6th European Conference on Computational Mechanics (ECCM 6)
7th European Conference on Computational Fluid Dynamics (ECFD 7)
11 – 15 June 2018, Glasgow, UK

UNDERSTANDING THE PATHOLOGY OF BLAST-INDUCED TRAUMATIC BRAIN INJURY

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Key words: Blast-induced Traumatic Brain Injury (bTBI), Intracranial Pressure, Shear Stress, Principal Stress, Injury Mechanism

Abstract. Many service members who have been exposed to blast shockwaves, suffer serious lifetime injuries, many of which are fatal. Treatment of such injuries requires a thorough understanding of the mechanisms leading to them. Hence, a computational study was developed to investigate the role of different injury mechanisms in bTBI. An open blast scenario for the detonation of 70 grams TNT at a stand-off distance (detonation to the head-front distance) of 60 cm was modeled using a finite element multi-material arbitrary Lagrangian Eulerian (MM-ALE) approach. The tissue response of the 50th percentile male North Dakota State University Head Model (NDSUHM) was evaluated in terms of intracranial pressure (ICP), maximum shear stress (MSS), and maximum principal strain (MPS) using two different blast injury models. In the first model, skull was considered rigid to account only for the head acceleration's contribution on the dynamic responses of the brain (Rigid-skull model) while in the second model skull was treated as an elastic material to also allow for the contribution of skull deformation to the injury metrics (Inclusive model). An overpressure of 520 kPa, representative of lung injury threshold, was generated and the head was exposed to blast waves from the front. Peak ICPs, MSSs, and MPSs, were recorded in both blast models for the unprotected head. While Rigid-skull model predicted a peak ICP (200 kPa) about 56% of the Inclusive model (356 kPa), both MSS and MPS values for the Inclusive model (2.82 kPa, 1.8%) were insignificantly lower than the ones for the Rigid-skull model (3 kPa, 2%). The maximum ICP for the inclusive was shown to occur at the parietal lobe while it occurred exclusively at the frontal lobe for the Rigid-skull model. Considering other injury mechanisms such as skull flexure and cavitation, our model predicted that shockwaves propagation had a significant and the highest contribution to the development of the hydrostatic response in the brain, while it had negligible effect on the deviatoric responses of the brain tissue.

1 INTRODUCTION

Increased use of improvised explosive devices (IEDs) has escalated the prevalence of blast-induced traumatic injuries (bTBIs). Blast TBI has been reported to be a major cause of life-threatening injuries as well as severe post-war neurotrauma among military personnel. Neuropathological changes such as neuronal degeneration, brain edema, and diffuse axonal injuries can occur as a result of bTBI. There are three major types of bTBI: primary resulting from shockwave propagation within the cranium, secondary which occurs when head is hit by objects propelled by blast wave, and tertiary, which occurs mainly when the person is accelerated or decelerated by the wind of the blast. Primary bTBI occurs through different mechanisms such as shockwave propagation inside the cranium, head acceleration, skull flexure, and cavitation [1-5]. While the contribution of each mechanism is different, the overall injury rate depends on the combination of all effects [6].

Many researchers have studied the biomechanical responses of the brain in terms of intracranial pressure (ICP), and shear stress and strains assuming the combination of different mechanisms but have not discussed their effects individually [7-10]. However, understanding the contribution of different injury mechanisms is critical for developing more effective protective tools. This has been the subject of many studies since understanding and improving the protection capability of military helmets can significantly contribute to the prevention of bTBI [11-14]

The main objective of this study is to investigate the role of shock propagation and head acceleration injury mechanisms in the bTBI pathobiology. This is the first study that takes advantage of two blast injury models to investigate contributions of these mechanisms to bTBI using a detailed 3D head model.

2 METHODS

A detailed 3D finite element model representing a 50th percentile male head was used for our study which includes major anatomical components of the human head (Figure 1). The details of this head model can be found in [15]. An open blast scenario for the detonation of 70 grams TNT at a stand-off distance (detonation to the head-front distance) of 60 cm was modeled using a finite element multi-material arbitrary Lagrangian Eulerian (MM-ALE) approach (Figure 2a) [16].

