation of knowledge that could contribute to improvements in catcher's mask design and performance. The second goal was to compare these head accelerations to established injury thresholds for MTBI. This comparison will provide insight to the mechanisms of these injuries.

2.2 Methods

Testing involved using a pneumatic cannon to shoot baseballs at an instrumented headform with and without a catcher's mask on the head (Figure 8). The head and neck assembly of a 50th percentile male Hybrid III anthropomorphic test device was attached to a 6.8 kg base that could translate with low friction along a pair of rails. The pneumatic cannon consisted of a high pressure air tank, a solenoid, and a 1.22 m long, 0.10 m inner diameter polyvinylchloride pipe. Prior to all tests, the end of the launch tube was 0.53 m from the head and was aligned so that the center of the ball would impact the tip of the nose. The ball speed was controlled from approximately 26.8 – 35.8 m/s (60 – 80 mph) by varying the pressure in the tank from approximately 172 kPa – 435 kPa. This range of speeds was selected to approximate the speed of a baseball impacting a catcher's or umpire's mask after a foul tip in high school, college, or professional baseball.

Regulation NCAA baseballs (mass = 142 ± 8.5 g, circumference = 22.9 ± 0.8 cm) were propelled at the head using a pneumatic cannon.

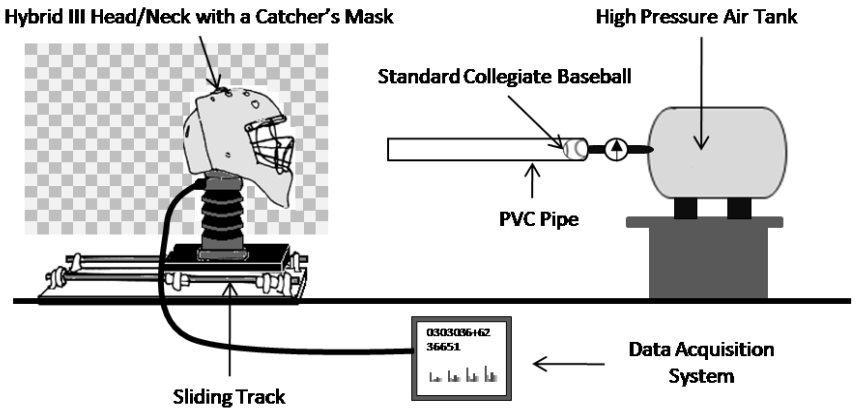

Figure 8. Experimental setup featuring the high pressure air tank, PVC pipe, head-neck-slider system with a catcher's mask, and the data acquisition system.

Several tests were first performed without a mask on the head for comparison with tests with a mask. Tests were then repeated using four catcher's masks. Two masks were one-piece hockey-style masks and two were two-piece traditional-style masks (Figure 9). All of the masks were available over-the-counter and were approved for use by the NOCSAE. Mask sizes were chosen based upon the manufacturer's recommendations and the size of the Hybrid III head.

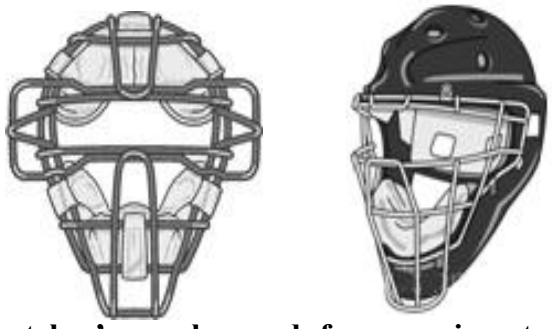

Figure 9. Types of catcher's masks used for experimentation; (*Left)* one-piece catcher's mask (*Right)* two-piece catcher's mask.

