Spring 4-29-2016

Pulmonary Primary Blast Injuries

Moe Uddin
University of Connecticut - Storrs, moe.uddin@uconn.edu

Pulmonary Primary Blast Injuries

Biomedical Engineering Honors Thesis
Date: April 29, 2016

By: Moe Uddin

Honors Advisor: Professor Ki Chon

Table of Contents

Abstract

Primary underwater explosions are particularly dangerous because of the water properties relative to air, and the resulting pathologies presents difficulty in determining their etiologies. The whole body is affected by the blast wave and the survivor is considered a trauma patient. The exact mechanism of injury is not well understood. In the literature, there are inconsistences between the explosive and the resulting outcome of the experimental animals. Primary lung blast injuries is the leading cause of mortality in underwater explosions and this explores the different pathologies, symptoms, and treatments that are currently available.

1. Introduction

At first glance, explosive damage may not appear to be widespread, but they are a large concern, particularly in wartime and from the casualties experience in Iraq by the United States [1]. The injuries experienced from a blast range from blunt force trauma to projectiles to radiation, and the danger comes from the uncertainty of the explosive and the environment. We will be looking into primary blast injuries, which only takes into account the pressure wave expelled by the explosive. The underwater environment we will be considering is a free, unobstructed environment and not an enclosed area. Further, in underwater explosions the primary injuries experienced are greater due to the difference in the properties of water relative to air. The survivor of an underwater blast will have whole body injuries and will be managed as a total body trauma patient. This work will look into primary pulmonary injuries, or lung injuries resulting from the blast wave. The devastation of the lung tissue where there is massive pulmonary hemorrhage due to the disruption of the alveolar structure and the resulting air embolism was termed “blast lung” [2]. The causality of the resulting pathologies is discusses as there are many different pathologies and inconsistences between the data available. The symptoms of those with blast lung varies and overlaps and exacerbates as time progresses, leading to a difficult diagnosis. The treatment is contingent upon proactive diagnosis of the survivor.

2. Background

Injuries from explosives range cover a wide spectrum of conditions, and how the injury occurred is classified in different ways. When an explosive is detonated, it causes the rapid, almost instantaneous, release of gas and heat causing rapid expansion. This rapid release of gas is the blast overpressure (BOP) of the explosive, which raises the atmospheric pressure [2] [3]. The pressure wave then travels radially outward from the epi-center of the explosion and interacts with materials in its way, notwithstanding humans [2]. The trauma induced by the explosive wave can be categorized into different categories: primary, secondary, tertiary, and quaternary injuries, see table 1.

Category	**Characteristics**	**Body Part Affected**	**Types of Injuries**
Primary	Unique to high energy explosives, occurs from the envelopment of the body in a blast overpressure wave.	Gas-filled structures are the most vulnerable because they are distorted to the greatest degree, such as the upper airways, lungs, GI tract, and middle ear.	Blast Lung (pulmonary barotrauma)
	Body surface and internal organs are rapidly distorted because the air-containing organs are	Internal distortions of air-containing organs cause the distortion of neighboring organs as well--heart, liver, spleen, and kidneys.	Tympanic membrane rupture and middle ear damage.

	compressible and undergoes volume changes.	Differential loadings in the body can be transmitted to other parts of the body.	Abdominal hemorrhage and perforation.
			Concussion
			Laceration of the liver, spleen, and kidneys.
			Contusion to the heart
			Distortion and rupture of the great vessels.
			Air emboli across the air-blood boundary of the lungs.
			Surge in blood flow and pressure that may lead to tissue injury in the brain.
Secondary	Impact on the body from projectiles.	Any body part can be affected.	Any injury associated with impact of high-speed objects. This type of injury is not unique to blasts.
		Depends on the speed, mass, and the shape of the impacting object.	Penetrating fragments.
Tertiary	Body acceleration caused by the blast wind.	Any body part can be affected.	Any injury associated with body motion and impact.
	Uneven forces on the body due to the blast winds.	Depends on the surface the body impacts.	The typical injuries that would occur with falls or car crashes.
		Primarily head or neck can be accelerated relative to the torso.	Traumatic amputation; muscle tears
Quaternary	All explosion caused injuries that are not associated with pressure or wind effects	Any body part can be affected.	Burns.
	High temperatures.	Body surface, eyes.	Asphyxia.
	Toxic gases.	Respiratory system.	Injury from inhaled toxic gases.
Collateral	Secondary responses to trauma.	Systemic responses from massive trauma.	Not unique to blast.
	Exacerbation or complications of conditions		Angina, hyperglycemia, and hypertension.
			Asthma, COPD, or other breathing problems.

Table 1. The different categories on blast injuries [2] [3] [4] [1].

The injuries are presented in figure 1 in scenario. Note that explosions are not sharply demarcated as we have explained, because the injuries occur in a complex system where there will be refractions of the wave off of surrounding objects.

