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Basic Neuroscience

Low-cost blast wave generator for studies of hearing loss and brain injury: Blast wave effects in closed spaces

Andrew J. Newman[a,1], Sarah H. Hayes[b,*], Abhiram S. Rao[a], Brian L. Allman[b,2], Senthilvelan Manohar[b], Dalian Ding[b], Daniel Stolzberg[b,3], Edward Lobarinas[b,4], Joseph C. Mollendorf[a], Richard Salvi[b]

[a] Department of Mechanical and Aerospace Engineering, University at Buffalo, The State University of New York, Buffalo, NY, United States
[b] Center for Hearing & Deafness, Department of Communicative Disorders and Sciences, University at Buffalo, The State University of New York, Buffalo, NY, United States

HIGHLIGHTS

- Many devices used in animal models of blast wave exposure are large and expensive.
- We developed a low cost blast wave generator easily housed in laboratory settings.
- The generator reliably produced blasts with peak pressures of up to 198 dB SPL (159.4 kPa).
- Exposure of rats to blast waves caused damage to the inner ear and hippocampus.
- This low cost device is useful for studying blast induced ear and brain injury.

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ABSTRACT

Background: Military personnel and civilians living in areas of armed conflict have increased risk of exposure to blast overpressures that can cause significant hearing loss and/or brain injury. The equipment used to simulate comparable blast overpressures in animal models within laboratory settings is typically very large and prohibitively expensive.
New method: To overcome the fiscal and space limitations introduced by previously reported blast wave generators, we developed a compact, low-cost blast wave generator to investigate the effects of blast exposures on the auditory system and brain.
Results: The blast wave generator was constructed largely from off the shelf components, and reliably produced blasts with peak sound pressures of up to 198 dB SPL (159.3 kPa) that were qualitatively similar to those produced from muzzle blasts or explosions. Exposure of adult rats to 3 blasts of 188 dB peak SPL (50.4 kPa) resulted in significant loss of cochlear hair cells, reduced outer hair cell function and a decrease in neurogenesis in the hippocampus.
Comparison to existing methods: Existing blast wave generators are typically large, expensive, and are not commercially available. The blast wave generator reported here provides a low-cost method of generating blast waves in a typical laboratory setting.
Conclusions: This compact blast wave generator provides scientists with a low cost device for investigating the biological mechanisms involved in blast wave injury to the rodent cochlea and brain that may model many of the damaging effects sustained by military personnel and civilians exposed to intense blasts.

Abbreviations: OHC, outer hair cell; IHC, inner hair cell; DPOAE, distortion product otoacoustic emission; IED, improvised explosive device; TBI, traumatic brain injury; PVC, poly vinyl chloride; PBS, phosphate buffered saline; DCX, doublecortin; MWM, Morris Water Maze; PCB, printed circuit board.

* Corresponding author at: Center for Hearing and Deafness, University at Buffalo, 137 Cary Hall, 3435 Main Street, Buffalo, NY 14214, United States. Tel.: +1 716 829 5310; fax: +1 716 829 2980.

E-mail addresses: ajnewman@ara.com (A.J. Newman), shhayes@buffalo.edu (S.H. Hayes), abhirams@buffalo.edu (A.S. Rao), brian.allman@schulich.uwo.ca (B.L. Allman), smanohar@buffalo.edu (S. Manohar), dding@buffalo.edu (D. Ding), dstolzbe@uwo.ca (D. Stolzberg), elobarinas@ufl.edu (E. Lobarinas), molendrf@buffalo.edu (J.C. Mollendorf), salvi@buffalo.edu (R. Salvi).

1 Present address: Applied Research Associates, Panama City, FL, United States.
2 Present address: Department of Anatomy and Cell Biology, Schulich School of Medicine and Dentistry, University of Western Ontario, Ontario N6A 5C1, Canada.
3 Present address: Department of Physiology and Pharmacology, Schulich School of Medicine and Dentistry, University of Western Ontario, Ontario N6A 5C1, Canada.
4 Present address: Department of Speech, Language, and Hearing Sciences, University of Florida, Gainesville, FL, United States.

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1. Introduction

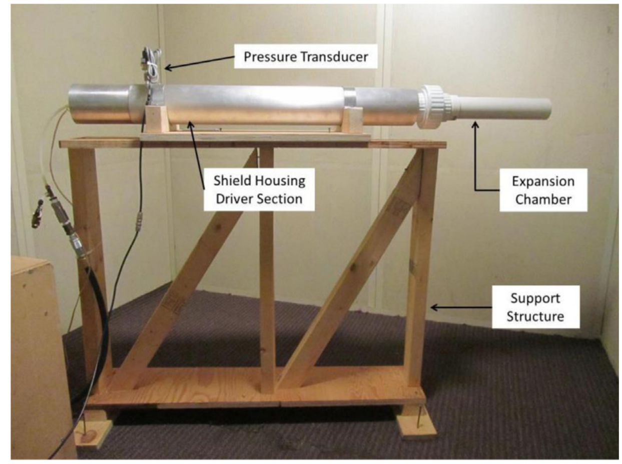
Combat personnel and civilians residing in war zones have increased risk of exposure to intense blast waves in open or closed spaces (Taber et al., 2006). These originate from many sources including muzzle blasts from artillery, exploding bombs and improvised explosive devices (IEDs). Blast wave exposure is a major cause of auditory dysfunction in military personnel. Because the sensory hair cells in the cochlea are especially vulnerable to loud sounds, blast wave exposure often leads to hearing loss and tinnitus. Not surprisingly, noise-induced hearing loss and tinnitus are the most prevalent service connected disabilities costing the Veterans Administration over $1 billion annually (Fausti et al., 2009). In addition to otologic injury, blast waves are a leading cause of traumatic brain injury (TBI) in the military; the number of blast-related TBI cases was estimated to be as high as 320,000 as of June 2011 (Mac Donald et al., 2011). Blast related TBI can result from blast wave overpressure (primary blast exposure) in addition to physical trauma from projectiles accompanying the blast wave (secondary blast exposure) (Wolf et al., 2009). Blast-induced TBI has been associated with learning deficits, memory loss, depression and post-traumatic stress disorder (Hoge et al., 2008; Nelson et al., 2009; Terrio et al., 2009; Cernak, 2010). The co-morbidity of blast-induced auditory damage and TBI is particularly common with 33%, 43% and 9% of veterans suffering from TBI showing acute, sub-acute and chronic hearing loss, respectively (Hoffer and Balaban, 2011).