The Hybrid III head was instrumented with nine accelerometers (7264-2000B, Endevco, San Juan Capistrano, CA) in a 3-2-2-2 orientation so that linear and angular acceleration could be calculated [50]. All acceleration data were sampled at 50 kHz. High speed video (V9, Phantom, Wayne, NJ) was also collected at 1800 frames per second with a 1440x720 resolution to visualize the impact, verify proper ball impact location, and to calculate ball speed. Ball speed was calculated by determining the displacement of the baseball over 7 – 8 frames and dividing by the elapsed time. Preliminary testing revealed no systematic trend in peak force and ball contact time when a single ball was propelled at a load cell at the same speed, suggesting no change in ball properties over this number of impacts. However, a different baseball was used for each of the trials without a mask, and one baseball used for all testing on a single mask (up to 12 trials). All trials occurred at a room temperature of 22.8 – 23.3 °C and a humidity of 22 – 23%, and the balls had sat in this environment for 24 hours prior to testing.

Acceleration data were processed using custom-written MATLAB software. Data were filtered in accordance with SAE J211 using channel frequency class (CFC) 1000, and the resultant linear and angular accelerations were determined. Tests with ball speed outside of 26.8 – 35.8 m/s (60 – 80 mph), or improper ball impact location, were removed from further analysis. HIC and SI [36] were calculated using the resultant linear acceleration from each test.

2.3 RESULTS

Four trials without a mask and twelve trials with a mask were selected for further analysis based upon ball speed being 60 – 80 mph and ball impact at the desired location. Peak resultant linear acceleration occurred approximately 0.8 ms after initial contact

without a mask and approximately 3 ms after initial contact with a mask (Figure 10). The ball remained in contact with dummy head/mask for approximately 3 ms, regardless as to whether or not a mask was worn (Figures 11 and 12).

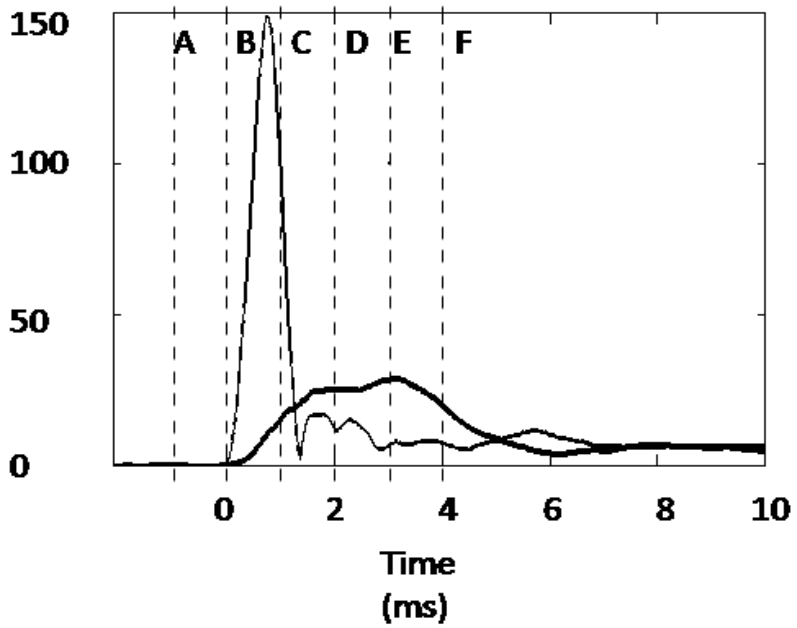

Figure 10. Resultant linear acceleration during a trial without a mask (thin line) and with a mask (thick line). During the trial without a mask, the ball speed was 67.1 mph (30.0 m/s), and peak acceleration was 149 g. During the trial with a mask, the ball speed was 67.6 mph (30.2 m/s), and peak acceleration was 29 g. Dotted lines labeled A-F indicate the instants in time at which photographs are shown in Figures 11 and 12.

