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Abstract

Objective: To determine the effect of impact direction on strains within the brain.

Research design: Laboratory drop tests of hybrid III head-form and finite element simulation of impacts.

Methods and procedures: A head-form instrumented with accelerometers and gyroscopes was dropped from 10 different heights in four orientations: front, rear, left and right hand side. Twelve impacts with constant impact energy were chosen to simulate, to determine the effect of impact location. A finite element head model was used to simulate these impacts, using 6 degrees of freedom. Following this a further set of simulations were performed, where the same acceleration profiles were applied to different head locations.

Main outcome and results: The angular accelerations recorded were up to 30% higher in lateral and rear impacts when compared to frontal impacts. High strains in the mid-brain (41%) were recorded from severe frontal impacts where as high strains in the corpus callosum (44%) resulted from lateral impacts with the same energy.

Conclusion: Impact direction is very significant in determining the subsequent strains developed in the brain. Lateral impacts result in the highest strains in the corpus callosum and frontal impacts result in high strains in the mid-brain.

Keywords

Concussion, Maximum principal strain, corpus callosum

Introduction

Concussion in sport is very prevalent with between 1.6 and 3.8 million sports related concussions in the United States each year (1). The diagnosis of concussion is particularly difficult with many studies reporting that approximately 50% of concussions go unreported (2)(3). Historically efforts have focused on using linear acceleration to predict injury; this has changed in the last decade to include rotational velocity and angular acceleration. It has been possible to capture acceleration data in US football, due to the space available within the helmet to mount sensors. The Head Impact Telemetry System (HITS), Simbex, Lebanon, was developed in 2002, and uses an array of accelerometers to measure linear and rotational accelerations. Rowson et al used data from this system to develop a probability theory to predict whether an injury has been sustained (4). They found that concussive impacts in the HITS database had average linear accelerations of 104 ± 30g and rotational accelerations of 4726 ± 1931 rad/s^2. Other researchers have used video data to recreate impacts in the laboratory using instrumented hybrid anthropomorphic dummies and hence determine head accelerations (5). A study of head impacts in rugby in Australia, used this method and found that there was a 75% probability of concussion from a rotational acceleration of 2296 rad/s^2 in the coronal plane, and again a 75% probability of concussion from linear accelerations above 88.5g (6). Data from these studies has been used in finite element models to determine strain within the brain (7)(8). Patton et al determined that maximum principal strain was significantly associated with concussion (8). Gilchrist et al investigated the relationship between linear and rotational acceleration and the durations of these accelerations (7). They found that as the duration increased the rotational acceleration becomes dominant and a rotational acceleration as low as 2500 rad/s^2, over 10 to 15ms, can result in a brain injury. A number of other studies have investigated the magnitude, shape (9) and duration (10) of the acceleration pulses but there has been little analysis, to date, on how the location and direction of the impact effects different regions of the brain.

In order to investigate strain within the brain, following an impact, finite element models have been utilised. There are a number of well-known finite element head models, among these are the models developed by KTH Royal Institute of Technology in Stockholm (KTH) (11), and the Wayne State University model (WSUM) (12), both of these models represent an average adult male. Both models use a finite element explicit solver, LsDyna (LSTC, Livermore, CA) and both model the scalp, skull, brain, meninges, cerebral spinal fluid, bridging veins, white and grey matter and the ventricles. The material properties of the models were developed from laboratory tests performed on a combination of human and animal tissue. The validation of these models has been performed using human cadaver experiments to determine brain displacement (13) and brain pressure response (14) (15). The Wayne State head model has now been incorporated into the Global Human Body Consortium (GHBC) model. Another model developed at Dartmouth College, Hanover, US is subject specific and has been validated against the same human cadaver experiments as the other two models. These models are being used to investigate the response of the brain and to determine injury thresholds. Large principal strains have been proposed as one of the causes of diffuse axonal injury (16), in particular high strains in the corpus callosum have been found to be associated with concussion (17). Margulies et al used a porcine model to investigate diffuse axonal injury (18). They applied a sudden coronal plane rotation without impact, from this they determined that a rotational acceleration above 9000 rad/s^2 may lead to a moderate to severe DAI but they state that there is a continuum of axonal injury and that concussion might be associated with much lower levels of rotational acceleration. In one of the few studies of the movement of the living human brain, subjected to small impacts, it was determined that DAI may also be caused by linear acceleration, as the motion of the brain is constrained by structures in the front and base of the skull, the tangential components of linear forces cause the brain to rotate about its centre of mass (19).

