

Development of Concussion Risk Curves Based on Head Impact Data from Collegiate Football Players

J. R. Funk, S. M. Duma, S. J. Manoogian, and S. Rowson

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ABSTRACT

Previous attempts to develop engineering risk curves for mild traumatic brain injury have been hampered by a paucity of data from living humans. A recent study by Pellman et al. (2003) presented risk curves for concussion based on data from reconstructed head impacts sustained by National Football League (NFL) players. These risk curves were flawed because the data were heavily biased towards injurious impact exposures. Unbiased head impact exposure data for collegiate football players at Virginia Tech (VT) have been collected using the Head Impact Telemetry System (HITS), which estimates head acceleration based on spring-mounted helmet accelerometers. Nearly 23,000 head impacts were recorded during the 2003 – 2005 seasons, including 3 impacts in which a player sustained a concussion. A concussion risk curve was estimated using the Consistent Threshold (CT) method. Head impact exposure was modeled using a Weibull distribution and normalized on a per player per play basis. An error deconvolution technique was developed to analyze the effect of measurement error in the HITS data on the distribution of head impact exposures. The expected incidence of concussion was estimated by combining the CT risk curve with the head impact exposure data. The CT risk curve derived from the HITS data provided a far more reasonable estimate of concussion incidence than the NFL risk curve.

INTRODUCTION

Biomechanical research on head injury has historically focused on characterizing serious injuries using cadaveric or animal models. These models are limited in their ability to predict concussion, or minor traumatic brain injury (MTBI), in living humans. Although typically only an AIS 1 injury, MTBI occurs far more commonly than severe head injury and is a major public health problem. Finding an experimental model with which to study MTBI is difficult; MTBI cannot be detected in cadavers, results from animal experiments cannot be directly applied to living humans, and it would be unethical to intentionally inflict MTBI on living humans in a laboratory setting. For these reasons, studying athletes in contact sports is a promising avenue of biomechanical research on MTBI.

An important goal of MTBI research is to establish biomechanical risk curves that can be used to predict the likelihood of injury based on head impact severity. MTBI risk curves would be useful in improving helmets, padding, and other countermeasures to reduce the incidence of MTBI in sports, car crashes, and other settings. Although several studies have provided important information about the magnitude of injurious and non-injurious head impacts sustained by living humans, an accurate concussion risk curve has yet to be established. Pellman et al. (2003) have provided the most extensive biomechanical incidence data concerning concussive head impacts in living humans to date. Based on video footage of National Football League (NFL) games, Pellman et al. (2003) reconstructed 31 head impacts, 25 of which resulted in a player sustaining a concussion, using helmeted Hybrid III dummies. Pellman et al. (2003) presented risk curves for MTBI based on logistic regressions of the NFL data. However, these risk curves were flawed, because the statistical analysis required unbiased exposure data, and the NFL data were intentionally biased towards injurious impacts. As a result of this improper statistical analysis, the NFL risk curves are far too conservative. For example, data collected by Pincemaille et al. (1989) showed that amateur boxers were able to sustain numerous head impacts without adverse consequences at severity levels that the NFL risk curves would associate with a near certainty of concussion. The objective of the present study was to establish biomechanical risk curves for MTBI using a large set of unbiased head impact data collected from collegiate football players. This paper describes a novel statistical methodology that has been developed for analyzing the head impact data and presents some preliminary results of the study.

METHODS

Biomechanics

Head impact data obtained from instrumented helmets worn by Virginia Tech (VT) football players during the 2003 – 2005 seasons were analyzed. Data were collected from 56 different players during games and full impact practices. The helmets were instrumented with the Head Impact Telemetry (HIT) System (Simbex, Lebanon, NH), which consisted of six spring-mounted linear accelerometers designed to stay in contact with the player's head. Data from the accelerometers were sampled at 1000 Hz and transmitted wirelessly to a laptop computer on the sidelines in real time. Only impacts in which the signal from at least one of the accelerometers exceeded a user-defined threshold of 10 g were recorded. The linear acceleration at the estimated center of gravity of the head was calculated from the six accelerometer signals using an algorithm described by Crisco et al. (2004). The Head Injury Criterion (HIC) was calculated from the resultant linear acceleration at the head center of gravity. Additional details regarding the data collection methodology are described by Duma et al. (2005).

MTBI risk curves were calculated as a function of impact severity. Impact severity was characterized in terms of both peak resultant linear head acceleration and HIC. An assumption was made that each player had a fixed injury tolerance. That is, it was assumed that any head impact having a peak acceleration or HIC greater than the player's injury tolerance would cause a concussion, and any impact having a severity less than the player's injury tolerance would not cause a concussion. The purpose of the statistical analysis was to characterize the variation in individual MTBI injury tolerances across the population. The MTBI risk curves were derived using a straightforward method that is commonly used when analyzing data from biomechanical experiments. All injury data points and maximal non-injury data points for each player were analyzed using the consistent threshold (CT) estimate for doubly censored data (Nusholtz and Mosier, 1999). The CT estimate is a non-parametric maximum likelihood estimate of risk. The CT risk curves were based on data from only 59 impacts (the maximal non-injurious head impact for each of the 56 instrumented players plus the 3 concussive impacts), which was obviously only a tiny fraction of the full data set. The vast majority of the biomechanical data was excluded based on the assumption that all submaximal noninjurious head impacts for an individual player contained no unique information. CT risk curves were generated using a custom-written FORTRAN program.