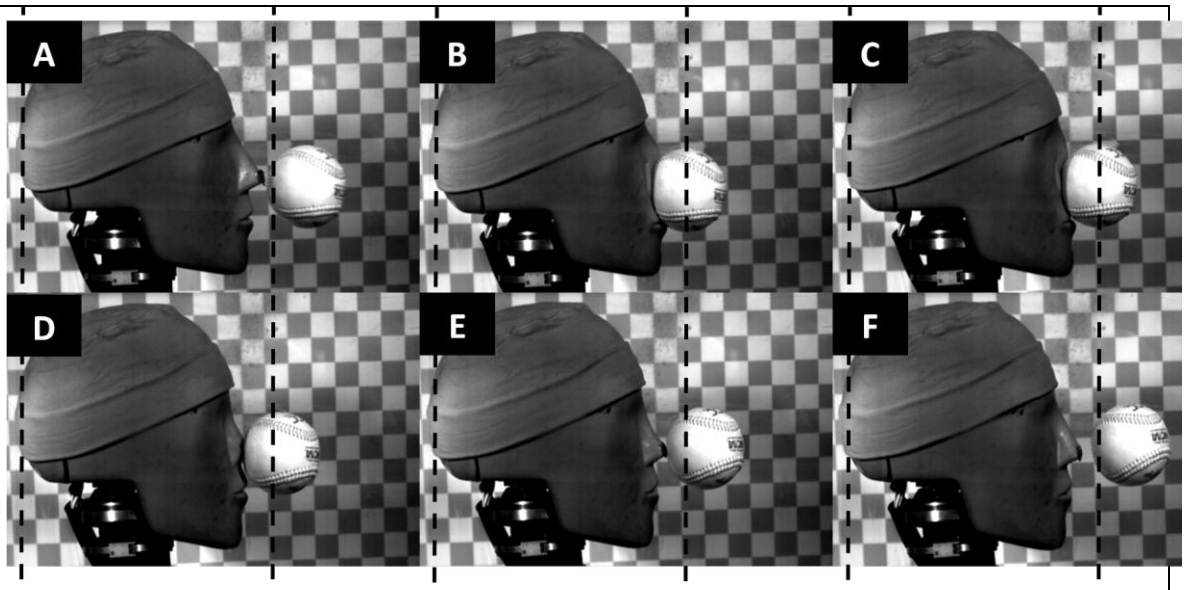

Figure 11. Photographs during a trial without a mask in which the ball impacted the head at 67.1 mph. The time interval between each photograph was 1.1 ms, therefore 5.5 ms elapsed from frame A to frame F. Vertical lines serve as reference points for initial position of the head, and help illustrate that only a small amount of head displacement occurred over this time.

