SAND2010-0371C

Simulation and Correlation of Blast-Induced, Early-Time Intracranial Wave Physics with Traumatic Brain Injury

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Abstract. Many U.S. troops in Iraq are suffering blast-related, traumatic brain injuries as a result of being within non-lethal distance of a detonated improvised explosive device (IED). Consequently, we are conducting modeling and simulation along with clinical analyses of brain injury to investigate the mechanisms of blast to the human head that give rise to traumatic brain injury (TBI). In particular, we are attempting to identify the specific conditions of focused stress wave energy within the brain, resulting from blast loading to the head, which define a threshold for persistent brain injury. Once this threshold is mapped and correlated with clinical magnetic resonance assessments of blast and blunt trauma victims, we will employ this information to investigate the effectiveness of various helmet designs in mitigating the blast-induced, intracranial stress conditions that otherwise lead to TBI.

1. INTRODUCTION

Recent combat statistics show that several thousand US soldiers have sustained TBI, with over two-thirds of those the result of blasts. Injuries sustained from blast exposure have been categorized into three major components, primary, secondary and tertiary. Primary blast injury is associated with direct exposure of the head and body to the blast wave. In secondary blast injury, debris is accelerated into the individual, while in tertiary injury the victim is thrown into stationary objects by the blast. Importantly, the role of primary blast exposure in the development of TBI is not well understood and is the focus of this work.

In this paper, we describe our modeling & simulation and clinical efforts to establish the role of stress wave interactions in the genesis of traumatic brain injury (TBI) from exposure to explosive blast. The hypothesis underlying our investigation of blast-induced TBI is that stress waves from blast exposure will transmit and reflect within the intracranial contents of the head, resulting in focal areas of elevated stress wave magnitude, energy, and power great enough to cause axonal injury within the brain. If we can establish a correlation between specific levels of the relevant wave physics variables (stress magnitude, energy, etc.) with the onset and severity of TBI, we will then be in a position to assess the effectiveness of various helmet designs in mitigating the conditions leading to TBI.

A description of our investigation into this topic is presented in two parts. The first part, already completed, reports on our work to develop and demonstrate a modeling & simulation (M&S) toolset necessary to simulate blast-induced intracranial wave mechanics in a human head without protection. The second part describes our current efforts to expand upon our preliminary model, establish a correlation between various levels of wave physics quantities and observed axonal injury, and to use these results to assess the effectiveness of various helmet designs in mitigating the blast conditions leading to TBI.

2. PART ONE: PRELIMINARY STUDY

2.1. Head Model & Simulation Methodology

Our first study was based on a high resolution (1 mm^3 voxel), 4 material model of the human head (see Figure 1), created by segmentation of color cryosections from the National Library of Medicine's Visible Human Female dataset [1]. This model consisted of skull, white matter, gray matter, and cerebral spinal fluid (CSF). We assigned equation-of-state and constitutive models to represent the four constituents of the head as well as the air that surrounds the head and occupies the sinuses.

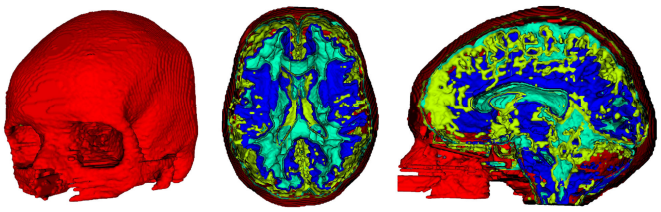

Figure 1. Head model depicting bone (red), white matter (light blue), gray matter (dark blue), and cerebral spinal fluid (yellow). Voxel resolution: 1 cubic millimeter.

The skull was represented by a compressible, elastic perfectly plastic model and an accumulated strain-to-failure fracture model, fit to data reported by Carter [2] for cortical bone. The white and gray matter were modeled as compressible, viscoelastic materials with constitutive properties reported by Zhang et al. [3]. The CSF, which resides in the ventricles and subarachnoid spaces within the head, was modeled as a slightly compressible, Newtonian fluid with density and viscosity similar to those of water [4]. The air surrounding the head and occupying the sinuses was represented by a nonlinear equation-of-state model for dry air specifically designed for shock wave applications [5].