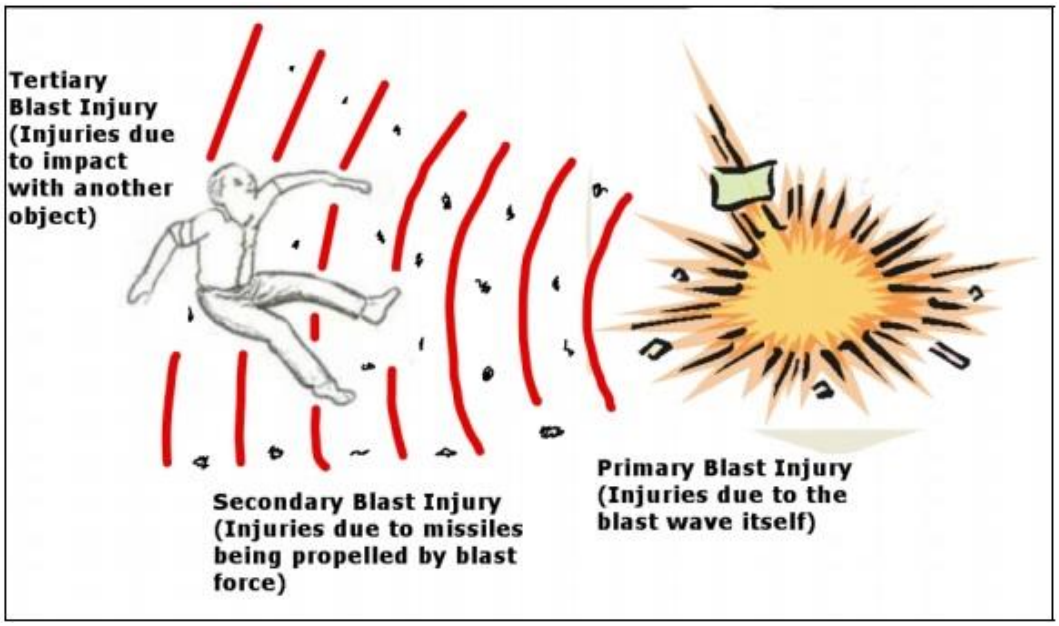

Figure 1. The defined explosive injuries from a blast [2].

As seen from figure 1, the individual caught in the blast will be damage from primary, secondary, tertiary, as well as quaternary blast injuries depending on the type of explosive. Furthermore, there are also collateral injuries which are caused by trauma on the body that disrupts the body's biochemical and neurological system [1]. Although injuries are incurred from these different sources, this work will investigate the effects of primary blast injuries.

2.1 Blast Wave Properties

The blast wave created by an explosive is affected by many different factors, such as the properties of the explosive, the medium, and the complexity of the environment.

Explosives can be categorized into either high-order explosive or low-order explosive [2] [4]. A high-order explosive is one where a shock wave is produced that is faster than the speed of sound and the wave is sustained as it goes through the blast medium. A low-order explosive creates a subsonic explosion where the shock wave is slower than the speed of sound. A low-order explosive releases its energy slower than a high-order explosive. The lethality of high-order explosives is attributed to a fast reaction rate and the charge combusts almost instantaneously [2] [4]. Examples of low-order explosives are black powder or gun powder, and high-order explosives are TNT, C4, and RDX [4].

The medium where the explosive occurs heavily affects the damage potential of the explosive and how the BOP will interact with the surroundings. The mediums that will be briefly discussed

will be air and water. In the literature, there was more research for blast in-air than underwater blasts. Air has a lower density than water, lower sound speed, and lower viscosity relative to water means that in-air blasts are less deadly than underwater explosives [5]. Because air has a lower density and higher elasticity than water, the positive pressure of the wave in air expands faster than sound, and the blast intensity decreases by the cube of the distance from the center [6]. On the other hand, water is essentially incompressible so the blast wave travels further and faster than in air. Water also has a higher density and lower elasticity relative to air, and the positive pressure wave has a higher intensity in water, but a shorter duration. The blast intensity decreases proportionally to the distance from the blast center [6]. In addition, an explosion in a free-field, which is defined as an open environment has been characterized as a Friedlander waveform [3] [4]. The Friedlander waveform is an idealized blast wave shown in figure 2.

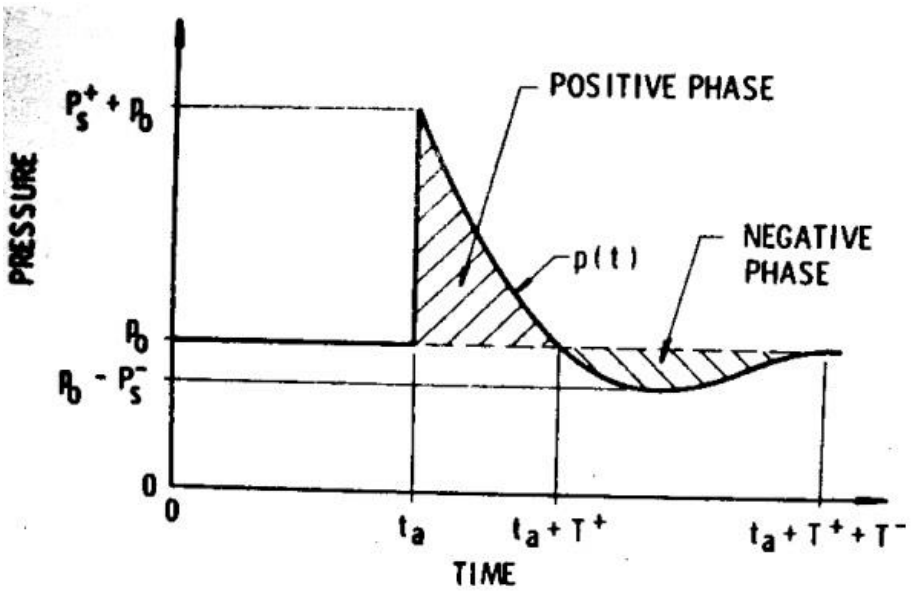

Figure 2. The idealized blast pressure vs. time (Friedlander) waveform in a free-field in air environment [4].

