CHAPTER 3

THE BIOMECHANICS AND PATHOMECHANICS OF SPORT-RELATED CONCUSSION

Looking at History to Build the Future

Kevin M. Guskiewicz, Jason P. Mihalik

Sports Medicine Research Laboratory, The University of North Carolina, CB #8700 Fetzer Gymnasium, South Road, Chapel Hill, NC 27599

Abstract: Sport-related concussion is still considered by many as a hidden epidemic in sports medicine. Despite the fact that this condition is not visible by neuroimaging, current research has allowed clinicians to better understand the condition. This chapter will discuss sport-related concussion in the context of the biomechanics and pathomechanics involved with injury. We will further explore how historical studies of concussion-related biomechanics research have paved the way for more novel, technologically advanced mechanisms by which head injury mechanics can be studied.

Key words: Acceleration-deceleration; Biomechanics; Concussion; Diffuse axonal injury; Focal injury; Mild head injury; Mild traumatic brain injury; Neuropsychology; Pathomechanics; Pathophysiology; Second impact syndrome.

1. INTRODUCTION

Sport-related concussion is a multifactorial disorder and, unlike severe head injury, the pathophysiology is less well understood. Sport-related concussion has received more attention in the medical literature since 2000, than it had in the previous 30 years combined. It has become a very popular topic within the medical community and lay media. It has also become an integral area of research for professionals in the fields of athletic training, biomechanics, and neuropsychology.

The work of Rimel et al. (1981) alerted the medical community to the high morbidity associated with mild head injury (MHI) and concussion. As a result, a much needed diagnostic criteria for defining MHI was developed. Second Impact Syndrome (SIS), as described by Saunders and Harbaugh (1984) and Cantu (1992), captured the attention of practitioners involved in

the management of sport-related concussion; SIS has continued to be an issue in managing sport-related concussion over the last 25 years.

A standard definition of mild head injury has gradually evolved and currently includes four related criteria: 1) Cranial trauma resulting in impairment of consciousness for 20 minutes or less, 2) Glasgow coma score between 13 and 15, 3) Hospitalization not exceeding 48 hours, and 4) Negative findings on neuroimaging (Levin, 1994; Rimel, Giordani, Barth, Boll, & Jane 1981). Most practitioners refer to mild head injury or mild traumatic brain injury as a concussion.

The term *cerebral concussion* is often interchanged with MHI when describing head injuries sustained in sport. It is best classified as a mild diffuse injury involving an acceleration-deceleration mechanism in which a blow to the head or the head striking an object results in one or more of the following conditions: headache, nausea, vomiting, dizziness, balance problems, feeling "slowed down," fatigue, trouble sleeping, drowsiness, sensitivity to light or noise, loss of consciousness (LOC), blurred vision, difficulty remembering, or difficulty concentrating (Guskiewicz, 2004; Practice Parameter, 1997). In 1966, the Congress of Neurological Surgeons proposed the following consensus definition of concussion, subsequently endorsed by a variety of medical associations: "Concussion is a clinical syndrome characterized by immediate and transient impairment of neural functions, such as alteration of consciousness, disturbance of vision, equilibrium, etc, due to mechanical forces" (Congress of Neurological Surgeons, 1966). Although the definition received widespread consensus in 1966, a more contemporary opinion (as concluded at the First International Symposium on Concussion in Sport, Vienna, 2001) was that this definition fails to include many of the predominant clinical features of concussion, such as headache and nausea. It is often reported that there is no universal agreement on the standard definition or nature of concussion; however, agreement does exist on several features that incorporate clinical, pathological, and biomechanical injury constructs associated with head injury:

1. Concussion may be caused by a direct blow to the head or elsewhere on the body from an "impulsive" force transmitted to the head.
2. Concussion may cause an immediate and short-lived impairment of neurological function.
3. Concussion may cause neuropathologic changes; however, the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.

4. Concussion may cause a gradient of clinical syndromes that may or may not involve LOC; resolution of the clinical and cognitive symptoms typically follows a sequential course.
5. Concussion is most often associated with normal results on conventional neuroimaging studies (Aubry, 2002).

2. MECHANISMS AND PATHOPHYSIOLOGY OF MILD HEAD INJURY

Hugenholtz and Richard (1982) reported that concussion and MHI result from a blow to the head that is equivalent to a linear acceleration 80 to 90 times the force of gravity for more than 4 milliseconds. This force represents a force several times that which causes discomfort in a football player wearing a helmet. Research has consistently identified the mechanical factors responsible for producing concussion involves acceleration and deceleration (Chason, Hardy, Webster, & Gurdjian, 1958; Gennarelli, 1993; McIntosh & Vink, 1989), and/or rotation of the skull (Gennarelli, 1993; McIntosh & Vink, 1989). Gennarelli (1993) explained that the application of force to the head led to a complex series of mechanical and physiological events. Loading of the force is initiated either by static forces or dynamic forces. Most head injuries are a result of dynamic loading. A very brief insult is initiated either by a direct blow to the head (impact) or by a sudden movement of the head (impulsive) produced by impacts elsewhere. The latter may occur when sudden changes in the motion of a person's head occur during a car crash (i.e. a whiplash mechanism). Thus, biomechanical mechanisms of head injury can be divided into two categories: those related to head-contact injuries and those related to head-movement injuries.