The model was inserted into the shock physics wave code, CTH [6], and subjected to a simulated IED blast wave of 1.3 MPa (13 bars) peak pressure (see Figure 2) from anterior, posterior and lateral directions.

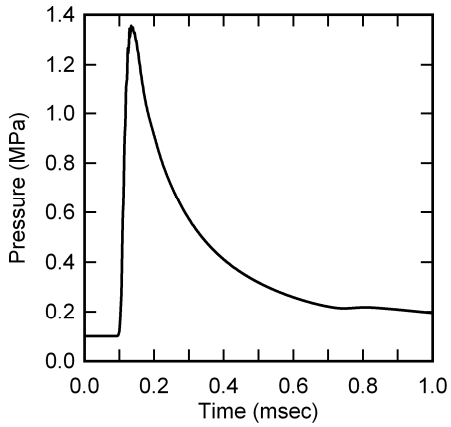

Figure 2. Wave form of approximated air blast of 1.3 MPa (13 bars) magnitude.

An example of our methodology is displayed in Figure 3 where a frontal (anterior) blast is simulated with the pressure plots in the mid-sagittal plane displayed as a function of time. Further details of our head model and simulation methodology can be found in reference [7].

2.2. Simulation Results

Three-dimensional plots of maximum pressure, volumetric tension, and shear stress demonstrated significant differences related to the incident blast direction. As an example, we plot maximum shear stress in mid-ventricular axial and mid-sagittal planes as a function of blast direction in Figure 4. In that figure, the axial images in row A show considerable differences in shear localization in all three blast scenarios. More focal areas of elevated shear were seen in the lateral blast scenario (multiple red areas within the red oval) whereas the anterior corpus callosum showed greater diffuse shear in the anterior blast (black arrow) but focal shear locations in the posterior blast (red arrows). The occipital area (white arrow) was most affected in the posterior blast scenario. In the mid-sagittal views of row B, all blast orientations generated higher shear stress in the sub-frontal regions (red ovals) and brainstem areas. Posterior blast was the only scenario generating high shear stress levels near the midline occipital region (white arrow).

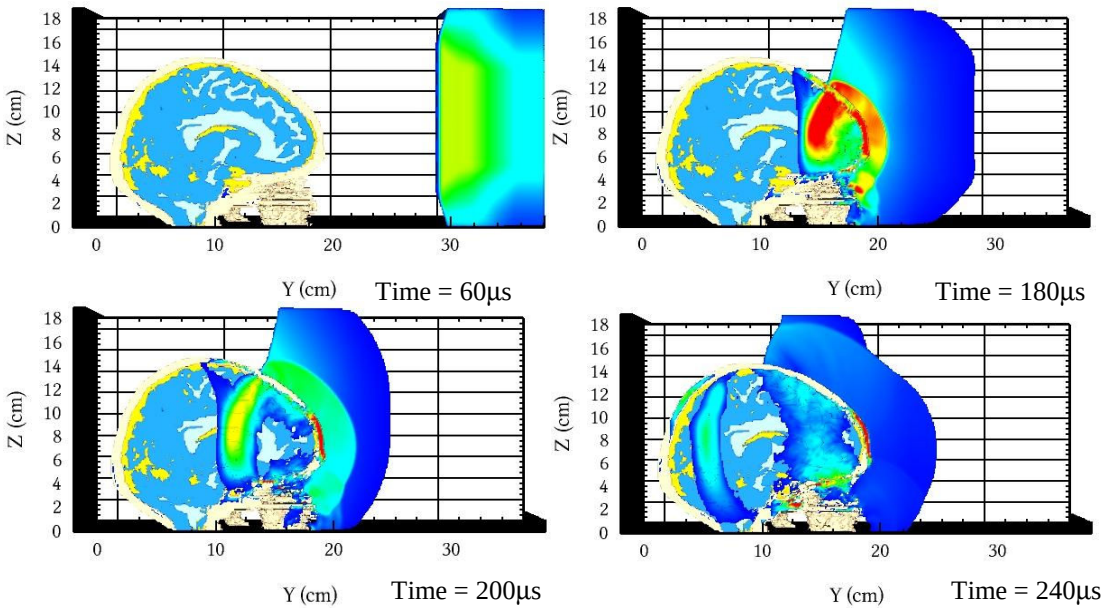

Figure 3. Pressure plots in mid-sagittal view of 1.3 MPa frontal air blast scenario at various times. Color scale: blue = 0.1 MPa, green = 0.5 MPa, yellow = 1.5 MPa, red = 5 MPa.