Animal models of blast wave exposure have been developed to study blast-induced auditory dysfunction and TBI in order to determine its biological basis in more controlled environments. Blast-induced otologic damage typically results from rupture of the tympanic membrane, damage to the ossicular chain and/or damage to the sensory hair cells in the cochlea that are particularly vulnerable to acoustic overstimulation. Intense blast wave exposures not only damage the sensory hair cells, but also the supporting cells, afferent dendrites and spiral ganglion neurons (Hamernik et al., 1984a,b; Roberto et al., 1989; Patterson and Hamernik, 1997; Cho et al., 2013).

Animal models of blast-induced TBI have identified the hippocampus – a brain region important for learning and memory and one of two regions in the adult brain where neurogenesis occurs – as particularly vulnerable to blast exposure (Carbonell and Grady, 1999; Sato et al., 2001). In rodents, blast wave exposure has been shown to alter expression of genes in the hippocampus including the down-regulation of genes involved in neurogenesis (Saljo et al., 2002; Risling et al., 2011). Furthermore, both single and multiple blast exposures produce evidence of hippocampal cell death and alters the level of hippocampal neurogenesis as reflected in doublecortin (DCX) immunolabeling (Kovesdi et al., 2011; Kwon et al., 2011; Kamnaksh et al., 2012). Similar to the learning deficits seen in human TBI patients, blast wave exposure induces cognitive deficits in rodents as measured with the Morris Water Maze (MWM) task, indicative of hippocampal damage (Hamm et al., 1996; Vandevord et al., 2012; Tompkins et al., 2013).

To date, few studies have investigated the co-morbidity of auditory damage and TBI following blast wave exposure. In order to investigate the concurrent effects of blast wave exposure on the auditory system and brain, a method of generating consistent and controllable blast waves is required. The equipment used to simulate blast overpressures in most laboratory settings was typically designed for aerodynamic compressible flow studies and is typically custom-constructed from large, heavy metal tubes (Hamernik et al., 1984a,b). In addition to being prohibitively expensive, many blast wave generators are extremely large with lengths upward of 19 feet, making them particularly inconvenient to house in biological laboratories. To overcome these space and fiscal constraints, we developed a compact, low cost blast wave generator that can be readily constructed largely from off the shelf components. Here we describe the methods and materials needed to construct the blast wave generator, its performance characteristics, and the effectiveness of the blast wave generator to induce cochlear damage and impair neurogenesis in the rodent hippocampus.

A



B

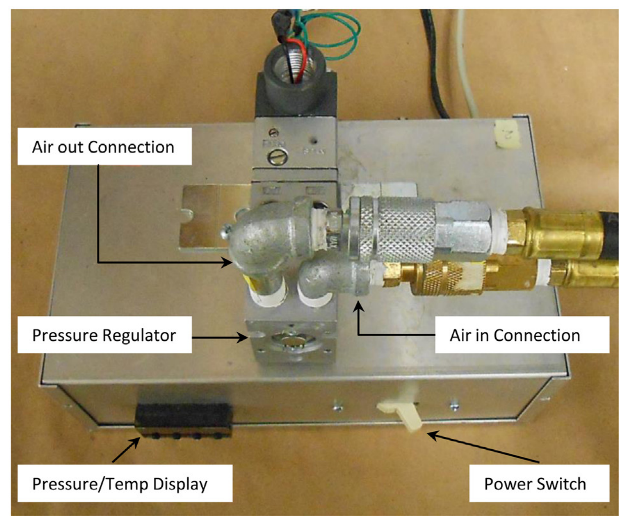


Fig. 1. Fully assembled shock tube (A) and controller (B).

2. Materials and methods

2.1. Shock tube assembly

The blast wave generator's major components, the shock tube and the control module, were constructed independently and connected to signal processing equipment and sensors in the laboratory. The fully assembled shock tube and control module are shown in Fig. 1. Fig. 2 shows an exploded view of the shock tube and its component. The device was primarily constructed from 2 in. (internal diameter 2.05 in.) Schedule 40 Poly Vinyl Chloride (PVC) pipe and related PVC components. Schedule 80 PVC has a pressure rating of 166 PSIG (1144 kPa gauge pressure), which is well above driver pressures used in the present experiments. The shock tube assembly consists of a high pressure chamber and low (ambient) pressure chamber separated by a thin, brass diaphragm. The 32-in. long high pressure driver section that houses compressed air was connected to the 15-in. low pressure expansion chamber through which the blast wave propagates by way of a 2-in. PVC union. The driver section was enclosed within a 4-in. aluminum