Assessment of Fluid Cavitation Threshold Using a Polymeric Split Hopkinson Bar-Confinement Chamber Apparatus

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ABSTRACT

Mild Traumatic Brain Injury (mTBI) has been associated with blast exposure resulting from the use of improvised explosive devices (IEDs) in recent and past military conflicts. Experimental and numerical models of head blast exposure have demonstrated the potential for high negative pressures occurring within the head at the *contre-coup* location relative to the blast exposure, and it has been hypothesized that this negative pressure could result in cavitation of Cerebrospinal Fluid (CSF) surrounding the brain, leading to brain tissue damage. The cavitation threshold of CSF, the effect of temperature, and the effect of impurities or dissolved gases are presently unknown. In this study, a novel Polymeric Split Hopkinson Pressure Bar and confinement chamber apparatus were used to generate loading in distilled water similar to the conditions in the vicinity of the CSF during blast exposure. Cavitation was identified using high-speed imaging of the event, and a validated numerical model of the apparatus was applied to determine the pressure in the fluid during the exposure. Increasing the water temperature resulted in a decrease in the 50% probability of cavitation from 21° C (-3320 kPa ± 3%) to 37° C (-3195 kPa ± 5%) in agreement with the theoretical values, but was not statistically significant. Importantly, the effect of water treatment had a significant effect on the cavitation pressure for water with wetting agent (-3320 kPa ± 3%), degassed water (-1369 kPa ± 16%) and untreated distilled water (-528 kPa ± 25%). Thus, reducing dissolved gases through degassing or the use of a wetting agent significantly increases the cavitation pressure and reduces the variability of the cavitation pressure threshold.

Keywords: fluid cavitation, Polymeric Split Hopkinson Pressure Bar, mild traumatic brain injury, negative pressure

INTRODUCTION

Mild Traumatic Brain Injury associated with blast exposure has been a prominent topic of study as a result of the increasing use of IEDs. Brain injury is categorized as mTBI when the individual experiences an alteration or loss of consciousness for up to 30 minutes [1–3]. There is currently no consensus on the mechanisms that can cause mTBI, with some suggesting: shearing damage of soft-tissue [4–7], distortion of brain cellular structures [5, 8–14], and intracranial fluid cavitation [5, 15–19]. With regard to cavitation, a number of studies have demonstrated that negative pressure can occur within the head at the *contre-coup* location relative to the blast exposure, as a result of pressure wave propagation initiated by a blast wave [16, 19–28]. There is a possibility of cavitation if the negative CSF pressure exceeds the tensile threshold, resulting in the sudden inception, growth, and collapse of a cavitation bubble. Theoretically, the implosive collapse of a near-vacuum bubble results in localized compressive pressures and temperatures several magnitudes above ambient pressure and temperature, respectively [29–31]. CSF cavitation *in vivo* has not been observed and the negative pressure threshold for cavitation is currently unknown. Several numerical studies investigating cavitation due to blast limit the minimum CSF pressure to -100 kPa or the equivalent of 1 atmosphere to simulate the occurrence of cavitation [16, 17, 26, 32]. The aim of this study was to determine the effects of fluid degassing treatment and temperature on the cavitation pressure threshold using distilled water.

METHODS

Tests with distilled water were performed using a Polymeric Split Hopkinson Pressure Bar (PSHPB) apparatus coupled with a confinement chamber developed for evaluating cavitation (Fig 1). The apparatus comprised a 25.4 mm diameter steel

sphere striker, a 25.4 mm diameter and 2.4 m long Polymethyl Methacrylate (PMMA) incident bar, and a 25.4 mm diameter and 61 mm long PMMA confinement chamber. Strain gauges (CEA-13-250UW-120, Micro-Measurements) were mounted at the midpoint of the incident bar. Measured strains were amplified (2210B Signal Conditioning Amplifier, Vishay) and recorded (BNC-2110, National Instruments) at 2 MHz. A Photon Doppler Velocimeter (PDV) (1550 nm wavelength, Ohio Manufacturing Institute) probe was directed at the free-end of the chamber, measuring surface velocity at a recording frequency of 10 MHz. A high-speed camera (FASTCAM SA5 Model 1300K-M1, Photron, 50,000 fps with 512x272 resolution) was oriented perpendicular to the transparent chamber, and used to determine the occurrence of cavitation [33–36].