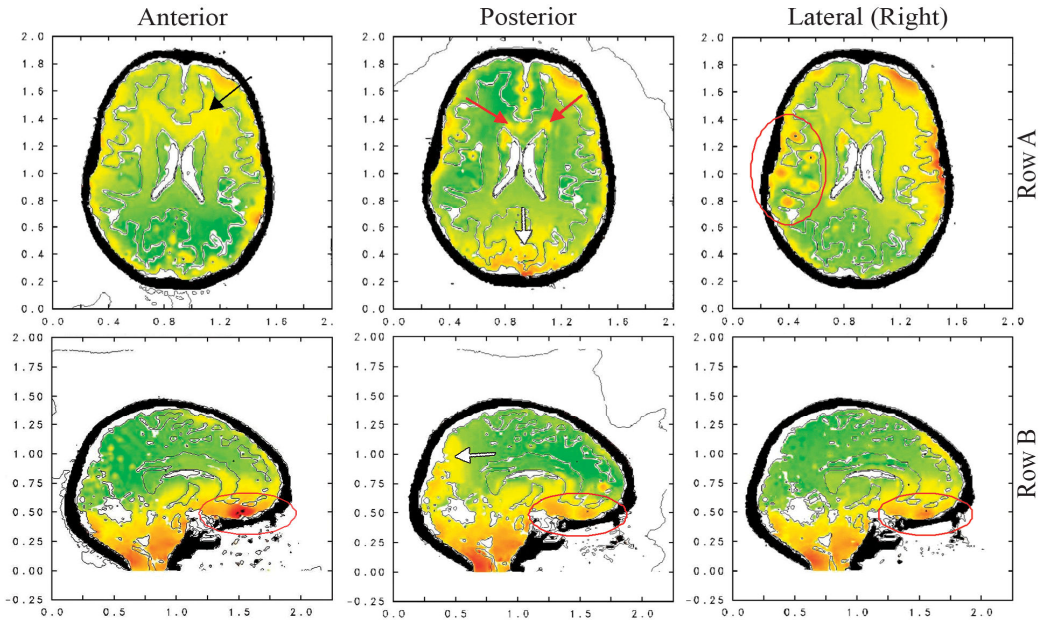

Figure 4. Maximum shear stresses in a mid-ventricular axial plane (row A) and mid-sagittal plane (row B) for anterior, posterior, and right lateral blast orientations. Color scale: Row A: green = 0.1 KPa, yellow = 0.6 KPa, orange = 2 KPa, red = 15 KPa; Row B: green = 0.1 KPa, yellow = 0.7 KPa, orange = 3 KPa, red = 25 KPa. Arrows and ovals indicate regions of focal or diffusely elevated shear stress levels.

The calculations further revealed focal brain regions of elevated pressure (not shown) and shear stress within the first 2 milliseconds of blast exposure. Calculated maximum levels of 15 KPa shear, 3.3 MPa pressure, and 0.8 MPa volumetric tensile stress (not shown) were observed before the onset of significant head accelerations. In fact, intracranial stress wave formation, propagation, and focusing were predicted to occur before the head moved 1 mm in response to the blast. Doubling the blast strength changed the resulting intracranial stress magnitudes but not their distribution.

The study by Zhang, et al. [3], of football collisions, predicted shear stress levels of 3.1-6.4 KPa in the thalamus that were associated with concussive injury and mild TBI. If those levels represent a basic injury threshold for shear stress, then we conclude that the stress levels we calculated in the brain from blast-induced intracranial wave motion alone will be significant and could contribute to the development of multifocal axonal injury underlying TBI. Consequently, our preliminary work suggests that traumatic brain injury may be caused from blast exposure alone, resulting from stress waves that focus in critical regions of the brain before the onset of significant head motion, and supporting the hypothesis that primary blast injury is a viable mechanism leading to TBI.

