Head Impact Exposure Sustained by Football Players on Days of Diagnosed Concussion

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Abstract

Purpose—This study compares the frequency and severity of head impacts sustained by football players on days with and without diagnosed concussion and to identify the sensitivity and specificity of single impact severity measures to diagnosed injury.

Methods—1,208 players from eight collegiate and six high school football teams wore instrumented helmets to measure head impacts during all team sessions, of which 95 players were diagnosed with concussion. Eight players sustained two injuries and one three, providing 105

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injury cases. Measures of head kinematics (peak linear and rotational acceleration, Gadd Severity Index (GSI), Head Injury Criteria (HIC_{15}), change in head velocity (v)) and the number of head impacts sustained by individual players were compared between days with and without diagnosed concussion. Receiver operator characteristic curves were generated to evaluate the sensitivity and specificity of each kinematic measure to diagnosed concussion using only those impacts that directly preceded diagnosis.

Results—Players sustained a higher frequency of impacts and impacts with more severe kinematic properties on days of diagnosed concussion than on days without diagnosed concussion. Forty-five injury cases were immediately diagnosed following head impact. For these cases, peak linear acceleration and HIC_{15} were most sensitive to immediately diagnosed concussion (AUC = 0.983). Peak rotational acceleration was less sensitive to diagnosed injury than all other kinematic measures ($p = 0.01$) which are derived from linear acceleration (peak linear, HIC_{15}, GSI, and v).

Conclusions—Players sustain more impacts and impacts of higher severity on days of diagnosed concussion than on days without diagnosed concussion. Additionally, of historical measures of impact severity, those associated with peak linear acceleration are the best predictors of immediately diagnosed concussion.

Keywords

HIT System; Sport; impact biomechanics; MTBI; TBI; injury threshold

INTRODUCTION

In 1999, the National Institutes of Health Consensus Development Panel declared the incidence of mild traumatic brain injury (mTBI) had reached epidemic proportions and concluded that reducing incidence, severity, and post-injury symptomology should be a national priority.(37) Four years later, in response to the increasing number of diagnosed cases, the young age of the at-risk population, and the possibility of long-term disability from repetitive injury, the National Center for Injury Prevention and Control declared mTBI occurring in sports, which is commonly diagnosed as concussion, an important public health problem that required an increase in research, treatment, and prevention efforts.(8) Since that time, sports-related concussion has become a prominently discussed topic in academic, public, and government forums due to an ever-growing body of evidence that concussion history may lead to a higher likelihood of developing mild cognitive impairment, clinical depression, and early onset of Alzheimer's disease.(23, 24) It has even been hypothesized that the repetitive subconcussive head impacts sustained in contact sports (e.g., football, hockey, lacrosse, etc.),(3, 10–12) even in the absence of diagnosed concussion, may potentially lead to the deleterious effects of chronic traumatic encephalopathy (CTE), a degenerative brain disease with clinical presentation similar to amyotrophic lateral sclerosis (ALS) and Alzheimer's disease.(18)

Developing strategies for preventing concussion has been challenging, primarily due to the difficulty in determining the causal relationship between head kinematics and injury. Traditionally, laboratory reconstruction techniques using surrogates (i.e., cadaveric specimens, animal models, and anthropomorphic test devices) have been employed to

replicate the human response to impact.(21, 27, 38) Three primary limitations exist when trying to relate measures of head kinematics obtained from laboratory impacts to those experienced by athletes who are diagnosed with concussion in sports: 1) sports-related concussion is typically diagnosed by signs of neurological or neuropsychological dysfunction and self-reported symptomology which cannot be easily deduced from surrogates, 2) surrogate tests do not account for the complex system of intrinsic and extrinsic variables (e.g., contact force and direction, player physiology at time of impact, equipment condition, player anticipation, etc.) that influence kinematic response to impact, and 3) single impact events created in the laboratory may be an insufficient injury model considering impact and/or injury history may modulate an athlete's tolerance to impact. These variables, which may vary widely for a representative cohort, are impractical to obtain and difficult to replicate using traditional laboratory reconstruction.