Epidemiology

The CT MTBI risk curves were validated using a novel epidemiological methodology that characterized the players' head impact exposure and estimated concussion incidence based on the risk curves. The HITS data were assumed to provide unbiased and mutually independent estimates of head impact exposure. This assumption was considered valid because of the prospective nature of the study and the fact

that the instrumented players were chosen to encompass a wide range of body types and player positions. The distribution of head impact severities was expressed in terms of a probability density function (pdf) and cumulative distribution function (cdf). Head impact exposure data from games and full contact practices were combined. The distribution of head impacts was modeled using a standard Weibull distribution:

$$pdf = f(x) = \frac{\alpha x^{\alpha-1}}{\beta^\alpha} e^{-\left(\frac{x}{\beta}\right)^\alpha} \quad (1a)$$

$$cdf = F(x) = 1 - e^{-\left(\frac{x}{\beta}\right)^\alpha} \quad (1b)$$

where α is the shape parameter, β is the scale parameter, and x is the impact severity in terms of either peak resultant head acceleration or HIC.

The HITS data were ordered and normalized to obtain an experimental cdf (cdf_{exp}) of the head impact exposure data. The experimental cdf was mathematically manipulated in two ways before curve fitting it to a Weibull distribution (Equation 1b). First, in order to maximize the accuracy of the curve fit for the higher severity impacts that were of most interest, all impacts below 40 g were excluded when fitting the peak acceleration data, and all impacts with a HIC < 50 were excluded when fitting the HIC data. These relatively low severity impacts were deemed unlikely to cause MTBI, based on the NFL data (Pellman et al., 2003). The equation form for the Weibull distribution was adjusted to obtain the pdf and cdf over the severity region of interest $\theta < x < \infty$, where $\theta = 40$ g for the analysis of the peak acceleration data, and $\theta = 50$ for the analysis of the HIC data:

$$pdf_\theta(\theta < x < \infty) = f_{adj}(x) = \frac{f(x)}{1 - F(\theta)} = \frac{\alpha x^{\alpha-1}}{\beta^\alpha} e^{-\frac{\theta^\alpha - x^\alpha}{\beta^\alpha}} \quad (2a)$$

$$cdf_\theta(\theta < x < \infty) = F_{adj}(x) = \frac{F(x) - F(\theta)}{1 - F(\theta)} = 1 - e^{-\frac{\theta^\alpha - x^\alpha}{\beta^\alpha}} \quad (2b)$$

Although the adjusted pdf and cdf contain the parameters α , β , and θ that are sometimes associated with a three-parameter Weibull distribution, the distribution in Equations 2a and 2b is not a Weibull distribution. It is a modified Weibull distribution that allowed the curve fit to be performed over a specific region of interest ($\theta < x < \infty$). Second, rather than curve fitting Equation 2b directly to the experimental cdf, the log transform of the complement of Equation 2b was fit to the log transform of the complement of the experimental cdf:

$$\frac{\theta^\alpha - x^\alpha}{\beta^\alpha} = \ln[1 - cdf_{exp}(\theta < x < \infty)] \quad (3)$$

This transformation was done in order to give greater weight to higher severity impacts when performing the least squares curve fit. It had the incidental effect of excluding the most severe impact in the data set from the curve fit, because the expression is undefined when the experimental cdf is equal to one.

Error Deconvolution

The curve fitting method described above assumes that there is no error in the experimental measurements. However, every measuring device has error, and the HIT system is no exception. A novel method of accounting for measurement error when fitting a distribution of data was developed and applied to the HITS data. Measurement error can be divided into two components: bias and scatter. Bias is typically characterized by the mean value of the measurement error in terms of a percentage error and/or an offset at various levels of measurement. Bias can be analytically removed from the data so that the average of the corrected measurements equals the true value. The correction process is straightforward. For example, if bias in the measurement system can be characterized by

$$x_{measured} = m \cdot x_{true} + b \quad (4a)$$

then the data can be corrected according to the following formula:

$$x_{corrected} = \frac{x_{measured} - b}{m} \quad (4b)$$

However, even if the measurements are unbiased, they may still contain error in the form of scatter. The mean value of the error due to scatter is zero. Individual data points cannot be corrected for scatter, because there is no way to know whether the measured value overestimates or underestimates the true value. However, the presence of error due to scatter can affect the distribution of a large number of measurements. To understand the effect of scatter on a distribution of measurements, first consider a hypothetical distribution of true values being measured (Figure 1). Now consider a small subset of the true distribution, say head impacts having a peak acceleration of 50 g. If this subpopulation of head impacts were to be measured, the error due to scatter might cause some of them to be measured at 40 g or 60 g, even though the average of the measurements would still be 50 g, the same as the true value. This phenomenon would occur at all measurement levels, depending on the measurement error at each level. Therefore, when measuring from a random sample of the distribution of true values, the distribution of measured values is actually the sum of the scatter from the distribution of true values. That is, the subpopulation of measured values having a peak acceleration of 100 g actually contains not only head impacts in which the true peak acceleration was 100 g, but also a percentage of impacts in which the true value was 90 g but the error due to scatter was +10 g, and a percentage of impacts in which the true value was 105 g with a scatter error of -5 g.

Mathematically, the distribution of measured values (f') can be calculated by convolving the distribution of true values (f) with the error distribution (g):

$$f'(x) = \int_0^{\infty} f(z)g(x-z)dz \tag{5}$$

where x is the measurement in terms of peak head acceleration or HIC, and z is a dummy variable. In order for Equation 5 to be a true convolution mathematically, the error function (g) must be the same at all levels of measurement. If the measurement error is assumed to be normally distributed, this condition corresponds to the assumption of homoscedasticity that is implicit in standard least squares linear regression. Most often, the measurement error of instrumentation is not constant, but increases at increasing levels of measurement. Typically, measurement error is expressed as a percentage of the value being measured, which corresponds to a normally distributed error function in which the coefficient of variation (c_v) is constant:

$$c_v = \frac{\sigma}{\mu} \tag{6}$$

where σ is the standard deviation and μ is the mean of the normally distributed error function. This error model assumes the measurement error is heteroscedastic. The parameter c_v can be obtained from validation data by fitting the squared residuals of the curve fit to a quadratic function. Modeling the error due to scatter as heteroscedastic requires an error function g that can be expressed as a function of both $(x-z)$ and (z) :