3. PART TWO: CURRENT STUDY

3.1. Description

In the current study, we expand the scope of our work to include modeling and simulation (M&S) as well as a clinical aspect to investigate the severity of TBI in human subjects. Our intent is to (1) – further develop the M&S toolset for greater fidelity, (2) – recruit and assess brain injury in TBI subjects, (3) – simulate injury scenarios of a select group of TBI subjects, (4) – attempt to identify and correlate magnitudes and rates of relevant wave physics quantities with specific brain injury as identified in the clinical analyses of the TBI subjects, giving rise to a Brain Injury Threshold Criterion (BITC), and (5) – employ the BITC to assess the effectiveness of various helmet designs in the mitigation of both blast and blunt impact.

3.2. Head Model & Investigative Methodology

Although the earlier study provided useful guidance for further investigation into blast-induced TBI, it was deficient in a number of anatomical components. For example, the head model, described in section 2.2, lacked a variety of internal and external tissues structures; specifically, the neck, lower brainstem, falx and tentorium (membranes within the brain), and soft tissue external to the skull (scalp, musculature, etc.). Our newest head model, depicted in Figures 4 and 5, is based on the National Library of Medicine's Visible Human Male dataset [1] and includes these structures as well as the components comprising the earlier model, i.e., bone, white and gray matter, and CSF, totaling 6 materials at a voxel resolution of 1 mm^3.

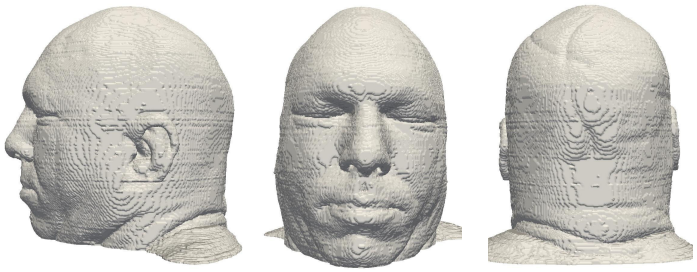

Figure 4. New head model showing external views.

The new head model exists in both finite volume (voxel) and finite element versions so that we may conduct blast and impact simulations by means of Eulerian or Lagrangian finite volume and finite element methodologies. The model also employs viscoelastic constitutive models to represent the various soft tissues and a model similar to the elastic-plastic-fracture model employed with the earlier head model to represent bone materials, including skull, mandible, and cervical vertebra.

As mentioned above, we are expanding our simulation methodology to include both Eulerian and Lagrangian simulation methods so that we can investigate blast loading as well as blunt impact. This is essential if we are going to attempt to establish a correlation between head insult and traumatic brain injury.

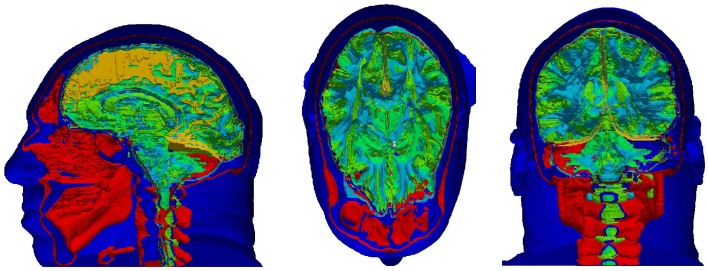

Figure 5. Head model depicting bone (red), membranes (orange), CSF (green), white matter (light blue), gray matter (medium blue), scalp & musculature (dark blue).

3.3. Clinical Methods

A modeling and simulation approach to developing mitigation strategies for impact and blast induced injury is attractive on many levels. A robust predictive tool would lessen the need to create multiple physical models to be subjected to damage scenarios, allow repeated simulations with variable geometry and loading levels, and provide insights for the properties of armor materials that might yield optimized protection. An important limitation of M&S methods is the possibility that the observations made do not reflect real world consequences, especially for biological systems. For this reason, we are currently engaged in specific studies of brain injured people to provide a clinical correlation with the M&S predictions.

The major goals of our study are to confirm the locations of brain injury in subjects with TBI from blunt impact and explosive blast, compare them with patterns predicted from the model and establish a threshold of wave energy deposition from simulations that predicts tissue damage to the brain. These clinical correlations and injury thresholds would help promote confidence in simulated mitigation strategies that reduce wave energies from blast or impact.

