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

Hesam S. Moghaddam[1], Mariusz Ziejewski[2], Ghodrat Karami[2]

[1] Northern Arizona University, Flagstaff, AZ 86005, hesam.moghaddam@nau.edu
[2] North Dakota State University Fargo, ND 58108, mariusz.ziejewski@ndsu.edu, g.karami@ndsu.edu

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

Increased use of improvised explosive devices (IEDs) has escalated the prevalence of blast-induced traumatic injuries (bTBIs). Primary bTBI occurs through different mechanisms such as shockwave propagation inside the cranium, head acceleration, skull flexure, and cavitation. While the contribution of each mechanism is different, the overall injury rate depends on the combination of all effects. The main objectives of this study are to investigate the role of shock propagation and head acceleration injury mechanisms in the bTBI pathology as key factors for developing efficient protective tools.

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. 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.