$$f'(x) = \int_0^{\infty} f(z)g(x-z, z)dz \tag{7}$$

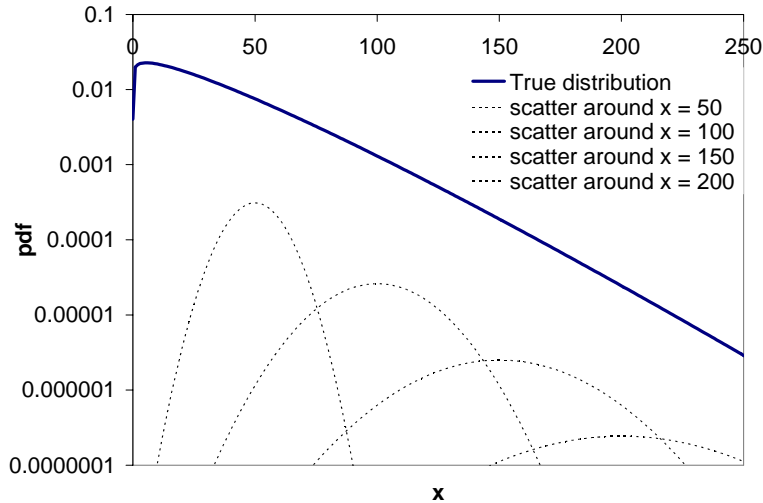


Figure 1: Illustration of the effect of scatter error ($c_v = 20\%$) on the distribution of measurements.

Equation (7) is no longer a true convolution integral mathematically, and might be more accurately called a modified convolution integral. However, it will continue to be referred to as a convolution integral for simplicity. The error function $g(x-z, z)$ can be expressed as an unbiased normal distribution with scatter error having a constant coefficient of variation:

$$g(x-z, z) = \frac{1}{\sqrt{2\pi}c_v z} e^{-\frac{(x-z)^2}{2c_v^2 z^2}} \quad (8a)$$

Alternatively, Equation 8a can be expanded to include the effect of measurement bias as expressed in Equation 4a:

$$g(x-z, z) = \frac{1}{\sqrt{2\pi}c_v z} e^{-\frac{(mz+b-x)^2}{2c_v^2 z^2}} \quad (8b)$$

Thus, bias in the measurements can be corrected either at the level of individual measurements, or at the level of the distribution of measurements. If individual measurements are corrected for bias, the error due to scatter must be recalculated from the validation test data to reflect the correction. Substituting Equations 1 and 8b into Equation 7 yields the final form of the error convolution:

$$f'(x) = \frac{\alpha}{\beta^\alpha \sqrt{2\pi}c_v} \int_0^\infty z^{\alpha-2} e^{-\left(\frac{z}{\beta}\right)^\alpha} e^{-\frac{(mz+b-x)^2}{2c_v^2 z^2}} dz \quad (9)$$

To account for measurement error when fitting to a distribution, it must be recognized that a curve can only be fit to the measured distribution $f'(x)$, not to the true underlying distribution $f(x)$. However, $f'(x)$ must first be manipulated before it can be fit to the experimental HITS data. Equation 9 has no closed-form solution, so the integration must be performed numerically. The cdf of $f'(x)$ was obtained by numerically integrating $f'(x)$, then adjusting $F'(x)$ by normalizing it over the region $\theta < x < \infty$:

$$F'_{adj}(x) = \frac{\int_\infty^x f'(x) dx}{\int_\theta^x f'(x) dx} \quad (10)$$

The values of α and β in Equation 9 were obtained by curve fitting the log transform of the complement of the adjusted cdf in Equation 10 to the log transform of the experimental cdf:

$$\ln[1 - F'_{adj}(x)] = \ln[1 - cdf_{exp}(\theta < x < \infty)] \quad (11)$$

Equation 11 is analogous to Equation 3, with the key difference being that the optimized α and β parameters are estimates of the true distribution in Equation 11, rather than the error-containing measured distribution, in Equation 3. In this way, the measurement error can be deconvolved from the true distribution. The operations necessary to perform this deconvolution were accomplished using a custom-written FORTRAN program. In order to estimate the importance of scatter error on the final results, a sensitivity analysis was conducted in which the HITS data were assumed to be unbiased with scatter errors having coefficients of variation of 10%, 20%, and 30%.

Normalizing Exposure Per Player

Once the overall distribution of head impacts was established, these exposure data were normalized on a per player per play basis by combining the HITS data with player information obtained from the Virginia Tech football team. Once the distribution of head impacts in the severity region of interest was characterized, the next step was to determine the probability that a player would sustain a head impact in that region of severity in a given play, which was denoted by p_θ :

$$p_\theta = p(x_{hit} > \theta) = \frac{\# \text{ recorded impacts} > \theta}{\# \text{ instrumented players} \times \text{plays}} \quad (12)$$

The number of recorded impacts greater than θ was obtained from the HITS data. The number of “players x plays” was the sum of the number of plays played by each instrumented player, which was obtained from team records. Preliminary estimates of p_θ were obtained by analyzing data from team records.

