

REPORT DOCUMENTATION PAGE			Form Approved OMB NO. 0704-0188		
The public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington VA, 22202-4302. Respondents should be aware that notwithstanding any other provision of law, no person shall be subject to any penalty for failing to comply with a collection of information if it does not display a currently valid OMB control number. PLEASE DO NOT RETURN YOUR FORM TO THE ABOVE ADDRESS.					
1. REPORT DATE (DD-MM-YYYY) 29-02-2012		2. REPORT TYPE Abstract		3. DATES COVERED (From - To) -	
4. TITLE AND SUBTITLE Brain Injury Risk from Primary Blast			5a. CONTRACT NUMBER W911NF-10-1-0526		
			5b. GRANT NUMBER		
			5c. PROGRAM ELEMENT NUMBER 611103		
6. AUTHORS Karin A. Rafaels, Cameron R. 'Dale' Bass, Matthew B. Panzer, Robert S. Salzar, William A. Woods, Sanford Feldman, Tim Walilko, Richard Kent, Bruce P. Capehart, Jay K. Shridharani, Amanda Toman			5d. PROJECT NUMBER		
			5e. TASK NUMBER		
			5f. WORK UNIT NUMBER		
7. PERFORMING ORGANIZATION NAMES AND ADDRESSES University of Pennsylvania Office of Research Services University of Pennsylvania Philadelphia, PA 19104 -				8. PERFORMING ORGANIZATION REPORT NUMBER	
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES) U.S. Army Research Office P.O. Box 12211 Research Triangle Park, NC 27709-2211				10. SPONSOR/MONITOR'S ACRONYM(S) ARO	
				11. SPONSOR/MONITOR'S REPORT NUMBER(S) 58155-LS-MUR.12	
12. DISTRIBUTION AVAILABILITY STATEMENT Approved for public release; distribution is unlimited.					
13. SUPPLEMENTARY NOTES The views, opinions and/or findings contained in this report are those of the author(s) and should not be construed as an official Department of the Army position, policy or decision, unless so designated by other documentation.					
14. ABSTRACT Objectives: Increased use of explosive devices in recent military conflicts have resulted in, blast overpressure is the primary cause of traumatic brain injury among combat veterans (Owens, 2008). Primary blast injury has been studied extensively in air-containing organs such as the lungs, gastrointestinal tract, and ear due to their increased susceptibility to primary blast (Hooker, 1924), but recent epidemiology shows an increase in the occurrence of head injuries (Martin, 2008). Since there is little information on the intensity of a blast wave needed to cause blast brain					
15. SUBJECT TERMS blast overpressure, blast injury, traumatic brain injury, shock waves					
16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT UU	15. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON David Meaney
a. REPORT UU	b. ABSTRACT UU	c. THIS PAGE UU			19b. TELEPHONE NUMBER 215-573-2726

Report Title

Brain Injury Risk from Primary Blast

ABSTRACT

Objectives: Increased use of explosive devices in recent military conflicts have resulted in, blast overpressure is the primary cause of traumatic brain injury among combat veterans (Owens, 2008). Primary blast injury has been studied extensively in air-containing organs such as the lungs, gastrointestinal tract, and ear due to their increased susceptibility to primary blast (Hooker, 1924), but recent epidemiology shows an increase in the occurrence of head injuries (Martin, 2008). Since there is little information on the intensity of a blast wave needed to cause blast brain injury, the goal of this study is to provide injury risk assessments for brain blast fatality, meningeal bleeding, and apnea as a function of blast intensity in a gyrencephalic animal model and to provide exposure guidelines for clinically relevant blast injuries.

Materials and Methods: Shock waves were generated that simulated blasts with charge sizes up to 1000 kg of high explosives. The blast exposure to a gyrencephalic animal model (ferret) was isolated to the head by combined abdominal and thoracic protection that reduced blast levels to an order of magnitude below pulmonary injury threshold. The results were scaled to a 70kg human using a biomechanical scaling technique. The outcomes including apnea, meningeal bleeding, and fatalities were analyzed using logistic regressions in terms of applied shock peak pressure and scaled duration.

Results: Increasing severity of blast exposures (either by increasing peak maximum pressure and positive phase duration or both) increases occurrence of injury. Gross necropsy revealed subdural, subarachnoid, and cerebral contusions typically on or around the brainstem though there were no skull fractures for any blast intensity. Risk functions were developed that relate blast intensity to the risk of developing one or more adverse clinical outcomes; mild and moderate/severe meningeal bleeding, initial apnea, and evoked potential signal loss and were scaled for human exposure conditions. For high explosives at close range, the human fatality injury risk for primary blast exposure to the brain was found to be more than twice the pulmonary fatality injury risk. However, the blast level for 50% risk of mild brain bleeding was found to occur at similar overpressure values as the 50% risk of unprotected pulmonary blast injury onset.

