

Characterization of Brain Material Properties following Brain Blast Injury

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Abstract

Improvised explosive devices (IEDs) have caused traumatic brain injury (TBI) in approximately 360,000 soldiers over the past decade. This injury requires immediate medical attention and victims can suffer from long-term neurological consequences when treatment is delayed. Early diagnosis of blast TBI (bTBI) may improve the clinical outcome of these patients. A better understanding of the mechanical response of the brain during and after these events may assist diagnosis of bTBI in both clinical and battlefield scenarios. Diagnostic methods using non-invasive stiffness techniques, such as field-deployable ultrasound, rely on detecting changes to the mechanical properties of brain tissue after injury; however, these tools require a priori information on how injured tissue correlates with mechanical changes. Moreover, changes to the brain mechanical properties with a blast injury may be a) region-specific, b) time-specific, and c) blast severity-specific. The goal of this study is to characterize changes in the mechanical response of brain tissue following blast injury. This will improve our understanding of the mechanical response of the brain following injury for assessment and diagnosis of blast TBI.

Thirty adult, male Sprague-Dawley rats were exposed to a primary blast wave generated by a compressed-gas driven shock tube. Exposed animals were placed inside the shock tube and subjected to a single ideal blast wave from one of two levels of blast severity: low (18-20 psi peak overpressure, 5 ms duration) or high (30-25 psi peak overpressure, 5 ms duration). Sham animals placed briefly inside shock tube but not exposed to blast injury. All animals were anesthetized before injury and euthanasia. Animals were sacrificed either at 2 or 24 hours following injury. Five animals were used for each of the six groups—sham 2 hour and 24 hour, blast low 2 hour and 24 hour, and blast high 2 hour and 24 hour. Whole brains were extracted immediately following death and sectioned in the coronal plane to extract 3 slices: frontal cortex, midbrain, and cerebellum. Ramp and hold indentation tests with a cylindrical indenter were performed at a depth of 0.6 mm and ramp hold of 30 s at 5 locations: frontal cortex, midbrain superior, midbrain aqueduct, midbrain inferior, and brainstem. Force-displacement data were analyzed using a 1-way anova followed by Tukey HSD multiple comparisons.

Significantly higher forces were measured in the midbrain inferior region in the blast high 24 hour when compared to sham group (+50%, $p < 0.05$). This stiffening could be due to edema and other physiologic responses to injury. In addition, we observed lower forces in the brainstem region (-43%, $p < 0.05$) of the blast low 24 hour as compared to the sham group. Bleeding is observed in the pons due to brain blast injury, which can contribute to this change. There were no significant changes in the 2 hour groups.

The results show a temporal, regionally-dependent mechanical response—stiffening in the blast high 24 hour, softening in blast low 24 hour—to injury. The mechanical changes can serve as correlates to injury to improve detection and diagnosis of bTBI.