Combining Equations 2a and 12, the probability of a head impact having a severity greater than x for a single player in a single play was calculated:

$$p(x_{hit} > x) = p(x_{hit} > x | x_{hit} > \theta)p(x_{hit} > \theta) = [1 - F_{adj}(x)]p_\theta = p_\theta e^{-\frac{\theta^\alpha - x^\alpha}{\beta^\alpha}} \quad (13)$$

It was assumed that the probability of sustaining a head impact of severity x is the same for each play. Therefore, each play was modeled as an independent Bernoulli trial. The probability of exactly k head impacts having a severity greater than x for a single player exposed to n plays is given by the binomial distribution:

$$p_n(x_{hit} > x, k) = \binom{n}{k} [p(x_{hit} > x)]^k [1 - p(x_{hit} > x)]^{n-k} \quad (14)$$

The probability that the most severe head impact for a single player over n plays was greater than x was obtained from the complement of the probability that no head impacts would be greater than x (binomial distribution with $k=0$):

$$p_n(x_{max} > x) = 1 - [1 - p(x_{hit} > x)]^n = 1 - \left(1 - p_\theta e^{-\frac{\theta^\alpha - x^\alpha}{\beta^\alpha}}\right)^n \quad (15)$$

Equation 15 gives the probability that one or more head impacts will be greater than x . The probability that the most severe head impact for a single player over n plays was exactly equal to x was calculated by taking the partial derivative of the complement of Equation 15 with respect to x :

$$p_n(x_{max} = x) = np_\theta \left(1 - p_\theta e^{-\frac{\theta^\alpha - x^\alpha}{\beta^\alpha}}\right)^{n-1} \frac{\alpha x^{\alpha-1}}{\beta^\alpha} e^{-\frac{\theta^\alpha - x^\alpha}{\beta^\alpha}} \quad (16)$$

Having established the exposure of head impacts experienced by a single player over n plays, it is now possible to investigate the relationship between the risk of injury and incidence of injury. From the assumption that each player has a fixed injury tolerance, it follows that if a player sustains a concussion, then that concussion must have been caused by the most severe impact that player experienced. There are two equivalent approaches that can be taken to determine a player’s risk of MTBI as a function of the number of plays to which he is exposed. Both involve calculating the probability of a player sustaining a concussion at a particular impact severity level x and then integrating over all x to obtain the overall risk of concussion for that player. In the first approach, the probability of a concussion incidence for a player having an injury tolerance $x_{inj} = x$ is given by:

$$p_{inc}(x) = p(x_{inj} = x)p_n(x_{max} > x) \quad (17)$$

In the second approach, the probability of a concussion incidence for an impact of severity $x_{hit} = x$ is given by:

$$p_{inc}(x) = p_n(x_{max} = x)p(x_{inj} < x) \quad (18)$$

The incidence probabilities calculated by these two approaches are not equivalent. In the first approach (Equation 17), the probability of a concussion incidence is given in terms of the injury tolerance of the player ($x = x_{inj}$). In the second approach (Equation 18), the probability of a concussion incidence is given in terms of the maximum severity impact experienced by the player ($x = x_{max}$). When a player is injured, it is because the severity of the concussive impact exceeded the injury tolerance of the individual by some amount ($x_{max} > x_{inj}$). Therefore, the probability incidence calculated from Equation 17 will be shifted to the left somewhat (lower values of x) compared to the probability incidence calculated from Equation 18. In spite of this difference, it can be shown from integration by parts that the area under each curve is the same:

$$\int_0^\infty p(x_{inj} = x)p_n(x_{max} > x)dx = \int_0^\infty p_n(x_{max} = x)p(x_{inj} < x)dx \quad (19)$$

Therefore, the overall probability of injury for a given player exposed to n plays can be calculated using either approach:

$$p_{inj}(n) = \int_0^{\infty} p_{inc}(x) dx \quad (20)$$

It should be noted that the probability of MTBI incidence (Equations 18 and 19) and the overall probability of injury (Equation 20) represent the probability of a player sustaining one or more concussions.

Although both approaches are equivalent, there are two advantages to the second approach (Equation 18). First, the CT risk curve is a discontinuous staircase function that cannot be differentiated to obtain the $p(x_{inj} = x)$ expression required in the first approach (Equation 17). Second, characterizing MTBI incidence in terms of the impact severity of the injurious impact allows for a meaningful comparison with the experimental data obtained in both the present study and the NFL study (Pellman et al., 2003). The overall probability per play that a player will sustain a concussion is found by substituting Equation 18 into Equation 20:

$$p_{inj}(n=1) = \frac{p_{\theta} \alpha}{\beta^{\alpha}} \int_0^{\infty} x^{\alpha-1} e^{-\frac{\theta^{\alpha} - x^{\alpha}}{\beta^{\alpha}}} p(x_{inj} < x) dx \quad (21)$$

where $p(x_{inj} < x)$ is the CT risk curve. Because the CT risk curve is a stepwise constant function having k_{steps} steps, Equation 21 can be integrated and summed over each step:

$$p_{inj}(n=1) = p_{\theta} \sum_{k=1}^{k_{steps}} \left(e^{-\frac{\theta^{\alpha} - x_k^{\alpha}}{\beta^{\alpha}}} - e^{-\frac{\theta^{\alpha} - x_{k+1}^{\alpha}}{\beta^{\alpha}}} \right) p(x_k < x_{inj} < x_{k+1}) \quad (22)$$

The expected number of concussions per game can be estimated by multiplying the above probability by the mean number of players x plays per game, which is approximately 1600.

RESULTS

Biomechanics

HITS data from 22,704 impacts in games and practices were collected. The distribution of impacts followed a roughly exponential distribution in terms of both peak acceleration and HIC. The distribution of head impacts was similar for games and practices (Figure 2), which justified combining the data. The vast majority of the impacts were of low severity. Nonetheless, there were 3,339 impacts in which the peak acceleration was greater than 40 g, and 1,376 impacts in which the HIC was greater than 50. There were three impacts in which an instrumented player sustained a concussion. In the three concussive impacts, the peak acceleration values were 81 g, 172 g, and 200 g, and the HIC values were 200, 589, and 859. In two out of the three concussed players, the injurious impact was the most severe impact experienced by that player in terms of both peak acceleration and HIC out of over 400 recorded impacts for each player. In the third concussed player, the injurious impact was the eleventh most severe impact in terms of head acceleration, the third most severe impact in terms of HIC out of over 200 recorded impacts, and the most severe impact from the front in terms of both head acceleration and HIC.