Many investigations of brain pathology after TBI show that death and poor outcome can occur as a result of injury to axons at the microscopic level. Called diffuse axonal injury or DAI, this pathology describes a multifocal process in white matter tracks and at tissue interfaces within the brain. With DAI, axons show disruption of their cytoskeletal structure, alterations in membrane permeability and cellular energetics, and the development of swelling. The culmination of this pathological cascade can be the transection of axons, analogous to cutting wires in an electrical circuit. All of the cognitive, motor, and behavioral activities of the human brain can be adversely affected by this injury mechanism.

Sometimes routine brain imaging by MRI or Computed Tomography (CT) methods show areas of the brain where tissue injury has occurred due to trauma. However, it is well known that severe TBI can exist in subjects with normal brain imaging in these routine scans. Fortunately, non-conventional imaging sequences, such as Diffusion Tensor magnetic resonance Imaging (DTI), can show areas of axon injury otherwise missed on standard scans [8,9].

Our hypothesis and justification for the clinical correlation studies is that stress waves from impact or blast exposure will transit and reflect within the intracranial contents of the head, resulting in focal areas of elevated wave energy great enough to cause axonal injury. We will attempt to detect areas of axonal injury by performing DTI on patients with a history of blast- or impact-induced TBI. Comparisons and possibly statistical correlations will be made between observed injury patterns from the two mechanisms and the predictions from M&S analyses of the events that gave rise to the brain injury.

3.3.1 Experimental design

We will identify two groups of subjects who have experienced a traumatic brain injury. The first group will have impact trauma from motor vehicle accidents, falls or assaults. The second group will be combat veterans with TBI resulting from blast exposure. Subjects will be at least 3 months from the date of injury and have sufficiently recovered to participate in the study procedures and to give informed consent. We will attempt to obtain medical records of their injury and hospitalization to confirm the severity of the initial injury, the results of prior imaging studies (e.g., MRI), and the pace of their recovery of consciousness and discharge from hospital care. When possible, we will try to ascertain the conditions

giving rise to their injury, for example the direction of the impact and the best estimate of intensity (e.g., the speed of the vehicle or magnitude of the explosion).

3.3.2 Study procedures

History and exam: Subjects will undergo a history and basic neurological exam to assess subtle focal injury signs. We will try to ascertain the history of injury and problems occurring in the post-injury period related to memory and cognitive problems, headaches, symptoms of post-traumatic stress disorder, and mood disorder.

Cognitive testing: To stratify TBI subjects and compare them to the control group, subjects will undergo a brief neuropsychiatric battery of tests to assess domains of memory and learning, attention, executive function, processing speed, and mood.

MR Imaging: MRI and DTI studies will be performed on the Siemens 3 Tesla Magnetom Trio at the MIND Research Network, located on the University of New Mexico Health Sciences Center campus in Albuquerque, New Mexico, USA. The following imaging sequences will be obtained:

1. Standard proton density and T2 weighted anatomic imaging to look for signal changes compatible with macroscopic brain injury
2. High resolution 3D T1 weighted sequence for segmenting the brain into major tissue types
3. DTI series to detect microscopic regions of brain injury to be correlated with simulation results
4. Resting state functional MRI as an exploratory measure of changes in brain activity related to TBI

3.3.3 Clinical data analysis

We will obtain anatomic imaging of the subjects to be reviewed by a neuro-radiologist for findings suggestive of TBI. The three-dimensional (3D) sequences will be segmented to allow data correlations with specific brain regions such as white matter tracks, cortex, nuclei and tissue interfaces. We will then attempt a correlation of voxel-based cluster peak locations, displaying changes in diffusion tensor anisotropy (a DTI measure of local damage), to regions of predicted stress wave magnitude, energy, and power deposition.

The overall clinical plan for this study is to acquire data from 20 subjects with blunt trauma and 20 from military personnel injured in blast exposures followed at the Veterans Administration Hospital in Albuquerque, New Mexico, or living in our community. An additional 10 normal controls will be studied for comparison. The baseline characteristics of our first 3 subjects are summarized in Table 1. Correlations with simulated data will be used to verify the predictions of the model. If the modeling studies are able to predict locations of observed injury, the relevant wave physics metric (e.g., stress magnitude, wave energy, etc.) will be computed in simulations of several injury scenarios in which the geometry and external forces are best known. For example, the injury of subject #3, listed in Table 1, who suffered an impact to the head from a thrown brick. This scenario could be simulated with relative accuracy. These experiments could provide direct correlations of prediction to observation and help identify the threshold wave energies needed to produce the damage seen on MRI and cognitive deficits measured by the neuropsychiatric battery.

