

Finite Element Analysis of Head Impact in Contact Sports

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Abstract: Traumatic brain injury (TBI) is one of the world's major causes of death and disability. To aid companies in designing safer and improved protective gear and to aid the medical community in producing improved quantitative TBI diagnosis and assessment tools, a multiscale finite element model of the human brain, head and neck is being developed. Recorded impact data from football and hockey helmets instrumented with accelerometers are compared to simulated impact data in the laboratory. Using data from these carefully constructed laboratory experiments, we can quantify impact location, magnitude, and linear and angular accelerations of the head. The resultant forces and accelerations are applied to a fully meshed head-form created from MRI data by Simpleware. With appropriate material properties for each region of the head-form, the Abaqus finite element model can determine the stresses, strains, and deformations in the brain. Simultaneously, an in-vitro cellular TBI criterion is being developed to be incorporated into the Abaqus models for the brain. The cell-based injury criterion functions in the same way as damage criteria for metals and other materials in that it predicts failure. Thus, for any given loading situation (blast, impact, or acceleration) a map showing the location and severity of tissue injury can be computed.

Keywords: Constitutive Model, Experimental Verification, Impact, Tissue, Traumatic Brain Injury

1. Introduction

Over the last two decades the rate of concussions in 15 NCAA sports has doubled, where football and ice hockey generally have had the highest rate of concussions per athletic exposure (Hootman, 2007). There have been many attempts to curb the rate of concussions and head injuries, such as new rules in football prohibiting certain types of tackles, and new regulations for helmets implemented by the American Society for Testing and Materials (ASTM) and the National Operating Committee on Standards for Athletic Equipment (NOCSAE) with standards for the ability to absorb energy (Crisco, 2011). Despite these efforts there are no standards to measure how well helmets protect against concussions because the mechanisms that cause brain injury are not well understood. It is known that brain injury is caused by the death or injury of neurons and neuronal networks, however the exact mechanisms of how an external impact force is transmitted is still an open research question. This paper is part of a larger investigation to increase the understanding of these mechanisms and aid in the development of a neuronal injury criterion.

Efforts have been made to measure the impact forces to which athletes are exposed during athletic events. Over the past years players on several NCAA football and hockey teams have had a series of accelerometers mounted in their helmets collecting data on impacts during games and practices.

One of the currently used systems is known as the Head Impact Telemetry (HIT) system (Simbex, Lebanon, NH). This system incorporates six single-axis accelerometers mounted non-orthogonally and oriented normal to the surface of the head (Rowson, 2011). Each time an impact occurs, a recording is triggered and the accelerometer data is wirelessly transmitted to a laptop that processes the data.

Currently Virginia Tech is using a different, but similar, system to measure impacts in college football. Their system utilizes twelve accelerometers mounted in six distinct locations in orthogonally mounted pairs (Rowson, 2011). They also use a fabric pad system that maintains constant pressure and orientation relative to the head throughout impact in order to ensure the acceleration measured is that of the head instead of the helmet. In order for an impact measurement to be triggered one accelerometer must read greater than 10 g. For each impact 40 ms of data is recorded, including an 8 ms delay before trigger and 32ms after trigger. Like the HIT system, this system transmits the data wirelessly to a computer. Using rigid body dynamics, the linear and angular accelerations about all three axes of the head's center of gravity are calculated.

Another technology currently being used to detect concussions is the X2 sensing devices (X2Impact, Seattle, WA). In helmeted sports they utilize a mouthguard with a series of imbedded sensors, which measure and transmit data about each impact. For non-helmeted sports, an impact-sensing headband has been developed that operates similarly.

Although such systems measure the acceleration levels experienced by a player, there are no conclusive guidelines on how the type (e.g. rotational or linear) and magnitude of acceleration correlates with injury. Thus, the ongoing research at Brown University plans to investigate the correlation between head accelerations with the death and injury of neurons, and hence develop a quantitative guideline for concussions and the onset of TBI.

1.1 Role of Finite Element Analysis

Many researchers have employed the use of finite element analysis (FEA) to explore the forces to which various portions of the brain are exposed during an impact. This is a difficult task for various reasons, the main one being that the head is composed of many distinct anatomical sections of tissue, bone, and cerebrospinal fluid. Obtaining an accurate representation of the intracerebral stresses and strains requires a model with well-defined geometries and meshes, accurate constitutive models, and accurate boundary conditions.

