Primary Blast Injury – the Often Overlooked Hyperbaric Oxygen Indication

E. George Wolf[1], Tom Fox[2]

[1] 59th Medical Wing, Undersea and Hyperbaric Medicine, Joint Base San Antonio - Lackland, Texas U.S.

[2]Centre Hyperbare de L'Île, Pincourt QC. Canada

CORRESPONDING AUTHOR: E. George Wolf – *earl.g.wolf.ctr@mail.mil*

ABSTRACT

Primary Blast Injury – the Often Overlooked Hyperbaric Oxygen Indication

Introduction: This opinion article presents the historical aspect of blast injury from early casualties, physician observations and pathophysiology from research and case reports.

Methodology: A literature search and review using OpenAthens, PubMed, and Google Scholar was done to obtain the evolution of blast injuries symptoms, etiology and treatment.

Conclusion: Primary blast injury pathophysiology has an air emboli etiology that should be initially treated using hyperbaric oxygen therapy to resolve the emboli and minimize resultant inflammation and subsequent nerve degeneration.

Primary Blast Injury – the Often Overlooked Hyperbaric Oxygen Indication

Introduction:

It has been called the shot without the bullet, the slash without the sword. It is present everywhere within its range and it cannot be shielded. This quote describing primary blast injury has been attributed to two physicians, Franchino Rusca in 1914 (1) and Theodor Benzinger (2) in 1952, as they attempted to explain combat deaths without apparent injuries they had treated. What is clear that changes in ambient pressure brought about by exposure to blasts, detonations, and weapons firing is capable of injuring soldiers. These injuries are not apparent and usually subordinated to the care of injuries that are more visible. Nevertheless, make no mistake these injuries are real and a direct result of extraordinary changes in ambient pressure.

Descriptions of the effects of extraordinary pressure on soldiers in battle predate World War I and appear in the writings of soldiers in the field. One such writing is provided by Lt. William A. Morgan of the 23rd Kentucky Regiment Union Force. Lt. Morgan was among a group of men exposed to a cannonade from an estimated fifty Confederate guns. “The bursting projectiles seem to compress the air and one’s head feels as if bound with iron bands.” (3)

Lt. Morgan’s insight into the environmental and physical changes in pressure, mirrors comments appearing in an April 18, 1885 Compressed Air Bath article where Dr. C.T. Williams (4) identified symptoms experienced upon pressurization as “a sensation of tightness, as if the head was bound with a band of iron.”

In World War 1 there were competing theories in how best to address the invisible wounds of war. The original term shell shock was used by the soldier in the trenches to describe the

impairment from exposure to extensive bombardment. Recognition of blast injury in World War I expanded physical, psychiatric and neurological symptoms. These resultant symptoms were described by psychologist CPT (Dr.) Charles Myers (5) who popularized the term “shell shocked” and neurologist Dr. Gordon Holmes (6) within their respective specialties. Throughout 1914–18 the term shell shock evolved with stark diagnostic choice was between traumatic neurosis (a physical injury), which gave pension rights, yet allegedly cost the nation dearly in both its finances and its moral and mental health, and traumatic hysteria (a psychiatric condition), which yielded no compensation but usually ended in return to employment (7). To address this great influx of wounded, Captain Myers was tasked to gather data in order to create a policy for classification and treatment of psychiatric battle casualties (8). Then as in today there was a great struggle to understand the nature of these silent injuries affecting the combatants. This struggle placed CPT (Dr.) in direct conflict with the position taken by his direct supervisor LTC (Dr.) Gordon Holmes who believed the origin of the injury was a physical injury (6). This struggle lead to the adoption of the term NYDN – Not Yet Diagnosed Nervous" in triage pending further investigation by medical specialists.

Following World War I, D.R. Hooker (9) undertook an investigation to determine a mechanism of injury from blast. In this investigation he established a relationship between the pressure and duration of the blast and the severity of the injury. Williams (10), as a result of experiments with human subjects at various distances from guns, felt there was a diminution of efficiency of those personnel exposed to pressures as low as 2.5 psi. This observation continued to be emphasized in US Army Medical Department publications following World War II and can be applicable to the silent injuries seen currently.