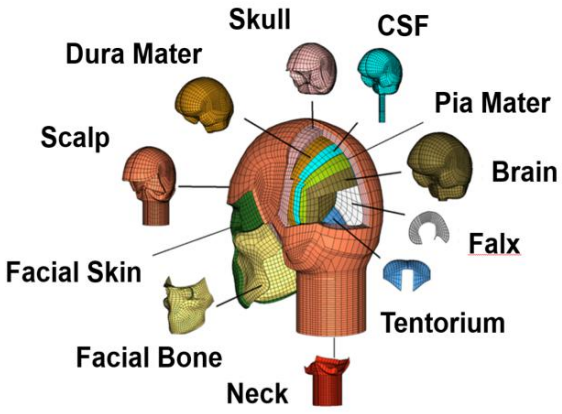

Figure 1: Finite Element model of the head

An overpressure of 520 kPa, representative of lung injury threshold [13], was generated and the head was exposed to blast waves from the front. The tissue response of the brain was evaluated in terms of intracranial pressure (ICP), maximum shear stress (MSS) and maximum principal strain (MPS) using two different blast injury models (Figure 2b).

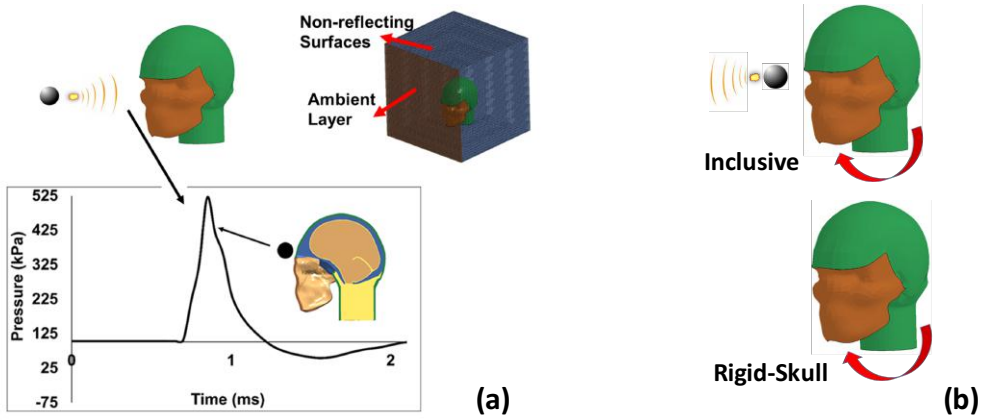

Figure 2: (a) Blast modeling and boundary conditions; (b) Blast TBI models

In the first model, skull was considered rigid to account only for the head acceleration's contribution to the dynamic responses of the brain (Rigid-skull model) while in the second model skull was considered deformable to also allow for the contribution of skull deformation to the injury metrics (Inclusive model). Brain tissue was modeled as a hyper-viscoelastic material using Mooney-Rivlin hyperelastic and Maxwell linear viscoelastic constitutive models, while other head components were considered as linear elastic [15]. Material properties for the hyper-viscoelastic brain tissue and linear elastic head components are presented in Table 1 and Table 2, respectively [15, 17, 18].

Table 1: Parameters of hyper-viscoelastic brain material.

C_{10}(Pa)	C_{01}(Pa)	G_1(kPa)	G_2(kPa)	$\beta_1(s^{-1})$	$\beta_2(s^{-1})$	K (GPa)
3102.5	3447.2	40.744	23.285	125	6.6667	2.19

Table 2: Mechanical properties of the elastic head components.

Head Component	Density (g/cm^3)	Elastic Modulus (GPa)	Poisson's Ratio	Bulk Modulus (GPa)
Scalp/ Skin	1.2	0.0167	0.42	
Skull	1.21	8.0	0.22	
Dura, falx, tentorium	1.133	0.0315	0.45	
Pia mater	1.133	0.0115	0.45	
Facial bone	2.10	5.54	0.22	
Cervical Vertebrae	2.5	0.354	0.3	
CSF	1.004	---	0.499	2.19

3 RESULTS

Primary blast-induced neurotrauma mainly occurs due as a result of wave propagation in the intracranial space and head acceleration. Peak ICPs, MSSs, and MPSs, were recorded in both blast models. While Rigid-skull model predicted a peak ICP (199.3 kPa) about 56% of the Inclusive model (356 kPa), both MSS and MPS values for the Inclusive model (2.82 kPa, 1.8%) were similar to the ones predicted by the Rigid-skull model (3 kPa, 2%). The maximum ICP for the Inclusive model was shown to occur at the frontal-parietal and temporal lobes while it occurred exclusively at the frontal lobe for the Rigid-skull model. However, maximum shear stresses occurred at the intersection of temporal lobe and parietal lobes in both models. Figures 3a & b show instantaneous variation of ICP and shear stress within the brain tissue for the Inclusive model, respectively, which highlights the regions undergoing the maximum and minimum ICPs and stresses.