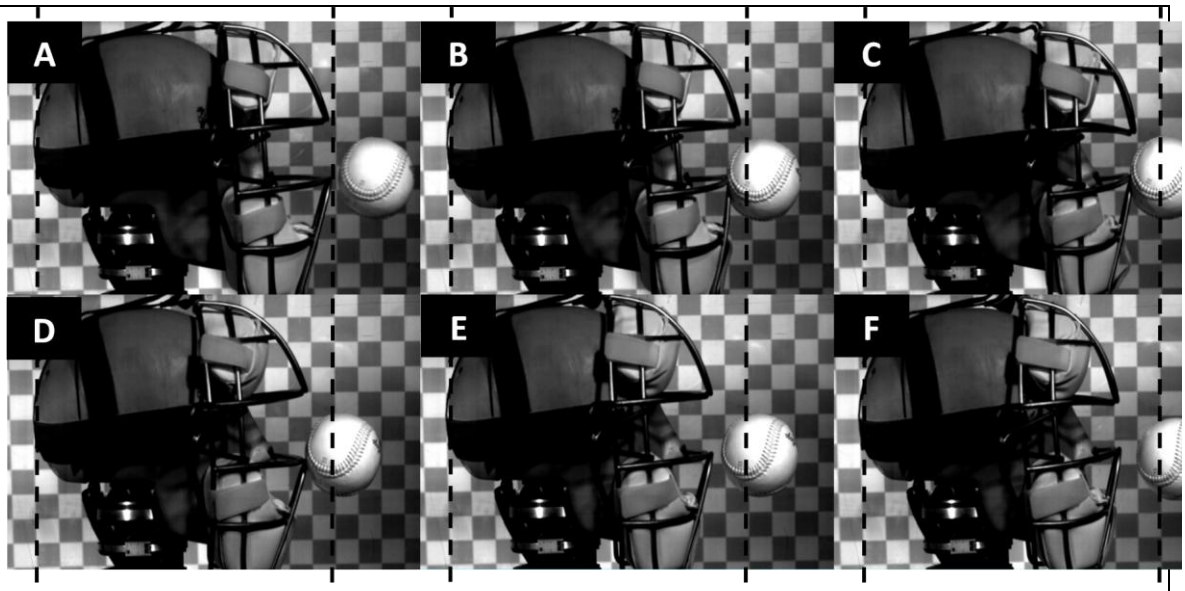

Figure 12. Photographs during a trial with a mask in which the ball impacted the mask at 67.7 mph. As in Figure 4, the time interval between each photograph is 1.1 ms and vertical lines serve as reference points for initial position of the head and mask. Although the mask in frame F has been displaced from its initial position approximately half the diameter of the ball, the head has not displaced an appreciable amount.

The catcher's masks reduced peak head accelerations (Figure 13). Peak linear resultant acceleration was 140 – 180 g without a mask and 16 – 30 g with a mask over the range of balls speeds investigated. Peak angular resultant acceleration was 19500 – 25700 rad/sec^2 without a mask and 2250 – 3230 rad/sec^2 with a mask. The masks reduced peak linear acceleration and angular accelerations to 11 – 16% and 11 – 12% of the trials without a mask, respectively. The HIC was 93 – 181 without a mask and 3 – 13 with a mask. The SI was 110 – 210 without a mask and 3 – 15 with a mask.