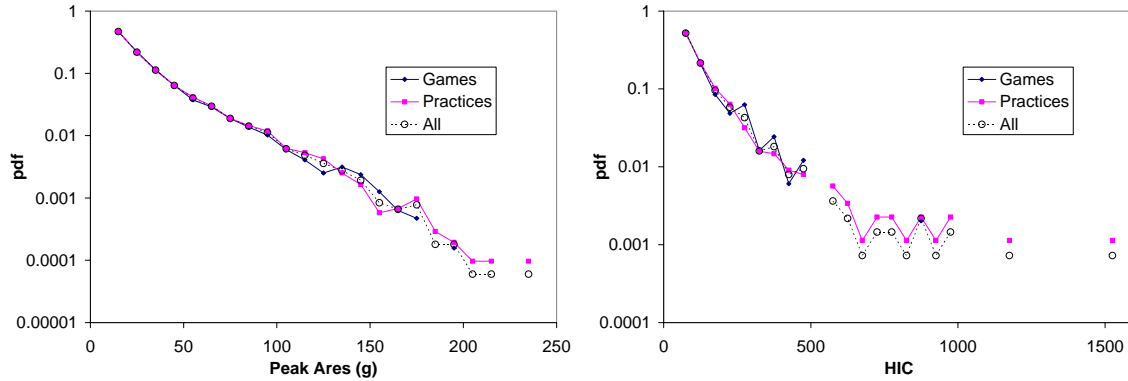


Figure 2: Probability density functions for all Virginia Tech HITS data.

The MTBI risk curves developed from the HITS data using the CT estimate are drastically different from the MTBI risk curves proposed by Pellman et al. (2003). The NFL risk curves (Pellman et al., 2003) predict a far higher risk of injury at a given impact severity than the CT risk curves derived from the HITS data (Figure 3). For example, for a head acceleration of 150 g, the CT risk curve predicts a 3% risk of concussion, while the NFL risk curve predicts a 98% risk of concussion. Curiously, the NFL risk curve predicts a 7% risk of concussion at a HIC of 0. A 7% risk of concussion corresponds to a HIC of 589 in the CT estimate.

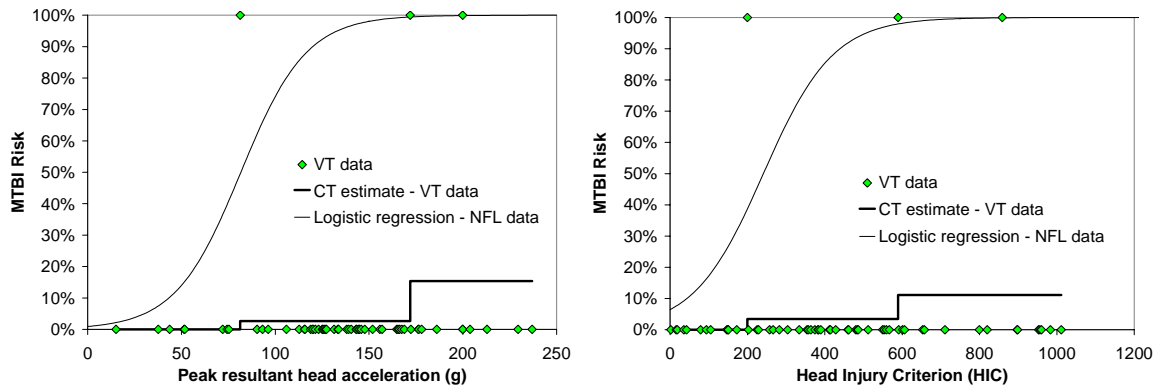


Figure 3: Comparison of MTBI risk curves from the present study (CT estimate – VT data) and from the NFL study (Pellman et al., 2003).

Epidemiology

The Weibull distribution effectively modeled the distribution of head impacts measured by the HIT system. The Weibull model produced a good fit both visually and in terms of the correlation coefficient R^2 , which exceeded 0.99 in all cases (Figure 4). The effect of measurement error due to scatter on the estimated distribution of head impacts was negligible for the HIC distribution but potentially important for the peak acceleration distribution. The main effect of scatter error was to inflate the high end tail of the measured distribution.

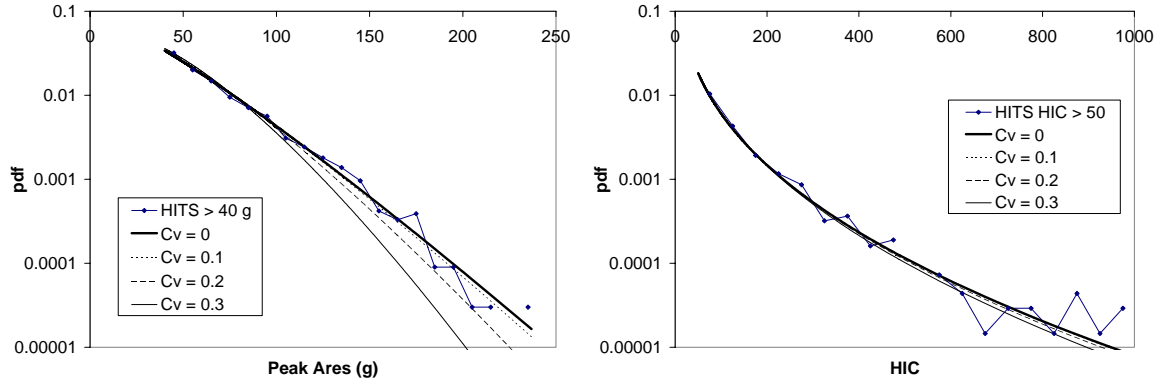


Figure 4: Effect of measurement error due to scatter on the true vs. measured distribution of head impacts.

