Blast exposure causes brain injury due to pressure waves

Improved body armour and medical treatment have resulted in an increase in troops surviving blast from improvised explosive devices (IEDs) during recent conflicts. However, these survivors exhibit the highest number of severe traumatic brain injuries (TBIs) since Vietnam. In addition, many returning veterans have experienced delayed symptoms similar to those of mild TBIs that were not diagnosed prior to discharge. This has focused recent research interest on the effects of blast on the brain.

Blast causes injury due to several factors, some of which are similar to those encountered in car accidents and sports injuries such as penetrating projectiles (secondary blast injury), or the whiplash and impact forces from being thrown (tertiary blast injury). However, blast also produces a pressure wave as the gases expand away from the detonation (Fig. 1). It is thought that at “far-field” distances from the blast, where secondary or tertiary blast injuries are less likely to occur, primary blast-induced TBI (PbTBI) may be caused by exposure to the blast wave. This study examined whether head-only exposure of rats to simulated blast waves causes brain damage.

Blast causes a unique type of pressure wave called a shock wave, characterized by an almost instantaneous increase in pressure, which then falls off over a time frame measured in a few thousandths of a second (Fig. 2). Blast experiments are difficult to carry out, and are also extremely technically challenging to simulate in the laboratory. This has made the study of the subtle effects thought to be caused by PbTBI very difficult. There is controversy as to whether blast-induced pressure waves actually play a role in the often delayed mild TBI seen in a large number of returning veterans.

For this study an “Advanced Blast Simulator” (ABS) was developed through extensive modification of a classical shock tube, which is a cylinder (test section) separated from a pressure vessel (driver) by a membrane. Breakage of the membrane causes a shock wave to propagate into the test section. Due to its unique design the ABS generates a shock wave that has all the specialized features of a free-field blast wave (Fig. 2).
Rats were exposed to simulated blast to assess brain injury. After exposure, the animals were removed from the ABS and all recovered in a very similar fashion to those not exposed to blast (controls). The effects of head restraint were examined. When the head was not restrained it experienced significant whiplash movement during exposure. This is an artifact due to the small size of the rat’s head and similar head movement would not occur in a human. In these animals an indicator of brain damage known as glial fibrillary acidic protein (GFAP) was very significantly elevated in the brain by one day post-blast (Fig. 2). This biomarker has been widely utilized as a diagnostic for the presence of TBI due to concussion and/or whiplash. In contrast, when head motion was minimized using netting, although several markers of brain damage were observed at one to seven days after exposure, GFAP was not elevated (Fig. 2). We believe that the GFAP increases observed were due to the artifact of whiplash movement and that blast-induced pressure waves do not impact this biomarker. We conclude that blast-induced pressure waves can directly cause changes in the brain. Furthermore, a widely used test using a protein called GFAP, will not diagnose this kind of brain injury. This work has implications for the diagnosis of mild to moderate TBI in individuals exposed to blast who may not exhibit overt signs of injury.


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