To overcome these limitations, sporting fields, particularly those for contact sports, have been identified as living-laboratories to explore the human response to impact since athletes routinely sustain head contact during play.(35) One of the most impact-rich sporting environments is American-style football. In this sport, concussion is the third most common game injury with nearly 5% of all players diagnosed with concussion per season and 15% of those injured athletes diagnosed with multiple concussions in the same season.(13, 26) To leverage the large sample size, high frequency of impacts, and high potential for injury, Head Impact Telemetry (HIT) technology (Simbex, Lebanon NH) was developed to record head impact exposure (frequency, location, and kinematics of head impact) sustained during play.(9, 15) Using this technology, Duma et al. first reported the aggregate head impact exposure of 38 collegiate football players over a single season, one of whom sustained a concussion.(15) Since that time, the same general methodology has been used in numerous studies to quantify the kinematics of head impact across several athletic populations.(5, 10–12, 33, 41)

While the pathomechanics of head impact exposure leading to concussion diagnosis still remains unclear, several studies have provided preliminary insight into the relationship between on-field measures of head impact and diagnosed injury. Guskiewicz et al. first postulated,[18] and recently concluded,[17] that it may be difficult to identify a threshold for concussion after observing wide variation in head acceleration recorded prior to 13 cases of diagnosed concussion in collegiate football players. Greenwald et al. observed similar variation in head acceleration when examining 17 impacts associated with diagnosed concussion; however, they demonstrated that injury predictions based on measures of head impact exposure could be improved by combining impact location, peak head acceleration, and impact duration into a single, independent metric through the use of principal component analysis.(20) Similarly, Broglio et. al found measures of head acceleration prior to diagnosed concussion were similar between high school and collegiate football players, and a combination of linear acceleration, rotational acceleration, and impact location best differentiated 13 impacts associated with injury from head impacts not associated with injury.(5) Alternatively, Rowson et al, suggested the lack of specificity of these measures to injury was due, in part, to under-reporting and were able to develop injury risk curves using estimates of injury prevalence and both linear and rotational acceleration distributions for impacts associated with and without injury.(39, 40) They went on to show that risk curves

based on simple biomechanical parameters have a high level of power for predicting concussions when compared to epidemiological data.(16) While insightful, one significant limitation of these initial studies is the relatively small sample of recorded head impacts associated with diagnosed concussion used in their respective analyses.

To overcome sample size limitations, we have collectively pooled the injury cases recorded with helmets instrumented with HIT System technology across multiple institutions and studies.(5, 20, 25, 41) With this much larger retrospective data set, we aim to elucidate the biomechanical basis of mild traumatic brain injury in American-style football. The purpose of this study is to compare two components of head impact exposure (frequency and kinematic response) on days with and without diagnosed concussion and to identify the sensitivity and specificity of single impact severity measures to diagnosed injury. Specifically, we tested the hypotheses that individual players will sustain more impacts and impacts of higher severity on days of diagnosed concussion than on days without diagnosed concussion. Through this analysis, we begin the process of identifying components of head impact exposure that best correlate with diagnosed injury, which provides quantitative data from which protective equipment and safety standards can be developed and could potentially lead to both rule changes to mitigate at risk behavior and new methods for identifying impaired athletes who, using current gold-standard methods, go undiagnosed.

PARTICIPANTS AND METHODS

Participants

Over a six year period (2005 – 2010), 1,208 players from eight collegiate and six high school football teams wore instrumented helmets (Head Impact Telemetry (HIT) System, Simbex, Lebanon, NH) to measure head impacts during practices, games and scrimmages – designated as team sessions. Yearly participation by each organization, as well as inclusion for each individual within the organization, was voluntary with no consideration given to a player's previous history of concussion or playing position. Because instrumentation was only available for Riddell helmets, participation was limited to subjects already wearing VSR-4 (24%), Revolution (72%), or Speed (4%) helmet models. A total of 673 players were instrumented during multiple years providing a yearly subject pool of 230 players from 6 teams in 2005, 330 and 550 players from 11 teams in 2006 and 2007, and 422, 426, and 352 players from 8 teams in years 2008–2010. At all institutions participating in the research, approval for data collection and reduction was received by an Institutional Review Board and informed consent was obtained, including parental consent in the case of minors.

Helmet Instrumentation

Instrumented helmets were used to continuously monitor the head during all competitive activity and record head acceleration in real-time following impact. The HIT System is comprised of an in-helmet data acquisition system, a sideline transceiver, and a laptop computer.(10, 15) The in-helmet unit positions six, single-axis accelerometers (Analog Devices, MA) against a player's head providing isolated head acceleration measures.(28) Prior to use, all helmet model and size (M, L, XL) combinations were tested to meet on-field use requirements that included meeting standards set by the National Operating Committee

for Standards of Athletic Equipment. During play, when any accelerometer exceeded a 14.4g threshold, 40 ms of data were recorded [8 pre-trigger and 32 post-trigger, 10 bit, 1000 Hz per channel], time stamped, and transmitted wirelessly (903–927 MHz) to a transceiver and laptop computer positioned on the sideline. Communication range typically exceeds 200 yards; however, in the event of poor communication, each unit was capable of storing up to 100 impacts in non-volatile memory to minimize the potential for data loss. To verify the accuracy of on-field data collection, processing, and reduction, a multi-phase validation process was conducted which included laboratory testing,(2, 9, 15, 28) video correlation of on-field events,(7, 15, 34) and multi-site field trials.(5, 10–12, 15, 33, 41)