Contact loading occurs directly as a result of an impact on the head itself; whereas, inertial loading results from head motion generated by either impact or impulsive forces. Contact loading can lead to local skull bending, volume changes, and propagation of shock waves. Translation, rotation and angulation, may all result from inertial loading. This is dependent, however, on the direction, speed, and duration of head movement, as well as the manner in which it moves (Gennarelli, 1993). Fig. 1 illustrates how

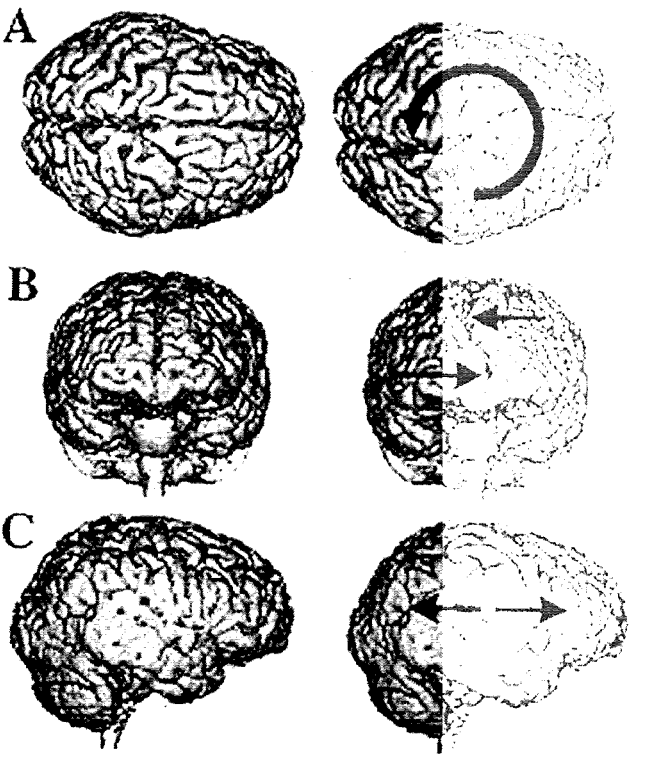

Fig. 1. Impacts to the head may cause rotational (A), shearing (B), or compressive (C) forces to act on the brain. It is not uncommon for a combination of these movements to occur.

different types of head movements can cause compression, shear, and/or rotation of the brain (Bigler, 1993).

Scott (1940) was the first to demonstrate that the principle factor in the production of concussion in animals was the sudden increase in intracranial pressure that accompanies head injury. According to Salazar (1994), mild traumatic brain injury (TBI) is not simply a faint reflection of moderate and severe injury. Not all of the brain damage attributable to head injury occurs at the moment of impact, and *immediate impact injury* (i.e. `primary' brain damage) may only be the start of an evolving process. Mild TBI is a dynamic process very similar to that which occurs in injury to soft tissue of the musculoskeletal system. Although the pathophysiologic factors mediating brain damage are poorly understood, it is now generally believed that MHI can produce irreversible brain damage (Mcintosh & Vink, 1989).

Several pathological components have been identified and discussed in the literature; however, MHI is usually the result of either *focal injury*, or

diffuse injury. Focal injuries are cerebral contusions or hematomas that form under the site of impact and thus result in focal neurological deficits referable to that area (i.e. aphasia, hemiparesis). The most common locations for contusions after acceleration-deceleration injuries are in the orbitofrontal and anterior temporal lobes, where the brain lies next to bony edges. Thus, behavioral and cognitive abnormalities referable to the frontal and temporal lobes are often seen (Gennarelli, 1993; Salazar, 1994).

Diffuse injuries are associated with widespread or global disruption of neurological function and are not typically associated with macroscopically visible brain lesions. These injuries result from shaking of the brain within the skull, and thus are lesions caused by the inertial or accelerative effects of a mechanical input to the head (Bruno, Gennarelli, & Torg, 1987). This very broad group of diffuse brain injuries includes all those injuries not associated with focal lesions. They typically include: 1) mild through severe concussion, and 2) diffuse axonal injury (DAI), which involves prolonged traumatic brain coma with LOC lasting more than six hours due to shearing of the axons and disruption of axonal flow. According to Levin (1994, 1988, 1979), results from animal models with DAI suggest that injury occurs along a continuum and that DAI may be involved in many cases of MHI.

