ABSTRACT

Head rotation as a mechanism for brain injury was proposed back in the 1940s. Since then a multitude of research studies by various institutions were conducted to confirm/reject this hypothesis. Most of the studies were conducted on animals and concluded that rotational acceleration sustained by the animal’s head may cause axonal deformations large enough to induce their functional disruption. Other studies utilized mathematical models of human and animal heads to derive brain injury criteria based on deformation/pressure histories computed from the models. This study differs from the previous research in the following ways: first, it uses a detailed mathematical model of human head validated against various human brain response datasets; then establishes physical (strain and stress based) injury criteria for various types of brain injury based on scaled animal injury data; and finally, uses dummy (Hybrid III, ES-2re, WorldSID; all 50th percentile male) test data to establish kinematically (rotational accelerations and velocities) based brain injury criterion (BRIC) for each dummy. Similar procedures were applied to the college football data where thousands of head impacts were recorded using a six degrees of freedom (6 DOF) instrumented helmet system. Since animal injury data used in derivation of BRIC were predominantly for diffuse axonal injury (DAI) which is an AIS 4+ injury, cumulative strain damage measure (CSDM) was used to derive BRIC risk curve for AIS 4+ brain injuries. The AIS 1+, 2+, 3+, and 5+ risk curves for CSDM were then computed using the ratios between corresponding risk curves for head injury criterion (HIC) at a 50% risk. The risk curves for BRIC were then obtained by setting its value to 1 such that it corresponds to 30% probability of DAI (AIS4+). The newly developed brain injury criterion is a complement to the existing HIC which is based on translational accelerations. Together, the two criteria may be able to capture most brain injuries and skull fractures occurring in automotive or any other impact environment. One of the main limitations for any brain injury criteria, including BRIC, is the lack of human injury data to validate the criteria against, although some approximation for AIS 2+ injury is given based on the estimate of average injurious (concussion) angular velocities and accelerations for the college football players instrumented with 5 DOF helmet system. Despite the limitations, a new kinematic rotational brain injury criterion – BRIC – may offer additional protection to an automotive occupant in situations when using translational accelerations based HIC alone may not be sufficient.

INTRODUCTION

According to the Centers for Disease Control (CDC) traumatic brain injury (TBI) is an important public health problem in the United States. TBI is frequently referred to as the “silent epidemic” because the complications from TBI, such as changes affecting thinking, sensation, language, or emotions, may not be readily apparent. The most recent CDC report (Frieden et. al, 2010) estimates 1.7 million people sustain a TBI annually, of them 52,000 die. The report finds that among all age groups, motor vehicle-traffic (MVT) was the second leading cause of TBI (17.3%) and resulted in the largest percentage of TBI-related deaths (31.8%).

Based on NASS-CDS analyses of frontal crashes (Eigen and Martin, 2005) fatalities attributable to head injuries are second only to fatalities attributable
to thoracic region (Figure 1) with societal costs exceeding $6 Billion.

![FIGURE 1. Cost and fatalities attributable to injuries in frontal crashes (Eigen and Martin 2005).](image)

Many attempts have been made in the past to reduce the occurrence and severity of TBI as a result of automotive crashes. Among them are design and development of improved safety systems governed by various Federal Motor Vehicle Safety Standards (FMVSS), requirements of the New Car Assessment Program (NCAP), tests of Insurance Institute for Highway Safety (IIHS), and others. However, despite of all these requirements TBI is still one of the most frequent injury types in MVC (Figure 1). The reasons for this may be multiple: (1) the mandatory and voluntary requirements may not capture some real world crash scenarios leading to TBI, (2) the test dummies used in the tests are not interacting with vehicle environment in the way humans do, and (3) the interpretation of the dummies’ measurements is not sufficient to capture all possible types of TBI.