Clinical Diagnosis

During the period of study, concussion was generally defined as an alteration in mental status, as reported or observed by the player or team's medical staff, resulting from a blow to the head which may or may not have involved loss of consciousness. For all cases of injury, a certified athletic trainer (ATC) or team physician at each respective institution diagnosed and treated the injury at their professional discretion. Following symptom resolution, the medical staff provided the date of injury, the suspected time of injury, the approximate time of diagnosis, day of symptom resolution, and player anthropometrics (age, height, and weight). Additionally, anecdotal descriptions of the events surrounding injury (e.g., description of the impact, method of identifying the injury, and on-field observations regarding clinical presentation) were provided by each team when available.

Data Reduction

Impact location and linear and rotational acceleration of the head center of gravity (CG) were computed for each impact from acceleration data collected with the instrumented helmets.(9, 40) Events recorded outside of an organized team session (practice, scrimmage, or game) or with peak linear acceleration below 10 g were removed prior to analysis, as these were considered to be outside of the measurement range of the device. (10, 33, 36) Other identified non-head impact related events, such as throwing a helmet, were also removed from the dataset.

From the processed acceleration data, measures of impact kinematics available for analysis included the peak magnitude of linear and rotational acceleration and three additional metrics calculated from the linear acceleration time series data: Gadd Severity Index (GSI), (17) Head Injury Criteria (HIC_{15}),(43) and change in head velocity (v). Additionally, the total number of daily head impacts sustained and the number of head impacts above the 50th and 95th percentile values of peak linear and rotational acceleration for all players were calculated. The percentile cutoff values for peak linear acceleration (50th = 20.5 g, 95th = 62.7 g) and peak angular acceleration (50th = 981 rad/s^2, 95th = 2,975 rad/s^2) were previously reported by Crisco et al(11) and Rowson et al,(40) respectively.

Statistical Analysis

Only impacts sustained by athletes diagnosed with concussion during the period of study were considered for the purposes of this analysis (Figure 1). For each kinematic measure, the median (50th percentile) and 95th percentile levels for these individual players on days

with and without diagnosed concussion were calculated. Results were expressed as median values and 25–75% interquartile range. A Wilcoxon signed-rank test for matched pairs was used to test the significance between the 50th and 95th individual player percentiles on days with and without diagnosed injury because the study variables were not normally distributed (Lilliefors test; p < 0.001). Similarly, distributions of impact frequency were skewed towards lower occurrence (Lilliefors test; p < 0.001), so the same method was used to test the hypothesis that a greater number of impacts per player occur on days of diagnosed concussion than those without diagnosis. This analysis was performed using all impacts as well as only with impacts greater than the 50th and 95th percentile acceleration levels to determine if differences exist even when only considering the highest magnitude impacts.

Receiver Operator Characteristic (ROC) curves were generated to evaluate the sensitivity and specificity of single impact severity measures to diagnosis of concussion. Impacts recorded immediately before a player was removed from participation and diagnosed with concussion were defined as immediate diagnosis impacts and used as positive ROC input. Negative ROC input cases were designated as all impacts for the concussed players occurring on days without diagnosis of concussion. For each ROC curve, the null hypothesis of the true area under the curve (AUC) equaling 0.5 (same as guessing), was tested and an asymptotic significance value (*P* value) is reported. Hanley's method for comparing area under ROC curves was used to test if any of single impact severity measures were more sensitive to diagnosed concussion than peak linear acceleration.

Binary logistic regression was conducted to determine the odds ratios for concussion risk relative to incremental increases of each impact severity metric. This method determines how much the potential for diagnosed injury increases based on the measured severity and the presence or absence of clinically-defined injury following impact. Again, immediate diagnosis impacts were used as positive input into the analysis and all impacts sustained by concussed athletes on days without diagnosis was used as negative input. Results of this analysis include the regression coefficients (α, β), standard error of the regression coefficient, the Wald statistic used to test the significance of each regression coefficient, the odds ratio, and the 95% confidence interval of the odds ratio.

All statistical analyses described above were performed with custom Matlab scripts (version 7.11, The MathWorks Inc., Natick. MA) in combination with built-in statistical toolbox functions. A significance level of $\alpha = 0.05$ was set *a priori* for each of the statistical tests.

