NeuroRehabilitation 26 (2010) 179–181
DOI 10.3233/NRE-2010-0553
IOS Press

Guest Editorial

Military traumatic brain injury and blast

David F. Moore[a,b,*] and Michael S. Jaffee[a]
[a]*Defense and Veterans Brain Injury Center, Walter Reed Army Medical Center, Washington, DC, USA*
[b]*Institute of Soldier Nanotechnology, Massachusetts Institute of Technology, Cambridge, MA, USA*

Abstract. The effects of blast on biological tissue are documented for some organ systems such as the lung. In the central nervous system (CNS) the mechanism of CNS injury following blast wave is unclear. For example is there a selective effect of blast on varying brain region or white matter bundles. The effect of blast on traumatic brain injury (TBI) has come into particular focus with the Global War on Terror and Operation Iraqi Freedom and Operation Enduring Freedom where TBI has become known as the signature injury of these conflicts. The reason for the prominence of TBI in these particular conflicts as opposed to others is unclear but may result from the increased survivability of blast due to improvements in body armor. In the current series of articles in the Journal some developments of current research concepts in relation to military traumatic brain injury (TBI) are highlighted together with many remaining unsolved questions.

The effect of blast in relation to traumatic brain injury (TBI) has been described following the current conflicts in Iraq and Afghanistan probably due to the asymmetrical nature of the conflicts and the extensive use of improvised explosive devices (IEDs). Understanding this mechanism of injury and its clinical implications compared to other mechanisms of injury such as acceleration-deceleration impact has become an important question in the care of our service members and veterans.

Blast may be defined as an explosion in the atmosphere characterized by the release of energy in such a short period of time and within such a small volume resulting in the creation of a non-linear shock and pressure wave of finite amplitude, spreading from the source of the explosion. The energy radiating from a conventional blast can be chemical, electrical, thermal and kinetic or pressure energy. This is seen in Fig. 1 where the kinetic energy associated with fragments results in their expulsion in front of the shock wavefront. The 'ideal case' of a blast pressure wave is the Friedlander waveform with a rapid rise-time to the peak positive pressure above atmospheric, the overpressure followed by an exponential pressure fall-off together with a relatively prolonged sub-atmospheric underpressure. Typically the time scale of the total explosive pressure event is tens of milliseconds. The prolonged underpressure component of the pressure waveform may exceed the critical tensile strength of the fluid component of a tissue allowing the development of cavitation.

Blast injury is categorized as primary where injury is related to the shock-wave overpressure and underpressure, secondary where the injury results from blast-associated fragments or shrapnel, tertiary where injury occurs secondary to falling debris or throwing of the dismounted soldier or vehicle, and quaternary where injury develops from a variety of physical processes associated with explosive detonation such as thermal, toxic detonation products. Quinary effects are sometimes included and refer to the environmental hazard remaining after an explosive detonation. The peak overpressure is most simply dependent on the distance from the blast source but approximately scales according to the standoff distance divided by the cube root of the

*Address for correspondence: Dr. David F. Moore, MD, PhD, Defense and Veterans Brain Injury Center, Walter Reed Army Medical Center, Building 1, Room B207, 6900 Georgia Avenue NW, Washington, DC 20309-5001, USA. E-mail: david.f.moore@amedd.army.mil.

