TOYOTA

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March 24, 2008

Ms. Kathleen C. DeMeter, Director
Office of Defects Investigation
National Highway Traffic Safety Administration
Room W45-302
1200 New Jersey Avenue, S.E.
Washington, D.C. 20590

Re: Toyota Siennas with Power Liftgates; EA06-020;
Supplemental Response to Recall Request Letter

Dear Ms. DeMeter:

I am writing to supplement my February 25, 2008 response to your letter of January 25, 2008, in which you requested Toyota to conduct a safety recall to address an alleged safety defect in the performance of certain Toyota Sienna minivans equipped with power liftgates. Toyota recently noticed an error in that response that we wish to bring to your attention.

In my letter, I explained why the inability of the liftgate struts to hold the liftgate in the open position was extremely unlikely to lead to any injuries other than minor bumps and/or bruises. Among other things, I described testing conducted by Toyota using a Hybrid III 50% percentile male dummy fitted with a triaxial accelerometer that was positioned in a manner such that the liftgate would strike its head with maximum energy in the event of a total failure of one of the struts. The letter stated (at page 6) that since the peak acceleration did not exceed 35 G, there was a "very low probability of sustaining a concussion," based on well-accepted injury curves.

Toyota engineers recently discovered that they had mis-identified the units in the acceleration measurements in that testing. The units were actually expressed in meters/sec^2, as opposed to Gs. Thus, the actual peak acceleration of the dummy head in those tests was 33.5m/sec^2, which equates to 3.4 G, rather than value referred to in my letter. As can be seen from the data depicted in Figure 12 of the article by King, et al., referenced in my earlier letter,[1] the first occurrence of even a "Mild Traumatic Brain Injury" (MTBI) did not occur until an acceleration of about 500 m/sec^2 – which is more than 10 times the maximum acceleration that can be generated by the Sienna liftgate. Further, non-injury data points were recorded with accelerations as high as 1000 m/sec^2. In this context, an acceleration of 33.5 m/sec^2 (3.4G) is

[1] King, et al., "Is Head Injury Caused by Linear or Angular Acceleration?" IRCOBI, 2003. A copy of this article is enclosed for your convenience.

negligible, furthering confirming our previous assertions that the risk of injury is extremely low, and does not constitute an unreasonable risk.[2]

I would like to thank you in advance for your consideration of the information in this supplemental response. I regret the error in our measurements and the fact that we did not identify it earlier.

Sincerely,

Christopher Tinto
Vice President

Enclosure

[2] The King article also discussed (at page 7) a study of mild concussive events that occurred during National Football League games. A total of 53 cases were studied, of which there were 22 cases of concussion, as diagnosed by the team physician on site. The average linear acceleration for concussed and non-concussed players was 94±27 and 55±21 G, respectively. This data provides a further demonstration that the risk of injury associated with the 3.4 G experienced by the dummy in the Toyota testing is virtually non-existent.

IS HEAD INJURY CAUSED BY LINEAR OR ANGULAR ACCELERATION?

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ABSTRACT

Currently, angular acceleration is believed to be more damaging to the brain than linear acceleration, even though both are present in any head impact. In a recent experiment, it was found that a helmeted head sustained the same degree of angular acceleration as the unhelmeted head for the same impact, but its linear acceleration was decreased significantly. So, if angular acceleration is the cause of brain injury, then how is the brain protected by the helmet? This paper proposes a new hypothesis of brain injury and suggests that input acceleration limits should be replaced by response variables.

KEYWORDS: Head injury, linear acceleration, angular acceleration, strain, strain rate

TRAUMATIC BRAIN INJURY continues to be a serious societal problem affecting some 1.5 million Americans each year. An estimated 2% of the US population lives with disabilities resulting from a traumatic brain injury. Recent automotive injury data (NHTSA Special Cases Investigation database) reveal the surprising fact that, even with the airbag, minor traumatic brain injury cases are still being reported among drivers. It is perhaps unnecessary to provide a lengthy discussion regarding the significance of this public health problem but it is important to state that the precise mechanisms of brain injury have not been fully established and methods of prevention cannot be fully effective if we do not know the cause.

REVIEW OF BRAIN INJURY MECHANISMS

Zhang et al. (2001a) summarized the state of knowledge regarding brain injury mechanisms at the turn of the century. Injury mechanisms described in this paper were those responsible for the acute injury sustained at the time of impact and are an attempt to explain the immediate mechanical and physiological damage that result in functional and anatomical changes. Although the proposed mechanisms are still principally at the hypothesis level, the more information we have on injury mechanisms, the better are we able to provide means of protection against brain injury. In general, an individual mechanism should produce a specific type of brain injury. Over the years, a number of studies have been performed that have improved our understanding of brain injury. The studies point to brain deformation or strain as a principal cause of injury. Unfortunately, the measurement of strain is almost impossible during an impact, particularly *in vivo*. Therefore, input variables, such as head acceleration, are used as alternate parameters to characterize the injury mechanism.