It has been established that following severe traumatic injury to the brain, secondary or delayed injury mechanisms such as brain swelling, hematoma, elevations in intracranial pressure, hypoxia, and vasospasm, are initiated; these may result in further tissue damage (Cooper, 1985; McIntosh & Vink, 1989). Brain damage secondary to increased intracranial pressure often leads to secondary complications as described by Adams and Graham (1972) and Adams (1975). The pathology of head injury continues to evolve over the first few hours and days after trauma, often with devastating secondary injury in more severe cases; however, the physiological and clinical aspects of the recovery process itself can continue for some time.

A series of physiological, vascular, and biochemical events is set in motion in injured tissue following head trauma. These include changes in arachidonic acid metabolites such as the prostaglandins and leukotrienes, the formation of oxygen-free radicals and lipid peroxidation, and changes in neuropeptides and neurotransmitters. These products can result in progressive secondary injury to otherwise viable brain tissue through a number of mechanisms such as altering the vascular activity and producing further ischemia or by causing increased brain swelling (Salazar, 1994). Metabolic alterations, including reductions in brain intracellular pH, increased cerebrospinal fluid (CSF) lactate concentrations (McIntosh & Vink, 1989), and decreased intracellular free magnesium concentrations

(Vink, McIntosh, & Demediuk, 1988), have been reported in clinical studies of mild to moderate head trauma. In many cases, the alterations resulted in irreversible brain damage. More recently, a cascade of neurochemical, ionic, and metabolic changes has been described following brain injury. Some areas of the brain have shown glycolytic increases, a state of metabolic depression from decreases in both glucose and oxidative metabolism, and a reduction in cerebral blood flow (Hovda, 1991; Giza, 2001). In terms of MHI, complications similar to those of severe head injury have been documented (Alves & Jane, 1985), and it is agreed that in order to interrupt the cycle of events that accompany head injury, removal from activity until symptoms subside is paramount (Cantu, 1986, 1992; Hugenholtz & Richard, 1982; Saunders & Harbaugh, 1984).

3. SPORT-RELATED CONCUSSION AS IT RELATES TO NEWTONIAN PHYSICS

Understanding the biomechanics of sport-related concussion involves consideration of several factors, but none more than that of *acceleration* and *deceleration* of the brain housed within its protective capsule – the cranium. The forces imparted to the brain are a result of the head's rapid deceleration from impacting a stationary force (e.g., the playing surface) or opposing force (e.g., opposing player making a tackle). Fundamental Newtonian physics may serve as a starting point for understanding the acceleration-deceleration mechanisms associated with sport-related concussion. These formulas can assist in better understanding the stresses and strains applied to the head and, ultimately, the brain tissue during these acceleration-deceleration head injuries. Deceleration must follow acceleration in the context of a head impact; therefore, it should be the key issue when considering the forces directed to the brain. Deceleration can be viewed as negative acceleration or a decrease in velocity over time. The formula for calculating acceleration or deceleration is as follows:

$$a = (v^2\text{-}v_o^2)/2sg \qquad (1)$$

where a is acceleration or deceleration; v_o is initial speed in a given direction before deceleration starts; v is the directional speed at the end of deceleration; and s is the distance traveled during deceleration. The use of g in Eq. (1) allows for the expression of results in terms of multiples of acceleration due to gravity or g force. One g force is equivalent to 9.812

m/s^2 (10.73 yd/s^2). Since v in a sports acceleration-deceleration model is generally calculated as zero, because the player is presumably brought to a halt, the formula can be simplified to the following (Varney, 1995; Barth, 2001):

$$a = -v_o^2/2sg \tag{2}$$

An interaction of several other factors such as mass, weight, hardness and surface area of the impacting object also play a role in determining the extent of the injury. This brings us to consider Newton's Second Law of Motion:

$$F = ma \tag{3}$$

As Barth (2001) points out, if a is nothing but the acceleration of gravity (1 g) or, for example, a player falling to the ground with no other forces acting on them, then Eq. (3) can be rewritten as the following:

$$F = mg \tag{4}$$

Thus, if a football player's head experiences an acceleration of 25 g, which approximates the average peak acceleration of players in our recent study of head impact biomechanics, the force on the brain is 25 times the force of what it would experience from gravity alone. The question being asked by many sport concussion researchers is: What is the tolerance level for brain tissue in terms of these biomechanical factors?

