

Mechanical Response and Brain Injury of Swine Subject to Free-Field Blast

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Abstract

*The mechanisms by which primary blast overpressure produce mild to moderate traumatic brain injury is still unclear. The assessment of the severity of injury following primary blast is still a great challenge clinically. The aim of this study is to explore and quantify information between mechanical response and brain injury on live anesthetized swine exposed to open-field blast. The specific aim of the study is to determine the relationship between intracranial pressure in the brain and its effect on axonal damage and cellular injury. **METHODS:** Mechanical Response: Nineteen open field blast tests were performed on 3 instrumented anesthetized or expired Yucatan swine under 3 different nominal incident pressure levels (150, 300 and 450 kPa) in 3 directions (front, side, rear) to obtain biomechanical data. Six Kulite piezoelectric pressure sensors were inserted in the swine brain to measure pressure at frontal, left and right temporal, parietal, occipital and central brain regions during blast. Three linear accelerometers and three angular rate sensors were mounted to the skull to record head kinematics during blast. Pressure and acceleration data were analyzed. Injury and biomarker data: Five non-instrumented anesthetized swine were exposed to frontal 300 kPa blast and survived for 7 days. An additional 4 sham animals survived for 7 days. Serum biomarker levels are being assessed 6, 24, 48 and 72 hours post blast and brain histology at 72 hours post-blast. Serum at the stated time points is being assessed for temporal changes in various brain biomarkers (S100B, NSE, MBP, NF-H, SBDP, IL-6, and HSP-70). The histology of brain injury is being assessed as follows: beta amyloid precursor protein, neurofilament light chain and silver staining for axonal injury, glial fibrillary acidic protein (GFAP) and microglial (Iba1) for inflammatory response, caspase 3 immunocytochemistry, fluoroJade B, TUNEL and H&E staining for cellular injury and Prussian blue staining for hemorrhage. **RESULTS:** For the instrumented tests intracranial pressure data was typically lower than the incident (pencil) pressure. In the front blast group, the highest intracranial pressures were recorded in the parietal region under 300 and 450 kPa incident pressure. In the rear and side blast groups, the highest intracranial pressure was in the occipital region under all three levels of incident pressure. **CONCLUSIONS:** Within each blast direction group, all the intracranial pressure increased with higher blast overpressures. Investigation of the relationship of blast pressure to quantitative changes in brain histology is ongoing.*