Head injury typically results from either a direct impact to the head or from an indirect impact applied to the head and neck when the torso is stopped or accelerated rapidly. In either case, the head sustains a combined linear and angular acceleration. Although skull fractures are generally absent in indirect loading, severe brain injury was found in experimental animals. Currently, impacts with head contact (predominantly linear acceleration) and inertial loading of the head (predominantly rotational acceleration) have been postulated as the two major mechanisms of head injury. Rotational acceleration is considered to produce both focal and diffuse brain injuries, while linear acceleration

produces focal brain injuries. These two theories of head injury are discussed below. In addition, hypotheses for the mechanism of concussion are also presented under a separate heading.

LINEAR ACCELERATION:

Gurdjian, Lissner and co-workers (1945, 1955, 1961,1963) attributed intracranial damage to deformation of the skull and pressure gradients caused by skull deformation and acceleration of the head due to direct impacts to the head. Linear acceleration was considered to be the most important mechanism, while rotational acceleration, negative pressure and cavitation were of minimal or no significance. Ommaya et al. (1966) indicated that rotation alone could not produce the levels of injury caused by direct impact. About twice the rotational velocity was required to produce cerebral concussion by indirect impact (whiplash). Later, Ommaya and Hirsch (1971) suggested that rotation could account for approximately 50 percent of the potential for brain injury, while the remainder was attributed to direct impact. Gennarelli et al. (1971,1972) demonstrated that translation of the head in the horizontal plane produced essentially only focal effects, resulting in well-circumscribed cerebral contusions and intracerebral hematomas, while diffuse injuries were seen only when a rotational component was present. In particular, some of the contusion seen under purely linear loading suggested that the intracerebral cavitation mechanism was most probably due to rupture of blood vessels. Unterharnscheidt (1971) studied the role of linear and angular acceleration in producing brain injury. The principal mechanism of purely linear acceleration appears to be pressure gradient, while that for purely rotational acceleration appears to be shear stress, which results from differential motion between the skull and brain. Ono et al. (1980) conducted a series of experiments with monkeys and found no correlation between the occurrence of concussion in monkeys and angular acceleration. It was concluded that the concussion could be produced by linear acceleration from a direct impact.

ROTATIONAL ACCELERATION:

Holbourn (1943) was the first to cite angular acceleration with or without direct impact as an important mechanism in head injury. It was hypothesized that shear strain and tensile strain generated by rotation alone could cause cerebral concussion as well as contrecoup contusion. Lowenheilm (1975) proposed angular acceleration as the cause of gliding contusion resulting from excessive strain in cerebral vessels. It was concluded that the site of maximum shear occurred at a constant distance from the surface of the brain. It was also stated that the deep brain could be injured while the surface was not injured and that the zone of maximum shear became deeper as the angular acceleration pulse duration increased. Gennarelli, Thibault, and co-workers, in a series of studies (Gennarelli et al., 1971,1981, 1982, Gennarelli and Thibault, 1982, and Thibault and Gennarelli, 1985), investigated the role of rotational acceleration in causing brain injury by using live subhuman primates and physical models. They concluded that angular acceleration contributes more than linear acceleration to the generation of concussive injuries, diffuse axonal injuries, and subdural hematomas. They hypothesized that these injuries were induced by the shear strain generated by angular acceleration and claimed that virtually every known type of head injury can be produced by angular acceleration. However, McLean (1995) argued that there were no cases of brain injury without head impact in his investigation of a series of more than 400 fatally injured road users. Even in non-fatal impacts, it is hard to imagine how the human neck can transmit enough energy to the head to cause brain injury without a direct impact to the head. Brain injury is not generally associated with neck injury.

CONCUSSION MECHANISMS:

The mechanism causing unconsciousness following an impact is not clearly understood. Various injury mechanisms have been proposed by researchers to explain their experimental results. Several hypotheses on the mechanism of cerebral concussion are listed below:

1) Shear strains generated by rotation cause cerebral concussion (Holbourn, 1943).
2) Extent of relative displacement due to impact from different directions (Pudenz and Sheldon, 1946).
3) Relative displacement between the brain and the skull produces coup/contrecoup cavitation (Gross, 1958).
4) Concussion occurs as a result of shear stress, distortion, or mass movement in the brain stem principally resulting from pressure gradients due to impact loading. Linear acceleration is the

most important mechanism while, rotational acceleration, negative pressure and cavitation are of minimal or no significance. (Gurdjian et al., 1955, 1961, 1963; Hodgson et al., 1969).

5) Pressure waves traveling through the brain cause cerebral concussion (Goldsmith, 1972).
6) Disturbance of consciousness is caused by strains affecting the brain in a centripetal sequence of disruptive effects on function and structure. The effects of this sequence always begin at the surface of the brain in the mild cases and extend inwards to affect the core at the most severe levels of trauma (Ommaya and Gennarelli, 1974).
7) Impact pulses containing frequencies that are close to the nodal frequencies of the skull/brain complex can cause injury due to resonance (Willinger et al., 1996).