FEA is a valuable tool that allows us to determine the response of the entire brain to an impact, which cannot be done through direct measurement. McAllister et al. used the HIT system to collect impact data corresponding to known cases of concussions in ten athletes. They developed a technique to create meshes of the players' heads through the use of MRIs. Using the meshes developed along with Abaqus/Explicit they were able to run finite element models for each impact scenario based on the data collected with the HIT system. They validated their model by comparing it to measurements from cadaver head impacts. They also used MRI and diffusion tensor imaging to show a correlation between strain and changes in both fractional anisotropy and median diffusivity (McAllister, 2012).

Cloots et al. used FEA to focus on the different length scales involved in TBI. They ran simulations of mechanical loads at a length scale typical for individual cells. They discovered that

the resultant axonal strains could be higher than the applied tissue level strains (Cloots, 2011). This is important to note since the FEA for our model will predict tissue level strains rather than axonal strains. Colgan et al. used FEA to investigate the effects of anisotropy in TBI that were solely caused by rotational acceleration. They applied rotational acceleration about the center of mass of both an isotropic and anisotropic FE model. They found that anisotropic models predicted greater levels of injury compared to the isotropic model (Colgan, 2010).

The current FEA investigation expands on these studies by using a realistic model of the brain derived from MRI scans, where as many as 12 anatomical sections can be defined by the user with varying material properties. The FEA mesh also includes neck muscles and vertebrae, which simulates more realistic angular accelerations, and thus provides a more realistic investigation of the injury-causing mechanisms. By isolating each region and specifying realistic constitutive models, one can better estimate the areas of the brain that are most affected by a particular mode (such as amplitude or direction) of an external force or impact.

1.2 Project Outline and Goals

The long-term goal of this investigation is to establish a quantitative measure of the severity of a player's head injury based on live in-game data. This will be accomplished via carefully calibrated laboratory experiments using the HIT system to gather raw impact data. Specifically, a head form equipped with the HIT system is impacted with a spherical mass of 5.5 kg moving at 1 m/s. We then process the laboratory data to determine the linear and angular accelerations of the head, which are then used as boundary conditions for the FEA.

Abaqus/Explicit version 6.11 is utilized to determine strain, pressure, and stress in various regions of interest in the brain. The goal for finite element analysis is to determine the typical magnitudes and behavior of these quantities that players are typically exposed to during athletic events. In particular, the strains and strain rates that are extracted from the FEA will be applied to live neurons in the laboratory in order to gain a further understanding of TBI injury mechanisms on a cellular level. Figure 1 illustrates the progression toward achieving the project goals.



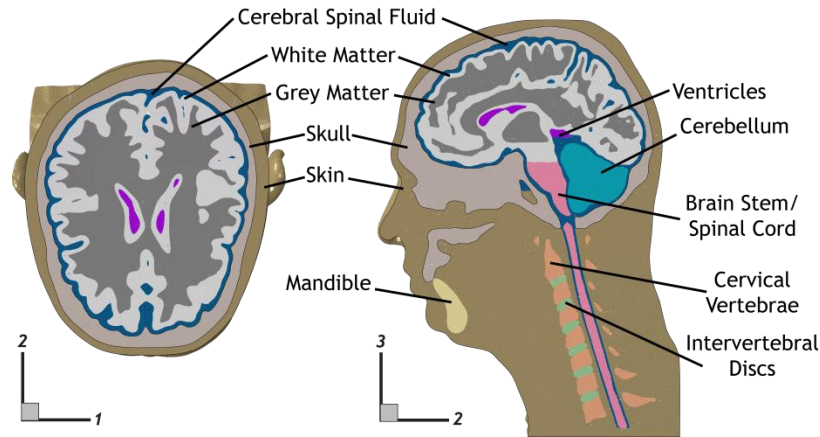
Figure 1. Workflow for project.