As we can see in figure 2, there's an instantaneous rise in pressure at time, t_a, which reaches pressure, $P_s^+ + P_0$, and then it decays back down to ambient pressure over time $t_a + T^+$ and then goes below the ambient pressure over time T^-. The positive phase is the time it takes for the pressure wave to reach back to ambient pressure from the peak overpressure. The negative phase is the time it takes for the negative pressure to return to the ambient pressure. This is described by the Friedlander equation below.

$$P(t) = p_0 + P_s^+ (1 - {}^{t}/_{T^+}) e^{\frac{-bt}{T^+}}$$

Equation 1. The modified Friedlander equation describing the free-field in-air explosive wave [3] [4].

In equation 1, p_0 is the ambient pressure, p_s^+ is the peak overpressure, T^+ is the positive phase duration, and b is the decay factor.

Therefore, there are a number of factors that affects the damage from an explosive. The peak overpressure, duration of the blast, the medium of the blast, the distance, the environment, and if there is protective equipment worn by the subject.

In real combat however, explosives are not always in an open environment, and when it occurs in an enclosed environment, then complex waves are encountered as opposed to simple waves. In an enclosed environment, there are reflections from the walls or other surrounding objects that amplifies the waves that are experienced by the subject. So the pressure vs. time waveform will look different than showed in figure 2. There will still be one large peak overpressure, but it will be followed by smaller peak overpressures instead of a decay that will compound to the overall force felt by the subject.

At this moment, there has not been an equation that has characterized an explosive underwater like the Friedlander equations as done for in-air explosives [5]. This work will look at the injuries incurred in underwater blasts, which are more dangerous than in-air blasts. Because water has a higher density than water, underwater blasts propagate unfettered because it is not dissipated and travels further. However, because of these water properties, there are only primarily primary blast injuries. Secondary injuries from projectiles are effectively nullified because the viscosity of water exerts a drag force on the projectile and prevents it from reaching the underwater subject in most cases, and prevents damage from quaternary damage if the subject is sufficiently away from the blast epicenter. In addition, there are additional factors to take into account, such as the charge depth, bottom depth of the body of water, gage depth, the reflectivity from the bottom, and the gas bubble fluctuations after detonations [5].

2.2 Anatomy

From explosives, irrespective of medium, there will be many injuries due to the malicious nature of intent in using explosives. However, prior research has shown that in underwater blast injury, the lungs are the most vulnerable and the most likely cause of mortality and morbidity [4]. In an underwater pressure wave, it propagates through the water, and because the body tissue is close to the density of water, the blast wave propagates through the water and into the tissue. The propagation characteristics change when the wave interacts with a gaseous medium, such as the lungs or gastrointestinal (GI) tract. So, the areas of injury are those filled with gas, such as the middle ear, lungs, and GI tract. The ears are the most vulnerable organ but they are the most easily protected with ear plugs, but the lungs and GI tract are not as easily protected because a protective vest will cause reflections of the wave and increase the injury [3]. The degree of injury is not as great in the muscles of body because the density is more uniform and does not have significant density changes [3] [7].

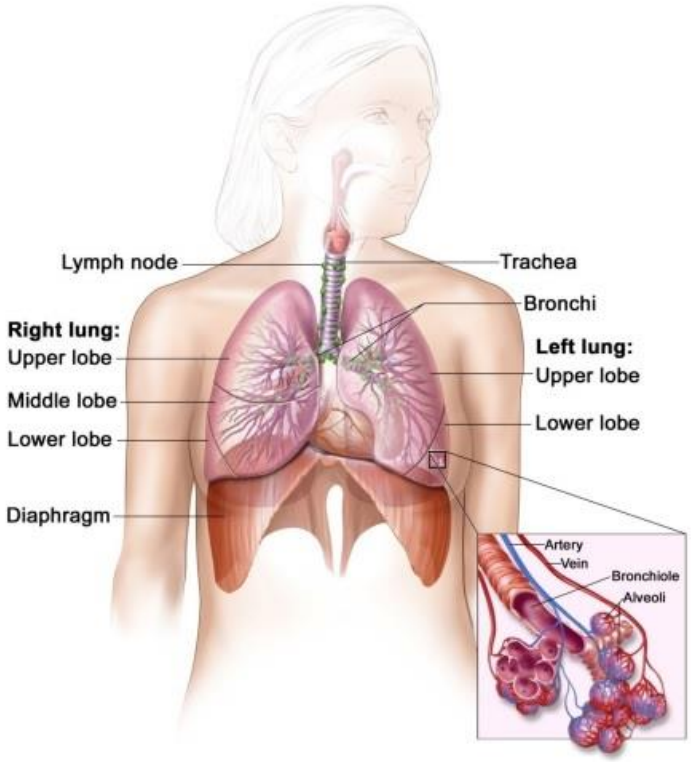

Figure 3. A representation of the respiratory system of a human [4].

The inherent nature of the function of the lungs leads to it being the most vulnerable organ behind the tympanic membrane. The lungs are charged with the gaseous exchange of oxygen and carbon dioxide for the body, and to accomplish this end, the cells of the alveoli are one cell thick to allow the rapid diffusion of the gases. Furthermore, the lungs essentially act as a balloon that fills and empties as the pressure difference between the inside and outside of the lungs not being too great. This modest pressure difference shows the overall resistance of the small alveoli pathways [1].