RESULTS

161,732 head impacts were recorded over 10,972 player days from 95 athletes clinically diagnosed with mTBI (Figure 1). Eight of the subjects sustained two diagnosed concussions and one had three, yielding 105 identified cases of injury. The median reported age, height, and weight of all concussed athletes was 19.2 ± 2.2 yr (15 – 23 yr), 183.5 ± 6.7 cm (165.1 ± 198.1 cm), and 94.6 ± 16.3 kg (63.5 – 138.8 kg) respectively. Collegiate athletes accounted for 68 of the diagnosed injuries with the remaining 37 sustained by high school players. Seventy of the cases (66.6%) occurred during games or scrimmages with the remainder occurring during practices The time of symptom resolution was reported for 89 of the 105

cases, and, of these, symptoms resolved in a mean of 5.9 ± 7.4 days (range: 15 min to 59 days) from the reported time of injury.

Kinematic measures for head impacts sustained on days with diagnosed concussion were higher than on days without diagnosed concussion (Table 1). Statistical significance was observed for both the 50th and 95th levels for all kinematic measures except 50th percentile rotational acceleration ($p = 0.08$; Table 1). On days when injury occurred, athletes also sustained a greater number of head impacts than on days when no injury was diagnosed (Figure 2). The difference was found to be significantly different when considering all impacts as well as those with peak linear acceleration greater than the 50th and 95th percentile of all impacts ($p < 0.001$; Table 2).

In 45 of the injury cases (43%), the player did not continue playing following an impact that directly preceded diagnosis of concussion. In the other 60 cases, the player was not immediately removed from play, and the diagnosis did not occur until either later that day or in the following days because the signs and symptoms of injury were either not immediately recognizable or the player did not self-report. These 60 cases of delayed diagnosis were excluded from both ROC and logistic regression analysis due to the potentially confounding factor of sustaining additional head impacts after onset of symptoms.

Impacts sustained prior to immediately diagnosed concussions had mean severity of 112.1 ± 35.4 g peak linear acceleration, $4,253 \pm 2,287$ rad/s^2 peak rotational acceleration, 321.5 ± 239.4 HIC$_{15}$, 439.3 ± 315.2 GSI, and 4.29 ± 1.71 m/s change in velocity. The area under the ROC curves generated for each severity measure ($0.921 – 0.983$) were statistically higher than 0.5 ($p < 0.001$), indicating that all measures of severity are better than guessing outcome of diagnosed concussion (Figure 3). Peak linear acceleration and HIC$_{15}$ were most sensitive to immediately diagnosed concussion (AUC = 0.983), but, this was not significantly different than either GSI (AUC = 0.982) or change in head velocity (AUC = 0.980). The only severity metric significantly different from peak linear acceleration was peak rotational acceleration, which had a lower sensitivity to immediately diagnosed concussion (AUC = 0.921; $p = 0.019$).

The odds ratios and associated 95% confidence intervals provided in Table 3 indicate the increase in odds of sustaining a diagnosed concussion for a single unit measure increase of each severity metric. For example, a 1g increase in linear acceleration corresponds to a 1.052 greater odds of sustaining an immediately diagnosed concussion, or, more practically, a player has 10.3 times greater odds of sustaining an immediately diagnosed concussion following a mean 95th percentile impact (84.9 g) than a mean 50th percentile impact (38.9 g) since the increase in odds equals the odds ratio raised to the power of change in a single unit measure (ex. odds increase = odds ratio $^{(\text{Top 95th} – \text{Top 50th})}$).

DISCUSSION

On days of injury, 95 athletes with one or more diagnosed concussions sustained impacts with higher associated kinematic response than on non-injury days. Because many individual factors (e.g., style of play, playing position, team tendencies, etc.) could influence

susceptibility to injury, it is interesting to note that on days without injury, these players sustained head impacts typical for all football players. The 50th and 95th percentile peak linear (20.7 g and 63.5 g) and rotational (848 rad/s^2 and 2,761 rad/s^2) accelerations recorded on non-injury days were nearly identical to those reported by Crisco et al. (20.5g and 62.7g) and Rowson et al (981 rad/s^2 and 2,975 rad/s^2) who employed similar methods to quantify the head impact exposure of collegiate football players who were not diagnosed with concussion from three collegiate football teams over three years. This comparison is especially compelling considering the injured athletes evaluated in this study came from a larger range of seasons, a higher number of teams, and is inclusive of both high school and collegiate players. Given the relatively large sample of injury cases presented in this analysis, it is clear that a significant distinction exists between kinematics sustained on days with concussion and other days of play. These in-vivo measures of head acceleration represent a foundation of quantitative data that can be used to develop future protective equipment and test standards for that equipment. Additionally, because head impact kinematics on days without concussion appear to be similar for all athletes (both those who were never diagnosed and those who were), the differences identified suggest that implementing a procedure to screen athletes for injury based on daily head impact exposure could lead to increased injury detection.