It is reason 3 that is investigated in this paper with the focus on the most frequent type of TBI – diffuse axonal injury (DAI). First, we make use of the scaled animal data (Abel et al., 1978; Gennarelli et al., 1982; Stalnaker et al., 1977; Nusholtz et al., 1984; Meaney et al., 1993) along with the NHTSA developed finite element (FE) model of human brain, e.g. the simulated injury monitor (SIMon) and its biomechanical injury criterion for DAI – cumulative strain damage measure (CSDM) (Takhounts et al., 2003 and 2008). Then, assuming DAI and its biomechanical equivalent - CSDM to be an AIS 4+ injury (AAAM, 2005), the risk curves for CSDM are scaled to AIS 1+, 2+, 3+, and 5+ using ratios between the risk curves similar to those developed for HIC (FMVSS 208) at 50% risk. These scaled CSDM risk curves represent various severities of concussive injuries. For example, AIS 3+ risk curve is a risk of severe concussion with the loss of consciousness 1-6 hours (AAAM, 2005). Finally, kinematic brain injury criteria (BRIC) were developed for each tested dummy (Hybrid III, ES2-re, and WorldSID) as well as human volunteers based on college football data.

**METHODS**

The SIMon model was tested using available experimental animal injury data, including rhesus monkeys (Abel et al., 1978; Gennarelli et al., 1982; Stalnaker et al., 1977; Nusholtz et al., 1984), baboons (Stalnaker et al., 1977), and miniature pigs (Meaney et al., 1993). A total of 114 animal brain injury experiments were simulated in the development of the biomechanical injury metric - CSDM. The experimental kinematic loading conditions were scaled in amplitude and time to satisfy the equal stress/velocity scaling relationship, i.e., translational velocity scaled as $1$, angular velocity as $1/\lambda$, and time scaled as $\lambda$, where $\lambda$ is the scaling ratio (Takhounts et al., 2003). Once correctly scaled, these loading conditions were applied to the SIMon model. The SIMon FE model consists of 42,500 nodes and 45,875 elements, of which 5153 are shell elements (3790 rigid), 14 are beam elements, and 40,708 are solid elements (Takhounts et al., 2008). Major parts of the brain were represented: cerebrum, cerebellum, brainstem, ventricles, combined CSF and pia arachnoid complex (PAC) layer, falx, tentorium, and parasagittal blood vessels (Figure 2).
It was assumed that the injury results from animal subjects were the same as that which would be observed from a human under the equivalent impact input.

CSDM is based on the hypothesis that DAI is associated with the cumulative volume of brain tissue experiencing tensile strains over a predefined critical level. The CSDM metric predicts injury by monitoring the accumulation of strain damage. This is accomplished by calculating the volume fraction of the brain which sometime during the event is experiencing strain levels greater than various specified levels. This strain level is based on the maximum principal strain calculated from a strain tensor that is obtained by the integration of the rate of deformation tensor (Bandak and Eppinger, 1995). The cumulative nature of the CSDM means that the strain damage at the end state of a calculation may be related to the DAI associated with a particular loading regime. To select the critical values of strain and volume for the CSDM injury metric, data from animal experiments conducted by Abel et al. (1978), Stalnaker et al (1977), Nusholtz et al., (1984), and Meaney et al. (1993) was used to relate the CSDM levels to the observed occurrence of DAI.

The risk curve for CSDM was constructed using survival analysis (Weibull distribution, left/right censored data):

\[ \text{Injury Risk} = 1 - e^{-\frac{\text{CSDM}}{\lambda}}^k, \]  

where \( \lambda \) is scale and \( k \) is shape parameter for Weibull distribution. In the case of CSDM, \( \lambda = 0.6162 \) (st. err. 0.0431), and \( k = 2.7667 \) (st. err. 1.0302), Max Loglikelihood = -31.7.