4. HISTORICAL BACKGROUND OF BIOMECHANICAL CONCUSSION RESEARCH

Concussion has been recounted in historical texts dating as far back as Hippocrates' precepts written circa 415 B.C. Dating even further in history is the biblical account of how David rendered Goliath unconscious with a rock from his sling shot. There have also been many accounts of work by medieval surgeons in this area. Despite the long-standing awareness of concussion as a medical condition, it was not until the end of the 18th century that medical professionals had enough information to generate a more refined working definition. As time progressed, neurologists became

increasingly concerned that observable bouts of severe paralysis of neural function could occur with no obvious signs of physical trauma.

It was not until the beginning of the 20th century, however, that considerable work was undertaken to begin modeling brain injuries in a biomechanical sense. This work was pioneered by Denny-Brown and Russell in 1941, when they continued the development of animal models as they related to mechanical brain injuries. They were also among the first to provide a number of theories in an attempt to explain what was occurring at the level of the brain. These theories included a wide range of domains including mechanical, molecular, and vascular hypotheses. Although their research revolved around general concepts of concussion and providing a more comprehensive understanding of the condition, their chief interest was investigating the biomechanics of concussion. Cats, for the most part, were concussed by a device that struck the posterior aspect of their heads with a pendulum-like motion. Although other investigators during their time were conducting similar research, Denny-Brown and Russell's research differed in that they inflicted their head impacts while the animals' heads were free to move, as opposed to secured to a hard surface (i.e. countertop). Further to this revolutionary approach of investigating the biomechanics of head injury, Denny-Brown and Russell were the first to describe the appearance of head trauma in relation to a sudden change in velocity; the term acceleration-deceleration injuries arose from their initial research. Although Denny-Brown and Russell have been credited with a lot of the initial work in this field, it is important to note that their studies do not come without a number of issues. The main issue stems from the fact that they anaesthetized their animals prior to impacting them with their pendulum hammer device and, as such, level of consciousness could not be directly assessed (Symonds, 1962). Although the work by Denny-Brown and Russell paved the way for future research by identifying the role that head movements had on a potentially injurious blow to the head, it was Holbourn who more accurately defined the biomechanics of head trauma.

Interestingly, Holbourn did not use animals in his studies. Instead, he constructed models of the cranium and brain; these models consisted of a wax skull filled with a gelatinous structure that represented the human brain. He then subjected these models to differing impacts, and ultimately concluded that a brain's resistance to compression was not matched to its ability to resist deformation. This confirmed his hypotheses that rotational movements within the brain were necessary to produce cortical lesions and very likely concussion. He was unable to assess the latter, as he performed his testing on physical models with the inability to collect subjective and

objective information following the infliction of impacts. This further supported his hypothesis that linear forces played no major role in the shearing forces required to sustain any amount of concussion and would more likely result in the types of injuries associated with closed head injuries (i.e. subdural hemorrhage).

The next critical step in the development of concussive impact biomechanical analyses was the work performed by Pudenz and Shelden in 1946. In their studies, Pudenz and Shelden used monkeys as test subjects. They removed the top half of the monkeys' skulls, and replaced them with a transparent plastic dome. They then imparted an accelerative impact and using high-speed cinematography, captured the motion of the brain's surface. They concluded that due to the brain's relatively low inertia, it was unable to "keep up" with the movement of the skull. These projects have been the pioneering studies for many quantitative investigations of head injury biomechanics.

The degree of complexity in quantifying the biomechanics of head injury has led some to question whether a comprehensive understanding of the dynamics of head injury could ever be achieved (Shetter & Demakas, 1979). The diversity of head and brain injury mechanisms all involve a near instant transfer of kinetic energy which requires either an absorption (acceleration) or release (deceleration). Although force is the product of mass and acceleration, little trade-off occurs between the two. For example, a high velocity bullet may penetrate the skull and brain but not cause a concussion since the mass of the bullet is too small to impart the necessary kinetic energy to the head and brain (Gurdjian, Lissner, Webster, Latimer, & Haddad, 1954). Although the overall force is the same in both conditions, if the head is struck by a somewhat larger projectile than the bullet (but one that is traveling at a lower speed), MHI may now ensue.

Another property of kinetic energy follows that if an athlete's head is not mobile or is in contact with a wall or other surface, the kinetic forces imparted on the head and brain will travel through it and be transmitted elsewhere, often leaving brain function intact. In football, an athlete may tense his neck muscles prior to collision to decrease the mobility of the head and, therefore, allow for the kinetic energy to be dispersed throughout the rest of the body (Cantu, 1992). This leads to the suggestion that athletes that are blindsided and not given sufficient time to prepare for the collision are more likely to experience concussive blows to the brain.

The brain can also be injured by acceleration or deceleration mechanisms. In either case, the end result is one caused by impact or impulse. An impact injury occurs when a direct blow is made with the head.