Similarly, and may be less intuitive because injured athletes are commonly removed from competition, athletes sustained more head impacts on days with diagnosed injury than on days without diagnosis. Athletes also sustained more impacts above the 50th and 95th percentile levels of peak linear and rotational acceleration on days of diagnosed concussion, with the median number of highest severity impacts ranging between 2.0 – 2.85 times higher on days of injury. These data indicate that players not only experienced more head impacts on days of diagnosed concussion, but they also sustained more high severity head impacts on these days. Previously, pilot studies have suggested a link between the number of head impacts sustained and in-season cognition;(1, 42) however, there has been little evidence suggesting that impact frequency is predictive of concussion. While it is still unclear if multiple impacts pre-dispose an athlete to injury (i.e. high number of impacts lowers a player's threshold of injury) or if the athlete simply has a higher risk of injury from a single event due to the higher number of impacts sustained, it is clear that the number of impacts a player sustains is a key measure to consider when evaluating the link between head impact and injury. This finding, combined with several previous studies showing impact frequency is related to several factors including team, playing position, skill level, and session type, (6, 10, 11, 33, 41) suggest that injury mitigation strategies, such as rule changes to limit head contact, can be developed to reduce the occurrence of concussion in sports.

In a 2007 report, Schnebel et al. provided a detailed description of two diagnosed cases of concussion and highlighted the difficulty of associating an injury diagnosis with a single impact.(41) Issues that confound this association include multiple impacts occurring within a short period of time, symptoms that either resolve quickly or only become pronounced over time, and, most importantly, the reliance on a player's self-report to initiate the medical evaluation. More than half of the diagnosed concussions reported in this study were not immediately identified by the team's medical staff, and, in the majority of cases, went undiagnosed until after play had ended. To mitigate uncertainty, we limited our analysis of

injury risk to only those impacts sustained immediately prior to diagnosis of concussion as positive cases and all impacts for those players sustained on days without diagnosis of concussion as negative cases. By focusing solely on impact events with clearly discernible outcome, risk estimates presented within this study most likely underestimate risk of sustaining any concussion due to the exclusion of cases with delayed diagnosis. In addition, it has been estimated that up to 50% all head injuries in football go un-diagnosed,(31) and preliminary findings have been presented that indicate a sub-set of athletes exists who experience in-season cognitive decline without experiencing abnormal symptomology.(1) Because of this, the risk estimates presented here are defined as the risk for sustaining an immediately diagnosed concussion rather than the risk of sustaining any concussion or signs and symptoms of a concussion. Differences in head impact exposure between cases of immediate and delayed injury diagnosis were not evaluated directly; however, this will be the subject of future communications.

While the risk estimates presented in this study are limited to those of immediately diagnosed concussion, it is still valuable to compare these results to historical studies of traumatic brain injury to evaluate commonly accepted theories. In the 1950–60's, Gurdjian et al. first observed a relationship between an impact event and clinical indicators of TBI through a series of tests conducted on anesthetized canines and human cadavers.(22) From these experiments and supplementary data, a brain injury tolerance curve was created, known as the Wayne State Tolerance Curve (WSTC) that relates brain injury to linear head acceleration and duration.(21, 27) The WSTC is the basis for both GSI and HIC_{15} which are still used today in the development and standardization of head injury protective devices for both the automotive and helmeted sports industries. Though it has long been accepted that both peak acceleration and duration play a role in brain injury, it is interesting to note that, in our study, no statistical difference was found between peak linear acceleration and either GSI, HIC_{15}, or v when assessed as a predictor of immediately diagnosed. One probable reason for this finding is that head impacts in football typically have very similar temporal characteristics (duration of 8.99 ± 3.01 ms)(6) and the associated injuries are less severe, making the data set described in this study more homogenous than the one used to develop the WSTC, which included injuries ranging between skull fracture and loss of consciousness following [linear] acceleration durations ranging between <0.001 – 0.60 ms.(27) While this does not discredit the role of impact duration to concussion in general, it does appear that the magnitude of linear acceleration without the inclusion of a temporal component is sufficient for differentiating impacts associated with concussion from those that are not when considering only head impacts sustained within a single helmeted sport.

While many studies have shown that rotational acceleration is the most likely cause of diffuse axonal injury, historical literature on this association is primarily derived from animal surrogates undergoing pure rotational acceleration (i.e., whiplash events).(19, 29) These data have been supported for humans by simulating brain tissue deformation following impact with finite element brain models and associating resulting measures of strain with the input kinematics.(44) The question still remains, however, if these surrogate data apply to the impact scenarios occurring in a sports environment. While the mechanism of injury cannot be assumed, studies focusing on impacts sustained during football have shown that combining rotational acceleration with other impact measures such as linear

acceleration and impact location increases the specificity of injury prediction;(5, 20, 44) however, for this analysis, we chose to treat each impact measure independently for the purpose of developing single measure ROC curves. While the predictive capabilities of peak linear acceleration and measures derived from the linear acceleration resultant were not found to be statistically significant, peak rotational acceleration was found to be the least sensitive of all evaluated severity measures to immediately diagnosed concussion. At this time it is unclear why a discrepancy exists between the evidence that rotational acceleration is the cause of brain injury and yet it is the least predictive measure of immediately diagnosed concussion. These results could indicate a discrepancy between the pathomechanics of injury previously explored in the laboratory and the injury being defined as a concussion in helmeted sports,(14) or that the association between individual kinematic parameters and injury could simply be masked by the correlated relationship of these parameters in a football head impact.(40)