By combining player data from team records with the HITS data, it was estimated that on average, players experienced head accelerations greater than 40 g in approximately 6% of plays, and HIC values greater than 50 in approximately 2.5% of plays in which they played. These values defined p_θ (Equation 12) and allowed the head impact distributions to be normalized on a per player per play basis. Specifically, the distribution of the maximum head impact for a player was characterized as a function of the number of plays the player was in. In any single play, a player was unlikely to experience a severe head impact, and often did not experience any head impact at all (Figure 5). The risk of experiencing a severe head impact rises with each subsequent play, but the rate of that rise decreases roughly logarithmically as the number of plays increases. The expected distribution of maximum head impact severities as a function of number of plays is only mildly affected by scatter error (Figure 6).

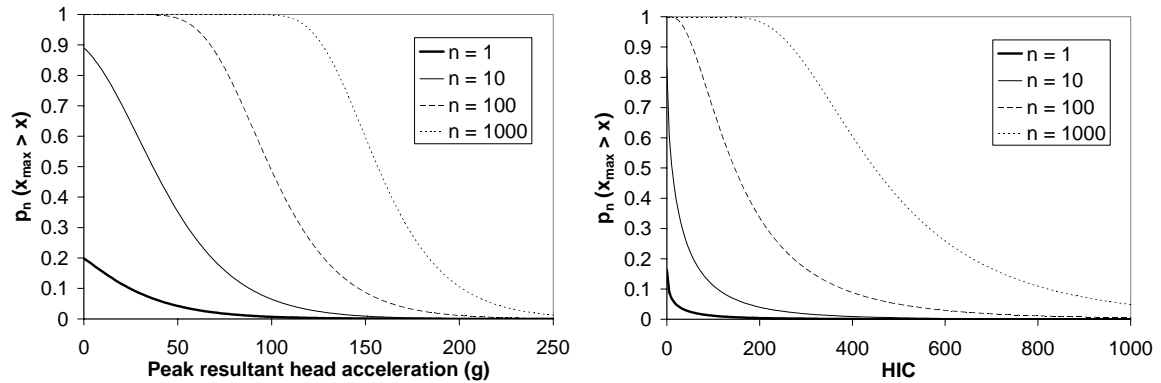


Figure 5: Effect of the number of plays on the probability of maximum head impact severity.

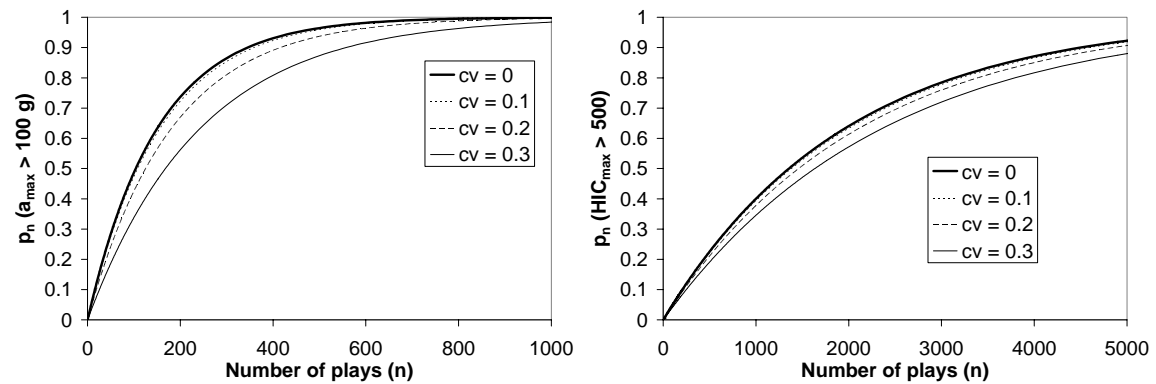


Figure 6: Effect of scatter on the distribution of maximum head impact severity.

The incidence of MTBI was evaluated more globally by comparing the cdf of the injury data set and the expected incidence of concussion to the NFL data. The injurious impacts contained in the HITS data occurred at generally higher impact severities compared to the biomechanical incidence data from the NFL study (Pellman et al., 2003) (Figure 7). The match was closer for the HIC data than the peak acceleration data. However, there were only three concussive impacts recorded, so the sample size was rather small.

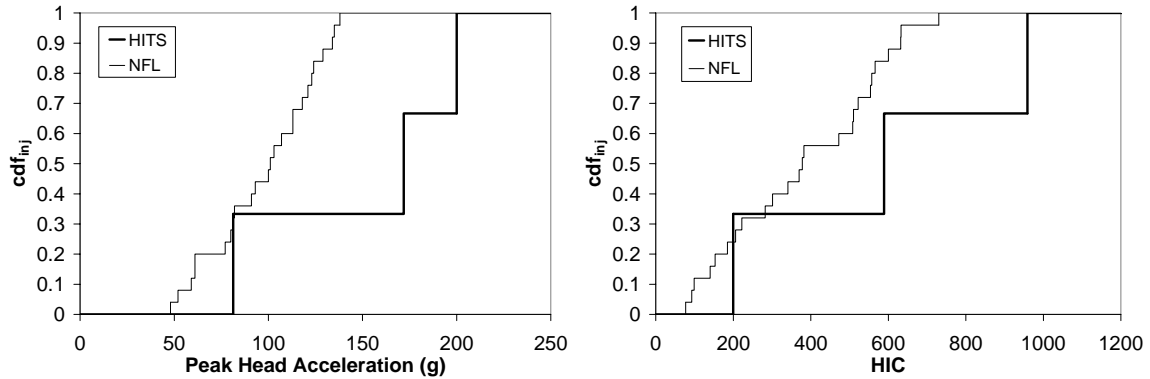


Figure 7: Cumulative distribution of concussive impacts.