Camarillo et al found that six degrees of freedom kinematics was a better criteria to indicate a possible injury compared to criteria such as peak linear acceleration and head impact criteria (HIC), but they also found that the best predictor of injury was strain in the corpus callosum (17). McAllister et al reported that peak principal strains in the corpus callosum of 0.28 ±0.089 resulted in concussion(20), this study recreated impacts in the laboratory and applied the recorded head accelerations to the Dartmouth head model. A similar study by Zhang et al , using the Wayne State University head model, found that shear stress in the brain stem provided the strongest correlation with the occurrence of concussion (21). Patton et al studied 27 concussive cases in helmeted and unhelmeted sports, the average linear acceleration for the injury cases was 103.8g and the average rotational acceleration was 7141 rad/s^2 , using the KTH model they concluded that the best predictor of injury was strain in the corpus callosum, thalamus and white matter (8). A comparison of brain strain injury criteria is shown in Table 1.

TABLE 1

This study investigated the relationship between impact, magnitude and direction, and the subsequent variation of strain levels in the central region of the brain. In particular the strains in the corpus callosum, mid-brain, thalamus, and brain stem are examined as these regions have been identified as '*hot spots'* during concussive impacts (22).

Methods

A number of drop tests were carried out and these were then simulated.

Drop tests

An anthropomorphic head was dropped in a purpose built tower from a range of heights, in a number of orientations to represent impacts to the front, rear and side of the head. Linear

acceleration was recorded using a triaxial accelerometer (Kistler 8688A) and rotational velocity was recorded using a three axis gyroscope (DTS ARS 12k), the instruments were mounted at the centre of gravity of the head. The accelerometer and gyroscope data were recorded using Labview Software (National Instruments) and were sampled at 10,000Hz over 200ms for each impact. Linear acceleration was filtered at 1000Hz and rotational velocity was filtered at 300Hz, these frequencies were verified as suitable filters using Fourier transformation to calculate the amplitude spectrum, a finite difference method was used to calculate rotational acceleration. The tests consisted of 400 impacts in four locations, left occipital, right occipital, front and rear impacts to the head, as shown in Figure 1.

FIGURE 1

The test conditions were designed to simulate impacts ranging from 20g to 200g peak linear acceleration. To create these conditions, the head form was released from heights ranging from 90mm to 610mm, and impacted a steel hemisphere of 120mm diameter. Ten tests were completed at each height to investigate the repeatability of the tests, see Table 2. Impacts at each height were also recorded using a high speed camera at 2000 frames per second. Impact velocities were calculated using TEMA analysis motion tracking software.

TABLE 2

Simulation

The drop tests were simulated using the Wayne State head model (Figure 2) from the Global Human Body Consortium, the model, which has been well documented (14,22,23), consists

of 33 parts, 165,093 elements and both linear and non-linear material properties. The solver used was LsDyna running on Amazon cloud computing services.

FIGURE 2

Two sets of simulations were performed. The first set of simulations kept the energy of the impact constant (i.e. the same drop height) and the impacts were performed to the front, side and rear of the head, six degree of freedom accelerations were applied to the model as recorded from the drop tests, see Table 3.

TABLE 3

The second set of simulations kept the peak linear and rotational accelerations the same, for impacts again, to the front, side and rear of the head-form. Accelerations in these cases were applied in two degrees of freedom as sinusoidal waveforms in the direction of impact and the principal plane of rotation (Table 4). The magnitudes ranged from 30g and 2000 rads/s^2 to 130g and 6000 rads/s^2 , these are similar to the ranges published in rugby and US football studies (25), (26). The profiles were determined by averaging the results of the head accelerations and time durations, acquired from the drop tests. Simulations were run for 30ms, thus ensuring that there was sufficient time to examine the brains response to the impact.

TABLE 4

Results

Constant Impact energy

The first set of results, where the impact energy remained constant, found that the linear peak impact accelerations were generally higher (up to 12%) during rear and lateral impacts of the head form compared to frontal impacts, the most severe lateral impact differed to this, see

Table 3. The angular accelerations were also generally higher (up to 30%) in lateral and rear impacts when compared to frontal impacts. Following the most severe lateral impact (150g, 7171rad/s^2) the strain in the corpus callosum was 44%, this reduced to 19% in the brain stem (Figure 3). Strain in the mid-brain increased linearly as the impact energy increased, also the mid-brain experienced higher strains during frontal impacts, and the least strain during lateral impacts.