The lungs can be broken up into the pleura, parenchyma, and the bronchovascular structures [8]. The pleura serves as a single layer of cells that work as a connective tissue that makes a protective lining for the lung parenchyma. It is relatively transparent due to its single cell layer thickness. The parenchyma is the functional portion of the lung where the gas exchange occurs. The air comes to the tiny respiratory units called acini [8]. The bronchovascular structures are within the parenchyma and perform the same function as the parenchyma [8].

3. Mechanism of Injury

The mechanism of pulmonary blast injury is not well understood but there are widely accepted ideas of the etiology of injury. Previously, it was thought that BOP caused injury by accessing the internal organs through the body's orifices, but that has been proven wrong [2]. Further, internal damage to the lungs are not readily observed externally and could be more lethal than typical in-air explosions, so that lends to the difficultly of diagnosing injuries without diagnostic machines. The BOP wave causes a physical impact on the body that causes internal mechanical

damage. It has been noted that the degree of damage is dependent on the depth of immersion of the diver [4] [5] [7]. Table 2 describes all the injury mechanisms that will be discussed.

Injury Mechanisms	
Mechanism	**Effect**
Volumetric Changes	The volume changes causes excessive stress on the tissue beyond its threshold.
Implosion Effect	The creation of gas bubbles at the air-gas interface, and the damage caused by the re-expansion of the bubbles.
Spalling Effect	The movement of liquid from more dense to less dense tissue.
Inertial Effect	The difference in densities of the tissues causes injury.
Pressure Differential Effect	Injury is caused by the difference between the tissue and the air-filled portion of the lung.
Negative Pressure Effect	This is where the negative phase of the wave also causes damage.
Hemodynamic Effect	The instantaneous increase in blood pressure overwhelms the blood vessels.
Free Radical-Mediated Oxidative Stress	The propagation of injury is due to the free-radical mediated oxidative stress which is caused by the decrease in oxygen partial pressure.

Table 2. Proposed primary lung blast injury mechanisms and the resulting physiological effects.

The most likely mechanism has to do with the fact that the lungs are hollow organs. Because the lungs are air-filled and when external pressures, such as the BOP, is forced onto it, it undergoes rapid compression and decompression due to the impact of the pressure wave on

the chest wall [3] [4]. This rapid change causes the delicate tissue and cells walls to rupture from the compressive and decompressive forces [3]. Stuhmiller noted that the volumetric changes in the lung causes extensive volume changes of the lungs and that distorts and damages the delicate alveolar tissues of the lungs [1]. Furthermore, the difference in tissue tensile strength and the speed of the BOP through the different tissues causes different degree of injuries. The damage that occurs when the BOP compresses the tissue and passes the tensile strength of the material causes shearing of the vascular beds and pulmonary hemorrhages as the tissue is compressed and decompressed [2].

It has also been suggested that blast injury are due to the interaction of the pressure wave at the tissue-air interface with the BOP. The human body can be considered to be air and water, and the positive pressure wave will be transmitted to the chest wall by raising the intraabdominal pressure and that causes the rapid elevation of the diaphragm [6]. The implosion effect caused the accumulation of gas bubbles. The water is not completely compressed as the BOP is transmitted, unlike gases that are completely compressed. After the BOP passes, the bubbles build up and causes further pressure waves and the air-containing alveoli gets injured as the bubbles build due to re-expansion, and it has been shown that they contribute to pulmonary lung injury [6] [9] [10]. It has been noted by Stuhmiller that an air emboli is created by forcing gas through the air-blood barrier of the lung and this creates large pressure differences in the body's vascular system [1].

Another proposed mechanism is the inertial effect, which is caused by the different densities of the tissue. The speed of BOP propagation is dependent on the density of the medium or tissue it is in; therefore, then the BOP enters a tissue that is looser, its velocity will increase and leads to the separation of different tissues, such as the ribs and the intercostal tissue [9]. The acceleration and deceleration forces from the BOP causes the tearing of the lungs and mesentery because there are inertial differences between the organs [10].

The pressure differential effect is causes by the difference in pressure between the tissue and the air-filled portion of the lungs [9]. Similarly, Cudahy et al. suggested that injury is caused by the non-uniform pressure distribution by the BOP that leads to the cells to be squeeze upwards and burst [7]. In addition when forced, the vessels will cause fluids to be forced into the less compressible adjacent structures causing internal damage [10]. Further, the compressibility of the tissue is like water, relatively incompressible, so the site of injury is where the differential in compressibility is the greatest, such as the air-tissue interface [7]. The air-containing organs are crushed under the high external load because they are hollow and compressible; therefore, they are distorted and produce local, really high stresses [1]. This mechanism is calling spalling, which is the forcible movement of fluids from more dense to less dense tissues, such as the lung [10].

There is a proposed mechanism where the negative pressure also causes injury similar to the positive pressure as we have seen in figure 2. In both pressures, there is excessive tension from the over expansion and speed of expansion during the compressive and decompressive forces [9].

It is also proposed that there are hemodynamical factors to injury. The hemodynamic perspective of injury shows that injury is caused by the instant increase in blood pressure [9]. The normal blood pressure is between 0.4 kPa and 0.8 kPa and the instantaneous increase in blood pressure can increase that by a 100 times, and such a sheer increase can damage the small blood vessels [9].