COMMENTS:

Considerable controversy exists within the biomechanics community regarding the validity of competing hypotheses because they do not always correlate with clinical or pathological observations. Although both linear and angular acceleration can individually cause brain injuries in test animals, the severity of impact needed to produce these injuries is much higher than that experienced by humans involved in automotive type crashes. In fact, there is rarely an impact that is purely rotational or linear in the real world. However, moderate levels of both forms of acceleration combined can often cause severe brain injuries. Thus, more research is needed to demonstrate the validity of these hypotheses. Since there is no direct way to verify these hypotheses on living humans the research will continue to be carried out on laboratory animals, cadavers and/or through the use of computer models.

RECENTLY ACQUIRED BRAIN MOTION DATA

While relative motion between the brain and skull has been observed previously (Pudenz and Sheldon, 1946, Hodgson et al., 1966, Nusholtz et al., 1984), these studies involved either substantial alteration of the cadaver or animal specimen and qualitative observation of global motions. Recently Hardy et al. (2001) described a new methodology to acquire local relative motion data between the brain and the skull in an intact skull, using a unique high-speed biplane x-ray system and neutral density technology to measure brain deformation in human cadaveric heads. The techniques used to acquire the data will not be repeated here as they have already been described in detail by Hardy et al. (2001). However, a comparison of the data from frontal and lateral impacts is presented in this paper.

COMPARISON OF RESULTS:

Two different types of test were conducted. The head was either struck by a moving impactor or it was accelerated into a stationary block made out of acrylic. The latter tests had both large angular as well as linear acceleration components. After each test, the data were obtained with the help of an automated image enhancement and target tracking software program. The data were further reduced to express the motion of the targets with respect to a fixed point within the head, namely, the center of gravity of the head. For an impact to the frontal region of the head when it struck the fixed acrylic block, the results of which are shown in Figure 1, the linear acceleration peak was in excess of 60 g and the angular acceleration was above 2500 rad/s^2. Additional tests were carried out to reach linear and angular accelerations in excess of 200 g and 10,000 rad/s^2 respectively. An additional example is provided in Figure 2, which shows brain motion due to a side impact to the head. The commonality in the data shown here and in the data presented by Hardy et al. (2001) is the fact that the NDT's execute a figure eight pattern and that the magnitude of the relative motion of the brain relative to the skull is limited to approximately ± 5 mm, regardless of the magnitude and direction of the angular acceleration. It should also be noted that the motion due to linear acceleration is minimal, on the order of ± 1 mm at most. Upon enlarging one of those figure eight patterns (Figure 3), it can be seen that there is a small loop in the center of the response path. This is the only portion of the response that may be attributable to linear acceleration. If the components of this displacement are plotted as a function of time, it can be seen from Figure 4 that the magnitude is about 1 mm at the beginning of the impact when linear acceleration was predominant.

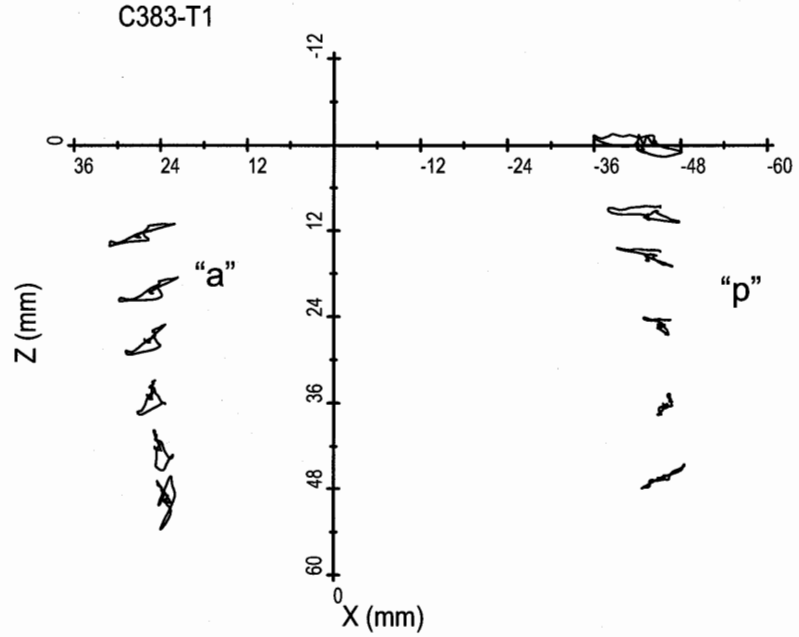


Fig. 1 - Sagittal plane brain deformation patterns at the NDT locations for test C383-T1, in which the frontal region of the moving head struck a fixed block.