FIGURE 3

Intracranial pressure was also examined using these simulations, see Figure 4, this was found to increase as impact energy increased and the maximum difference between pressure following impacts to different locations was 22%. Only the severest impact exceeded the limit of 235kPa for concussion, suggested by Ward et al (27).

FIGURE 4

Same acceleration profiles applied to different locations

The second set of results relate to simulations where the same acceleration profiles were applied to represent impacts to the front, rear and side of the head. Again the strains in the corpus callosum and mid-brain were larger than in the thalamus or brain stem, see Figure 5. Frontal impacts resulted in higher strains (up to 35%) in the mid-brain than impacts from the other directions. Severe lateral impacts resulted in very high strains of 45% in the corpus callosum with even moderate impacts (57g, 3500rad/s/s) resulting in strains of 31%. A maximum strain of 45% was recorded in the corpus callosum following a lateral impact of 130g and 6000 rad/s^2, while a 24% strain was recorded following a frontal impact of the same magnitude, an difference of 48%. On average lateral impacts resulted in a strain increase of 43% in the corpus callosum compared to frontal impacts. The strain thresholds determined by

Patton et al (8), were met or exceeded in at least one of the brain regions investigated for the severe and serious impacts, regardless of the impact direction. The moderate impact indicated the possibility of an injury in the mid-brain during a frontal impact. Ward et al(27) pressure criteria was only exceeded in the most severe frontal impact, this would indicate that strain provides a lower threshold to indicate injury (27).

FIGURE 5

Discussion

The mechanism of concussion is not fully understood, as it is not possible to measure the response of a living brain following a severe impact to the head. Several studies have used animal models and scaled the effect to represent a human brain (18) (28)(29), while others have conducted cadaver studies (13), (30). Finite element (FE) simulations are important to help understand the mechanical response of the brain following an impact. Using FE analysis it has been shown that increases in intracranial pressure are associated with linear accelerations while shear strain and diffuse axonal injury (DAI) is associated with rotational acceleration (18)(21).

The simulation results, from the impact tests in which the impact energy was kept constant, indicate that only lateral impacts exceeded the strain based criteria for injury within the corpus callosum. These high strains in the corpus callosum were also found when the same acceleration profiles were applied to represent frontal lateral and rear impacts, see Table 4. The strain following a lateral impact was widely dispersed within this region, see Figure 6.

FIGURE 6

Elkin et al also reported a significant increase in strain from lateral impacts in their study of head impact in helmeted sports (31). Angular acceleration in the coronal plane has also been shown to have a strong correlation with concussive injuries in un-helmeted sport, this is possibly due to an injury in the corpus callosum (6). The corpus callosum plays a vital role in the brain's function as it is the communication hub between the left and right of hemispheres and has been linked with traumatic brain injury (20). The high strains in the corpus callosum may be partially explained by the movement of the corpus callosum as it follows the motion of the skull more closely, than the more compliant grey matter that surrounds it (19). Previous studies which have examined the relationship between the falx and the corpus callosum have identified this region as mechanically vulnerable due to the connection between the longitudinal falx and corpus callosum (17). It was found that the areas of high strain following lateral impacts are concentrated in the superior sections of the brain core. This study provides evidence that the regions anatomical closer to the centre of the brain (corpus callosum and thalamus) are more vulnerable to strain during lateral impacts.

In contrast frontal impacts produced higher strains than rear and lateral impacts in the brain stem and mid-brain. An impact to the front of the head produced, on average, 47% and 37% more strain in the mid-brain than a lateral or rear impact, respectively. The analysis of the brain stem following a rear impact found that there was a noticeable strain response in the lower section of the brain stem during the initial phase of the impact. The strains were similar whether the impact was to the front or rear of the head. It was found that the strain response in the brain stem was due to the rotational accelerations (the linear acceleration was removed and the simulation re-run). This response to rotational acceleration may indicate a likelihood of DAI (18)(28).