The expected rate of concussions per team per game was calculated by applying the CT estimates for MTBI risk curves to the impact exposure data recorded by the HIT system. The expected concussion rate predicted by peak acceleration was considerably higher than the actual incidence in the NFL (Pellman et al., 2004), indicating that the CT risk curve based on peak acceleration was too conservative. The expected concussion rate predicted by HIC compared favorably to the epidemiological data from the NFL study (Figure 8, left). Scatter error in the measurements had no significant effect on the predicted incidence of concussion based on HIC. However, scatter in the peak acceleration measurements had a significant effect on the predicted number of concussions per game, but only for large coefficients of variation (20% – 30%). The concussion incidence predicted by applying the NFL MTBI risk curves proposed by Pellman et al. (2003) to the exposure data measured by the HIT system were more than 100 times higher than the actual concussion incidence in the NFL (Pellman et al., 2004) (Figure 8, right).

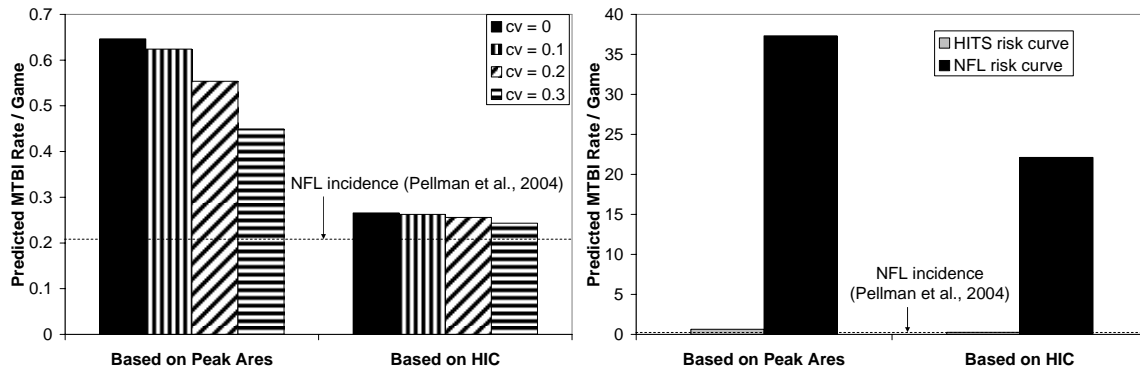


Figure 8. Number of concussions per game predicted by various MTBI risk curves.

DISCUSSION

Risk curves for MTBI were derived using the Consistent Threshold (CT) estimate, which is a standard technique for analyzing biomechanical data. The CT risk curves were validated using a novel epidemiological analysis of a large set of head impact data collected from collegiate football players. The CT risk curves developed in the present study are drastically different from the NFL risk curves reported by Pellman et al. (2003). MTBI risk curves from the present study are more accurate because they utilized a very large database that provided an unbiased estimate of head impact exposure. The data were unbiased because the present study was designed as a prospective cohort study. The experimental design of the NFL study (Pellman et al., 2003) was a case control study. As such, a reasonably large set of concussion cases

were studied, which provided good biomechanical data about concussion incidences. However, comparatively few non-injury control cases were studied, and therefore the head impact exposure could not be characterized biomechanically. Risk curves require accurate incidence data and accurate exposure data. There was insufficient information regarding the overall distribution of head impacts in the NFL to calculate MTBI risk curves. Nonetheless, Pellman et al. (2003) analyzed their data using logistic regression, which implicitly assumed that their data set was representative of the head impacts players were exposed to in the field.

The NFL data set was intentionally biased towards injurious impacts, such that almost all of the highest impacts in their data set were injurious. In the present study, very few of the highest severity impacts were injurious. In light of these more complete exposure data, it was determined that the risk of injury at the severity level where most injuries occur (peak head acceleration ~ 100 g, HIC ~ 400) is actually low, not high. This same phenomenon occurs and is widely recognized in the field of traffic safety. For example, although half of all fatal crashes occur at velocity changes of roughly 30 mph or less, the risk of fatality in a 30 mph delta-V crash is actually quite low (Evans, 2004). The number of car crashes decreases roughly exponentially at increasing delta-V levels, just like the number of head impacts on the football field decreases roughly exponentially at increasing peak acceleration and HIC levels. Because there are so many low severity car crashes and head impacts, the incidence of injury at these levels is amplified, which belies the fact that they are actually low risk events. However, the only way to uncover this epidemiological peculiarity is to obtain both unbiased incidence data and unbiased exposure data. The NFL study obtained the former; the present study obtained the latter (and a little bit of the former). In the present study, the head impact exposure for each player was not broken down by player position. However, the HITS data showed that the distribution of head impacts is notably different for different players and positions, and Pellman et al. (2004) noted significantly different concussion rates for different positions. Therefore, future work will attempt to refine the analysis by accounting for player positions.

It should be noted that determining concussion incidence from the HITS data involved more than simply multiplying the raw exposure data by the risk curve. For example, the CT estimate of injury risk for a peak acceleration of 85 g was 2.6%. There were 672 head impacts recorded with a severity of 85 g or greater. If the 2.6% risk were simply multiplied by the 672 exposures, then at least 17 concussions would be expected amongst those 672 head impacts. In actuality, only 2 occurred. It was therefore necessary to account for the fact that each head impact did not represent an independent exposure with respect to injury risk because many of the same players experienced multiple impacts. This problem was dealt with by assuming that each player had a fixed injury tolerance that could be characterized in terms of peak resultant linear head acceleration or HIC. Head impact exposure was therefore recalculated to reflect the distribution of the most severe impact experienced by each player, rather than the distribution of all impacts. The experimental data generally supported the assumption of a fixed injury tolerance for each player, in that two of the three injurious impacts recorded by the HIT system were the most severe impact experienced by the concussed player, and the third was among the most severe. It is possible that an individual's tolerance to head impact may vary somewhat from game to game, depending on physiological and environmental conditions, but the assumption of a fixed injury tolerance provides the most conservative estimate when calculating injury risk. One would also expect a continuous gradation of brain injury severity as a function of head impact severity, rather than a jump from no injury to injury at a particular severity level. In fact, clinicians do recognize a graduated progression in brain injury severity (e.g., from AIS 1 – 6). However, diagnoses at each severity level (i.e., AIS 1) are categorical, and therefore concussion has been treated as a binary event in the present study.