One mechanism is that the internal mechanical damage causes subtle biochemical changes, such as free radical-mediated oxidative stress, and that contributes to BOP injury [3]. Injuries from in-air explosions is associated with alveolar hemorrhage, interstitial edema, and rupture of the alveoli, which then impairs the ability to exchange gases at the one-cell interface of the alveoli. As a result of the pulmonary hemorrhage and alveolar edema by the blast, there is flooding of the alveoli that diminishes the gas exchange capability. Because there is a decrease in the amount of oxygen and carbon dioxide exchange, the partial pressure of oxygen (P_aO_2) decreases, although the carbon dioxide partial pressure (P_aCO_2) does not change [3]. This decrease in oxygen causes hypoxemia, and the degree of hypoxemia depends on the severity of the explosive. It was found by Elsayed that as the lung injury increased in severity, the P_aO_2 concomitantly decreased as well.

It was found that with decreasing P_aO_2 there was increasing lipid peroxidation and hemoglobin association. Further, the BOP causing lipid peroxidation was associated with antioxidant depletion and a disturbance of the ATP-dependent calcium transport across the cell membrane [3]. These biochemical changes correlated well with the BOP exposure. This effect of the lipid peroxidation continued for hours after the exposure, which could be due to pulmonary hemorrhage caused by the rupture of the capillaries and alveolar septa [3]. The lipid oxidation propagate for hours possibly due to hemoglobin, its oxidation products, such as metHb or oxoferrylHb, or its degradation products, heme or free iron, interacting with oxidants or antioxidants [3]. When the hemoglobin-derived products interact with oxidants or antioxidants, free-radical reactions occur, and that is thought to cause the propagation of injury for an extended period of time. Further, it was found that nitric oxide (NO) was found to interact with the hemoglobin mediated free-radical reactions and quenches them [3]. Elsayed found that NO exposure in rats increased following BOP exposure, which could mean that the NO functions to negate the hemoglobin-mediated reactions and indicate that injury occurred or to signal the repair process [3].

4. Causation

It must be noted that all of the research indicates that there is a *correlation* between the changes seen and the effects of an explosive. To confidently propose a causal relationship we must look at Bedford Hill criteria for causation. The reason it is difficult to determine the cause of the injuries is due to the fact that there are many different factors involved, which is why there are so many different proposed injury mechanisms. Due to ethical reasons, we cannot expose experimental subjects to a range of explosives and look at the resulting effects, and using animal models is not perfect either. As done by Bowen et al. 1968 and others, animal

models are scaled to human parameters and this may not accurately represent how the blast will actually affect an average 70 kilogram human [5]. With that said, we cannot know for a fact with absolute proof that the blast *causes* which pathology; "we can never *know* absolutely that exposure X causes disease Y" [11]. Therefore, the Bedford Hill criterion is a set of associations between the blast and injuries to decide the causal inference.

Criteria	Explanation
Strength	A strong association is more likely to be causal than a weak association.
Consistency	There's more confidence in a causal explanation for an association if the same answer is achieved in different situations.
Specificity	This criterion means that any exposure will give rise to only a single outcome.
Temporality	This is a necessary criterion for a causal association between exposure and outcome. The exposure must precede the outcome.
Dose Response Relationship	The likelihood of a causal association is increased if a dose-response curve can be demonstrated.
Plausibility	The causal association needs to be plausible in terms of the biology knowledge of the time.
Coherence	The cause and effect interpretation needs to fit the known knowledge, and should not contradict it.
Experiment	The strongest support for a causal relationship. Allows for control and randomization to find the true association between exposure and outcome.
Analogy	Clear-cut analogies can show the relationship for an otherwise weak association.

Table 3. The Bedford Hills criteria for a causal association [11].

The criteria seen in table 3 do not need to be satisfied to determine a causal relationship, but the more criteria is satisfied, the more confidence can be placed in the association. Some of the criteria are more important, or crucial, in determining a causal relationship than others. The temporality condition is the strongest case.

The first criteria is that strong associations were more likely to be causal than weak associations. In other words, the stronger the relationship between the blast and resulting

pathology, the less likely it will be due to a extraneous variable, such as a preexisting health condition that rendered the diver more likely to injury, and this pre-existing condition is what lead to the pathology [12]. This points out that a blast with a BOP of 100 PSI is more likely to *cause* a pathology than one at 30 PSI as shown by [2].

The second criteria is the consistency in the findings of the BOP injuries. This notes that different observations of the associations under different situations will lead to the same injuries. This presents a problem because as seen in Richmond et al., because he tested different animals at the same explosive parameters and observed different outcomes [13]. For example, using a 0.5 pound charge at a depth of 10 feet and 10 feet away from the animals resulted in different resulting pathologies. Some had no lung hemorrhages, others had petechial hemorrhaging, or intact or ruptured eardrums [13]. See appendix A for further differential results from exposives. There are more examples of the non-consistency in Richmond et al. 1973 in the outcome animal pathologies in response to the same explosive charge.

The third criteria is specificity of the cause of the injury. In an ideal case, the outcome would only have one cause, and to add credibility to a causal relationship to predict with one primary factor [12]. But as Lance explains, there is no definitive primary factor in predicting injury [4] [5]. Richmond suggests that impulse is a good predictor of injury because the total integral of the pressure vs. time waveform seen in figure 2 [13]. But others suggest that peak pressure can predict injury [3] [4]. It is difficult to determine a causal one-to-one relationship because there are different factors that lead to the same outcome. In this case, both impulse and peak pressure of the explosive are factors that both lead to lung pathologies.