Fig 1 Polymeric Split Hopkinson Pressure Bar with confinement chamber apparatus for measuring fluid cavitation threshold and instrumentation

The apparatus generated an incident pressure pulse comparable to that observed propagating in the brain originating from the *coup* of blast exposure [16, 19–28, 34]. The incident pulse propagated along the incident bar, through the chamber and fluid, and reflected in tension at the free-end of the chamber, generating negative fluid pressure localized adjacent to the fluid-chamber interface. It was concluded in a previous study that the implementation of a pressure transducer resulted in reduced cavitation thresholds due to discontinuous geometries and large impedance mismatches [34]. Therefore, a numerical model was developed to predict the apparatus and fluid dynamics. The axisymmetric numerical model consisted of 203,000 axisymmetric quadrilateral elements (~0.4 mm), following a mesh refinement study. A previous study by the authors describes the apparatus and numerical model in full detail and demonstrates the efficacy of the model in predicting strain wave propagation, surface velocities, and fluid pressure changes [34, 36].

Experimental cavitation threshold tests were performed with increasing striker velocities. The tests started at sub-cavitation loading, confirmed through high-speed imaging, and increased until cavitation was observed. After cavitation, the water was discarded, a new water sample was prepared, and the process of increasing striker velocities was restarted. The experimental striker velocities were used with the numerical model to predict the negative pressure occurring during the experiment. Lastly, a sigmoid-shaped probability curve was calculated with the procedure outlined in ISO/TS 18506 using the dataset of cavitation occurrence versus the peak predicted negative water pressure. The pressure corresponding to the 50% probability of cavitation was identified as the cavitation pressure threshold [37].

Three different water conditions (wetting agent, degassed, and non-degassed) and two different temperatures (room temperature, 21°C, and body temperature, 37°C) were tested. Non-degassed samples were created by using distilled water asis without any other treatments. For tests with a wetting agent, a thin film of ~1% concentration wetting agent (Jet–Dry Rinse Agent, Finish) and distilled water solution was applied to the inner-walls of the chamber before filling with non-degassed water. For the degassed condition, an open-chamber filled with water was degassed with an ambient pressure of -93 kPa for at least 20 minutes. The ambient pressure was slowly decreased to avoid vigorous agitation of the water. Samples requiring room temperature were left to sit at room temperature for several hours before testing. Samples requiring body temperature were prepared by placing a sealed-chamber filled with water into a 37°C water bath and verifying the test sample temperature with an infrared thermometer immediately before testing.

RESULTS AND DISCUSSION

Fifteen tests were performed with distilled water at 37° C with a wetting agent applied to the inner-walls of the chamber to minimize surface tension, and therefore minimize entrapped air. A cavitation pressure threshold of -3195 kPa \pm 5% (Fig 2) was calculated (ISO/TS 18506), demonstrating an expected decrease in threshold relative to -3320 kPa \pm 3% measured in a previous study for distilled water at 21°C with the same wetting agent (Fig 2) [34]. The measured 4% threshold decrease was comparable to a predicted 3% decrease calculated with the assumption that surface tension is the driving mechanism resisting cavity growth [30, 38, 39]. It should be noted that when considering the variability in the data, the average threshold decrease was not statistically significant.

Thirty-nine cavitation tests were performed with degassed distilled water at 37° C without a wetting agent, resulting in a calculated threshold of -1369 kPa ± 16%. Similarly, thirty-nine tests were performed with non-degassed distilled water at 21°C without a wetting agent, resulting in a calculated threshold of -528 kPa ± 25%. Lower variability and a higher cavitation threshold were observed in the degassed water relative to the non-degassed water. Both observations were attributed to the reduction of entrapped air at the water-chamber interfaces, resulting in a higher tolerance for tension. There is further support for this conclusion when both thresholds were compared to non-degassed distilled water with a wetting agent (-3320 kPa ± 3%), which demonstrated a much higher threshold and lower variability [34]. The effect of the wetting agent can be seen in the 21°C water tests, yielding an increase of approximately 630% in negative threshold relative to non-degassed conditions. With the assumption that the 37°C water tests exhibit similar effects as the 21°C tests, albeit lesser negative thresholds, degassing yields an increase of approximately 270% in negative threshold relative to non-degassed conditions.



Fig 2 Cavitation probability curves for distilled water including the effects of temperature and degassing method (error bars show one standard deviation)

CONCLUSIONS

Cavitation tests were undertaken for distilled water with varying temperature and pretreatment using a PSHPB coupled to a confinement chamber, high-speed imaging to identify cavitation, and a computational model to determine the fluid pressure during the exposure. It was determined that increasing the temperature of the distilled water with a wetting agent from 21°C to 37°C resulted in a small decrease in cavitation threshold, in agreement with theoretical calculations albeit not statistically significant. Pretreatments to the distilled water including degassing and the use of a wetting agent had the largest effect in terms of increasing the cavitation threshold while also reducing variability in the experimental data. It is concluded that the preparation of fluid samples for cavitation tests can have a large effect on the measured cavitation threshold.