Strain, for brain regions located anatomically lower, reduce significantly, due to the tethering effect of the vascular, neural and dural elements that bind the brain to the base of the skull

(19). Front and rear impacts had a higher strain response than lateral impacts lower within the brain structure (mid-brain and brain stem), due to the stretching of the lower structures during the coup, contra coup response following a frontal or occipital impact (32).

Strain based injuries such as diffuse axonal injury have been linked to rotational acceleration (33) during an impact while linear acceleration is thought to be related to an increase in intracranial pressure and more focal injuries (24). These simulations indicate that the impact location is significant in determining, the brain region effected and extent of an injury that may be sustained.

There is considerable variation in the linear and rotational accelerations reported to cause concussion, this study found that accelerations of 57g and 3500rad/^{2} resulted in a 50% likelihood of concussion. The magnitude of the rotational acceleration is similar to that found by Zhang et al in their reconstructed 24 cases of concussion in American football (21). It must be borne in mind that these NFL impacts would have had a longer duration to those in this study or in unhelmeted sports. Duration of the acceleration pulse has been shown to influence the strain response (34). Patton et al study of head impacts in rugby found that impacts with rotational accelerations as low as 1747 rad/s^2 in the coronal plane, may cause concussion. This contrasts with Margulies et als study using porcine models which determine that rotational accelerations above approximately 9,000rad/s^2 would cause a moderate to severe DAI, but they do acknowledge that accelerations much lower than this may cause a concussion (18).

This study has shown that the mid-brain and the superior area of the brain stem are ‘hot spots’ for strain during frontal and rear impacts (Figure 7), this is similar to a finite element study which recreated 25 cases of concussion US football impacts (22). This is significant as studies which have analysed head impacts in American Football determined that the highest

numbers of impacts are sustained to the front of the head (35). Blurred vision and motor control have been reported to be common symptoms in concussive injuries (5) within American football, these symptoms may relate to the role the mid-brain plays in the central nervous system. This study also found that moderate (Table 3) lateral impacts causing rotation in the coronal plane are likely to cause injury, these are of particular importance in rugby as half of all concussions are reported to occur following this type of impact (36).

Limitations

The head model only represents the 50th percentile male, it is also only partially validated using human cadaver tests. Published injury thresholds vary widely and a precise injury threshold has not yet been determined.

Conclusion

This study analysed the effect of the location of an impact and the resulting strain response within the central region of the brain. The study simulated six degree of freedom drop tests in which the impact energy was kept constant but the impact location was varied. This investigation found that impact location was significant. To investigate this further the study simulated impacts in three locations, the same accelerations in two degrees of freedom were applied to each location.

The study found that the superior regions of the brain (corpus callosum and thalamus) produced a higher strain response during lateral impacts while the inferior regions (mid-brain and brain stem) produced a higher strain response following frontal and rear impacts. Overall

the study highlights the need to consider the location of the impact as a criterion when discussing brain injuries.

Declaration of interest

The authors report no declaration of interest. The study was partly funded by the President's award at the Institute of Technology Tallaght , Dublin.