An impulse injury causes an accelerative or decelerative force, setting the head in motion, without directly contacting the head. Impulse injuries are best suited for biomechanical reconstructions of accelerative or decelerative MHI since there is no contamination by impact mechanics (Ommaya & Gennarelli, 1974). Regardless of whether the injury occurred via impact or impulse forces, the severity of the MHI is often related to the accelerative forces exerted on the brain. These forces are identified as linear or translational acceleration or deceleration; and angular or rotational acceleration or deceleration. Translation of the brain may be defined as movement in a straight line through the brain's center of mass (Shaw, 2002). In terms of athletics, this concussive mechanism of injury would be observed as a direct blow to the face. Rotation of the brain occurs when the head accelerates on an arc about its center of gravity. This mechanism is elicited by a contact such as the uppercut in boxing.

The contribution of the translational and rotational accelerative forces to the concussive insult remains a topic of divergence. In terms of sport-related concussion, it is accepted that a combination of the two accelerative forces play a role in the concussive injury (Shetter & Demakas, 1979). Although both accelerative forces are passed on the brain when imparted on the head, studies on primates have shown that it is only the rotational forces that invoke a loss of consciousness (Ommaya & Gennarelli, 1974); whereas, translational forces are more likely to result in contusion or hemorrhage.

5. HISTORICAL PERSPECTIVE OF HELMET-RELATED RESEARCH

In an attempt to reduce the amount of forces imparted on the head during contact sports such as American football and ice hockey, helmet manufacturers have sought to redesign their respective equipment. In the 1970s, studies were developed that implemented the use of instrumented suspension-type football helmets. These studies used frequency-based modulated accelerometers (Moon et al., 1971; Reid et al., 1971). The findings of these studies were questioned in Morrison's dissertation work (1983). Although his work was limited by a relatively small sample size, he used a similar system to the Moon and Reid studies, and examined whether the accelerometers were assessing movement of the helmet itself or movement of the head. In 2000, Rosanne Naunheim and colleagues compared impact data in ice hockey, football, and soccer. Her study, although the first of its kind to investigate the nature of head accelerations in

competition, was limited to 4 subjects: one ice hockey defenseman, one football offensive lineman and one defensive lineman, and one soccer player. To further question the results, the soccer player was fitted with a football helmet and asked to simulate heading tasks. Naunheim et al. found that the soccer player exhibited significantly higher accelerative forces than the ice hockey player and the football players. Furthermore, she also identified higher magnitudes of acceleration in the ice hockey player compared to the two linemen. The unrealism of having a soccer player perform a heading task while wearing a football helmet poses a significant limitation to her conclusions. Naunheim et al. discuss the limitation of having used two linemen who were more likely to sustain a number of repetitive low-level impacts as opposed to more dynamic positions such as running backs or receivers. Initial efforts have used video footage of on-field player impacts and recreated these impacts in controlled laboratory environments where these dynamics could be more closely investigated. In a study published in 2003, Pellman et al. used video surveillance and laboratory reconstruction of game impacts to evaluate the biomechanics of concussions sustained in the National Football League. They found that the peak head linear acceleration in concussion was 98 ± 28 g as compared to 60 ± 24 g for the uninjured struck players (Pellman, Viano, Tucker, Casson, & Waeckerle, 2003). Pellman et al. (2003) emphasized the need to develop new tests to assess the ability of helmets in reducing concussion risks. Current National Operating Committee on Standards for Athletic Equipment (NOCSAE) standards address impacts to the periphery and crown of the helmet. Of relevant concern, out of 174 concussions analyzed in the Pellman et al. study, 51 resulted from impacts to the facemask.

The approach of analyzing video footage followed by laboratory impact reconstructions was employed by Biokinetics & Associates, an independent consulting firm in Ottawa, Ontario (Canada). One result of their study was the proposal to use a new Head Impact Power (HIP) index rather than the traditional criteria of Severity Index and peak *g*'s to quantify head impacts (Newman, Beusenberg et al., 2000; Newman et al., 1999; Newman, Shewchenko, & Welbourne, 2000). Severity Index is computed using only linear information; whereas, HIP takes into account rotational forces as well. Their studies resulted in the report of linear and rotational risk curves. Although a great addition to the current knowledge of head impacts, their analyses were limited to specific impacts retrospectively and not to real-time data. The Riddell Corporation (Elyria, OH, USA) funded the development of a laboratory test device which could best simulate the most severe on-field impacts. They have since designed the Revolution® helmet which they

claim is intended to reduce the risks of concussion. The University of Pittsburgh Medical Center (UPMC) Sports Concussion Program is currently comparing the neurocognitive performance of high school football players that have sustained a concussion while wearing the Revolution to those that wear traditional football helmets in attempt to justify the Riddell Corporation's claim. Preliminary data in this regard have yet to be published.