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REFERENCES

- 1. Ruff R (2005) Two Decades of Advances in Understanding of Mild Traumatic Brain Injury. J Head Trauma Rehabil 20:5–18. doi: 10.1097/00001199-200501000-00003
- 2. McCrea M (2008) Mild traumatic brain injury and postconcussion syndrome: The New York evidence base for diagnosis and treatment. Oxford University Press, New York
- 3. Gardener RC, Yaffe K (2015) Epidemiology of mild traumatic brain injury and neurodegenerative disease Raquel. Mol Cell Neurosci 66:75–80. doi: 10.1016/j.mcn.2015.03.001.Epidemiology
- 4. Alley MD, Schimizze BR, Son SF (2011) Experimental modeling of explosive blast-related traumatic brain injuries. Neuroimage 54:S45–S54. doi: 10.1016/j.neuroimage.2010.05.030
- 5. Sarntinoranont M, Lee SJ, Hong Y, et al (2012) High-Strain-Rate Brain Injury Model Using Submerged Acute Rat Brain Tissue Slices. J Neurotrauma 29:418–429. doi: 10.1089/neu.2011.1772
- 6. Nie X, Sanborn B, Weerasooriya T, Chen W (2013) High-rate bulk and shear responses of bovine brain tissue. Int J Impact Eng 53:56–61. doi: 10.1016/j.ijimpeng.2012.07.012
- 7. Sosa M a G, De Gasperi R, Paulino AJ, et al (2013) Blast overpressure induces shear-related injuries in the brain of rats exposed to a mild traumatic brain injury. Acta Neuropathol Commun 1:51. doi: 10.1186/2051-5960-1-51
- Ling G, Bandak F, Armonda R, et al (2009) Explosive blast neurotrauma. J Neurotrauma 26:815–825. doi: 10.1089/neu.2007.0484
- 9. Bo C, Balzer J, Brown K a., et al (2011) Development of a chamber to investigate high-intensity compression waves upon live cell cultures. Eur Phys J Appl Phys 55:31201. doi: 10.1051/epjap/2011110052
- 10. Risling M, Plantman S, Angeria M, et al (2011) Mechanisms of blast induced brain injuries, experimental studies in rats. Neuroimage 54:S89-97. doi: 10.1016/j.neuroimage.2010.05.031
- 11. Bolander R, Mathie B, Bir C, et al (2011) Skull flexure as a contributing factor in the mechanism of injury in the rat when exposed to a shock wave. Ann Biomed Eng 39:2550–2559. doi: 10.1007/s10439-011-0343-0
- 12. Ryu J, Horkayne-Szakaly I, Xu L, et al (2014) The problem of axonal injury in the brains of veterans with histories of blast exposure. Acta Neuropathol Commun 2:153. doi: 10.1186/s40478-014-0153-3
- 13. Heldt SA, Elberger AJ, Deng Y, et al (2014) A novel closed-head model of mild traumatic brain injury caused by primary overpressure blast to the cranium produces sustained emotional deficits in mice. Front Neurol 5 JAN:1–14. doi: 10.3389/fneur.2014.00002
- 14. Kamnaksh A, Budde MD, Kovesdi E, et al (2014) Diffusion tensor imaging reveals acute subcortical changes after mild blast-induced traumatic brain injury. Sci Rep 4:4809. doi: 10.1038/srep04809
- 15. Lee CS, Frizzell LA (1988) Exposure levels for ultrasonic cavitation in the mouse neonate. Ultrasound Med Biol 14:735–742. doi: 10.1016/0301-5629(88)90029-4
- 16. Goeller J, Wardlaw A, Treichler D, et al (2012) Investigation of Cavitation as a Possible Damage Mechanism in Blast-Induced Traumatic Brain Injury. J Neurotrauma 29:1970–1981. doi: 10.1089/neu.2011.2224
- 17. Panzer MB, Myers BS, Capehart BP, Bass CR (2012) Development of a finite element model for blast brain injury and the effects of CSF cavitation. Ann Biomed Eng 40:1530–1544. doi: 10.1007/s10439-012-0519-2
- Hong Y, Sarntinoranont M, Subhash G, et al (2015) Localized Tissue Surrogate Deformation due to Controlled Single Bubble Cavitation. Exp Mech 97–109. doi: 10.1007/s11340-015-0024-2
- 19. Singh D, Cronin DS, Haladuick TN (2014) Head and brain response to blast using sagittal and transverse finite element models. Int j numer method biomed eng 30:470–489. doi: 10.1002/cnm.2612
- 20. Zhu F, Mao H, Dal Cengio Leonardi A, et al (2010) Development of an FE model of the rat head subjected to air shock loading. Stapp Car Crash J 54:211–225. doi: 2010-22-0011 [pii]
- 21. Bir C (2011) Measuring Blast-Related Intracranial Pressure Within the Human Head. Detroit, MI
- 22. Ganpule S, Alai A, Plougonven E, Chandra N (2013) Mechanics of blast loading on the head models in the study of traumatic brain injury using experimental and computational approaches. Biomech Model Mechanobiol 12:511–531. doi: 10.1007/s10237-012-0421-8
- 23. Grujicic M, Arakere G, He T (2010) Material-modeling and structural-mechanics aspects of the traumatic brain injury problem. Multidiscip Model Mater Struct 6:335–363. doi: 10.1108/15736101011080097
- 24. Hua Y, Kumar Akula P, Gu L, et al (2014) Experimental and Numerical Investigation of the Mechanism of Blast Wave Transmission Through a Surrogate Head. J Comput Nonlinear Dyn 9:031010. doi: 10.1115/1.4026156
- 25. Moss WC, King MJ, Blackman EG (2009) Skull flexure from blast waves: A mechanism for brain injury with implications for helmet design. Phys Rev Lett 103:4–7. doi: 10.1103/PhysRevLett.103.108702
- 26. Panzer MB, Bass CR, Myers BS (2010) Numerical Study on the Role of Helmet Protection in Blast Brain Injury. In: Pers. Armor Syst. Symp. Quebec City, Canada, p 11
- 27. Sayed T El, Mota A, Fraternali F, Ortiz M (2008) Biomechanics of traumatic brain injury. Comput Methods Appl Mech Eng 197:4692–4701. doi: 10.1016/j.cma.2008.06.006