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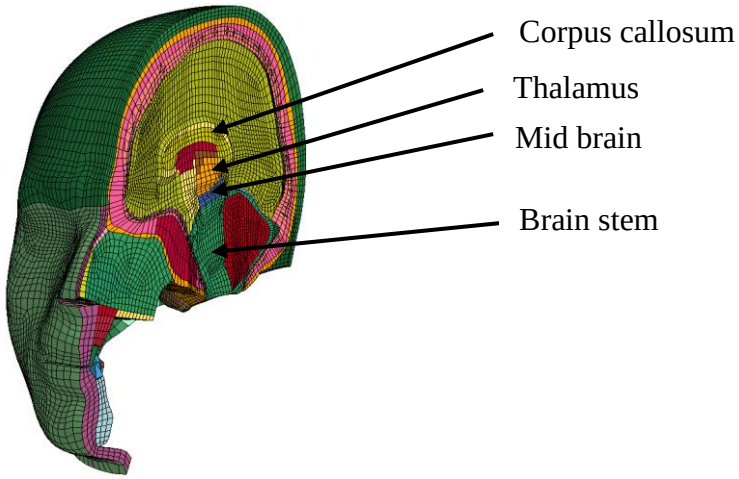
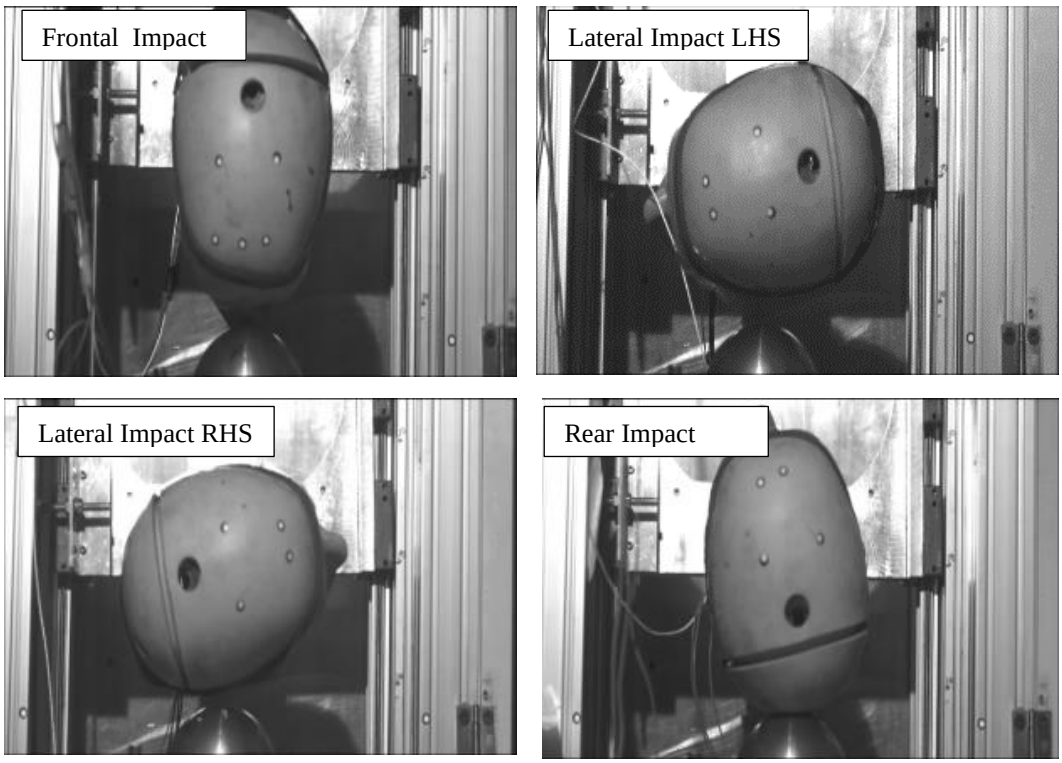
Region	Corpus Callosum		Mid brain		Thalamus		Model
	No Injury	Concussive injury	No Injury	Concussive injury	No Injury	Concussive injury	
McAllister (18)		0.28					Dartmouth
Patton (8)	0.12	0.31	0.13	0.25	0.1	0.26	KTH
Viano (20)			0.23	0.34	0.21	0.38	WSUMl