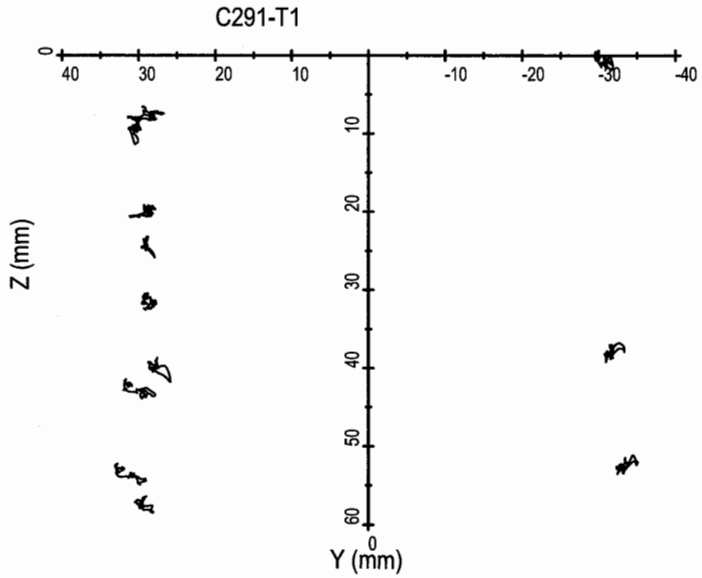


Fig. 2 - Coronal plane brain deformation pattern for test C291-T1, which was a right lateral impact to the head. The left side of the head (left side of the figure) has two columns of NDT's. One column is located anteriorly and one posteriorly. The right side of the head has a single column located posteriorly. Two targets in the middle of the right column could not be tracked. The displacement patterns are similar to those found in the sagittal plane.

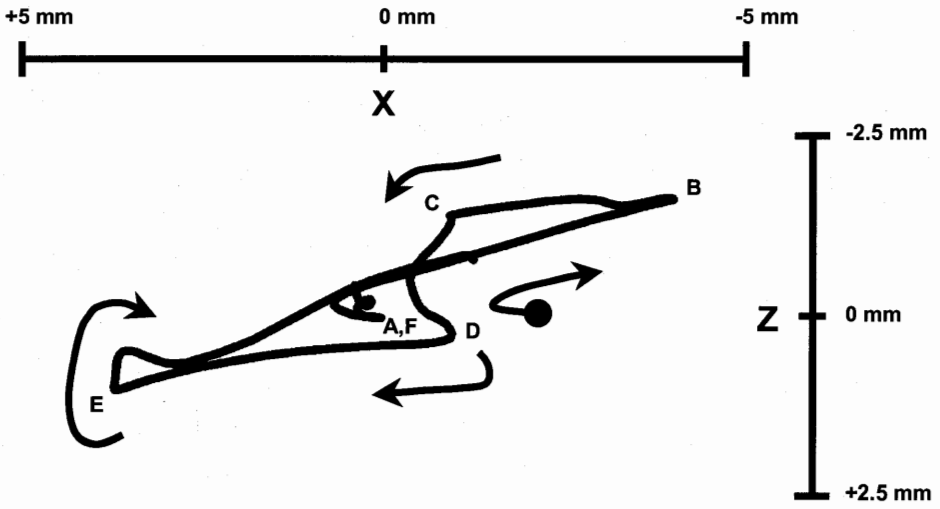


Fig. 3 - Details of the motion of a single NDT during a frontal impact (C383-T1). Arrows show the direction of motion and the letters along the path correspond to the letters in the time plot in Figure 4.

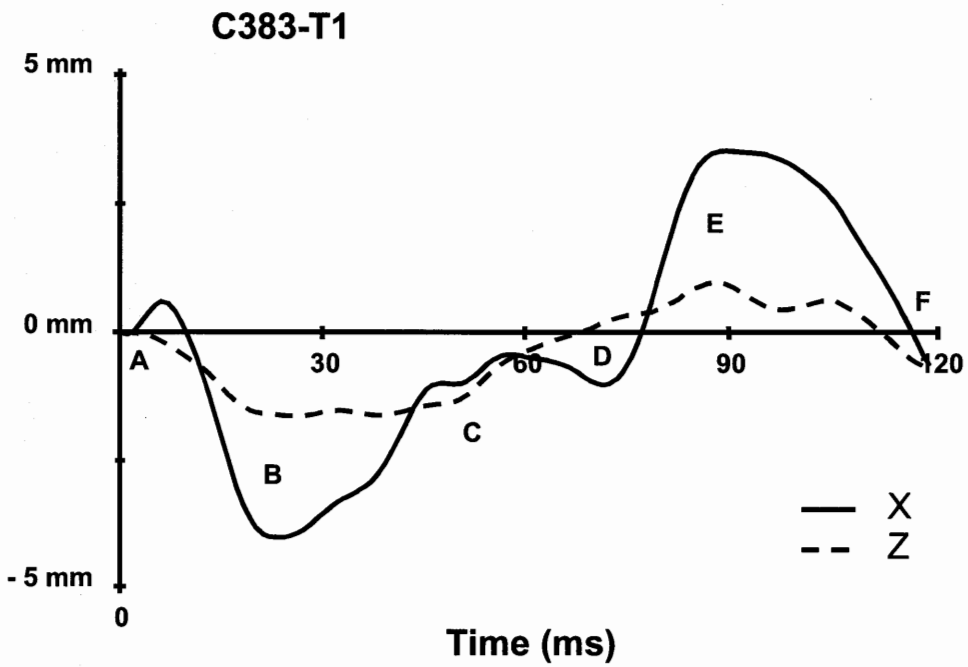


Fig. 4 - Time plot of X and Z relative displacement components of the NDT described in Figure 3.