There are several potential limitations of this study. First, all concussions were diagnosed by a trained medical professional using their clinical judgment and best practice guidelines at the time of injury; however, it has been well established that concussion symptoms frequently go unreported.(8, 31) It has also been shown that some players may experience cognitive change without any perceived symptomology.(1, 30, 42) While we can be reasonably assured that athletes diagnosed with concussion sustained an injury, the converse cannot be assumed. By limiting our analysis to head impacts sustained by players with at least one sustained concussion, we limit the potential for underestimating concussion while maintaining a large "control" sample of impacts not associated with injury, thus providing what we believe to be a robust estimate for risk of diagnosed concussion. To overcome this limitation in the future, additional methods for screening athletes on a consistent basis throughout the season could be implemented to evaluate in-season clinical presentation (i.e., presence / absence of signs and symptoms typically associated with concussion) rather than focusing solely on clinical diagnosis. Secondly, data were not analyzed separately by subject demographic information (e.g., playing position, high school vs. college, helmet type, etc.). While it has been shown that demographic-specific trends for head impact exposure exist between and within athlete populations,(3, 6, 15, 33, 41) it is important to note that frequency, location, and the kinematic response to head impact is highly dependent on an individual player.(10–12) For example, collegiate football players tend to sustain more impacts over the course of a season and impacts resulting in higher head acceleration more frequently than high school players; however, the range of head impact exposure athletes experience is quite large, so it is quite common for individual high school players to sustain impacts at a frequency and acceleration level that is on par with collegians.(4) Because sports-related concussion is a highly individualized and complex pathophysiological process,(32) our initial focus was to determine the head impact exposure measures most associated with injury for individual players. This within-subject design provides a better understanding of the biomechanical variables most related to diagnosed concussion, independent of these extrinsic factors that may have contributed to the level of exposure each athlete experienced. Moving forward, the results from this study can be combined with typical head impact exposure profiles already established for non-diagnosed athletes to determine if different conditions of participation place an athlete more at risk for injury.

Through this approach, strategies for injury mitigation can be developed. Finally, the study design employed was not epidemiological in nature and only tracked cases of diagnosed concussion for athletes while wearing instrumented helmets. Because of this, readers should be careful not to estimate concussion rates from the data presented or interpret concussion risk based on the occurrence of injury by specific demographic information alone.

To the authors' knowledge, this work presents the largest collection of *in vivo* biomechanical head impact data associated with diagnosed concussion to date. The key findings of this initial communication indicate that players sustain both a greater number of impacts and impacts of higher severity on days of diagnosed concussion than on days without diagnosed concussion. Additionally, kinematic measures associated with peak linear acceleration are similar predictors of immediately diagnosed concussion while predictive capability of rotational acceleration is significantly lower. While further analysis is required, the data introduced in this study provides a foundation for identifying the biomechanical basis of head injury from which future communications will build upon.

