Cerebral Blood Pressure Rise during Blast Exposure in a Rat Model of Blast-Induced Traumatic Brain Injury

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Abstract

Blood-brain barrier (BBB) is the mechanism that isolates the delicate environment of brain from circulating blood flow. The BBB dysfunction, present in almost all cases of traumatic brain injury (TBI), including blast-induced TBI (bTBI), triggers a cascade of events leading to cognitive and behavioral impairments. However, it is still not known if the rupture of BBB in bTBI is due to the pressure wave transferred directly to brain parenchyma through the skull and the resulting strains, or a sudden cerebral blood pressure rise transferred from large torso blood vessels exposed to the blast. The aim of this study was to investigate the underlying mechanism by measuring cerebrovascular blood pressure during blast exposure and comparing two scenarios of head-only or chest-only exposures to the blast wave.

This study was performed using a rodent blast injury model (Sprague-Dawley rat) utilizing a shock tube. In order to measure cerebral blood pressure, a miniature pressure sensor was inserted in the internal carotid artery (ICA), and then the animal was exposed to blast waves in front of the shock tube, in two exposure scenarios of chest-only or head-only. The ICA pressure was recorded during exposure for blast peak overpressures of 65, 110, 160, and 185 kPa. Immunohistochemical staining was also performed to investigate the macrophage infiltration to brain through the cerebrovasculature utilizing anti-CD68.

The results showed that the cerebrovascular pressure rises 2 to 10 times higher than physiological pressure and lasts for about 2ms depending on the peak overpressure of blast wave. Cerebral blood pressure rise was significantly higher in chest-only exposures, and caused infiltration of blood-borne macrophages into the brain. It is concluded that a significantly high pressure wave transfers from torso to cerebrovasculature due to chest exposure to a blast wave, which leads to blood-brain barrier disruption and triggers a secondary neuronal damage.