EFFECT OF HELMETS ON ANGULAR ACCELERATION

A series of experimental studies was conducted to determine the effect of a football helmet using the head-neck complex of a Hybrid III dummy (Zhang et al., 2003b). The impact tests were conducted by mounting the head and neck of a Hybrid III dummy onto a custom-designed fixture that was attached to a pneumatically driven mini-sled (Figure 5). This fixture was designed to allow the head-neck complex to be oriented in three major impact directions (frontal, lateral, and front-boss or 45 degrees oblique) to simulate typical head collisions involved in football accidents. Tests were conducted both with and without a helmet on the dummy head, which impacted an angled foam block.

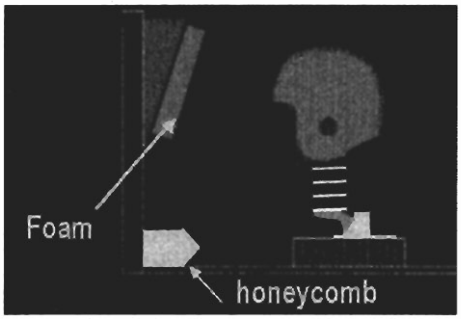


Fig. 5 - Schematic diagram of the mini-sled test configuration.

A Bike® and a Riddell® helmet were mounted on the Hybrid III head to study their performance. The pressure level for propulsion of the mini-sled was carefully adjusted so that it traveled at the desired impact speed. A laser velocity trap was used to record the impact velocity just prior to the head contacting the foam. Impact velocities of 5 and 7 m/s were selected for the bare head impacts and velocities of 7 and 10 m/s were selected for the helmeted head impacts. To stop the moving sled after the head has impacted the foam, a piece of appropriately sized Hexcel® honeycomb was used to absorb the kinetic energy of the sled. The foam block was attached firmly to a rigid barrier. The foam surface was sloped rearward at a 30-degree angle to the vertical or Z-axis of the head coordinate system to avoid engagement of the facial part of a Hybrid III head during impact. Five different types of foam material with varying densities were tested in order to compare their energy attenuation capabilities. The Hybrid III head was instrumented with a 3-2-2-2 nine-accelerometer array to measure head angular acceleration. The upper neck force and moment were also measured. The motion of the head during impact was captured by a high-speed video camera run at 1000 fps.

Of all types of foam material tested, it was clearly demonstrated that the acceleration measured at the center of gravity (c.g.) of the dummy head underwent a significant reduction, averaging 21% when the Bike helmet was used and 29% when the Riddell helmet was used (Figure 6). On the other hand, reduction of angular acceleration due to the addition of a helmet was not as obvious as that observed for linear acceleration. Figure 7 shows a comparison of angular accelerations for tests with and without a helmet. Instead of being reduced, angular acceleration increased in four of the nine foam materials tested using a Bike helmet and increased in two of the 9 foam materials tested using a Riddell helmet. Considering these results and the fact that the football helmet is capable of greatly reducing the frequency of head injury (Mueller, 1998), we are forced to conclude that the mechanism of head injury may not be linked to rotational acceleration as strongly as that suggested by early researchers (Gennarelli et al., 1972, Gennarelli and Thibault, 1982, Margulies and Thibault, 1992). Again, the numerical model we have developed may shed some light on the understanding of head injury mechanisms.

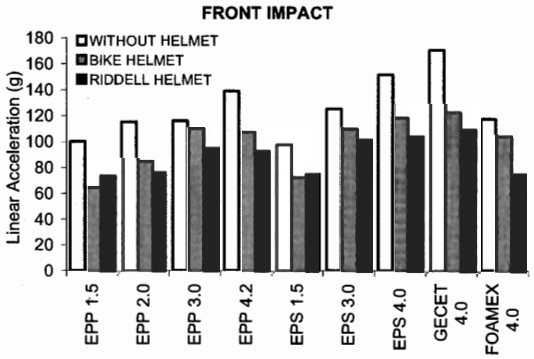


Fig.6 - Comparison of linear acceleration measured at the head c.g. without a helmet and with a Bike and Riddell helmet.