Despite how advanced laboratory testing has become, athletic environments offer a rich opportunity for collecting data on large numbers of head impacts. In order to do so, a mechanism by which large-scale data collection and real-time monitoring of head impacts in both practices as well as competitive events is critical to furthering our understanding of the pathomechanics of concussion. As technological advancements continue to progress in this regard, the ability to monitor head accelerations during all practice and game situations in football has been developed and validated by Simbex LLC. The Head Impact Telemetry System (HITS) has the ability to measure head acceleration and not helmet acceleration.* This technology is the first of its kind to enable prospective studies that combine biomechanical, clinical, and neuropsychological data in human subjects with sport-related concussion, allowing for direct measurement of injury parameters and their clinical consequences.

The HITS is comprised of six spring-loaded single-axis accelerometers. They are positioned in such a way that the data collected can be introduced into an algorithm, provided in Fig. 2, that is able to calculate head acceleration data. In order for head acceleration data to be recorded, the acceleration of any individual accelerometer must exceed a desired threshold; this threshold is usually set at 10 g. Once this occurs, information from the six accelerometers is collected at 1 kHz for a period of 40 ms; 8 ms are recorded prior to the data collection trigger and 32 ms of data are collected following the threshold trigger. Information from 100 separate head impacts can then be stored in non-volatile memory built into the accelerometer device (i.e. resides in the helmet proper). The collected data undergoes resident filtering to remove any DC offsets from the accelerometer signals. The data is then encoded, stored, and transmitted to a Sideline Controller (SC) via a radiofrequency telemetry link. The SC time

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* Head Impact Telemetry System was developed in part with funding thru the SBIR program from the National Center for Medical Rehabilitation Research in the National Institute for Child Health and Development (NICHD at the National Institutes for Health(NIH 2R44HD40473). The technology has been commercialized by Riddell Sports Group (Chicago, IL) as part of the Sideline Response System.

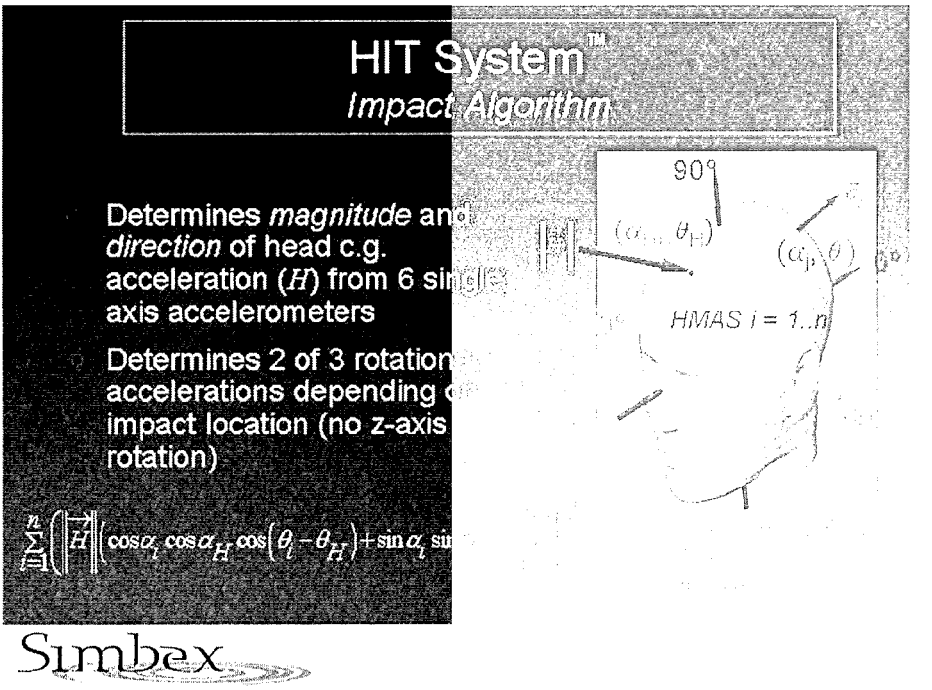

Fig. 2. The HITS uses an impact algorithm to determine the magnitude and direction of head center of gravity linear acceleration and rotational acceleration about 2 axes. The mathematical algorithm is also provided.

stamps the impact and computes standard measures of head acceleration such as linear acceleration, Gadd Severity Index (GSI), and Head Impact Criterion (HIC). The telemetry system is capable of transmitting accelerometer data from as many as 64 players over a distance well in excess of the length of a standard American football field. A screenshot of the Sideline Response System is depicted in Fig. 3.