Acknowledgments

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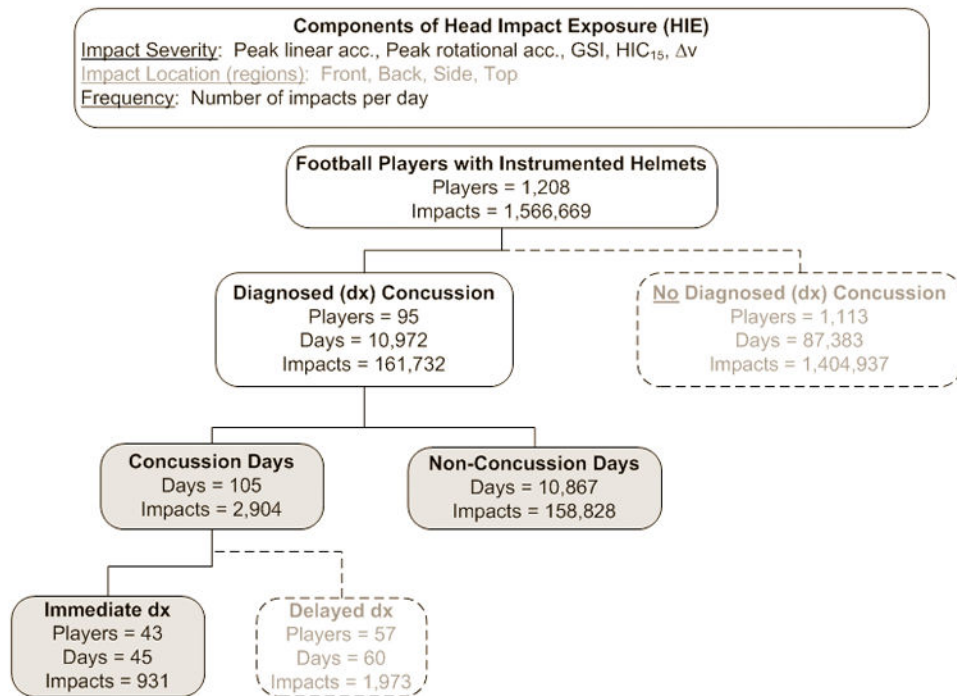
Figure 1.
Hypotheses tested within this communication are based on a subset of biomechanical and clinical data that was collected as part of a longitudinal study to investigate the biomechanical bases of mild traumatic brain injury. Data reported in this study are derived from the samples highlighted in the above flowchart.
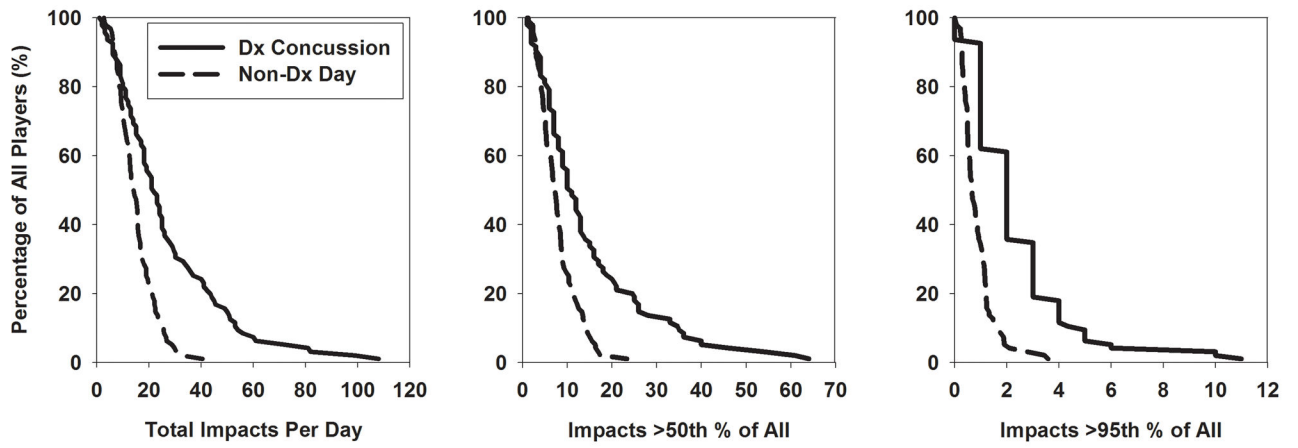
Figure 2.
Number of impacts per day (all, > 50th percentile, > 95th percentile linear acceleration) for players on days with and without diagnosed concussion.
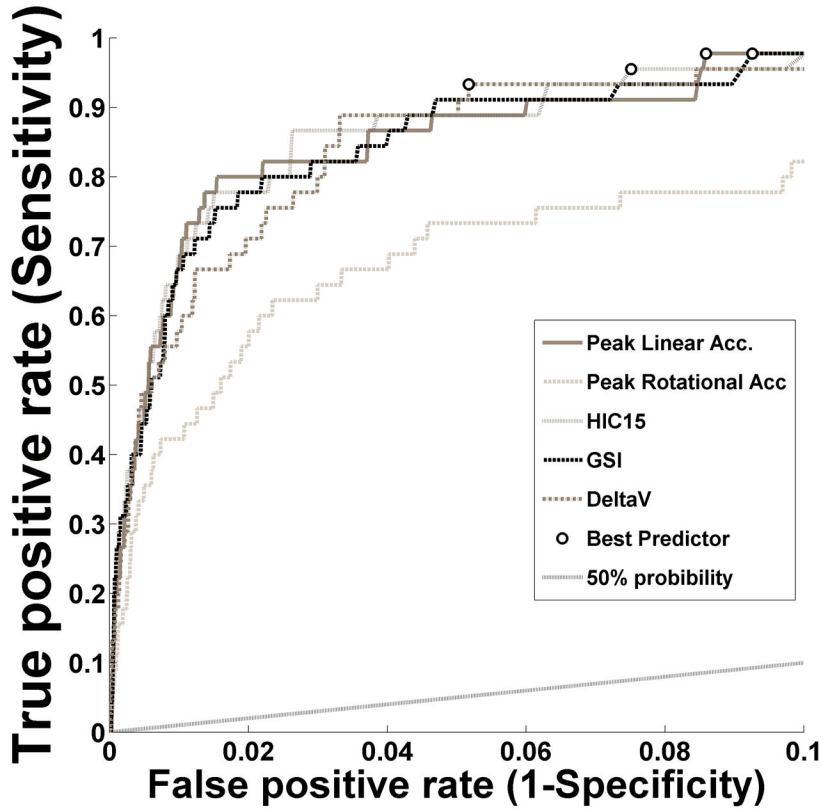
Figure 3.
Receiver Operating Characteristic (ROC) curves indicating the sensitivity and specificity of historical measures of impact severity. A 50% probability line is included to indicate the level of guessing (50 – 50 chance). Peak linear acceleration and HIC_{15} are the most sensitive single impact measures to immediately diagnosed concussion followed by GSI, change in velocity, and peak rotational acceleration. The horizontal axis is reduced to highlight only the top 90th percentile of all impact events which is inclusive of most impacts associated with injury.