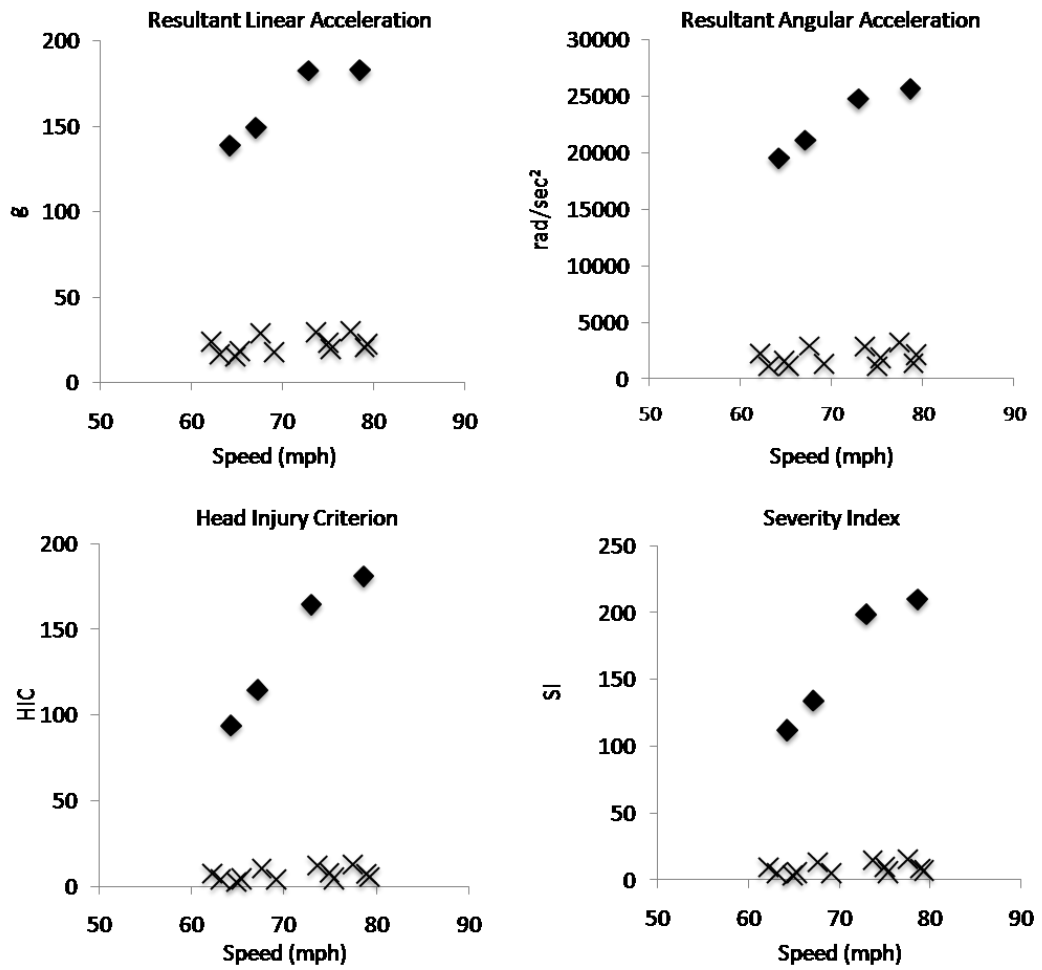

Figure 13. Summary of experimental data. (*Top Left*) Resultant Linear Acceleration (*Top Right*) Resultant Angular Acceleration (*Bottom left*) Head Injury Criterion (*Bottom Right*) Severity Index.

2.4 Discussion

The first goal of this study was to measure the ability of catcher's masks to attenuate head accelerations upon impact with a baseball. The masks we tested substantially reduced peak head accelerations. Peak linear resultant and angular resultant accelerations were reduced 83.3 – 88.6% and 87.4 – 88.8%, respectively, over the ball speeds we investigated. In addition, the HIC and SI were reduced 92.8 – 96.8% and 92.9 – 97.3 %, respectively. The second goal of this study was to compare these head accelerations to established injury thresholds for MTBI. Peak linear resultant and angular resultant accelerations were 16 – 30 g and 2250 – 3230 rad/sec^2 with a mask. In addition, the HIC and SI were 3 – 13 and 3 – 15, respectively, with a mask. These values were substantially lower than commonly accepted thresholds for MTBI, suggesting that baseball impacts under the conditions tested do not present a significant risk for MTBI. However, anecdotal and epidemiological evidence indicate that these injuries do occur. This apparent discrepancy is discussed in more detail below.

Several injury thresholds have been reported for MTBI. Unfortunately, the lack of understanding of what causes MTBI, the use of varying methods to develop these injury thresholds, and potential differences in injury thresholds between individuals result in a fairly large range of the reported thresholds. Approximately 30 reconstructed NFL impacts using Hybrid III dummies produced the following injury threshold values representing 50% risk of MTBI: 79 g for peak linear acceleration, 5757 rad/s^2 for peak angular acceleration, 250 for HIC, and 300 for SI [33-35]. Funk et al. [51] analyzed head acceleration data from over 27,000 impacts collected from instrumented collegiate football players and reported the following injury threshold values representing 10% risk

of MTBI: 165 g for peak linear acceleration, 9000 rad/s^2 peak angular acceleration, and 400 for HIC. Focusing on rotational kinematics, Ommaya [30] reported 4500 rad/s^2 with a change in angular velocity below 30 rad/s as an injury threshold for rotation about a horizontal axis in the frontal plane that passes through the center of mass of the head. It is also possible that these single value acceleration thresholds may not be the best predictors of MTBI. Greenwald et al. [52] showed that a composite variable of head kinematics may be a better predictors of MTBI than any single value. Other studies have shown that using computer models to predict the response of the brain itself to head acceleration, rather than global measures of head acceleration, provided improved injury prediction [35, 53, 54].