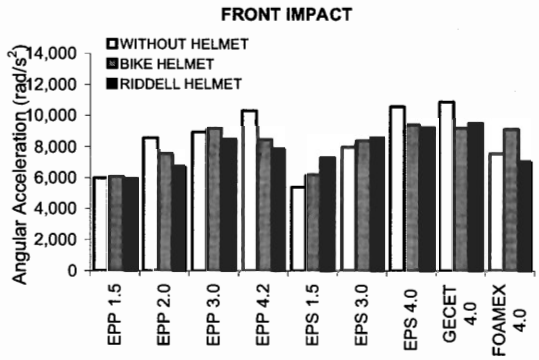


Fig.7 - Comparison of rotational acceleration without a helmet and with a Bike helmet, and Riddell helmet.

In view of the results presented above, we are forced to ask this question:
Just how does the helmet protect the brain if the prevailing thought is that angular acceleration is responsible for brain injury?

Associated with this question one can also ask the following:

Why is the relative motion limited to ± 5 mm, even at angular accelerations in excess of 10000 rad/s^2?

If the helmet protects the brain by lowering the peak linear acceleration and by increasing the impact duration and if the relative motion is on the order of ± 1 mm, then what mechanical parameter is the cause of brain injury?

A PROPOSED NEW HYPOTHESIS FOR BRAIN INJURY

We would like to propose a hypothesis regarding the cause of brain injury. That is, brain response governs injury and not the input acceleration. One response parameter which should be investigated is strain rate, particularly, in view of test results on single axons (LaPlaca et al., 1997 and Galbraith et al., 1993). For stretch, the experimental rate was found to be on the order of 500%/s. This is compared with a rate on the order of 400 to 900%/s based on our brain injury model (Zhang et al., 2001b). The measured strain rate was based on the larger motion due to rotation. The strain rate due to strain induced by linear acceleration may be very difficult to measure accurately because of the small magnitude of the strains involved. In the clinical literature (Strich, 1961), shear strain was considered to be a cause for diffuse axonal injury. It is not clear how an axon can be directly injured by shear but it is quite obvious that the measurement of shear strain and shear strain rate is a difficult task. The data presented in this paper should challenge researchers to delve further into the mysteries of brain injury.

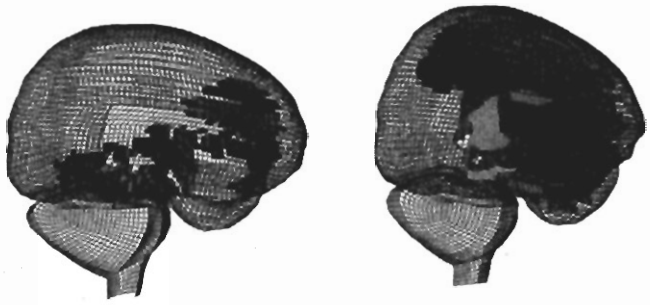
Another hypothesis is the old theory about pressure as being the cause of brain injury. We see surface contusions at the coup and contrecoup sites and computer models predict the transit of pressure waves travel through the brain upon impact. It is not clear what the physiological effect of transient pressure might have on axons and neurons and further study is warranted.

RECENT RESULTS

BRAIN RESPONSE:

Through a collaborative study with Biokinetics and Associates of Ottawa, Canada, mild concussive events that occurred during NFL games were quantified and duplicated in the laboratory (Newman et al., 1999). Helmeted dummies were used in the reconstruction during which head linear and angular accelerations were measured. These data were used as input into a comprehensive computer model of the human head, the Wayne State University Head Injury Model developed by Zhang et al. (2001b). A variety of brain response parameters were computed for both the concussed and non-concussed players (Zhang et al., 2003a). A total of 53 cases were studied of which there were 22 cases of concussion, as diagnosed by the team physician on site. The average linear acceleration for concussed and non-concussed players was 94±27 and 55±21 g respectively while the average angular acceleration for these two groups of players was 6398±1978 and 3938±1406 rad/s^2. Response parameters that were computed included intracranial pressure, shear and normal strain, strain rate and the product of strain and strain rate.

Figure 8 shows predicted maximum principal strains (ε) located in the brain for a non-injury case and an injury case. The strain response limit was set at 10% to show regions of the brain that experienced strains above 10%. As demonstrated in the figures, high strains were located in the midbrain and the posterior portion of the corpus callosum for a non-injury case. In comparison with an injury case, the model predicted a larger proportion of the elements experiencing strains over 10%. The high maximum principal strains were concentrated at the central core region of the brain, more specifically, located in the midbrain, upper brain stem and most of the diencephalon. The white matter of the frontal lobe sustained high strains as well. The corpus callosum region, where diffuse injury is commonly reported, did not experience significant strain.



(A) (B)

Fig. 8 - The highlighted elements are those experiencing maximum principal strains of over 10% from (A) non-injury case (B) injury case.