3.3.4 Examples of clinical data

Clinical histories, cognitive testing and imaging data have been acquired for 3 subjects with impact trauma from motor vehicle accidents and assaults (see Table 1). This initial group has helped to establish the clinical protocol and the overall impact data set will provide an important comparison to the blast injured cohort. It will be more common to find impact TBI patients whose mechanism of injury is sufficiently known to allow for a corresponding simulation of the event. Examples of the imaging data are also shown for subject 1, who was struck on the left face with a bat resulting in loss of consciousness and a skull fracture. His anatomic imaging, illustrated in Figure 6, did not show any areas of macroscopic brain injury. The DTI sequences were used to create tractography images shown in Figure 7. These images demonstrate how the diffusion tensor from each image voxel can be used to reconstruct the direction of axon fiber tracks

in the brain. It is difficult to visually detect fiber loss in these images but focusing the tractography on specific structures, like the corpus callosum, can provide evidence of axon loss (Figure 8).

A better way to analyze DTI data is to create maps of diffusion variables like fractional anisotropy or apparent diffusion coefficient that can be overlaid on anatomic images and quantitatively analyzed for evidence of axon injury. This data can be averaged across subjects and subjected to statistical correlations with our simulation predictions. The experimental plan includes the use of sophisticated voxel-based and cluster analysis methods to quantify brain regions with abnormal diffusion properties caused by TBI. These studies will provide the clinical connection that we hope will validate the predictions of the simulation experiments and allow the computer model to serve as a useful tool for mitigation designs.

Table 1. Summary of TBI subjects tested and scanned to date.

Subject	Injury	Cognitive Deficit	Imaging	Simulation of Event Possible?
1) 40 year old male	Assault with bat; left skull fracture	Mild decreased working memory	Displayed in Figures 6-8	Mechanism and geometry known
2) 18 year old female	Rear end motor vehicle accident; Head hit steering column	Impaired attention, learning, & processing speed	No focal defects on standard MRI; DTI in process	Possible; Vehicle speed must be ascertained
3) 24 year old male	Brick thrown; Right frontal skull fractured	Scheduled (to be determined)	No focal defects on standard MRI; DTI in process	Direction, location of impact known; Velocity to be estimated

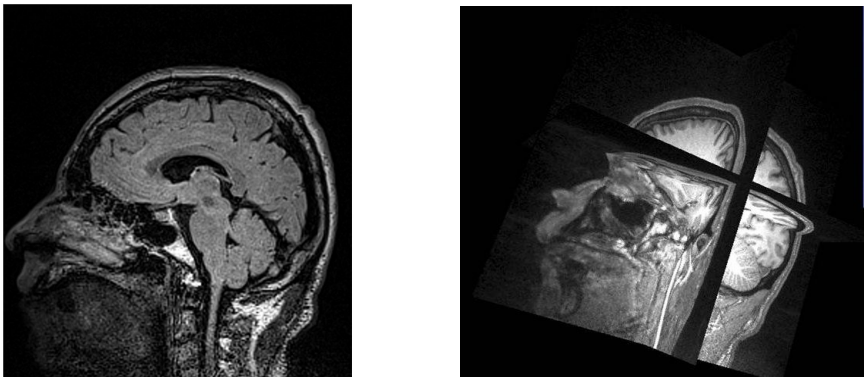

Figure 6. Subject #1 anatomic MRI (left) and 3D reconstruction for segmentation (right).