Pathophysiology

Barrow and Rhodes described 200 individuals who had air blast injuries during World War II (11). Their summary included “Those patients who survived sixty minutes recovered. Damage to the ears by blast and to the eyes from foreign bodies occurred in a high percentage of those who recovered. Periods of unconsciousness, convulsions, chest pain, pulmonary complications and abdominal distress were minimal in this group. Intestinal perforation did not occur. Shock, bradycardia, hypotension, decreased psychomotor activity and slow respiration were the outstanding clinical signs and symptoms of this group.” Another interesting comment was “In 2 of the 3 patients who died within thirty minutes after the explosion without external evidence of injury, generalized convulsions were noted. They were not unlike those sometimes associated with anoxia during anesthesia or too many "G's."

In examining the post mortem findings from the bombings in England in 1941 Sher suggested the creation of air emboli from exposure to the pressure from blast.[(12)] During the development of nuclear weapons, the effects of blast resulted in many studies in animal and human reports recognizing the creation of air emboli during the blast. Clemedson, (13) in 1954, entertained air embolism is a potential cause of death. Damon (14), in 1971 demonstrated key benefits of using hyperbaric pressure to treat air emboli in animal studies resulting in survivability of the hyperbaric exposed animals compared to the control animals. The presence of microbubbles in individuals exposed to blast waves, brought about by explosions and detonations, is documented by numerous sources. Rosenfeld (15) acknowledges, “The blast wave induces sudden changes in intracranial pressure that result in bubble formation, particularly at interfaces between cerebral

spinal fluid (CSF) and the brain, resulting in penetration and cavitation of brain tissue, disruption of axonal pathways and damage to capillaries." There is also evidence that primary blast injury from the blast wave can lead to neurologic injury from fractures around sinuses, air embolism in cerebral vessels, cerebral contusion, and intracranial hemorrhage.

The pressure wave created by the blast will travel through the body at different speeds depending on the tissue density. Particularly in the blood and vasculature, the blast wave frequency will create rapid cycles of compression and decompression that can result in air emboli injury in tissue or within the vasculature. These emboli can travel anywhere within the vasculature and is particularly concerning the brain (16), heart (17), intestinal tract (18) and also been found to lodge in kidneys (19). In addition, damage is also caused by stretch, strain and rupture of the brain parenchyma and damage blood vessels. One must recognize that primary blast injuries are caused by barotrauma from the pressure wave. The intensity of the blast wave is dependent upon the initial overpressure; the overpressure duration; and the environment density in which the blast occurred. This includes water versus air; the distance from the explosion; the reflection of the blast wave energy i.e. walls or confined environments (energy can be up to 9 times greater than similar blast in an open space (20). Secondary blast injuries result from projectiles and shrapnel from the blast source. Tertiary blast injuries are caused by the blast wind that may throw victims against fixed objects resulting in blunt trauma. Quaternary blast injuries result from fire and building collapse resulting in burns and crush injury (21) as well as carbon monoxide, dirty bomb radiation, and biological/clinical materials.

Cernak (22) identifies acute neurotrauma from blast including energy metabolism failure, diffusion and perfusion deficits, diffuse axonal damages resulting in impaired information

processing, increased blood-brain barrier permeability, and cognitive deficits. Zbesko (23) suggests that in stroke, that glial scar formation is critical to healing as seen in other nervous system injuries. However; necrotic liquefaction results in glial scar permeability. These scars when located near the hippocampus results in degeneration of hippocampal neurons.

Ma (24) suggests that multiple chronic neurological conditions are the result of calpains, "There has been growing awareness of the contribution of axonal pathology to the morbidity and mortality of many human diseases, including central nervous system and peripheral nervous system trauma, cerebral ischemia, neurodegenerative disorders, and peripheral neuropathies. Axonal dysfunction and degeneration in these seemingly disparate disorders likely share common molecular injury mechanisms, one of which is dysregulated intraaxonal calpain activity."

Sources of blast waves

In preparing to deploy to combat zones and in the wartime theater, members of combat forces are exposed to substantial changes of pressure through exposure to explosions i.e., IED's, and detonations including breaching operations. Other equipment that generally require hearing protection, are also capable of producing blast waves. Firing of crew-served weapons such as the Karl Gustav Recoilless Rifle MAAWS/RAWS weapons system and sustained firing of the individual combat weapon as in firefights. Hoge (25) was the first to highlight the positive correlation between "PTSD" or signature wounding and firefights.