Strain rate was hypothesized to be a key biomechanical parameter to explain the cause of brain injury and concussion. It is being introduced for the first time as a measure of injury at the macroscopic level. Strain rate ($d\varepsilon/dt$) was manually calculated by differentiating the maximum principal strain vs. time curves for those elements that have the highest values of strain. The rate varied from 23 to 140 s^{-1} with an average value of 84 s^{-1} for injury cases and from 11 to 67 s^{-1} with an average value of 38 s^{-1} for non-injury cases. The product of strain and strain rate is another local tissue response measure that could be a mechanical parameter for neural injury. It is based on a study by Viano and Lövsund (1999) who analyzed brain injury data from ferrets subjected to a linear impact. This product was computed by taking the product of the instantaneous strain and strain rate. For injury cases, the average $\varepsilon \bullet d\varepsilon/dt$ was around 36 s^{-1} while, for non-injury cases, the average $\varepsilon \bullet d\varepsilon/dt$ was as low as 10 s^{-1} in the midbrain region. Typically, the peak $\varepsilon \bullet d\varepsilon/dt$ occurred at least 5 ms after the linear and angular accelerations have peaked, due to the viscous nature of the brain material.

LOGISTIC REGRESSION:

Logistic regression was carried out to determine effective injury predictors and to estimate the probability of injury. To form the regression model, the dependent variable or outcome was the occurrence of concussion (MTBI). The independent or predictor variables were divided into two groups. One group consisted of input variables and head injury assessment functions, such as the Head Injury Criterion (HIC), Head Impact Power (HIP), which represents the rate of change of kinetic energy, and Head Impact Jerk (HIJ) or the rate of change of head input acceleration. Group two contained all predicted brain response parameters. The significance tests of –2 Log Likelihood ratio, Score and Wald Chi-Squared were performed to quantify whether or not relationships between outcome and the predictor variables were statistically significant using SPSS Version 8.0 (SPSS Inc. Chicago, Illinois). The results are shown in Table 1.

Table 1 - Results of logistic regression analysis

Rank Order	Predictor Variable	-2 Log Likelihood χ^2	p	Score χ^2	p	Wald χ^2 for M.L.E.	p
1.	$_{max} \bullet d/dt_{max}$ (s^{-1})	34.1	0.0000	23.8	0.0000	12.1	0.0008
2.	d/dt_{max} (s^{-1})	30.1	0.0000	23.6	0.0000	12.9	0.0003
3.	HIC_{15}	26.3	0.0000	21.9	0.0000	12.7	0.0004
4.	$Lin.Accel_{max}$ (m/s^2)	20.9	0.0000	20.8	0.0000	14.2	0.0002
5.	$Ang.Accel_{max}$ (rad/s^2)	20.7	0.0000	17.1	0.0000	12.1	0.0005

Based on values of p and chi-squared from all three significance tests, the product of strain ($_{max}$) and strain rate ($\varepsilon \bullet d\varepsilon/dt_{max}$) at the midbrain region provided the strongest correlation with the occurrence of MTBI ($p<0.0000$, $\chi^2=34.1$). Strain rate was also a good injury predictor. This was supported by the magnitudes of the three test statistics. A multivariate analysis revealed that no other multivariate model was significantly better than the univariate models because the p value for one of

the variables in the model was not statistically significant. A Logist plot of MTBI probability versus the product of strain and strain rate is presented in Figure 9. Logist plots for the other variables are shown in Figures 10 through 13.

The thresholds for a 25%, 50% and 75% probability of sustaining a MTBI in terms of the product of strain and strain rate are shown in Figure 9 and in Table 2. The thresholds for the other four best injury predictors are also tabulated in Table 2.

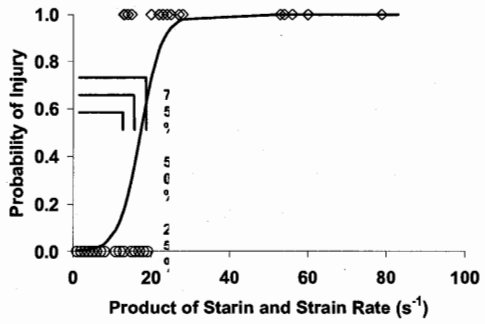


Fig. 9 - The probability of MTBI as predicted by the product of strain and strain rate in the midbrain region.

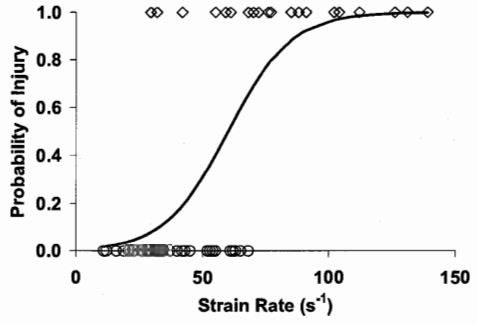


Fig. 10 - The probability of MTBI as predicted by strain rate in the midbrain region.

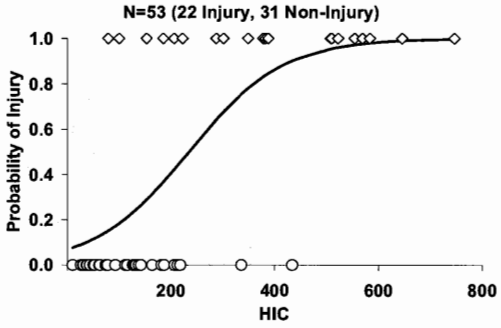


Fig. 11 - The probability of MTBI as predicted by HIC.