2. Numerical Methods

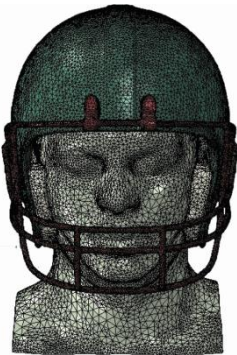
2.1 Segmentation and Meshing of Brain

A MRI of a healthy adult male head was used to create the STL geometry for the model. The mesh was then generated by using ScanIP (Simpleware, Exeter, UK) and imported into Abaqus for

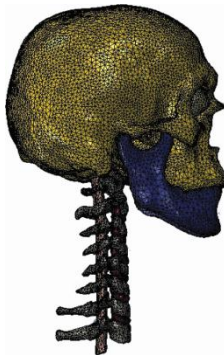
simulation. The mesh is composed of approximately 1.5 million elements and is broken up in to 15 sets. The helmet is composed of three sets, the brain, which consists of white matter, grey matter, ventricles, and cerebellum, is composed of four sets, and the other parts of the head including the vertebrae, skull, skin, mandible, cerebrospinal fluid, spinal cord, eyes, and intervertebral discs are composed of the remaining eight sets. It is important to include the neck in the model because the neck's resistance to rotational motions plays a crucial role in TBI. The sets that are touching each other share nodes so there is no need to create a tie between sets. Figure 2 shows several parts of the mesh color-coded by element set and an anatomical map of the sets used in the model.



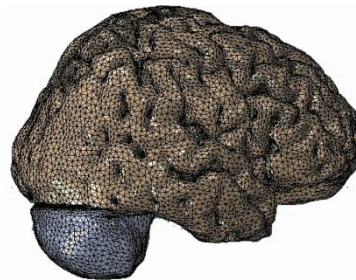
(a)



(b)



(c)



(d)

Figure 2. (a) A mid-sagittal cut showing all of the anatomical sections of the head with the exception of the eyes. The finite element mesh, which consists of (b) the helmet and skin sets, (c) the skull, eye, mandible, cervical vertebrae, intervertebral discs, and spinal cord sets, (d) and the white matter and cerebellum sets.

2.2 Constitutive Model

Choosing an appropriate set of constitutive models is a difficult task due to complex anatomy inside the head. These difficulties arise not only from the complex material properties of the brain, i.e. nonlinear viscoelastic and anisotropic, but also in the difficulties in experimentally determining these properties (Shen et al, 2005). In these preliminary simulations, we are utilizing an isotropic Neo-Hookean model for all sections of the brain and soft tissue defined by

$$U = \frac{\mu}{2} (\bar{I}_1 - 3) + \frac{K}{2} (J^{el} - 1)^2$$

where U is the strain energy density, \bar{I}_1 is the first deviatoric strain invariant, J^{el} is the Jacobian of the deformation gradient (or elastic volume ratio), μ is the shear modulus, and K is the bulk modulus. The values for the bulk modulus and the shear modulus are the same as those used by Moore et al. and are displayed in Table 1. In our model, the white matter, gray matter, brain stem, cerebellum, eyes, and Cerebral Spinal Fluid (CSF) were defined with identical material properties and are displayed in Table 1 under general brain.

Table 1. Material properties of the soft tissue used in the isotropic Neo-Hookean model

Brain Section	Density (kg/m ³)	K (Pa)	μ (Pa)
General Brain	1,040	2.19×10^9	2.253×10^4
Ventricles	1,040	2.19×10^9	2.225×10^4
Skin	1,040	3.47×10^7	5.880×10^6

For the skull, cervical vertebrae and intervertebral disks, we used a linear elastic model with the same values used by Moore et al. The values for density, Young's Modulus, and Poisson's ratio are $1,412 \text{ kg/m}^3$, $6.50 \times 10^9 \text{ Pa}$, and 0.22 respectively.

2.3 Simulation Parameters

We ran two different simulations to better understand the CSF's affect on the stresses during a traumatic event. For both simulations, we rigidly fixed the neck and shoulders, which includes the cervical vertebrae, brain stem, and CSF to the base. In this way, the neck stiffness is primarily attributed to these boundary conditions and not due to the skin's resistance to head motion. In future models a more realistic simulation would include the effects of the supporting muscles that play a major role in determining the stiffness of the neck.

In the bonded model, the nodes that are shared between the skull and CSF are always connected. In this way, the mesh between the skull and the CSF are constrained and not able to separate during deformation. This boundary condition is illustrated in Figure 3(b top), where the interface nodes (red dots) do not disconnect during deformation.

In the free model the meshes are allowed to freely disconnect during deformation (Figure 3(b bottom)). This boundary conditioned was produced by importing the skull as a completely different part and subsequently not tying the mesh of the skull and CSF together. All other anatomical sections are fully connected and are considered one mesh with different material

properties.