This injury risk curve (Eq. 1) would correspond to AIS 4+ brain injury according to the recently published AIS scale (AAAM, 2005) for DAI. To obtain other levels of the abbreviated injury scale, the risk curves for HIC were used (The U.S. Department
To achieve Transportation’s FMVSS No. 208 Final Economic Assessment, assuming equal severity ratios between corresponding risk curves for HIC and CSDM at 50% risks. For example, to obtain AIS3+ risk curve for CSDM, the ratio ($\beta_{34}$) of AIS3+/AIS4+ risk curves at 50% for HIC was found, and then AIS4+ risk curve for CSDM at 50% was multiplied by this ratio to find 50% risk point for the AIS3+ CSDM:

$$\text{CSDM AIS3+ (50%) } = \beta_{34} \times \text{CSDM AIS4+ (50%)}. \tag{2}$$

Using Eqs. 1 and 2 together the CSDM risk curve for AIS3+ was found. Other risk curves for CSDM were found in the similar fashion.

Next, frontal impact tests with the Hybrid III dummy (43 NCAP tests - drivers and passengers - available from NHTSA database), 31 side impact tests with ES-2re test dummy, and eight side impact tests with WorldSID test dummy (all were 50th Percentile male sized) were used to develop BRIC for each dummy. To do that, first, CSDM values were calculated for each test. Then optimization was carried out to obtain the best linear fit between CSDM and BRIC (in the form of equation 3) using critical values of angular velocity and acceleration $\omega_{cr}$ and $\alpha_{cr}$ as design variables and subjected to the constraint that BRIC =1 when CSDM =0.425 (30% probability of DAI/AIS4+).

$$\text{BRIC } = \frac{\omega_{max}}{\omega_{cr}} + \frac{\alpha_{max}}{\alpha_{cr}}, \tag{3}$$

where $\omega_{max}$ and $\alpha_{max}$ are maximum angular velocities and accelerations for each test respectively. The linear relationship between CSDM and BRIC was then utilized to obtain risk curves for each dummy.

Similarly to the procedure above, BRIC was developed based on translational and rotational data obtained from the college football players. Between 2007 and 2008, the helmets of 19 Virginia Tech football players were instrumented with a custom 6 degree of freedom (6DOF) head acceleration measurement device (Rowson et al, 2009). The measurement device consists of 12 accelerometers and recorded linear and angular acceleration about each axis of the head using a novel algorithm (Chu et al, 2006). Any time an accelerometer exceeded 10 g during play, data acquisition was automatically triggered and data were collected for 40 ms (including 8 ms of pre-trigger data). Once data collection was complete, data were wirelessly transmitted to a computer on the sideline. Linear and angular head accelerations were recorded for a total of 4709 head impacts of which 362 had peak resultant linear accelerations greater than 40 g. To determine resultant angular velocity, angular acceleration about each individual axis of the head was numerically integrated throughout the entire acceleration trace. Resultant angular velocity was then calculated. Each impact was visually inspected so that the angular acceleration (and resulting angular velocity) pulse of interest could be examined and peak values identified. Once peak angular acceleration and peak angular velocity were determined for each impact, a linear regression analysis was performed using a least squares technique. The regression model was constrained so that an angular acceleration of 0 rad/s² resulted in an angular velocity of 0 rad/s. Although none of the 6 DOF impacts resulted in brain or other head injury, CSDM and BRIC curves were computed to assess the potential for TBI.

To evaluate BRIC for college football players, concussive data were generated using the commercially available 5 DOF HIT System (Simbex, Lebanon, NH). This head acceleration device consisted of 6 accelerometers and measure resultant linear acceleration of the head. This device is limited in that only peak angular acceleration can be estimated from an assumed pivot point in the neck. Resultant angular velocities for concussive data points were estimated from resultant angular accelerations using a regression model. Details of the methods used for data collection can be found in the literature (Duma et al, 2005, Duma et al, 2009). Using the HIT System, head acceleration data were recorded for 6 concussions between 2003 and 2008 (Duma et al, 2009). These 6 concussions were combined with concussive data collected from published studies that utilized identical data collection methods (Broglio et al, 2010, Guskiewicz et al, 2007). This resulted in a dataset of 32 concussions.
RESULTS

Figure 3 illustrates the probability of DAI as a function of CSDM along with the 95% confidence intervals.