A recent paper published by Duma et al. (2004) was based on work conducted at Virginia Polytechnic Institute and State University (Blacksburg, VA, USA) using the HITS. Eight football helmets were fitted with the HITS and were worn by 38 different players over the course of the 2003 season. Data from over 3,000 head impacts were recorded and researchers have tracked head accelerations in excess of 100 times the acceleration due to gravity (i.e. 100 g). Although this data has very interesting clinical implications, their study was simply descriptive in nature, and no conclusions to the clinical manifestations of these impacts were identified.

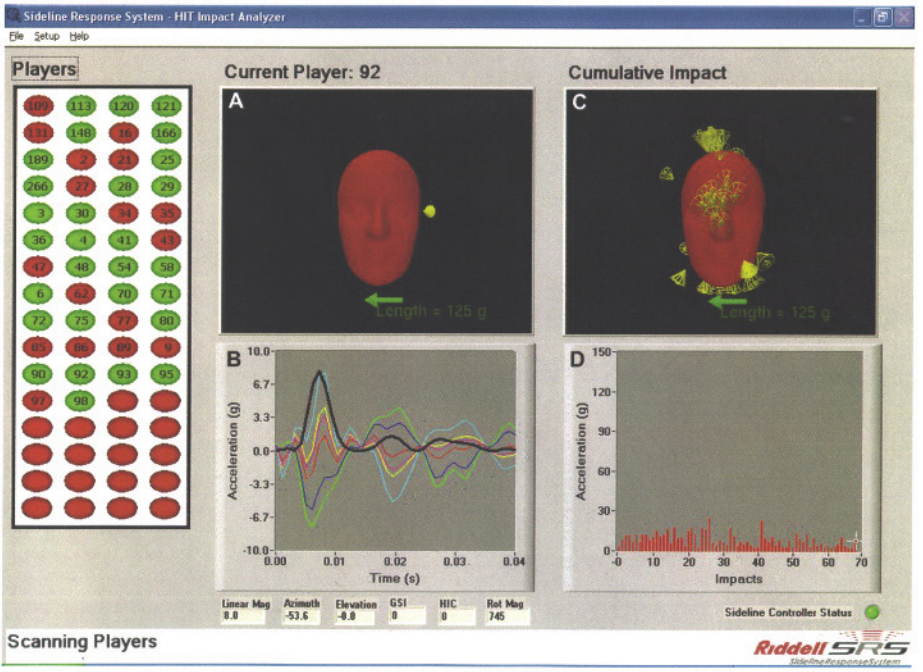

Fig. 3. The Sideline Response System depicts player identification numbers (left panel), as well as information pertaining to current impact location (A) and magnitude (B). The Sideline Response System also presents cumulative impact location (C) and magnitude (D) information.

The University of North Carolina (Chapel Hill, NC, USA) was awarded a grant from the Centers for Disease Control and Prevention to prospectively investigate how biomechanical, neuroanatomical, and clinical factors all relate to sport-related concussions. During the 2004 football season, 16 varsity football players were fitted with the helmet accelerometers at our institution. There were a total of 14,462 impacts collected over the course of the entire season, including the training camp. Although data from the 2004 season has yet to be published, preliminary findings suggest that there are both event (i.e. game versus practice) and positional differences. For example, offensive backs sustained the largest magnitudes of linear acceleration across the entire season. Surprisingly, defensive backs sustained a significantly lower magnitude of accelerations compared with defensive linemen and linebackers. Although our data suggest positional differences, we would like to emphasize that due to a limited sample size, we are unable to provide any more support with this data.

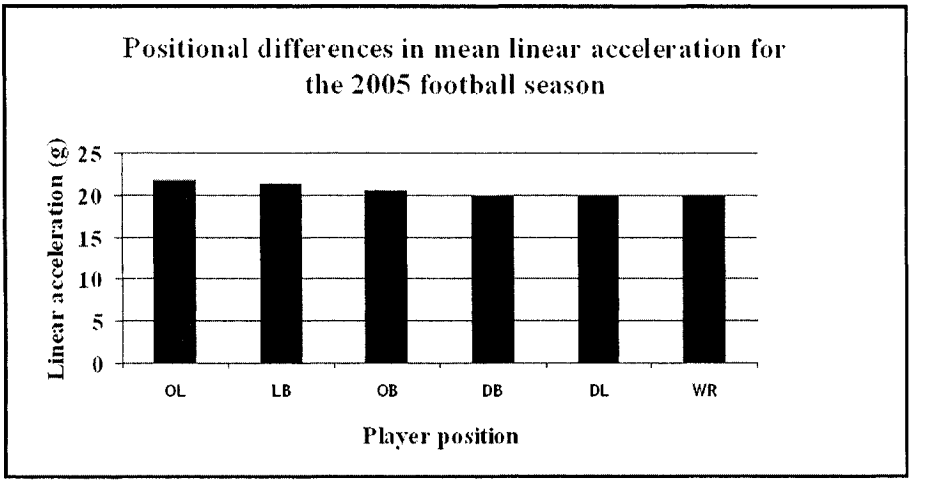

Fig. 4. Based on real-time data collection over an entire season, we observed that offensive linemen (OL), linebackers (LB), and offensive backs (OB) sustained higher magnitude impacts than defensive backs (DB), defensive linemen (DL), and wide receivers (WR).