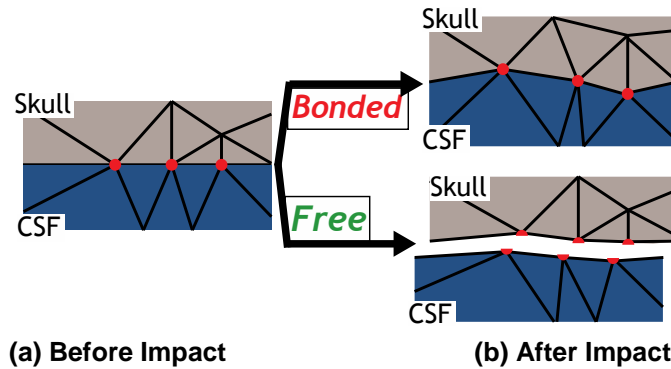


Figure 3. A comparison of the mesh connectivity between the bonded and free model for the interface between the skull and the CSF. (a) A mesh before impact showing node sharing (red circles) between the skull and CSF. (b top) In the bonded model, the skull and CSF are fully connected and continue to share nodes during deformation. (b bottom) In the free model, the skull and CSF meshes are free to separate during deformation.

The type of impact that we chose was aimed at mimicking a common hit seen in the National Football League; e.g. a helmet-to-helmet collision between a cornerback and his target. For this reason we chose a rigid cylinder weighing 80 kg moving at a velocity of 8 m/s impacting the forehead directly above the glabella. Figure 4 shows the rigid cylinder used to impact the head as well as the boundary condition of a constrained neck and shoulders.

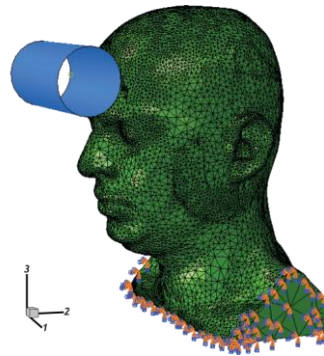


Figure 4. Mesh of the head showing the impact from a rigid cylinder while the neck and shoulders are rigidly constrained to the base.

We ran the simulations with a dynamic explicit step for a total time period of 6 ms with 80,139 increments using non-linear geometry. To ensure that there is no penetration of the cylinder into the head we created a bounding surface on the skin near the impact site and defined a state of general contact between this surface and the cylinder. We used a target time increment of 10^{-8} , and introduced mass scaling aimed at increasing the stable time increment. Each simulation took approximately three hours to run using a Windows HPC cluster running on 24 cores in a 144 core machine (12 nodes).

3. Results and Discussion

An important parameter to extract from these simulations is the total contact time, i.e. the time that the rigid cylinder is in contact with the head. In Figure 5, the contact area from the rigid cylinder impacting the forehead is shown in grey. A graph of the contact pressure over the simulation time shows a contact time of approximately 2 ms. In comparison, HIT system data produces typical contact times of approximately 10 ms. This discrepancy in contact time is small considering the large differences in the boundary conditions, material properties, and impact objects between the two models.

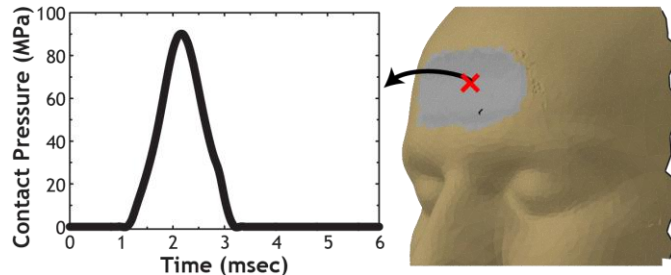


Figure 5. The time history of the contact pressure imposed on the forehead (grey area) due to the cylinder impact. The plot of the contact pressure shows that the total contact time is approximately 2 ms.

It is important for the model to exhibit head motion dynamics similar to those found in real life impacts. In the model, the head undergoes a rapid acceleration and deceleration suddenly after impact. This type of head motion is medically called cervical-acceleration-deceleration (CAD) and commonly known as whiplash. It is hypothesized to be a major factor in traumatic brain injury (Foreman, 2001). The dynamics of this motion can be seen in Figure 6 where the head accelerates from rest to a maximum speed of 26 m/s in 4.8 ms. This acceleration is equivalent to 550 g.