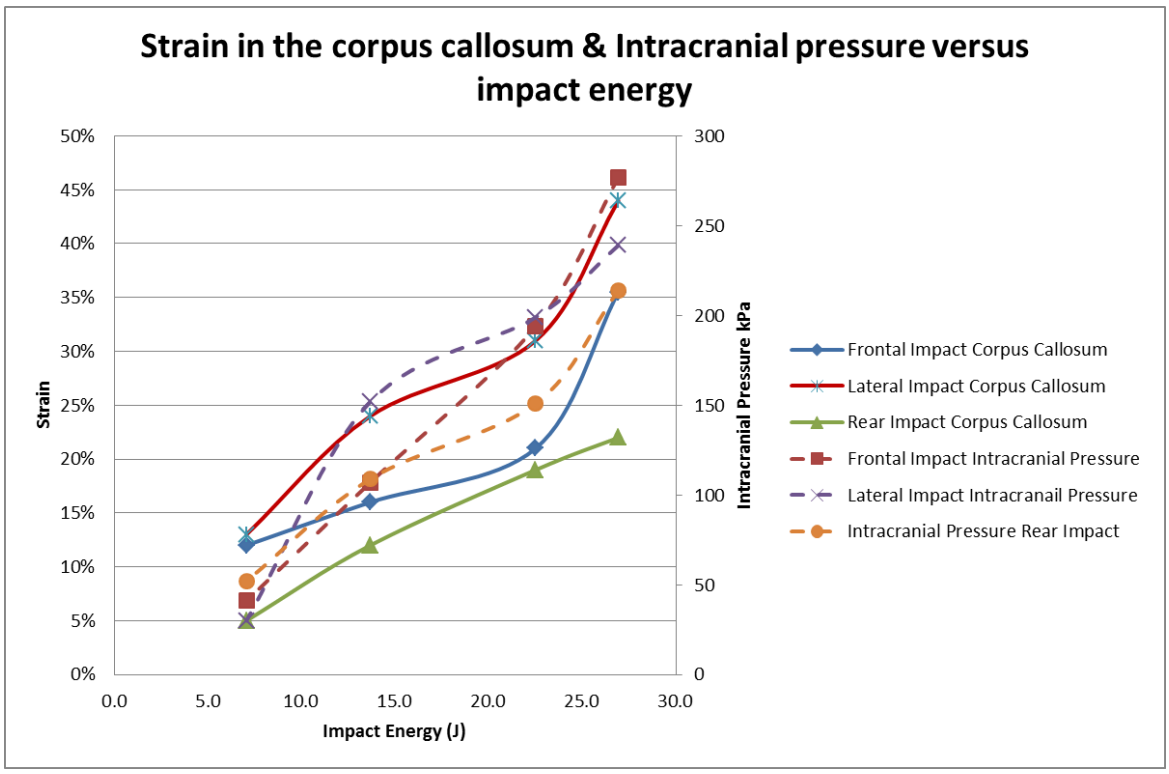
Frontal Impacts				
Drop Height mm	**Impact Energy Joules**	**Linear Acceleration g**	**Angular Velocity rad/s**	**Angular Acceleration rad/s/s**
610	26.93	127.6	25.5	7426.5
560	24.72	107.6	25.2	7857.4
510	22.51	97.8	23.9	6570.7
460	20.31	85.1	23.1	7494.3
410	18.10	75.3	21.9	5786.9
360	15.89	62.8	20.1	5135.8
310	13.68	53.7	16.8	3930.6
260	11.48	43.2	16.1	3102.3
210	9.27	36.2	15.6	3289.9
160	7.06	31.7	14.0	3981.2

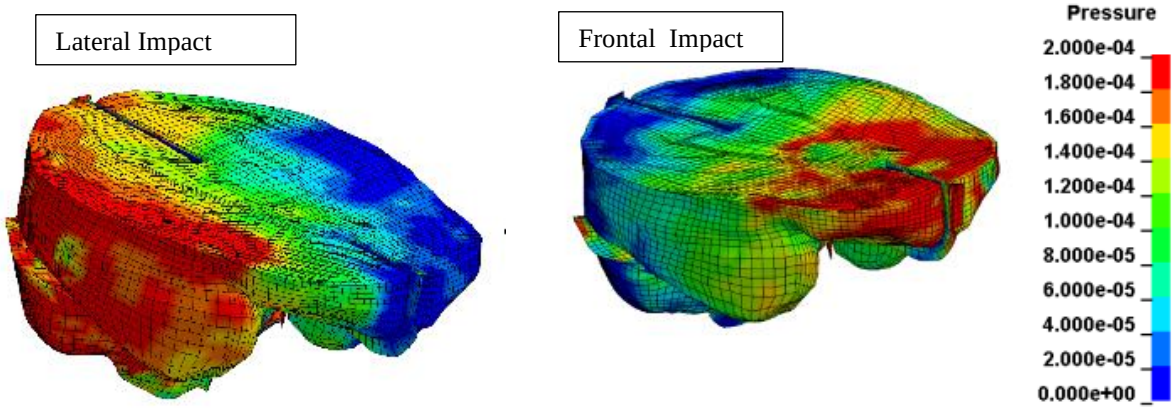
Frontal Impact								
	Impact Energy (J)	Linear Acceleration	Rotational Acceleration	Corpus Callosum	Mid-brain	Brain Stem	Thalamus	Intracranial Pressure
		g	rad/s/s	Stain				kPa
Severe	26.9	169	5501	36%	41%	32%	26%	277
Serious	22.5	93	5302	21%	34%	29%	23%	194
Moderate	13.7	57	3421	16%	26%	16%	14%	107
Mild	7.1	22	1641	12%	20%	17%	10%	41
Lateral Impact								
Severe	26.9	150	7171	44%	20%	19%	24%	239
Serious	22.5	105	5020	31%	15%	16%	17%	199
Moderate	13.7	78	4267	24%	12%	14%	15%	152
Mild	7.1	31	2346	13%	7%	9%	8%	30
Rear Impact								
Severe	26.9	169	6292	22%	29%	26%	23%	214
Serious	22.5	124	5097	19%	24%	19%	17%	151
Moderate	13.7	87	3568	12%	20%	15%	15%	109
Mild	7.1	25	1819	5%	9%	7%	8%	52