Conclusions: This study presents the first injury risk functions for primary blast injuries to the brain over a range of realistic blast exposures. Though the risk assessments show that the blast intensity necessary for 50% risk of fatality from brain injury is much greater than that needed to cause 50% risk of fatality from pulmonary injury in unprotected persons, mild meningeal bleeding may occur at blast intensities below those for threshold pulmonary injury. This injury risk combined with the strong pulmonary protective effects of body armor may explain the recent incidence of mild/moderate TBI in returning service members. The risk functions provided by this study provide the first realistic brain injury risk assessments that can be used to guide clinical assessments of blast injury, design protective equipment, and guide future blast brain injury research.

Objectives: Increased use of explosive devices in recent military conflicts have resulted in, blast overpressure is the primary cause of traumatic brain injury among combat veterans (Owens, 2008). Primary blast injury has been studied extensively in air-containing organs such as the lungs, gastrointestinal tract, and ear due to their increased susceptibility to primary blast (Hooker, 1924), but recent epidemiology shows an increase in the occurrence of head injuries (Martin, 2008). Since there is little information on the intensity of a blast wave needed to cause blast brain injury, the goal of this study is to provide injury risk assessments for brain blast fatality, meningeal bleeding, and apnea as a function of blast intensity in a gyrencephalic animal model and to provide exposure guidelines for clinically relevant blast injuries.

Materials and Methods: Shock waves were generated that simulated blasts with charge sizes up to 1000 kg of high explosives. The blast exposure to a gyrencephalic animal model (ferret) was isolated to the head by combined abdominal and thoracic protection that reduced blast levels to an order of magnitude below pulmonary injury threshold. The results were scaled to a 70kg human using a biomechanical scaling technique. The outcomes including apnea, meningeal bleeding, and fatalities were analyzed using logistic regressions in terms of applied shock peak pressure and scaled duration.

Results: Increasing severity of blast exposures (either by increasing peak maximum pressure and positive phase duration or both) increases occurrence of injury. Gross necropsy revealed subdural, subarachnoid, and cerebral contusions typically on or around the brainstem though there were no skull fractures for any blast intensity. Risk functions were developed that relate blast intensity to the risk of developing one or more adverse clinical outcomes; mild and moderate/severe meningeal bleeding, initial apnea, and evoked potential signal loss and were scaled for human exposure conditions. For high explosives at close range, the human fatality injury risk for primary blast exposure to the brain was found to be more than twice the pulmonary fatality injury risk. *However, the blast level for 50% risk of mild brain bleeding was found to occur at similar overpressure values as the 50% risk of unprotected pulmonary blast injury onset.*

Conclusions: This study presents the first injury risk functions for primary blast injuries to the brain over a range of realistic blast exposures. Though the risk assessments show that the blast intensity necessary for 50% risk of fatality from brain injury is much greater than that needed to cause 50% risk of fatality from pulmonary injury in unprotected persons, *mild meningeal bleeding may occur at blast intensities below those for threshold pulmonary injury.* This injury risk combined with the strong pulmonary protective effects of body armor may explain the recent incidence of mild/moderate TBI in returning service members. The risk functions provided by this study provide the first realistic brain injury risk assessments that can be used to guide clinical assessments of blast injury, design protective equipment, and guide future blast brain injury research.

Bowen, I., E. Fletcher, et al. (1968). Estimate of man's tolerance to the direct effects of air blast. Technical Progress Report. Washington, DC.

Eppinger, R. H., J. H. Marcus, et al. (1984). Development of dummy and injury index for NHTSA's thoracic side impact protection research program, Society of Automotive Engineers, 400 Commonwealth Dr, Warrendale, PA, 15096, USA.

Hooker, D. (1924). "Physiological effects of air concussion." American Journal of Physiology--Legacy Content 67(2): 219.

Martin, E. M., W. C. Lu, et al. (2008). "Traumatic brain injuries sustained in the Afghanistan and Iraq wars." AJN The American Journal of Nursing 108(4): 40.

Owens, B. D., J. F. Kragh Jr, et al. (2008). "Combat wounds in operation Iraqi Freedom and operation Enduring Freedom." The Journal of Trauma 64(2): 295.

Wood, G. W., M. B. Panzer, et al. (2010). Attenuation of blast overpressure behind ballistic protective vests. Personal Armour Systems Symposium. Quebec City, QB, Canada.

BRAIN INJURY RISK FROM PRIMARY BLAST

Karin A. Rafaels, Cameron R. 'Dale' Bass, Matthew B. Panzer, Robert S. Salzar, William A. Woods, Sanford Feldman, Tim Walilko, Richard Kent, Bruce P. Capehart, Jay K. Shridharani, Amanda Toman

Additional Text (removed from abstract)

In recent military conflicts, ballistic protective body armor is very commonly used and has been shown to reduce the pressure seen by the thoracic cavity up to a factor of 50 (Wood, 2010) increasing the pulmonary system's tolerance to blast exposures. Wood et al. hypothesizes this increase in the pulmonary system's tolerance to be the cause of the increased occurrence of brain injury.