Adapting to urban environment on the modern battlefield, modifications in barrel length of the individual combat weapon were made. For example, M-16 innovation of the direct impingement system results in a portion of the discharge pressure was redirected toward the firer to eject the

expended round casing and chamber a new round. In sustained firing, repeated pressure spikes deliver subconcussive impacts result in a diminished efficiency that can be likened to dementia pugilista. Impulse pressure equivalent of 2.4 times the atmospheric pressure can occur in a matter of milli-seconds (170 decibels or 242.862 kilopascals) for each round fired. The threshold for pain is 160 decibels. An impulse of 170 decibels is equivalent to that produced by the detonation of a single stun (flash-bang) grenade. The effects of this explosion are powerful enough to confuse and disorient. Repetitive or sustained firing produces multiple pressure impulses, the optimal environment for growth of microbubbles in bi-lipid structures throughout the body and the brain. The impairment created by this exposure is also acknowledged by Hoge, "The exposure of Service Members to 1 to 2 firefights doubles the occurrence of "PTSD", from what has been observed in individuals who have not been exposed to firefights. This increase continues, even with limited subsequent exposures. With exposure to 5 firefights, the incidence rises to 19.3 percent, without an indication that it would plateau at this point. Hadanny (26) relates to several studies that demonstrate the blast wave tissue changes results in bubble formation between the cerebrospinal fluid and the brain and cavitation of brain tissue causing axonal pathway disruption, capillary damage and decreased regional blood flow.

Suggested Treatment Protocol

Most blast injuries occur with military operations, unfortunately, terrorist attacks as well as industrial accidents and building gas explosions are becoming more commonplace. Once an explosion occurs, the management of the neurological abnormalities in the blast casualty should follow a comprehensive multidisciplinary treatment plan. Zajtchuk (28) identifies hyperbaric oxygen as a definitive treatment. Emergency Medicine textbooks recommend the use of

hyperbaric oxygen as part of the treatment of the blast victim. Wolfson (29) relays that HBOT can be effective for cerebral air emboli up to 12 hours after the acute event this allows patient trauma, including pneumothorax, to be stabilized before hyperbaric oxygen therapy.

Pressure wave only with no evidence of other trauma but continued loss of consciousness is suspect for an air embolism etiology. The use of ultrasound to the chest can evaluate the existence of a pneumothorax. Treat the pneumothorax, if present, using a device that can go into the hyperbaric chamber. Air embolism is typically treated with a U.S. Navy (USN) treatment table six. However, there is a possibility of a traumatic brain injury from coup-contracoup in addition to air emboli. Consequently a Hart table can be considered that will use a short duration, high pressure protocol to decrease bubble size and dissolve nitrogen to where the bubble dissipates. The protocol then decreases the pressure to levels used in acute traumatic brain injuries.

The Hart protocol is as follows:
Descent rate 1-3 psi per minute to 29.4 psig pressure (3.0 ATA); initiate (multiplace) breathing 100% oxygen for 30 min; decompress to 14.7 psig (2.0 ATA) over 15 min (about 1.0 psi/minute); maintain 14.7 psig (2.0 ATA) for 60 min; decompress to surface over 15 min. The total elapsed time, not counting descent is 120 min.

If the patient is unconscious, tympanic membrane rupture is possible. Pressure equalization tubes or myringotomy can be considered prior to the treatment. If any continued concussion symptoms are present, the patient can be treated daily for 60 minutes at 2.0 ATA as acute brain injury is possible.

Blast injury with secondary or greater category: stabilize injuries and use ultrasound to rule out pneumothorax. Treat pneumothorax as above, if present. Although a higher level of TBI may be present, potential air embolism in the brain, heart, or solid organs take precedence and the Hart protocol is recommended. If a TBI is suspected, then the patient should continue daily treatment at 2.0 ATA for 60-90 minutes particularly for other injuries such as compartment syndrome or compromised flaps or grafts.

Conclusion:

Primary blast injury has an air emboli etiology that was eventually recognized by observations, case reports and research over a 150 years period. Hyperbaric oxygen therapy has been recommended by civilian and military textbooks and publications, yet remains overlooked in blast injury cases presenting in emergency facilities in spite of available hyperbaric facilities. Hyperbaric facilities need to actively insert their capability into local disaster response options regarding blast events. Primary blast injury treatment should include hyperbaric oxygen therapy to resolve the emboli and minimize resultant inflammation and subsequent nerve degeneration.