The ratios $\beta_i$, where $i$ is the level of AIS of interest, are given in Table 1.

Table 1. Ratios for computing risk curves for AIS 1, 2, 3, and 5 based on known risk curve for AIS 4.

<table>
<thead>
<tr>
<th>$\beta_{14}$</th>
<th>$\beta_{24}$</th>
<th>$\beta_{34}$</th>
<th>$\beta_{54}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1003</td>
<td>0.5003</td>
<td>0.8156</td>
<td>1.0411</td>
</tr>
</tbody>
</table>

Probability of brain injuries as functions of CSDM for various AIS levels are shown in Figure 4.

The following three charts show the probabilities of brain injury as functions of BRIC for each AIS level for Hybrid III (Figure 5), ES-2re (Figure 6), and WorldSID 50th percentile male dummies (Figure 7).

The critical values of angular velocity and acceleration for the Hybrid III dummy were found to be $\omega_{cr} = 46.41 \text{ rad/s}$ and $a_{cr} = 39,774.87 \text{ rad/s}^2$, $R^2 = 0.38$. 

FIGURE 3. Risk of DAI (AIS 4+) as a function of CSDM based on animal injury data.

FIGURE 4. Risk of brain injuries as a function of CSDM for various AIS levels.

FIGURE 5. Risk of brain injuries as a function of BRIC for various AIS levels for Hybrid III.
The critical values of angular velocity and acceleration for the ES-2re dummy were found to be $\omega_{cr} = 65.68 \text{ rad/s}$ and $\alpha_{cr} = 23,063.90 \text{ rad/s}^2$, $R^2 = 0.70$.

The critical values of angular velocity and acceleration for the WorldSID dummy were found to be $\omega_{cr} = 153.18 \text{ rad/s}$ and $\alpha_{cr} = 11,527.92 \text{ rad/s}^2$, $R^2 = 0.94$.

For college football players, peak angular acceleration and peak angular velocity correlated strongly ($R^2 = 0.96$), proving to be a linear relationship. This suggests that most impact pulses in football are similar in duration and acceleration shape. For non-injury data points, the average angular acceleration was $2,404.00 \text{ rad/s}^2$ and the average angular velocity was $10.00 \text{ rad/s}$. The average concussive angular acceleration was $6,572.00 \text{ rad/s}^2$ and the average concussive angular velocity was $28.00 \text{ rad/s}$.

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Figure 8 shows BRIC criterion for the college football players. The critical values of angular velocity and acceleration for the college football players were found to be $\omega_{cr} = 42.05 \text{ rad/s}$ and $\alpha_{cr} = 363,268.91 \text{ rad/s}^2$, $R^2 = 0.81$.

It should be noted that the high intercept value for angular velocity for WorldSID and high value of intercept for angular acceleration for human football data compared to those of Hybrid III and ES-2re, are due to high correlation between the angular velocities and angular accelerations for these two datasets. In these cases, the optimizer usually chooses one of the parameters in the optimization process and “makes” the other one irrelevant (very high value).

DISCUSSION

The importance of head rotational kinematics as a mechanism for brain injuries has been discussed in the scientific literature since the 1940s (Holbourn, 1943, Gennarelli et al. 1972, Ueno and Melvin 1995). More recently, Hardy et al, 2001 and 2007, in the experiments describing the motion of brain with respect to the overall motion of the skull, noted that
angular velocity was the most “convenient” measure in describing relationship between brain and skull kinematics. Takhounts et al (2008) described that one of the ways to deform/strain a soft, nearly incompressible material (brain) contained within an almost undeformable shell (skull) is to rotate the shell.