The fourth criteria is temporality of the blast lung injury. For a cause-and-effect association to be established, the pathology must occur after the blast [12]. This association is simple to see because a blast lung pathologies cannot occur if there has not been an explosion yet.

The fifth criteria is dose response relationship that correlates the direct relationship between the risk factor (the explosive event) and the diver's resulting pathology [12]. This relationship of increasing exposure to explosive events leads to a greater susceptibility of injury as seen in [14]. [14] shows that sheep repeatedly exposed to 10 kilogram TNT explosions reduced their threshold value before injury.

The sixth criteria is the theoretical plausibility of the association between blasts and the injuries. The proposed mechanisms above questions the rational and theoretical basis for injury, and once that is accepted, then a causal relationship will be easier to argue [12].

The seventh criteria is the coherence of the cause and effect interpretation to the known body of knowledge. The direct association is clearest when it does not conflict with the known variables that are being studied and fits into our general understanding of the situation [12]. In this case, the association between injury and blasts are clear but which variable is causing the

injury? Peak pressure or impulse? What's the mechanism of injury? These questions are still actively being explored.

The eighth criteria is experimental evidence. This is the strongest indicator of a causal relationship between two variables. If manipulating the independent variable (the blast parameters) leads to different injuries or different severities of injuries then we can be more confident in the causal relationship. Furthermore in experiments, we can account for confounding or extraneous variables and maintain a more direct cause-and-effect relationship [11].

The ninth criteria is the analogy of an accepted phenomenon in one area and it can be applied to another [12]. These work to show the relationship between weak associations. This is directly important because we cannot perform explosive experiments on humans, but they have been performed on animals, as covered by [5]. Because the experiments have not been conducted on humans, they are analogous in nature because biology between humans and sheep are similar enough to make inferences about human injuries.

This criteria is meant to be viewed as a way to determine cause and effect and some of the criteria hold more weight than others, and is meant to aid in judgement, not replace it.

5. Blast Injuries

Since BOPs causes trauma to the body due to a physical force there can be multiple parts of the body that can be injured, but the parts of the body that can get injured are the lungs, ear, eye, abdomen, and brain [15] [16]. The reason the ear, lungs, and abdomen are injured is due to the air-tissue interface, and the proposed mechanisms of injury as covered above. The respiratory tract and tympanic membrane (TM) are the most vulnerable to primary blast injury [16]. Brain injuries are analogous to concussions or mild traumatic brain injuries from blows to the head, because the BOP wave exerts a force when it meets the body. All of the air-containing organs mentioned are all injured at similar blast exposures, so there will be multiple organ damages, which could result in multiple organ failure [1]. It is suggest by Elsayed that there is a free radical-mediated injury to the lungs and blood in rats, rabbits, and sheep after BOP exposure. In the case of primary injuries underwater, the extremities are safe from injury relative to the 4 areas above because they do not have as much gas-tissue interfaces. However depending on the situation, it can only be a part of the problem as there may be other, more dangerous factors such as extreme heat, chemicals, or radiation. Furthermore, there is no widely accepted criterion for injury severity as different authors have different criteria for extent of injury [2] [5] [9].

5.1 Respiratory Tract

The respiratory tract can be injured at high or low pressures. At low pressure petechial hemorrhaging can occur in the hypopharynx and larynx, but at low pressures it is unlikely that breathing will be compromised.

5.2 Tympanic Membrane

The TM is the most vulnerable as injury can happen even at 5 PSI [2], but it is also the least lethal and most easily protected relative to the other injury sites. Therefore, injury to an unprotected TM can signal to check for other areas for injury, but if the unprotected TM is still intact, then the blast was sufficiently weak enough to prevent damage. The rupture of the TM can lead to tinnitus (ringing or a sensation of noise [17]), pain, and hearing loss. A physical examination by a clinician will show blood in the external canal and evidence of perforation of the TM [16]. If there was a more severe blast, then vestibular damage could occur, which would then cause equilibrium problems.

5.3 Lungs

Blast trauma to the chest impacts the lungs. The resulting pulmonary contusion will look like blunt force trauma because there will be no rib fractures or chest wall injury [16]. The particular vulnerability of the lungs is due to the lungs being composed of more than 500 million alveoli, with tens of millions of connecting airways as we can see in figure 3 [1]. When the lungs are met with a BOP the air inside cannot flow fast enough through the airways due to the rapid motion of the chest, and a foam is made [1]. The physical properties of foam are different than air as the speed of sound of foam is 30 to 40 m/s, which is 1/10 of air and 1/5 of water [1]. This difference in the speed of sound causes the lung parenchyma to be crushed by the faster moving chest wall, if the BOP is great enough [1]. The resulting injury will produce a characteristic surface hemorrhage pattern as seen in the figure 4 below.