The project was further expanded during the 2005 season, whereby 52 varsity football players were followed over the course of the season. A total of 46,342 head impacts were recorded throughout the entire 2005 football campaign; 27,057 head impacts were above 10 g. Our data continued to suggest event and positional differences. Interestingly, we found that the mean linear acceleration of the head was greater in helmets-only practices when compared to both full pad practices and games or scrimmages. This may indicate that the typical Division I collegiate football player does not have any "light" days when it comes to reducing the extent of head trauma they sustain. We also identified positional differences, which are represented in Fig. 4. We also found that offensive linemen, defensive backs, and linebackers, as a group, were almost 2 times more likely of sustaining an impact greater than 80 g when compared to defensive linemen. These numbers dropped to 1.57 and 1.25 when compared to offensive backs and wide receivers, respectively. Another interesting finding from the work performed in the 2005 season was the discovery that just over 20% of all the impacts to the head occurred at the top of the head. Furthermore, we also observed that football players were almost 7 times more likely to sustain an impact greater than 80 g to the top of the head than to the right or left sides of the head. They were also almost 4.5 and 2.5 times more likely of higher impacts to the top of the head than to the front and back, respectively. This data, represented by Fig. 5, is suggestive that collegiate football players still

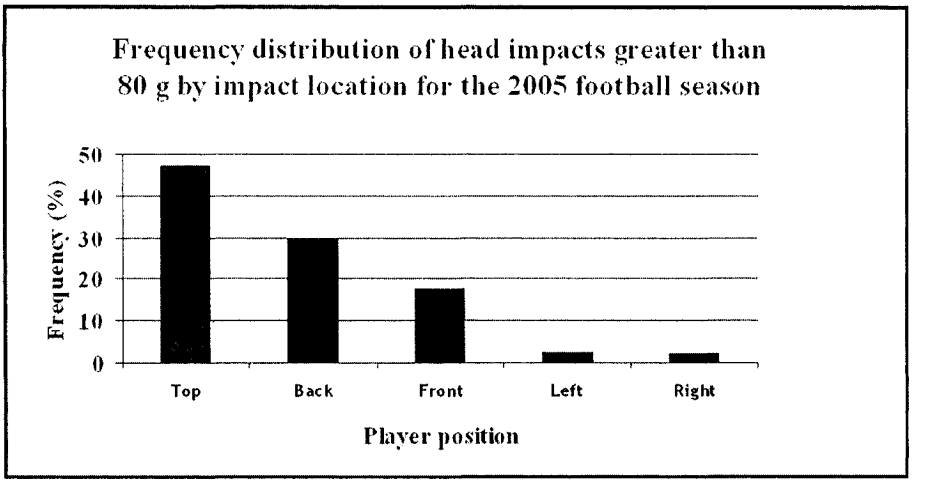

Fig. 5. Impacts occurring at the top of the head accounted for almost half of the 715 head impacts resulting in linear accelerations greater than 80 g. It was found that football players were almost 7, 4.5, and 2.5 times more likely to sustain an impact greater than 80 g to the top of the head than right or left sides, front, and back, respectively.

lead with the head, and these impacts are often significantly higher than those sustained to other areas of the head. Certified athletic trainers should continue to educate athletes on proper tackling techniques to decrease the risk of cervical injuries and concussions. Additional cases involving head and neck injuries will eventually help us to better understand the effect of impact magnitude and location on injury severity

Regardless of the role linear or rotational forces play on the results of the concussion, it is important that we recognize that the forces that are imparted on the head cause the brain to be set in motion. Methods of determining the extent of such forces have already been developed. Research in this regard has yet to be published, but results of such studies will lead to a better understanding of the pathomechanics behind MHI.

CONCLUSIONS

The study of impact biomechanics is leading researchers and clinicians to a better understanding of the mechanisms and forces that cause injury to the brain during sport-related activities. Technological advancements in research have allowed us to better predict the effects of linear and rotational

acceleration-deceleration on athletes sustaining sport-related concussion from both stationary forces (e.g., the playing surface) or opposing forces (e.g., opposing player making a tackle). Continued investigation in this area will help determine how higher impact collisions, as well as recurrent injury, affects threshold for future injury and recovery on clinical measures such as neuropsychological function and postural stability.

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