Despite the overwhelming evidence of the head rotational kinematics to be a mechanism for brain injuries, the difficulty was in relating animal injury data (Abel et al., 1978; Gennarelli et al., 1982; Stalnaker et al., 1977; Nusholtz et al., 1984; Stalnaker et al., 1977; Meaney et al., 1993, Ommaya, 1985) to the potential for brain injuries in humans. One possible way to accomplish this is to find injury criteria for animals and then scale it to humans using various scaling relationships (Ommaya, 1985). The advantage of this approach is in its simplicity – it is straightforward and a criterion is easily computed. The disadvantage of the approach is also in its simplicity as it doesn’t necessarily address the equivalency of the brain deformations (believed to be the primary cause of TBI) inside the brains of animals and humans. Another approach for relating animal injury data to humans is to develop FE models of animals and humans, find a scaling relationship between the two (Takhounts et al, 2003), and then develop a deformation/strain based criterion (CSDM) that would be equally applicable for both animals and humans. The advantage of this approach is that it gives a link between deformation fields inside the brains of animals and humans and thus may be more physically/biomechanically justifiable. The disadvantage of the approach is that it requires a powerful computer and several hours of run time to calculate CSDM. Both approaches suffer, however, from the lack of knowledge of how the injury severity in animals would translate to the injury severity in humans given equivalent loading conditions.

A second approach was adapted in this paper where an already developed and validated finite element model of human head – SIMon – was employed along with its injury criterion for DAI – CSDM (Takhounts et al, 2008). Once CSDM was computed and scaled for various AIS levels (Eq. 2), BRIC was calculated for each tested dummy and college football players in the form of equation 3 where it was set to the value of 1 to correspond to 30% probability of DAI (AIS 4+ injury). There are many different ways of obtaining BRIC from CSDM, but the chosen value of 1 corresponding to 30% of DAI indicates that the closer the BRIC is to the value of 1 a worse outcome can be expected.

BRIC is a correlate, not a fundamental property of a system (like CSDM), hence it was anticipated that different dummies (and humans) will have different relationships of BRIC to CSDM (figures 5 – 8) even when they all are “forced” through the same point in the BRIC vs. CSDM relationship, e.g. point (0.425, 1), where CSDM = 0.425 corresponds to 30% probability of DAI (Fig. 3). This difference is due to different values of slopes and intercepts in the assumed linear relationship between CSDM and BRIC for different dummies and humans, which, in turn may be caused by the difference in impact conditions and properties of the neck.

If concussion is assumed to be a mild form of DAI, then figures 5 – 8 could be used in assessing concussion as an AIS 2+ injury (AAAM, 2005). For example, 30% probability of concussion in college football players will give BRIC equal to 0.67, which at the same time gives approximately 5% chances of DAI. Substituting the average values of angular acceleration and velocity for concussed players into BRIC for football players gives the concussed value of BRIC equal to 0.68. Referring to Figure 8 this value of BRIC gives about 33% probability of concussion (AIS 2+ risk curve) or 5% probability of DAI (AIS 4+ risk curve). From the same Figure 8, the risk of AIS 3+ TBI for BRIC = 0.68 is approximately 10%. This risk is probably the upper limit of what a regular human (not a trained athlete) should be allowed to experience when protecting against concussion. The risk of AIS 3+ TBI for BRIC = 0.68 when using the Hybrid III dummy as an assessment tool (Figure 5) is approximately 1%, when using ES-2re dummy it is approximately 7% (figure 6), and when using the WorldSID it is about 8% (figure 7). This illustrates that the values of BRIC should be used in conjunction with the injury assessment device (dummies or humans) it is measured with.
Ommaya (1985) gave an overview of the rotational injury tolerance values for the onset of concussion based on the research conducted on rhesus monkeys and chimpanzees. The human rotational tolerances were obtained using a mass scaling relationship for angular accelerations (inversely proportional to the two-thirds power of the brain mass) giving angular velocity and acceleration tolerances for human of 20 – 30 rad/s and 1,800 rad/s^2 respectively. Inserting these tolerance values into Eq. 3 and using critical values obtained from college football data for humans will give BRIC values between 0.48 (for angular velocity of 20 rad/s) and 0.72 (for angular velocity of 30 rad/s). Referring to the AIS2+ risk curve for humans (Figure 8) will give a risk of concussion ranging from 3% - 41% depending of the chosen tolerance value of angular velocity. Taking an average angular velocity of 25 rad/s will give BRIC equal to 0.60 and 17% risk of AIS2+ injury. The BRIC of 0.68 obtained from football data is within the range of those obtained from scaling animal data and is closer to the upper limit of 0.72.