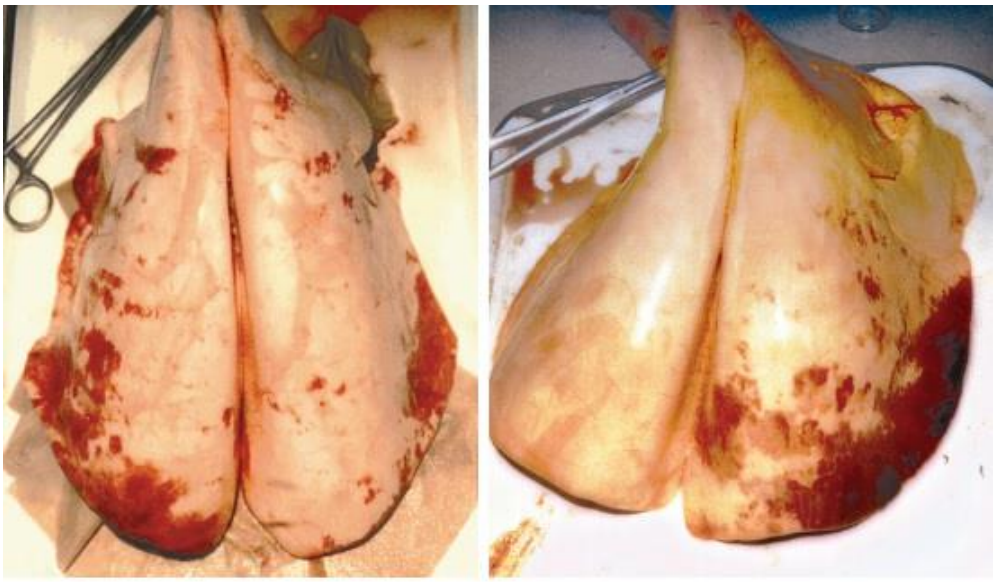

Figure 4. The resulting lung injury from a complex blast on the left and free field blast on the right [1].

The hemorrhaging of the lung tissue will predominately occur on the side facing the blast, because the brunt of the BOP will hit that side, but the rest of the lung can still be injured because the entire lung is enclosed in the rib cage [1].

As mentioned above, the lung parenchyma can be crushed by the chest wall if the BOP is great enough, and the resulting pathological changes exacerbates the survivor's condition. Damage to the parenchyma changes the ventilation to perfusion ratio, where ventilation is analogous to breathing and perfusion is the ability to deliver blood to the capillaries [18]. As we can see from the symptoms chart below in table 4, dyspnea or tachypnea changes the ventilation and the perfusion may be disrupted by the hemorrhaging or presence of gases in the body cavity. There is also increased shunting of the venous-arterial blood, which results in hypoxemia [18]. There are also hemodynamic changes after a BOP. It has been shown that arterial blood pressure decreases and there is an elevation on the central venous pressure [18]. This could be due to the fact that the alveolar destruction after the BOP reduces the diffusion ability of the gases that results in hypoxemia and blood is built up in the veins trying to get oxygenated but either cannot or is blocked by gases in the chest cavity, and that accumulation of the deoxygenated blood causes a lower arterial blood flow rate. The arterial blood pressure decreases because as time progresses there will be less and less oxygenated blood, and the blood is being pooled in the veins trying to get re-oxygenated.

The patient complaints will be about pain, hemoptysis (expulsion of blood from a part of the respiratory tract [17]), and chest tightness [16]. There are various symptoms of pulmonary blast injury, such as retrosternal pain, dyspnea, bradycardia, tachypnea, cyanosis, hypotension, haemoptysis, and rales or rhonchi on exhalation [2] [7]. Furthermore, the survivor may have sudden, acute abdominal pain, nausea that may be caused due to injury to the ear's equilibrium ability, vomiting with or without blood, or the sensation of an electric current [18]. The explanation for each symptom is presented in table 4. Hypotension may seem odd at first as a symptom, but given the physiological state of the survivor it may occur; it may be brought on by blood loss, compression of the blood vessels and heart by pneumothorax, the effects of an air embolism, or due to vagal effects [2].

Pulmonary Lung Blast Injury Symptoms

Symptoms	Description
Retrosternal pain	Pain behind the sternum.
Dyspnea	Difficult or labored breathing.
Bradycardia	Slow heart action.
Tachypnea	Increased rate of breathing.

Cyanosis	Bluish discoloration due to lack of oxygen.
Hypotension	Abnormally low blood pressure.
Hemoptysis	Release of blood from part of the respiratory tract.
Rales/Rhonchi	Abnormal sounds made with normal breathing.
Hypoxemia	Low oxygen concentration in the blood.

Table 4. Presented symptoms after primary lung blast injury [7].

Wright performed experiments with volunteer divers at sub-lethal blasts and recorded their sensations. At the lower charge weights and intensities, there were intense sounds and a blow to the chest. When the impulse raises higher than 95 psi-msec, then there was paralysis of the body and progressively more severe sensations. Table 5 below shows the varying outcomes of the divers as the range progressively decreases.

Range	**Sensations**	**Estimated Shock Levels**
Meters	Subjective Comment	Impulse (psi-msec)
33.5	Sound of intense bang.	75
30.5	Intense bang. Mild blow on chest.	85
27.4	Severe blow on chest.	95
24.4	Blow to head and torso. Body shaken. Brief paralysis of arms and legs.	105
22.9	Violent blow. Brief paralysis of limbs. Substernal pain for 1/2 to 1 hour.	110

21.3	Violent blow. Temporary paralysis of limbs. Substernal pain lasting several hours. Aural damage. Tongue lacerated. Mask blown off. Mild concussion	115

Table 5. The subjective comments of divers exposed to 5lb of TNT at varying distances [19].

It has also been found that the injuries can be sorted into either perforating or non-perforating injuries. If there is a non-perforating injury, then the body will have a whole body response with increasing leukocytes, rectal bleeding, and pyrexia (abnormal elevation of body temperature) [18]. If it is a perforation injury then there will be a variety of symptoms, such as tenderness, rectal bleeding, distention (swollen), increasing pain, and many other symptoms found in [18]. The resulting pathologies are worse when the lung laceration is present as gases end up inside the chest cavity in different location and can worse as seen in figure 5. The non-laceration injuries are not as severe as the laceration injuries, but they are dangerous as hemorrhage and pulmonary edema is still a possibility [18].