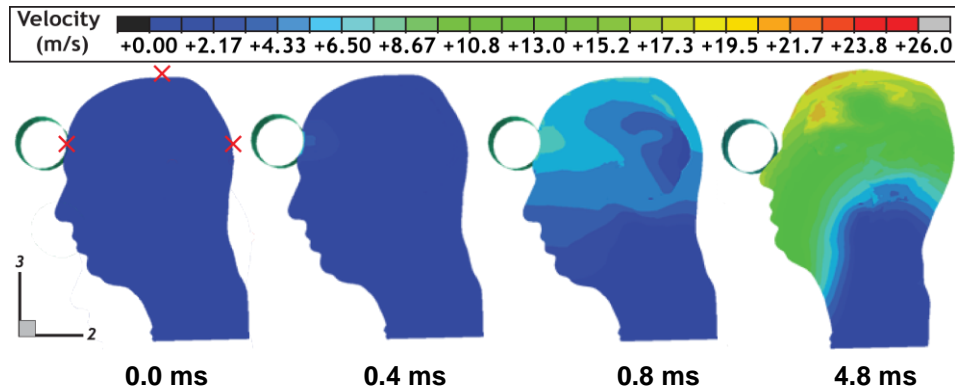


Figure 6. Contour plots of the velocity at different time points after impact in the free model.

Analyzing the intracerebral stresses in our model allows us to begin answering how tissue scale stresses translate to neurons on the micro scale. In both models, the normal stresses or the

pressure terms are significantly larger (on the order of 10^4) than the shear stresses found within the brain. This large difference is explained by the fact that the ratio of the bulk and shear modulus for the brain in our model is approximately 10^4 .

The time history at different points in the brain were probed to gain further insight on how the pressure propagates within the brain. Figure 7 shows the time history of the pressure at linearly spaced locations between the anterior and posterior of the brain. As expected, for both the bonded and free models, the pressure wave dissipates over distance away from impact site. Also, in both models, the pressure wave requires approximately 1 ms to travel from the anterior to the posterior of the brain. A difference between the models is that the maximum intracerebral pressure is larger in the free model. However, it is important to note that the difference between the maximum and minimum pressure is the same for both models. This occurs in the bonded model because the magnitude of the pressure tends to negative towards the posterior of the brain.

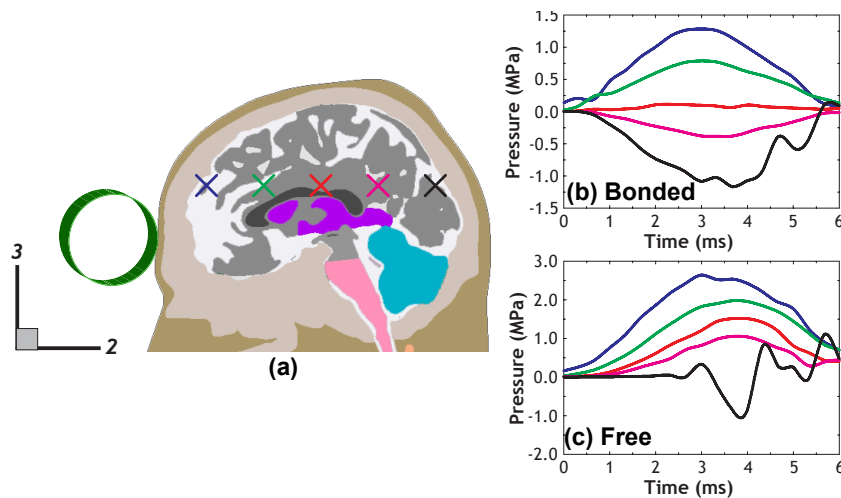


Figure 7. Time histories of the pressure at (a) different locations in the brain (shown by X) for the (b) free and (c) bonded model.

This behavior is clearly seen by looking at the spatial distribution of the pressure at different points in time. In Figure 8, the contour plots of the pressure 0.9 ms after impact provide a comparison between the two models. Furthermore, Figure 8(c-d) shows the rising phase (time of impact to 3 ms) of these pressure contours across the brain, which is denoted by the black line in Figures (a-b). For the bonded model, the pressure magnitude starts at 1.7 MPa and ends at minimum of -1 MPa at the posterior of the brain. This behavior is due to the constrained boundary condition that enforces a tensile pressure at the posterior of the brain. This pressure profile is starkly different than that of the free model. In the free model, the maximum pressure is 2.7 MPa and approaches zero as a function of distance from the anterior of the brain. Since the brain is free to move at the boundary, the pressures must be equal to zero. These differences in the pressure profiles highlight the importance of applying realistic boundary conditions to the simulation

