Facial, Hearing, and Visual Injuries

Assessment and Treatment of Blast-Induced Auditory and Vestibular Injuries

Researchers at the 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 investigators at the National Institute for Deafness and Other Communicative Disorders and the Lieber Institute for Brain Development at the Johns Hopkins School of Medicine, these investigators are developing strategies for mitigating or reversing auditory/vestibular injury that originates from damage to mechanosensory hair cells and brain structures. The most common symptoms after blast exposure are headaches, hearing loss, balance problems, and dizziness, which strongly suggest impairment of blast waves to the structure of inner ear and neuronal encoding of sound. Using an advanced blast simulator that produces a high fidelity recreation of blast overpressure 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, quantification and characterization of the auditory and vestibular injuries and efficacy of therapeutic interventions are judged by a battery of functional assessments, including auditory brainstem response, distortion product otoacoustic emission testing, vestibular sensory evoked potentials, and vestibulo-motor functional measurements. Blast exposure causes dramatic disruptions in these functional parameters in association with progressively worsening hair cell loss and neuropathological changes in brain structures involved in central auditory and vestibular signal processing. 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.