1. Fox TM; A Valuable Therapeutic Treatment Option in a Multi-Disciplinary Approach to Signature Wounding from Combat; https://intranet.txcouncil.com/wp-content/uploads/2016/11/Hyperbaric-_-Fox.pdf; Retrieved 1 Feb 2020

2. Bellamy, R.F., Conventional Warfare Ballistics, Blast, and Burn Injuries; Textbook of Military Medicine 1991 Office of the Surgeon General

3. Garrison, Webb, Curiosities of the Civil War: Strange Stories, Infamous Characters & Bizarre Events; 2011 ISBN 978-1-59555-359-1)

4. Williams CT, Lectures on the Compressed Air Bath and Its Uses in the Treatment of Disease; The British Medical Journal April 18,1885 pg 769-772.

5. Myers CS; the contribution to the study of shell shock; the Lancet, 23 February 2015, p316

6. MacLeod AD; Shell shock, Gordon Holmes and the Great War; J R Soc Med 2004;;(97):86–89

7. Leese, P; Shell Shock, Traumatic Neurosis and the British Soldiers of World War I; Springer, 12 Jul 2002 Pg 17

8 Jones, E , Shell shock and Mild Traumatic Brain Injury: A Historical Review; American Journal of Psychology,164 9 November 2007 1641-45

9. Hooker, D. R.: Physiological effects of Air Concussion, Am. J. Physiol, 1924, 67:219-274

10. Williams, E. R. P.: Blast Effects in Warfare, Brit. J. Surg., 30: 38-49, July 1942.

11. Barrow DW and Rhodes HT; Blast concussion injury; JAMA 29 Jul 1944, p 900 -902

12. Sher, S.; Air Embolism from Blasts; The British Medical Journal 11 May 1941, p 797

13. Clemedson CJ, Holter I, Hultman BM. Air embolism and the cause of death in blast injury. The Military Surgeon, June 1954 424-437).

14. Damon EG, Jones R. Comparative effects of hyperopia and hyperbaric pressure in the treatment of primary blast injury. Technical Report DASA 2708. Headquarters, Defense Nuclear Agency, Washington, DC. 1971

15. Rosenfeld, JV et al, Blast- related traumatic brain injury, Lancet Neurol 2013;12:882-93)

16. Clemedson CJ, Holter I, Hultman BM. Air embolism and the cause of death in blast injury. The Military Surgeon, June 1954 424-437

17. Phillips YY. Primary blast injuries. Ann Emerg Med 1986 Dec, 15 (12) 1446-1450

18. Wolf SJ, Bebarta VS, Pons PT, et al. Blast injuries. Lancet Aug 2009, 374 (9687) 405-415

19. Freund U, Kopolovic K, Durst A; Compressed air emboli of the aorta and renal artery in blast; Injury, 12 (1) July 1980, 37-38.

20. White CS. The scope of blast and shock biology and problem areas in relating physical and biological parameters. Ann. NY Acad. Sci. 152(1), 89–102 (1968)

21. ((Nichols, D, Shaffner D. Mass casualty events, Section: Bombings and blast injuries. Rogers' Textbook of Pediatric Intensive Care, 5th; Lippincott Williams & Wilkins, 2016. Retrieved 15 Jan 20 https://www.r2library.com/Resource/detail/1451176627/ch0034s1144.

22. Cernak I; Understanding blast-induced neurotrauma: how far have we come?; Concussion, 2017 2(3)

23. Zbesko, JC, et al, Glial scars are permeable to the neurotoxic environment of chronic stroke Infarct. Neurobiology of Disease, April 2018

24. Ma M. Role of calpains in the injury-induced dysfunction and degeneration of the mammalian axon. Neurobiol Dis. 2013 Dec; 60:61-79

25. Hoge, CW et al, Combat Duty in Iraq and Afghanistan, Mental Health Problems, and Barriers to Care, The New England Journal of Medicine Vol 351 No.1, July1 2004, pages13-22

26. Amir Hadanny & Shai Efrati. Treatment of persistent post-concussion syndrome due to mild traumatic brain injury: current status and future directions. Expert Review of Neurotherapeutics 23 Jun 2016 http://dx.doi.org/10.1080/14737175.2016.1205487

27. Zajtchuk,R et al, Conventional Warfare; Ballistic, Blast, and Burn Injuries. Text of Military Medicine- Office of the Surgeon General, Department of the Army, United States of America, 1998, pg 313

28. Wolfson, A. Blast injuries, section Differential diagnosis. Harwood-Nuss' Clinical Practice of Emergency Medicine, 6th; Lippincott Williams & Wilkins, 2015. Retrieved 4 Jan 20 from https://www.r2library.com/resource/detail/1451188811/ch0051s0926