Without a catcher's mask, the highest head accelerations measured in the present study (peak linear acceleration = 180 g; peak angular acceleration = 25700 rad/sec^2) exceeded injury thresholds from NFL reconstructed impacts (peak linear acceleration = 79 g; peak angular acceleration = 5757 rad/s^2) [33, 35]. However, HIC and SI values from these same trials (HIC = 181; SI = 210) did not exceed injury thresholds from these same studies (HIC = 250; SI = 300). With a catcher's mask, head accelerations measured in the present study (linear acceleration = 30 g; angular acceleration = 3230 rad/s^2; HIC = 13; SI = 15) were substantially lower than the injury thresholds from NFL reconstructed impacts, or any other thresholds to our knowledge. This would suggest a low risk of MTBI upon frontal impact with a baseball while wearing a mask. Evidence shows, however, that catchers sustain concussions while wearing a mask. This apparent discrepancy may result from the fact that a direct comparison of injury thresholds from the literature to the accelerations measured here is not straightforward. First, although

the above noted injury thresholds have been reported, MTBIs have occurred with linear and angular accelerations as low as 48 g and 3476 rad/s^2 [33]. Concussions in baseball may occur on the lower end of human head acceleration tolerance, perhaps due a cumulative effect of multiple impacts. Second, differences in measured head acceleration between various headforms might lead to different injury thresholds. Heald and Pass [38] compared the Severity Index upon baseball impacts to the side of the head at 28.6 m/s (60 mph) using cadavers (SI = 2187), the humanoid head model used by NOCSAE for equipment certification (SI = 2300), and the Hybrid III (SI = 490). These results showed Hybrid III values to be an order of magnitude lower than those measured in a cadaver and humanoid head model. Though the Hybrid III head may have limited biofidelity, it considered the gold standard in the automotive industry for cranial impact response and is commonly used in sports biomechanics applications [55]. It should also be noted that Pellman et al [33] used the Hybrid III for his NFL study and had the same impact duration of 14 ms as Rowson et al [56]. As such, a direct comparison between these studies and the current study is possible. Third, brain injury is thought to be a function of both acceleration magnitude and duration [57]. Baseballs impacting a headform have a duration on the order of 3-4 ms; while the football impacts, for example, have an impact duration on the order of 15 ms. When comparing the values presented in this study for baseball impacts to other types of head impacts, it is important to make note of the impact duration.

Head accelerations measured during athletic activities provide a useful basis for comparison with head accelerations measured here. During collegiate football practice and games, 172 out of 1712 measured impacts involved linear acceleration of the head

greater than 40 g (average 22.3 g, median 17.5 g), and 143 impacts involved angular acceleration of the head greater than 3000 rad/s^2 (average 1355 rad/s^2, median 1017 rad/s^2) [56]. During soccer, 25-26 year old experienced soccer players heading a soccer ball experienced mean (± standard deviation) translational and rotational accelerations of 16.1 ± 1.9 g and 1302 ± 324 rad/s^2,respectively. The range of HIC values was 10 – 18, SI values was 12 – 21, and impact duration was an average of 25 ms [37]. During collegiate hockey practice and games with a helmet, 95 % of all the impacts resulted in peak translational and angular accelerations of the head of less than 50 g and 5183 rad/s^2 respectively. The highest reported peak linear resultant acceleration (150 g) and peak angular resultant acceleration (15000 rad/s^2) represented less than 1% of all impacts [58]. It should be noted that these measurements were collected from the head of athletes during athletic activities, not collected during a simulated athletic scenario with an anthropometric dummy as performed here.

Several limitations of our methodology to investigate the effect of catcher's masks on head accelerations in a Hybrid III dummy warrant discussion. First, only one ball impact location was investigated. This location was selected because it was near the center of the mask (to minimize glancing blows on the mask for maximum energy transfer to the head/mask) and was associated with an easily identifiable anatomical landmark (tip of the nose). It is likely that the head acceleration upon impact and the ability of the catcher's mask to attenuate the impact are both highly sensitive to the ball impact location. Additional work is needed to more fully understand the effect of these variables. Second, only ball speeds 60-80 mph were investigated. Head acceleration and the ability of catcher's masks to attenuate head acceleration are dependent upon ball

speed. As such, caution should be used when generalizing these results to ball speeds above 80 mph. Third, there was insufficient data to investigate differences in impact attenuation between one-piece and two-piece masks or between masks from different manufacturers. While all masks substantially attenuated head accelerations to levels well below current injury thresholds, recognizing which types of masks and mask characteristics best attenuate head accelerations could be useful in improving safety equipment.