The accuracy of the HITS data and of the statistical analysis was assessed in various ways. The error deconvolution technique demonstrated that error due to scatter can alter the measured distribution of head impacts, in spite of the fact that the scatter error is unbiased. This technique quantifies the intuitive notion that the very highest of a large number of impacts are statistically more likely to be due to high end scatter than low end scatter. The implication is that the very highest measurements in the HITS data are more likely to be overestimates of the true value than the rest of the data. However, it was shown that error due to scatter must be large to substantially alter the final results of the analysis.

In addition, the HITS data were validated against biomechanical and epidemiological incidence data. The validation against epidemiological incidence data is primarily a test of internal consistency rather than a test of accuracy (Figure 8). For example, it would be possible for the HITS data to be erroneous and

yet accurately predict the expected number of concussions per game, because a shift towards lower or higher severities in the exposure data would be offset by a shift to a much more or less conservative risk function. However, the only way to reconcile the NFL risk curves with the NFL concussion incidence is to assume that the HITS data overpredicted head impact severity by a factor of roughly 100. Given that the HITS data have been shown to be reasonably accurate in validation testing, it must be concluded that the NFL risk curves are far too conservative.

The biomechanical incidence data were also compared to the NFL study, which is an external source of data. The injurious impacts in the HITS data were associated with significantly higher peak accelerations than the injurious impacts in the NFL data ($p < 0.05$), and with nonsignificantly higher HIC values than the NFL data ($p > 0.05$). This finding is interesting, because validation testing of the HIT system with Hybrid III dummies has shown that the results of the two measurement systems generally agree. The disparity between the HITS injury data and the NFL injury data could be due to error in the HITS data, error in the NFL data, and/or a true difference in the injury tolerance between college and professional football players. On the one hand, the helmet-mounted accelerometers in the HIT system may not measure head CG acceleration as accurately as the CG accelerometers in a dummy. Validation testing suggests that this error is small. On the other hand, the HIT system records the actual injury event as measured on the actual injured person, as opposed to Pellman et al.'s approach of measuring a reconstructed event in a dummy. The NFL study was subject to errors inherent in reconstructing a head impact from 30 Hz video using Hybrid III dummies, which have a different size, body mass, head shape, and neck response than the injured individuals. The errors associated with the video reconstruction aspect of the methodology were analyzed in depth by Newman et al. (2005) and found to be small. Lastly, there is the possibility that the disparity in the two injury data sets reflects the fact that college football players truly do have a higher concussive injury tolerance than professional football players, perhaps due to their youth or a less extensive history of previous concussions.

The implication of the MTBI risk analysis performed in the present study is that living humans, at least in the setting of collegiate football, can sustain much more severe head impacts without apparent injury than previously thought. Although the current CT risk curve is a coarse staircase function that only characterizes the low end of the risk curve, it is clear that future risk curves that will be refined with more injury data will also be radically different from the NFL risk curves. In fact, the CT risk curve for AIS 1+ injury as a function of HIC is not very different from the AIS 3+ HIC risk curve developed by Prasad and Mertz (1985). This unexpected similarity may be explained by the difference in the sources of the biomechanical data used to derive each risk curve. The CT risk curve is based on concussion data from helmeted football players, whereas the traditional HIC risk curve is based largely on cadaver head impacts into unpadded surfaces that resulted in skull fracture. The results of the present study imply that for a given HIC level, the risk of concussion in a padded head impact may be similar to the risk of skull fracture in an unpadded impact. Although the primary benefit of padding is that it attenuates the impact pulse and reduces the HIC level for a given head impact, padding has the additional benefit of distributing the impact load over a broad contact area, thus reducing the peak stress on the skull. Therefore, it is reasonable to expect that for the same level of head acceleration and HIC, a padded impact may be less likely to cause skull fracture than an unpadded impact.

CONCLUSIONS

The risk of MTBI in living humans was characterized by analyzing a large set of head impact data taken from college football players using the HIT system. The methodology of collecting head impact data in real time from players with instrumented helmets allowed for an unbiased estimate of the distribution of head impacts that players are exposed to on the field. A newly developed error deconvolution method showed that measurement error in the HITS data due to scatter probably does create significant error in the distribution estimate. The HITS data suggest that living humans can sustain much more severe head impacts without apparent injury than previously thought. Although the risk curves developed in the present study specifically reflect the risk of MTBI in helmeted college football players, they may have applicability to padded impacts in the general population, as well. These risk curves may be useful in improving helmets, padding, and other countermeasures to reduce the incidence of MTBI in sports, car crashes, and other settings.

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DISCUSSION

PAPER: **Development of Concussion Risk Curves Based on Head Impact Data from Collegiate Football Players.**

PRESENTER: ***J. R. Funk, Biodynamic Research Corporation***

QUESTION: *Guy Nusholtz, Daimler Chrysler*

Your results are extremely interesting, but if I—when I look at your acceleration and you HIC data, you've got HICs way out with no injury and you've got low HICs without concussion and low HICs with concussions. It almost looks like HIC and acceleration are not explanatory variables. They just happen to randomly show up under certain conditions. Have you considered that that might be the case? Even though you've got a prediction of the number of concussions, have you thought that maybe you've got more—your results are a little more profound than what you're saying that you may show that in this case HICs and g's are not really explanatory variables?

ANSWER: Well, that's a good point and that's one explanation. This is a statistical study. It's not a biomechanical study per se. It's only going to show you a correlation. It's not going to prove causality, you know, whether peak or g or rotational or HIC is the best predictor. Another explanation is that there's a range of tolerances out there, and the guys that get concussed have a lower tolerance than the ones that don't and therefore, you know, you see this a lot of times in other biomechanical data, too.

Q: Yes. Thank you.

A: Thanks.