Injury to Sensory Systems

Assessment and Treatment of Blast-induced Auditory and Vestibular Injuries

The most common symptoms after blast exposure are headaches, hearing loss, balance problems, and dizziness which strongly suggest blast waves caused injury/impairment to the structure of the inner ear and neuronal encoding of sound. Researchers at the Walter Reed Army Institute of Research (WRAIR) have developed complementary rodent models to characterize the effects of blast exposure on both the auditory and vestibular organs of the inner ear in conjunction with assessments of the disruptions in connections among the brain structures involved in auditory and vestibular signal processing. In collaboration with the National Institute for Deafness and Other Communication Disorders and the Lieber Institute for Brain Development at the Johns Hopkins School of Medicine, researchers at WRAIR are developing strategies for mitigating or reversing auditory/vestibular injury that originates from damage to mechanosensory hair cells and brain structures. Using an advanced blast simulator (ABS), which produces a high fidelity recreation of blast overpressure (BOP) in the laboratory; rodents are exposed to shock waves to characterize the etiology of blast-induced hearing loss and balance disorders. Along with histopathological and neurochemical assessments and the quantification and characterization of the auditory and vestibular injuries, the efficacy of therapeutic interventions are judged by a battery of functional assessments, including auditory brainstem response (ABR), distortion product otoacoustic emission (DPOAE) testing, vestibular sensory evoked potentials, and vestibulo-motor functional measurements. Mice that were exposed to blast shock waves of 19 pounds per square inch static pressure showed significant elevation of ABR threshold in wide-ranging acoustic frequency spectra and disappearance of DPOAE. Compared to low frequency (8 kilohertz) hearing, the impact to high frequency (40 kilohertz) hearing was severe. Those changes persisted through a month post-injury. Pathological evaluations by immunohistochemistry revealed a significant increase in the expression of glial fibrillary acidic protein (GFAP) and ionized calcium binding adaptor molecule 1 (Iba1) in the brainstem and cerebellum. Immunostaining of myosin VIIa (Myo7a) and Phalloidin in whole mount cochlea revealed appreciable damage to outer hair cells, inner hair cells, as well as to other structures in the inner ear. These data indicate that both peripheral and central auditory systems are vulnerable to blast injury and also point to neuroinflammation as a pivotal contributor to the secondary neuronal damage underlying these debilitating injuries. By revealing the neurobiological mechanisms that underlie BOP-induced auditory impairments and vestibular disruptions, these experiments will provide valuable insights into mitigation strategies and therapeutic countermeasures for affected Service Members.