Injury Models

Untangling the Effect of Head Acceleration on Brain Response to Blast Waves

Several mechanisms have been postulated to contribute to blast-induced TBI, including wave propagation, skull flexure, cavitation (bubble formation and subsequent collapse), and head acceleration. Despite numerous experimental and computational studies that have examined how these mechanisms potentially contribute to blast-induced TBI, there is not yet a quantitative description of the specific contribution of each of these mechanisms. Such a quantitative understanding could be key not only in understanding the etiology of blast-induced TBI, but also in guiding the development of equipment that will offer better protection to Service Members from explosive events. BHSAI, a subordinate organization of USAMRMC TATRC, has initiated computational investigations on the interaction of a blast wave with an animal head in order to determine the contribution of head acceleration to blast-induced TBI. BHSAI scientists developed a shock tube and two-dimensional rat head models and performed blast simulations to quantify the contribution of head acceleration to the biomechanical responses of brain tissues when exposed to blast waves in a shock tube. Biomechanical responses, such as pressure, stress, and strain, are correlated to brain tissue damage, and they provide valuable information on the response of the brain tissue to external mechanical loading, such as blast overpressure. By comparing pressure between the head model that captured all the mechanisms of blast-induced TBI (i.e., wave propagation, skull flexure, cavitation, and acceleration) to an acceleration-only model, BHSAI was able to calculate the relative contribution of head acceleration to blast-induced TBI. In addition, BHSAI investigated the response of the brain tissues to the different orientations of the blast wave. These simulations determined that head acceleration contributes 36 to 45 percent, depending on head orientation relative to the blast wave, of the maximum brain pressure at the coup region (area of the brain directly hit by the explosion), had a negligible effect on the pressure at the middle region, and was responsible for the low pressure at the contre-coup region (the region of the brain across from the coup region). These findings provide important guidance when performing blast-induced TBI experiments on small animals, as they demonstrate that the current practice of measuring rat brain pressure close to the center of the brain would record only two-thirds of the maximum pressure observed at the coup region. Therefore, it is recommended that in order to accurately capture the effects of acceleration in experiments, a pressure sensor be placed near the coup region, especially when investigating the acceleration mechanism using different experimental set-ups.