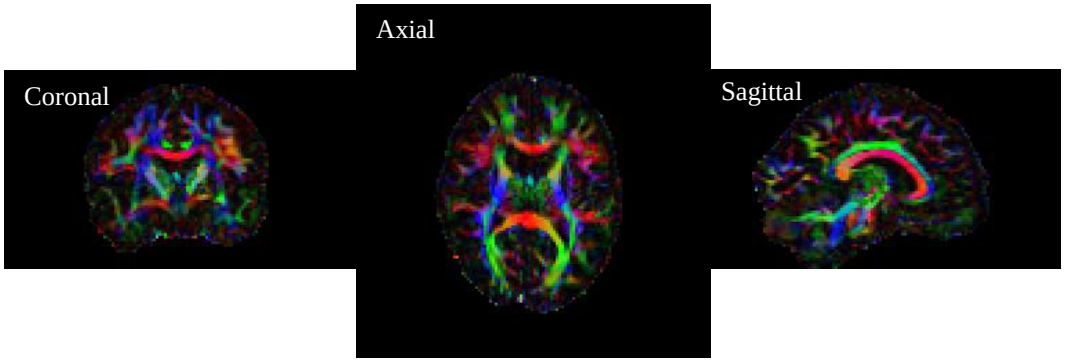

Figure 7. Diffusion tensor tractography of subject #1. Red tracts run left-right, green run anterior-posterior, blue run inferior-superior through the brain.

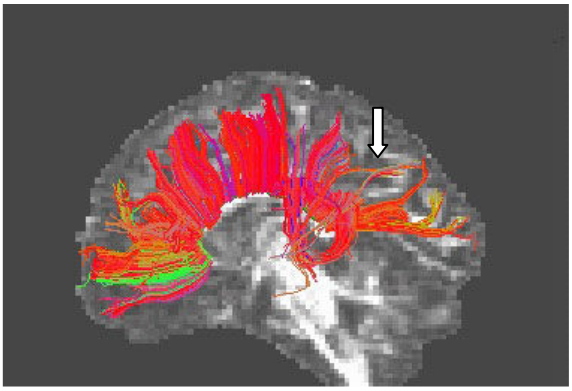

Figure 8. Subject #1 diffusion tensor tractography of axon fibers in the corpus callosum. White arrow points to area of possible fiber loss, thereby identifying localized axonal injury.

4. ASSESSMENT OF HELMET DESIGN

Once we correlate our modeling and simulation (M&S) approach with clinical DTI analyses of brain injured subjects and have established a set of criteria for localized brain injury as outlined in Section 3, our follow-on task will be to apply these results to assess various military helmet designs. In particular, we are interested in assessing the effectiveness of helmet designs to protect the warfighter against blast that would otherwise lead to TBI. This task will rely extensively on the use of our M&S tools, developed for the current study, to simulate various blast scenarios involving our head model wearing protective gear.

One of the first tasks of this effort will be the assessment of existing helmet designs, such as the Army ACH helmet [10] depicted on our head model in Figure 9. Some aspects of existing helmet designs worth investigating would be the helmet's baseline performance as well as modifications to the existing design. Helmet modifications that would merit study include the selection of replacement materials for the interior cushion pads as well as the placement configuration of the pads.

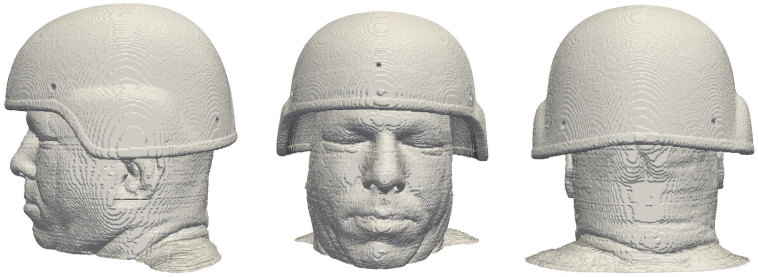

Figure 9. Head model wearing the Army ACH helmet.

New helmet design aspects worthy of investigation would include shape and composition of the outer shell, cushion pad composition and placement, and the influence of add-on protective devices for mission-specific applications.

By demonstrating a modeling and simulation approach to the task of helmet design, we hope to significantly reduce the amount of laboratory and field testing required in developing new designs that can effectively protect our warfighters from exposure to explosive blast.

Acknowledgments

This work is funded through the Naval Health Research Center, Office of Naval Research.

Sandia is a multiprogram laboratory operated by Sandia Corporation, a Lockheed Martin Company, for the United States Department of Energy's National Nuclear Security Administration under Contract DE-AC04-94AL85000.

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