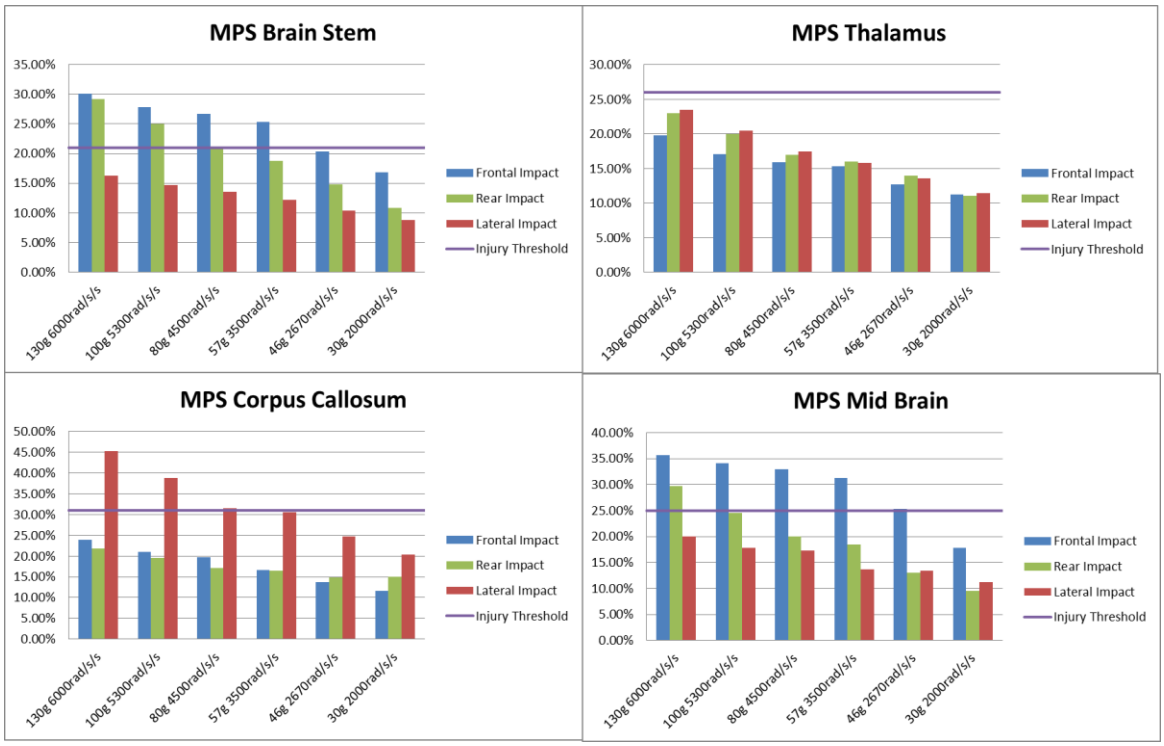
Acceleration Data					
Classification	**Impact no**	**Linear Acceleration (g)**	**Duration of linear accel ms**	**Rotational Acceleration (rad/s^2)**	**Duration of rotational accel ms**
Severe	1	130	8	6000	6
	2	100	9	5300	7
	3	80	11	4500	8
	4	57	13	3500	9
	5	46	12	2670	10
Mild	6	30	15	2000	12