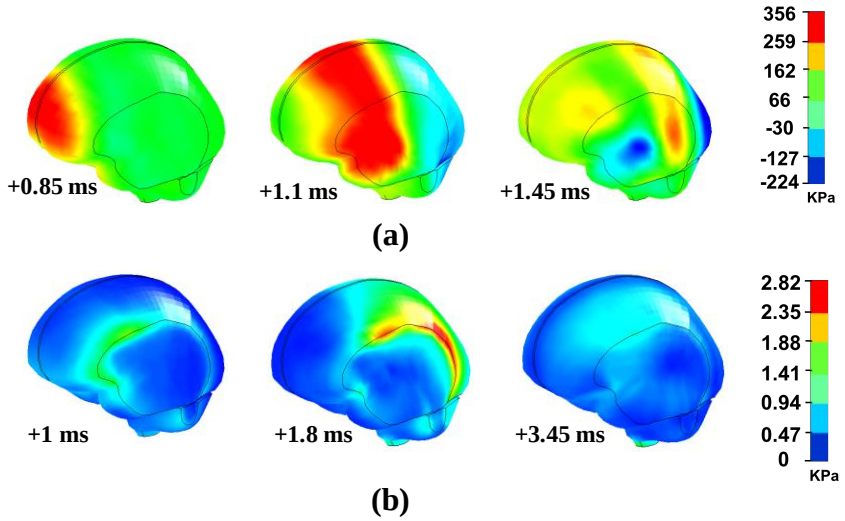

Figure 3: Variation of (a) ICP and (b) shear stress in the brain tissue for the Inclusive model

Figures 4a & b compare the temporal variation of the ICP and shear stress between two injury models at the region of maximum pressure and shear stress, respectively (generated by averaging the time histories of several elements in these regions).

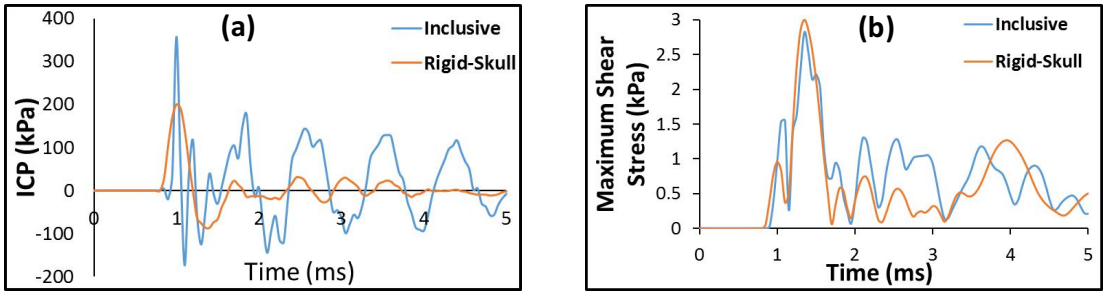

Figure 4: Comparison of time history of (c) ICP and (d) shear stress in the regions of maximum pressure and shear stress between inclusive and rigid-skull models

4 DISCUSSIONS AND CONCLUSIONS

Considering other injury mechanisms such as skull flexure and cavitation, our TBI models predicted that shockwaves propagation had a significant and the highest contribution to the development of the hydrostatic response in the brain, while it had negligible effect on the deviatoric responses of the brain tissue. Two major findings of this study are the difference in the value and the location of the maximum ICP between TBI models. While the Inclusive model predicted ICP levels higher than the mild TBI (mTBI) threshold [15], exclusion of the wave propagation in the Rigid-Skull model resulted in extreme understatement of injury risk [19]. Interestingly, both models predicted shear stresses well below the mTBI level of 7.8 kPa as reported by Zhang *et al*. [18].

Relocation of the maximum ICP region in our models suggested that shockwave transfer inside the cranium could extend the increased ICP region to different lobes of brain such as temporal and parietal lobes which could be associated with injury mechanisms other than those in the frontal lobe. Based on these findings from our case studies, we speculated that rigid-skull models may be able to give information on the risk of concussive and diffuse injuries that are mainly associated with shear response of the tissue. Such models could significantly save computational costs. However, this should be further verified by both more computational as well as experimental analyses.

The outcomes of this study could be used toward further improvement of current protective tools by evaluating the mitigating potential of them with respect to each injury mechanism. Future works could investigate the contribution of other injury mechanisms to bTBI, as well as inclusion of different blast scenarios.

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