In conclusion, catcher's masks reduced head acceleration metrics by approximately 85% when baseballs were impacted with the head of a Hybrid III anthropometric dummy. Head accelerations upon impact with a baseball with a catcher's mask were significantly lower than contemporary injury thresholds, yet evidence indicates that baseball impacts to the mask still result in MTBIs. This work provides a foundation of knowledge for future research aimed at understanding the cause of these injuries, improving safety equipment, and ultimately reducing the occurrence of MTBI in catchers and umpires due to facial impact with a baseball.

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Appendix A

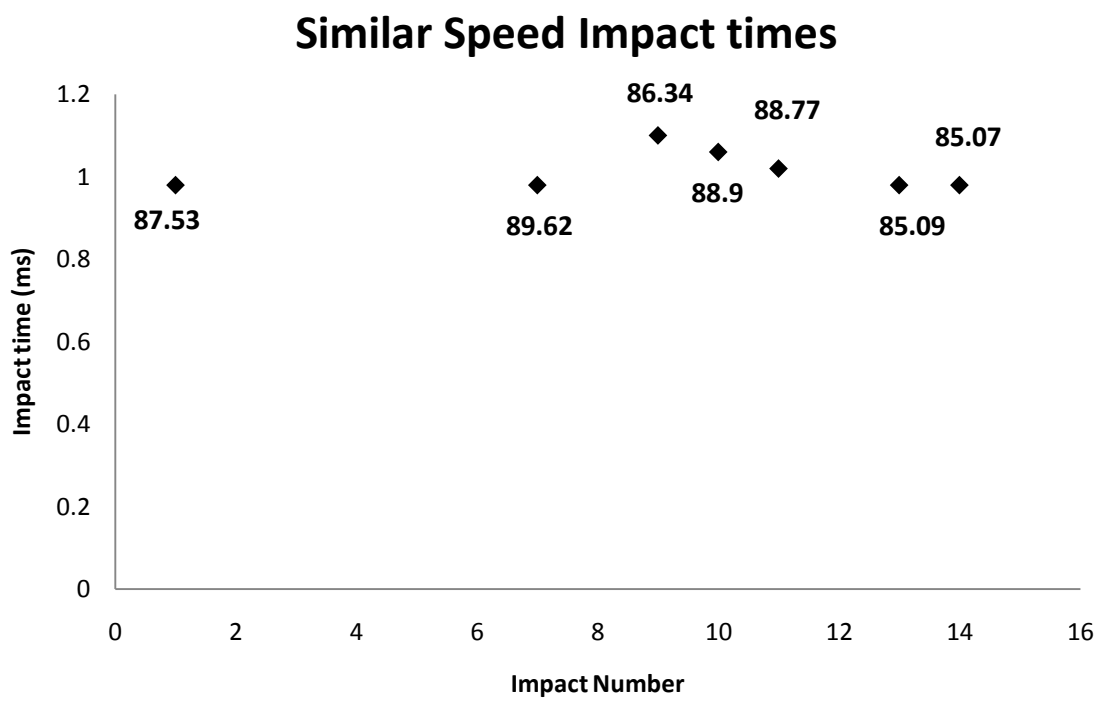

Figure 14. Shows the impact time in ms for a given impact number. Only impacts with a speed of 87.3 ± 1.9 mph were included.

To determine how many baseballs were needed for experimentation, a single NCAA regulation baseball was repeatedly shot at a wall-mounted force plate. Impact time, i.e. the time that the baseball was in contact with the force plate, was determined from the force recorded by force plate during each trial. Impact time versus impact number for similar speed impacts (Figure 14) shows that there was no substantial change in impact time as the impact number on a single baseball increased. These results give evidence that there is no substantial change baseball deformation as the impact number increases. It was determined from these results that one NCAA baseball per mask could be used.