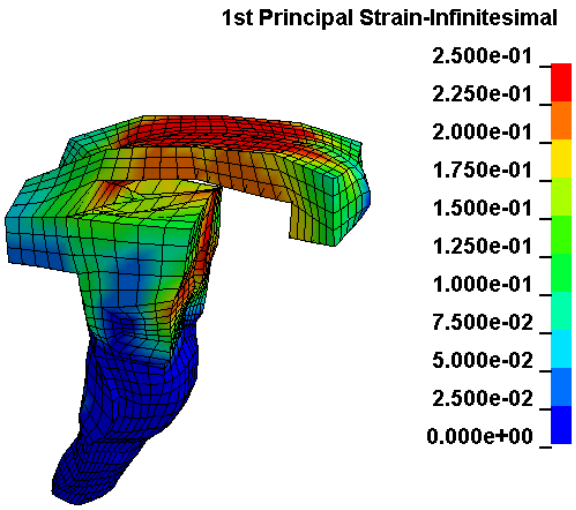

Corpus callosum
Thalamus
Mid brain
Brain stem

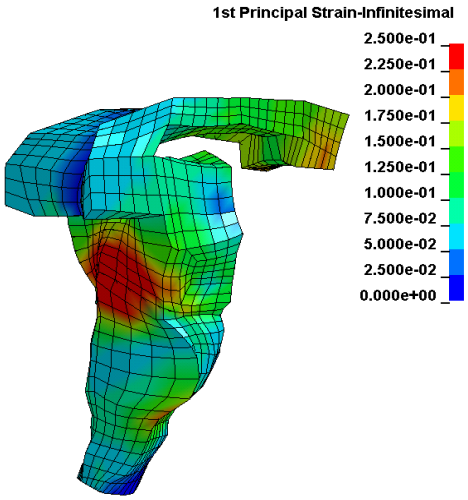

Frontal Impact
Lateral Impact LHS
Lateral Impact RHS
Rear Impact


Strain in the corpus callosum & Intracranial pressure versus impact energy
Strain
Intracranial Pressure kPa
Impact Energy (J)
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
300
250
200
150
100
50
0
0.0
5.0
10.0
15.0
20.0
25.0
30.0
Frontal Impact Corpus Callosum
Lateral Impact Corpus Callosum
Rear Impact Corpus Callosum
Frontal Impact Intracranial Pressure
Lateral Impact Intracranail Pressure
Intracranial Pressure Rear Impact


Lateral Impact
Frontal Impact
Pressure
2.000e-04
1.800e-04
1.600e-04
1.400e-04
1.200e-04
1.000e-04
8.000e-05
6.000e-05
4.000e-05
2.000e-05
0.000e+00


MPS Brain Stem
35.00%
30.00%
25.00%
20.00%
15.00%
10.00%
5.00%
0.00%
130g 6000rad/s/s
100g 5300rad/s/s
80g 4500rad/s/s
57g 3500rad/s/s
46g 2670rad/s/s
30g 2000rad/s/s
Frontal Impact
Rear Impact
Lateral Impact
Injury Threshold
MPS Thalamus
30.00%
25.00%
20.00%
15.00%
10.00%
5.00%
0.00%
130g 6000rad/s/s
100g 5300rad/s/s
80g 4500rad/s/s
57g 3500rad/s/s
46g 2670rad/s/s
30g 2000rad/s/s
Frontal Impact
Rear Impact
Lateral Impact
Injury Threshold
MPS Corpus Callosum
50.00%
45.00%
40.00%
35.00%
30.00%
25.00%
20.00%
15.00%
10.00%
5.00%
0.00%
130g 6000rad/s/s
100g 5300rad/s/s
80g 4500rad/s/s
57g 3500rad/s/s
46g 2670rad/s/s
30g 2000rad/s/s
Frontal Impact
Rear Impact
Lateral Impact
Injury Threshold
MPS Mid Brain
40.00%
35.00%
30.00%
25.00%
20.00%
15.00%
10.00%
5.00%
0.00%
130g 6000rad/s/s
100g 5300rad/s/s
80g 4500rad/s/s
57g 3500rad/s/s
46g 2670rad/s/s
30g 2000rad/s/s
Frontal Impact
Rear Impact
Lateral Impact
Injury Threshold


1st Principal Strain-Infinitesimal
2.500e-01
2.250e-01
2.000e-01
1.750e-01
1.500e-01
1.250e-01
1.000e-01
7.500e-02
5.000e-02
2.500e-02
0.000e+00


1st Principal Strain-Infinitesimal
2.500e-01
2.250e-01
2.000e-01
1.750e-01
1.500e-01
1.250e-01
1.000e-01
7.500e-02
5.000e-02
2.500e-02
0.000e+00