- 28. Zhang L, Makwana R, Sharma S (2013) Brain response to primary blast wave using validated finite element models of human head and advanced combat helmet. Front Neurol 4 AUG:88. doi: 10.3389/fneur.2013.00088
- 29. Thiruvengadam A (1974) Handbook of Cavitation Erosion. [Laurel, Md.] Hydronautics, Inc.
- 30. Brennen CE (1995) Cavitation and bubble dynamics. Annu Rev Fluid Mech. doi: 10.1017/CBO9781107338760
- 31. Knapp RT, Daily JW, Hammitt FG (1970) Cavitation. McGraw-Hill
- 32. Wardlaw A, Goeller J (2010) Cavitation as a possible Traumatic Brain Injury (TBI) damage mechanism. IFMBE Proc 32 IFMBE:34–37. doi: 10.1007/978-3-642-14998-6_9
- Salisbury C, Cronin D, Lien F-S (2015) Deformation Mechanics of a Non-linear Hyper-viscoelastic Porous Material, Part I: Testing and Constitutive Modeling of Non-porous Polychloroprene. J Dyn Behav Mater 1:237–248. doi: 10.1007/s40870-015-0026-2
- 34. Bustamante MC, Singh D, Cronin DS (2017) Polymeric Hopkinson Bar-Confinement Chamber Apparatus to Evaluate Fluid Cavitation. Exp Mech 1–20. doi: 10.1007/s11340-017-0323-x
- 35. Bustamante M, Singh D, Cronin DS (2016) Modified Hopkinson Apparatus to Investigate Fluid Cavitation as a Potential Source of Injury. In: Conf. Proc. Soc. Exp. Mech. Ser. pp 43–51
- 36. Bustamante M, Cronin DS, Singh D (2018) Experimental Testing and Computational Analysis of Viscoelastic Wave Propagation in Polymeric Split Hopkinson Pressure Bar. In: Dyn. Behav. Mater. Vol. 1 Proc. 2017 Annu. Conf. Exp. Appl. Mech. Dynamic Behavior of Materials, Volume 1: Proceedings of the 2017 Annual Conference on Experimental and Applied Mechanics, pp 67–72
- 37. ISO/TS (2014) ISO/TS 18506 Procedure to construct injury risk curves for the evaluation of road user protection in crash tests (). International Organization for Standardization (ISO), Geneva, Switzerland
- Vargaftik NB, Volkov BN, Voljak LD (1983) International Tables of the Surface Tension of Water. J Phys Chem Ref Data 12:817–820. doi: 10.1063/1.555688
- Kayser W V. (1976) Temperature dependence of the surface tension of water in contact with its saturated vapor. J Colloid Interface Sci 56:622–627. doi: 10.1016/0021-9797(76)90130-2