Table 1

Median head kinematic measures for individual players (n = 95) on days with and without diagnosed concussion. Players diagnosed with concussion sustain impacts with higher associated kinematics on days of diagnosed concussion than days without.

Impact Kinematics	Non-Concussion Days		Concussion Days		P Value
	Median	25–75% interquartile range	Median	25–75% interquartile range	
50th Percentile					
Peak Lin Acc (g)	20.7	19.5 – 22.0	22.5	19.3 – 26.5	< 0.001
Peak Rot Acc (rad/s²)	848	799 – 922	874	723 – 1,040	0.08
HIC₁₅	4.0	4.0 – 5.0	5.0	3.1 – 7.0	< 0.001
GSI	6.0	5.1 – 7.0	8.0	5.6 – 11	< 0.001
v (m/s)	0.77	0.71 – 0.81	0.79	0.69 – 0.97	0.002
95th Percentile					
Peak Lin Acc (g)	63.5	56.4 – 69.0	82.0	66.5 – 95.4	< 0.001
Peak Rot Acc (rad/s²)	2,761	2,448 – 3,011	3,376	2,632 – 4,154	< 0.001
HIC₁₅	62.7	48.9 – 79.3	130.0	75.1 – 195.8	< 0.001
GSI	94.4	73.8 – 121.6	190.2	116.6 – 277.8	< 0.001
v (m/s)	2.31	2.08 – 2.50	2.93	2.47 – 3.70	< 0.001

Table 2

Median [25–75% interquartile range] number of head impacts for individual players sustaining at least one diagnosed concussion on days with and without injury diagnosis. Athletes sustained more total impacts and more impacts with high kinematic response (top 50th and 95th percentile) impacts on days with diagnosed concussion than days without diagnosed concussion.

Number of Daily Head Impacts	Clinical Diagnosis		P Value
	No Concussion	*Concussion*	
> Linear 95% [a]	0.7 [0.4 – 1.2]	2.0 [1.0 – 3.0]	< 0.001
> Rotational 95% [b]	0.5 [0.3 – 0.8]	1.0 [1.0 – 2.0]	< 0.001
> Linear 50% [a]	7.2 [4.6 – 10.2]	10.0 [6.0 – 18.8]	< 0.001
> Rotational 50% [b]	5.9 [3.7–7.9]	9.0 [4.3 – 16.0]	< 0.001
All Impacts	13.8 [9.4 – 18.9]	21.0 [12.0 – 36.8]	< 0.001

[a] 50th and 95th percentile linear acceleration = 20.5 and 62.2 g

[b] 50th and 95th percentile rotational acceleration = 981 and 2,975 rad/s^2

Table 3

Statistical summary of binary logistic regression analysis used to determine probability of injury based on impact kinematics. Odds Ratio indicates the increased odds of sustaining a concussion per single unit of measure (ex. 1g).

Kinematic Measure	Coefficient		Standard Error	Wald (χ^2)	p	Odds Ratio	95% CI	
	(α)	(β)					Lower	Upper
Linear Acc.	−10.028	0.0509	0.00279	332.03	< 0.001	1.052	1.046	1.058
Rotational Acc.	−8.942	0.0008	0.00005	269.06	< 0.001	1.001	1.001	1.001
HIC$_{15}$	−7.825	0.0078	0.00052	226.93	< 0.001	1.008	1.007	1.009
GSI	−7.869	0.0059	0.00037	252.17	< 0.001	1.006	1.005	1.007
DeltaV	−9.628	1.2148	0.06948	305.64	< 0.001	3.369	2.940	3.861