Several approaches may be taken if BRIC is used in an automotive environment. One of them is to restrict BRIC for each injury assessment device to be no greater than the value at the respective 30% risks of AIS 3+ TBI (similar to HIC). This approach will give critical value of BRIC for the Hybrid III equal to 0.92, for ES-2re and WorldSID equal to 0.89.

The limitations of this study are multiple.

- First, all the limitations that were applicable in the development and validation of SIMon finite element head model (Takhounts et al, 2003, 2008) are applicable to this paper as well. In addition, correlation between CSDM and BRIC is not perfect that will add additional errors to the injury risk estimates. It should be noted, however, that similar limitations are applicable to any research – computational and/or experimental.
- Second, only DAI was investigated in this study. Inclusion of other types of TBI, such as focal lesions, contusions, or hematomas, may change the relationship for BRIC. However, BRIC is not an “ultimate” head injury criterion that captures all possible brain injuries and skull fractures, but rather a correlate to TBI with head rotation being a primary injury mechanism.
- Third, deriving CSDM and BRIC risk curves for various AIS levels based on ratios between 50% risks for different AIS levels for HIC assumes that rotationally induced injury severities change proportionally to those induced translationally. This assumption may or may not be correct, but due to lack of any data on rotational based changes in injury severity this assumption provides a “first approximation” of these changes.
- Fourth, although very valuable, the college football data has its own limitations: athletes are trained to sustain higher loads, the average concussed angular velocity and acceleration were calculated from the 5 DOF measuring system rather than measured directly by the 6 DOF system, thus the accuracy of these values may be questioned.
- Fifth, regarding scaling of the animal tolerances to those of humans, it is interesting to give a quote from Ommaya (1985): “It should be reemphasized that this information (rotational tolerances) is considered to be reliable for the Rhesus, sketchy for the chimpanzee, and completely speculative for man.” He then suggests revising the human rotational tolerances when the data from accident reconstruction in humans become available. College football data may be considered as one of these “accident reconstruction” data.
- Finally, BRIC is a rotational injury criterion (see second limitation), while HIC is a translational injury criterion (calculated using translational accelerations only), and combining the two may offer better protection from head injuries. However, a human head is rarely experiencing just rotational or just translational motion. It usually is experiencing both. This paper does not address this combination of both modes of motion and corresponding injury mechanisms. This has been proposed by
others, but additional work to derive a relationship is required.

Despite the limitations that are inherent in any research, this paper provides valuable information on the importance on limiting rotational kinematics of the human head that may be beneficial to both – athletes and general driving population.

CONCLUSIONS

A kinematic rotational brain injury criterion – BRIC – was developed for three 50th percentile test dummies (HIII, ES-2re, and WorldSID) and human athletes. Following are the conclusions:

- BRIC is different for different dummies and human athletes.
- Concussive (AIS 2+) values of BRIC for humans varied from 0.60 when scaled directly from animal data (Ommaya, 1985) to 0.68 when obtained directly from the college football players.
- The risk of AIS 3+ TBI for BRIC = 0.68 when using the Hybrid III dummy as an assessment tool is approximately 1%, when using ES-2re dummy it is approximately 7%, and when using the WorldSID it is also about 8%.
- BRIC for the 30% risk of AIS 3+ TBI is 0.92 if measured with HIII dummy, 0.89 if measured with ES-2re and WorldSID dummies.
- BRIC should be used in combination with HIC. However, the risk of TBI for combination of rotational and translational loading modes should be investigated in the future.

REFERENCES


The U.S. Department of Transportation’s FMVSS No. 208 Final Economic Assessment.