ISSN 1053-8135/10/$27.50

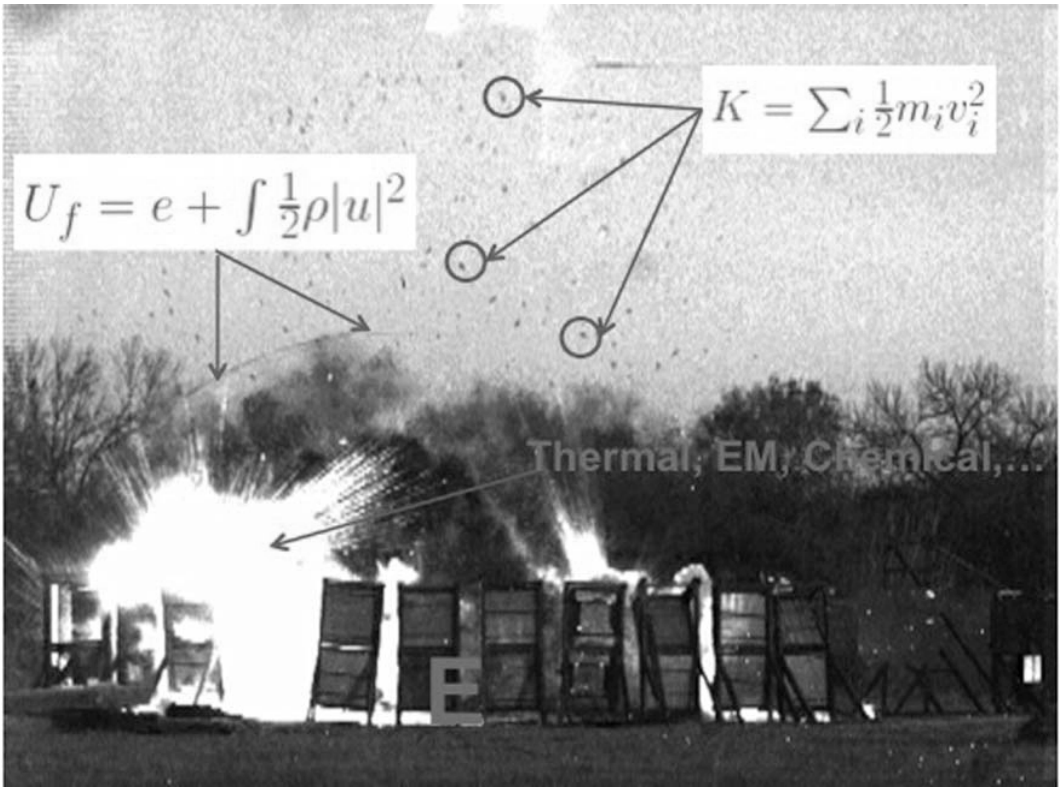

Fig. 1. The total energy conversion from detonation of an explosive charge resulting in kinetic energy of fragments (K), and a shock adiabat (U_f) together with excess thermal, chemical and electromagnetic pulses (EM). The shock wave and the associated blast wind are responsible for primary blast injury.

explosive weight (Hopkinson Rule). The coupling of the nonlinear blast wave into biological tissue results in increased energy deposition at high strain rates in fractions of microseconds. The biological effect will depend on the constitutive tissue properties together with the largely unknown high strain rate material properties for brain tissue. Ongoing work is establishing material properties of brain across the strain rate domain from low strain rates seen in impact injury to intermediate and higher strain rates seen in ballistic and blast injury. The above concepts lead to a frame of reference debate in relation to blast induced concussion or mTBI suggesting that lethal injury would occur from fragments or damage to other organs such as lung before sufficient blast pressure exposure could occur. Such a conceptualization has undoubted validity but probably has failed to factor-in the significant mitigation of current personal protective equipment such as body armor and helmets [1–4]. There has also been evidence suggesting that the blast waves do not behave in a free and open frame of reference but may be reflected from the ground or other objects in the battlespace (*Textbook of Military Medicine*, Part 1, Chapter 7, The Physics and mechanisms of primary blast injury).

Explosives detonation results in the formation of a detonation wave of altering chemical composition with the rapid formation of a propagated, nonlinear shockwave representing a large discontinuous increase in pressure, temperature and density in the gas flow. The propagation of the shockwave results in a 3D complex flow field that is altered by ambient conditions and environmental boundaries. This may result in multiple wave reflections and potentially pressure field intensification up to eightfold. A simulated propagation of a blast wave interaction through the brain with a bio-fidelic head model based on advanced computer modeling has been recently described [5].

The blast waveform can be regarded as a combination of compressive and tensile components that impose a stress on the tissue in a manner that is dependent on the strain rate together with the constitutive properties of the tissue. This combined with the potential for CNS injury from ballistic fragment, acceleration-deceleration impact injury as well as chemical, ther-

mal and electromagnetic radiation results in a highly complex problem where dominating effects become difficult to parse in terms of their biological effects on the CNS. Future analyses may consider the potential for combined and synergistic effects of some of these contributing etiological factors associated with a blast explosion. Furthermore, a significant amount of combat traumatic brain injuries associated with blast as a contributing component has the added complexity of having more than one mechanism of injury often with the blast component combined with acceleration-deceleration impact or fragment injury. This combination has been referred to as "blast-plus" injuries.

There is a great deal of ongoing work to better understand the clinical correlation of blast across the entire continuum of care. This special issue includes contributions from a variety of investigators affiliated with the Defense and Veterans Brain Injury Center (DVBIC), a congressionally mandated collaboration between the Department of Defense and the Department of Veterans Affairs spanning across more than 17 sites. The primary missions of the DVBIC include collaborating on clinical care, research, and education for our service members and veterans with TBI. These articles includes considerations of these clinical correlations in the context of screening and understanding comorbidities such as sensory impairment and psychological syndromes. There are articles addressing a variety of aspects of management of these patients to include neurorehabilitation models, community monitoring, and driving assessment. The results of an important Department of Defense consensus conference on cognitive rehabilitation for this patient population is included.

Our understanding of blast has been advanced by ongoing partnerships and collaborations between government and civilian partners. It is expected that these advances will also have application and benefit for the civilian TBI population. It is imperative that as we advance our understanding of blast, we are able to rapidly translate this knowledge to the direct care and improved outcomes of our service members and veterans. They deserve nothing less.

Acknowledgements

The authors thank Professor Raul Radovitsky, MIT Institute of Soldier Nanotechnology and Dr. David F. Moore, DVBIC for allowing the use of the image displayed in the Figure. The views expressed in these articles are those of the authors and do not reflect the official policy of the Department of the Army, Navy or Department of Defense, Veterans Affairs or US Government.

References

[1] I. Cullis, *J R Army Med Corps* **147** (2001), 16–26.
[2] R. DePalma, D.G. Burris, H.R. Champion and M.J. Hodgson, *N Engl J Med* **352** (2005), 1335–1342.
[3] M.A. Mayorga, *Toxicology* **121** (1997), 17–28.
[4] D.F. Moore, R. Radovitzky, L. Shupenko, A. Klinoff, M.S. Jaffee and J. Rosen, *Future Neurology* **3** (2008), 243–250.
[5] D.F. Moore, A. Jérusalem, M. Nyein, L. Noels, M.S. Jaffee and R.A. Radovitzky, *NeuroImage* **47**(Suppl 2) (Aug 2009), T10–T20.