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Neurological Effects and Mechanisms of Blast Overpressure Injury

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Description

Exposure to blast overpressure from explosive devices during warfare results in considerable neurotrauma known as blast-induced traumatic brain injuries (bTBIs). These injuries occur when shock waves issued from explosive devices impact the brain, causing shearing of tissues, destruction of axons, edema, hematomas, and various long-term neuropsychological effects associated with cellular and biochemical alterations. Although a majority of bTBIs have a good prognosis, in a warfare situation difficulties in diagnosis and inadequate treatments often result in multiple exposures to blast overpressure. The accumulated bTBIs cause a sequela of events resulting in cognitive and behavioral deficits, which may persist for years. This chapter describes the current knowledge and advancements of bTBI research, including blast physics, bTBI diagnostics and research methodologies, neurological effects and mechanisms, and emerging biomarkers. Finally, current therapeutic treatments are briefly discussed [1].

Explosive devices are common in modern warfare, leading to an increase in blast-induced traumatic brain injuries (bTBIs). bTBIs are almost identical to other TBIs in terms of neuropathology and clinical symptoms. Unique aspects of bTBIs arise from the injury setting and the behavior of explosive devices. bTBIs tend to occur in traumatic settings such as war zones or terrorist attacks, creating a strong negative emotional component. Explosive devices do not target the head specifically, leading to peripheral injuries. Both the emotional and peripheral aspects of bTBI can exacerbate symptoms. The majority of bTBIs are mild, and have good prognosis but can be difficult to diagnose as symptoms can be transient and mild. The inability to properly detect mild bTBI can expose victims to further injuries, worsening prognosis. This chapter describes the mechanism of injury, clinical features, neuropathology, animal models, and potential fluid biomarkers for bTBI [2].

Neurological Effects and Mechanisms

Over the last few years, thousands of soldiers and an even greater number of civilians have suffered traumatic injuries due to blast exposure, largely attributed to improvised explosive devices in terrorist and insurgent activities. The use of body armor is allowing soldiers to survive blasts that would otherwise be fatal due to systemic damage. Emerging evidence suggests that exposure to a blast can produce neurological consequences in the brain, but much remains unknown. To elucidate the current scientific basis for understanding blast-induced traumatic brain injury (bTBI), the NIH convened a workshop in April, 2008. A multidisciplinary group of neuroscientists, engineers, and clinicians were invited to share insights on bTBI, specifically pertaining to: physics of blast explosions, acute clinical observations and treatments, preclinical and computational models, and lessons from the international community on civilian exposures. This report provides an overview of the state of scientific knowledge of bTBI, drawing from the published literature, as well as presentations, discussions, and recommendations from the workshop. One of the major recommendations from the workshop was the need to characterize the effects of blast exposure on clinical neuropathology. Clearer understanding of the human neuropathology would enable validation of preclinical and computational models, which are attempting to simulate blast wave interactions with the central nervous system. Furthermore, the civilian experience with bTBI suggests that polytrauma models incorporating both brain and lung injuries may be more relevant to the study of civilian countermeasures than considering models with a neurological focus alone [3].

Traumatic brain injury (TBI) is a well-known consequence of participation in activities such as military combat or collision sports. But the wide variability in eliciting circumstances and injury severities makes the study of TBI as a uniform disease state impossible. Military Service members are under additional, unique threats such as exposure to explosive blast and its unique effects on the body. This review is aimed toward TBI researchers, as it covers important concepts and considerations for studying blast-induced head trauma. These include the comparability of blast-induced head trauma to other mechanisms of TBI, whether blast overpressure induces measureable biomarkers, and whether a biodosimeter can link blast exposure to health outcomes, using acute radiation exposure as a corollary. This examination is contextualized by the understanding of concussive events and their psychological effects throughout the past century's wars, as well as the variables that predict sustaining a TBI and those that precipitate or exacerbate psychological conditions.

Traumatic Brain Injury

Traumatic brain injury (TBI) is a brain dysfunction caused by an external force that may have short- and long-term effects on Service members and their units, families, and caregivers. Per Defense and Veterans Brain Injury Center1 (DVBIC) statistics, 383 947 individuals within the Department of Defense (DoD) sustained a TBI from 2001 to 2018, more than one-third of whom were exposed to a blast event. 2,3Management of TBI in the acute and chronic phases have shifted over time and will continue to change as operational constraints and medical advances evolve. Researchers should have a basic understanding of the blast mechanisms of injury as well as the first- through fifth-order effects that are possible with any blast. The physiologic effects of primary blast are most understood for the pulmonary and auditory systems, but the effects of blast on the central nervous system are less established. Explosion energy outside the body is transformed into biokinetic energy that causes damage to the brain and structures of the cranium from the overpressure [4].

Direct or indirect exposure to an explosion can induce traumatic brain injury (TBI) of various severity levels. Primary TBI from blast exposure is commonly characterized by internal injuries, such as

vascular damage, neuronal injury, and contusion, without external injuries. Current animal models of blast-induced TBI (bTBI) have helped to understand the deleterious effects of moderate to severe blast forces. However, the neurological effects of mild blast forces remain poorly characterized [5].

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