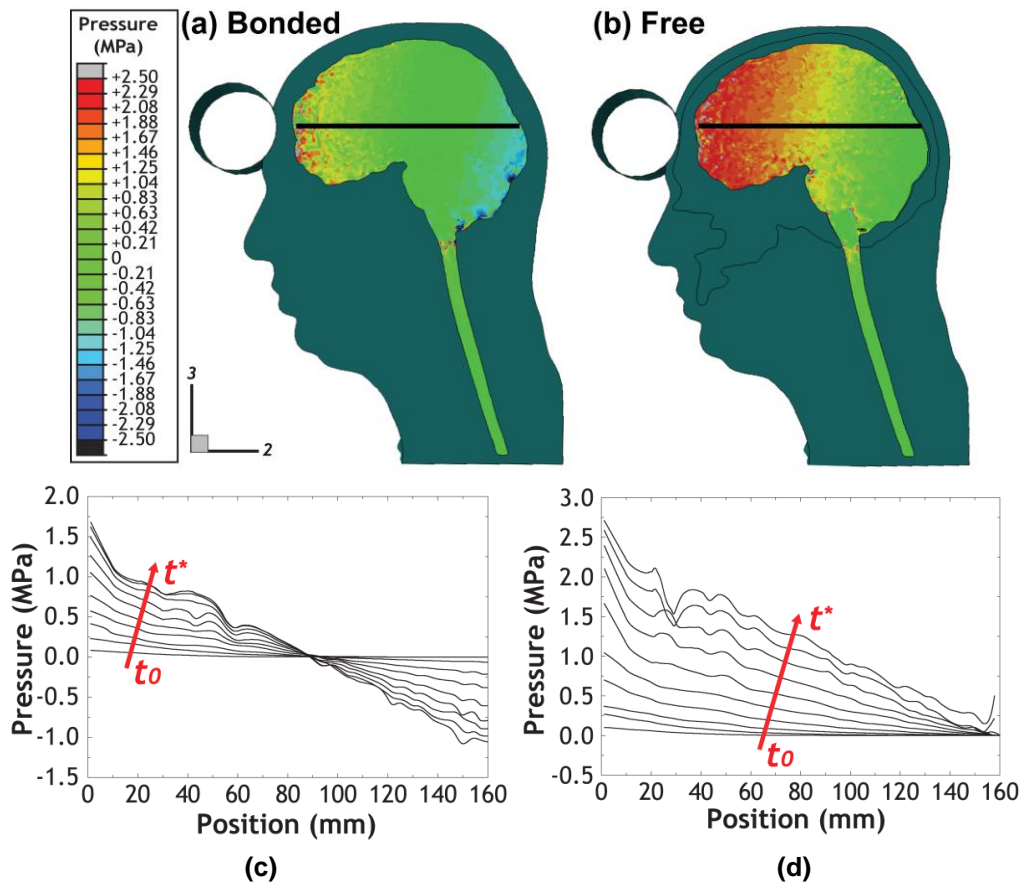


Figure 8. Contours of the pressures in a mid-sagittal cut of the brain 0.9 ms after impact for the (a) bonded and (b) free model. (c) Pressure along the black line in the (a) bonded and (b) free model of the brain from time of impact (t_0) to 3 ms post impact (t^*).

4. Conclusion

A FE model for traumatic brain injury provides a means of overcoming the difficulties associated with experimentally determining the intracranial stresses after a detrimental event. Additionally, a FE model allows the flexibility of testing different boundary conditions, material properties, and impact modes in a cost effective way. In this preliminary study, two different boundary conditions were simulated on a head form that was impacted by a rigid sphere. In both models, free and bonded, the head exhibited a form of whiplash, which is commonly seen in events that cause symptoms associated with TBI (Foreman, 2001). Furthermore, the contact time in the simulation is comparable to the contact time acquired during HIT experiments performed in a laboratory. It was also found that that the boundary conditions play a large role in determining the intracranial pressure distributions.

To provide better tools and parameters for medical professionals, future work will include: 1. more realistic constitutive models for the anatomical sections in the model, 2. Solid-Fluids-Interaction (FSI) to analyze the interaction of the brain, CSF, and skull during deformation, 3. an in-depth analysis of the stress waves traveling in the brain and skull, 4. tissue damage criteria for the model, and 5. the effects of neck muscle stiffening during impact. Additionally, these future models should be continually compared to and further validated by experimental data.

5. Acknowledgements

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