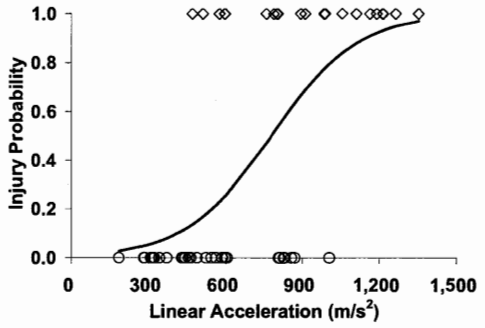


Fig. 12 - The probability of MTBI as predicted by head linear acceleration.

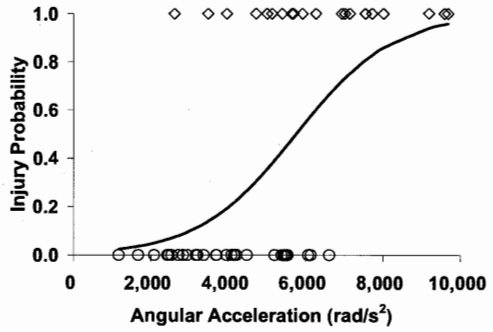


Fig. 13 - The probability of MTBI as predicted by head angular acceleration.

Table 2 - MTBI Tolerance Estimates for the Best Injury Predictors

Predictor	Probability of MTBI		
	25%	50%	75%
$_{max} \bullet d/dt_{max}$ (s^{-1})	14	19	24
d/dt_{max} (s^{-1})	46	60	80
HIC_{15}	136	235	333
Lin. Accel (m/s^2)	559	778	965
Ang. Accel (rad/s^2)	4384	5757	7130

DISCUSSION

The study of brain injury began at a time when measurement techniques were rather limited. It was not possible to measure impact force accurately half a century ago and the only convenient parameter that could be measured was linear acceleration of the head. The use of linear accelerometers became an accepted means of quantifying impact severity and linear acceleration is now used as an input variable that is correlated with observed injuries in many impact situations, including head injury. At about the same time, a theory was advanced by Holbourn (1943) suggesting that brain injury was dependent on angular acceleration of the head, even though there was no device that could make that measurement with precision at that time. In view of these two opposing hypotheses, researchers were almost forced to be in the linear acceleration camp or the angular acceleration camp, and an intellectual feud ensued. This led to the entrenchment of the idea that head injury was due either to linear or angular acceleration. Research was also directed toward producing injury using purely linear or purely angular acceleration, even though we know full well that such pure impact conditions do not exist in real life. However, the automotive safety standard for head injury was promulgated at a time when the only known limits for head injury were based on linear acceleration. At present, we have the Head Injury Criterion (HIC), which is an integral function of the resultant head acceleration. HIC was, and continues to be, criticized for not taking into account the angular acceleration of the head. However, HIC appears to have some validity because automotive related head injuries have been kept in check over the last 30 years. On the other hand, research on the effects of angular acceleration was pursued more vigorously than that on the effects of linear acceleration in an attempt to find a tolerance limit for angular acceleration. One unfortunate consequence of this schism among researchers is their focus on injury due to these two types of accelerations and their failure to consider other parameters that may be a more direct cause of brain injury. It is hoped that the availability of new techniques in experimental measurements, the use of validated comprehensive finite element models of the brain, and the results presented in this paper will stimulate researchers to change their focus and look at the wider picture of head injury. Our hypothesis that strain rate can play a role in brain injury is designed to move research in a new direction so that we can find the direct mechanism of how the brain is injured.

CONCLUSIONS

1. Many current researchers in head injury biomechanics consider angular acceleration to be the principal cause of brain injury.
2. Unique results of brain motion relative to the skull during a blunt impact have been acquired, using a biplane high-speed x-ray system.
3. Linear acceleration induced brain motion is very small, on the order of $\pm$ 1 mm.
4. Angular acceleration induced brain motion is limited to $\pm$ 5 mm, even if the magnitude of the acceleration is in excess of 10,000 rad/s^2.
5. Wearing a helmet does not change head angular acceleration appreciably but does reduce linear acceleration significantly.
6. In view of these results, the question of how the brain is protected by a helmet can be answered by considering brain response instead of input to the brain.
7. Strain rate and the product of strain and strain rate in the midbrain region appeared to be the best injury predictors for concussion.
8. Strain rate was proposed as a cause of brain injury in order to challenge researchers to move away from their focus on either linear or angular acceleration.
9. To study injury mechanisms, it is best to focus on brain reaction to complex inputs of linear and angular acceleration.
10. The inevitable conclusion is that, if we are to define tolerance in terms of brain response, we will need a computer model to describe this response. Intelligent helmet design will also need such a computer model so that it can afford omni-directional protection to the brain.
11. Injury is intimately related to the local response of the brain and not to the global input to the head.

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