There may be the immediate complaints after the blast but the blast lung condition may develop over 24-48 hours, which will make it difficult to diagnose with x-ray or physical examination early on as the blast lung condition may progress further in time. In addition, the pulmonary damage can be either acute or delayed. The acute pulmonary damage can be due to alveolar disruption due to the BOP therefore causing hemorrhage. Or, it can be acute due to pneumomediastinum or pneumothorax [7]. The pulmonary damage can be delayed if there are multiple small pulmonary emboli or pulmonary edema is present, which will cause further complications as time progresses [7]. For a clinician it may be difficult to determine the distresses because of all the different etiology, and the only way to make a determination is to take arterial blood gas measurements periodically [16].

The evidence of PBI to the lungs is where the patient is using accessory muscles to breath, tachypnea (fast breathing), pulmonary consolidation, and other signs of respiratory distress. If there is pulmonary consolidation, then it can progress to either hemothorax or contusion. The contusion may not be readily apparent immediately after the blast, but it can develop over a period of 24-48 hours. The contusion will look like local or diffuse infiltrate, which is fluid coming into the alveolar structures. The only reliable way to detect a PBI in the lungs early on is with an x-ray, which will show the characteristic butterfly shape [16]. X-rays can show a range of different blast pathologies; it can show: pneumomediastinum, subcutaneous emphysema, pulmonary interstitial emphysema, pleural blebs, hemothorax, and pneuomothorax [16]. Lung PBI is also associated with alveolar hemorrhage, pulmonary hemorrhage, and interstitial edema. Pulmonary hemorrhage then goes on to cause alveolar edema, and then alveolar flooding, which impairs gas exchange [3]. See table 6 for a description of the pathologies.

Pathology	**Description**

Pneumomediastinum	An abnormal state where there is gas in the mediastinum.
Subcutaneous emphysema	Air-filled expansion in the subcutaneous tissue, which can lead to rupture and loss of pulmonary elasticity.
Pulmonary interstitial emphysema	Air-filled expansion in the internal tissue, which can lead to rupture of the alveoli.
Pleural blebs	A small blister in the pleura of the lungs.
Hemothorax	Blood in the pleural cavity.
Pneumothorax	Air or another gas is in the pleural cavity.
Pneumopericardium	Abnormal state where there is gas in the pericardium.
Pneumoretroperitoneum	Presence of gas In the retroperitoneal space.
Pneumoperitoneum	Abnormal state where there is gas in the peritoneal cavity.
Contusion	Injury to a tissue without laceration.
Interstitial edema	An excessive accumulation of serous fluid in the interstital tissue.
Pulmonary edema	An abnormal accumulation of fluid in the lungs.
Alveolar hemorrhage	Acute blooding from the alveoli.
Pulmonary hemorrhage	Acute bleeding from the lung, such as the upper respiratory tract or trachea.
Pulmonary embolism	An obstruction of the pulmonary artery.

Table 6. Description of different PBI.

Pulmonary hemorrhage is concentrated at the junction between the alveolar tissue and the bronchioles, because the shearing force caused by the BOP occurs between the air-filled spaced and the tissues, and everything accelerates at different rates [7]. Pulmonary hemorrhage can be located either in the pleural or subpleural space and it can be diffuse throughout the parenchyma or have multiple points of damage, and this can be seen in the figure below [8].

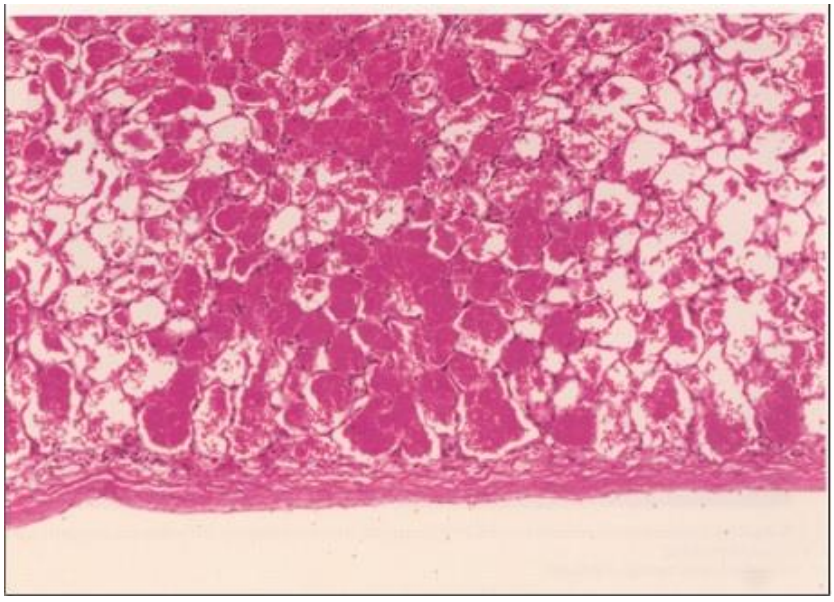

Figure 5. A histology of a hemorrhaged tissue which shows it at the pleural surface of the sheep's lung [8].

The difficulty in diagnosing the blast injuries is due to two factors. The first one is that different pathologies can progress over the period of 24-48 hours. The other factor is that all of the pathologies are related and can lead to another, compounding the injury severity of the blast survivor [2].

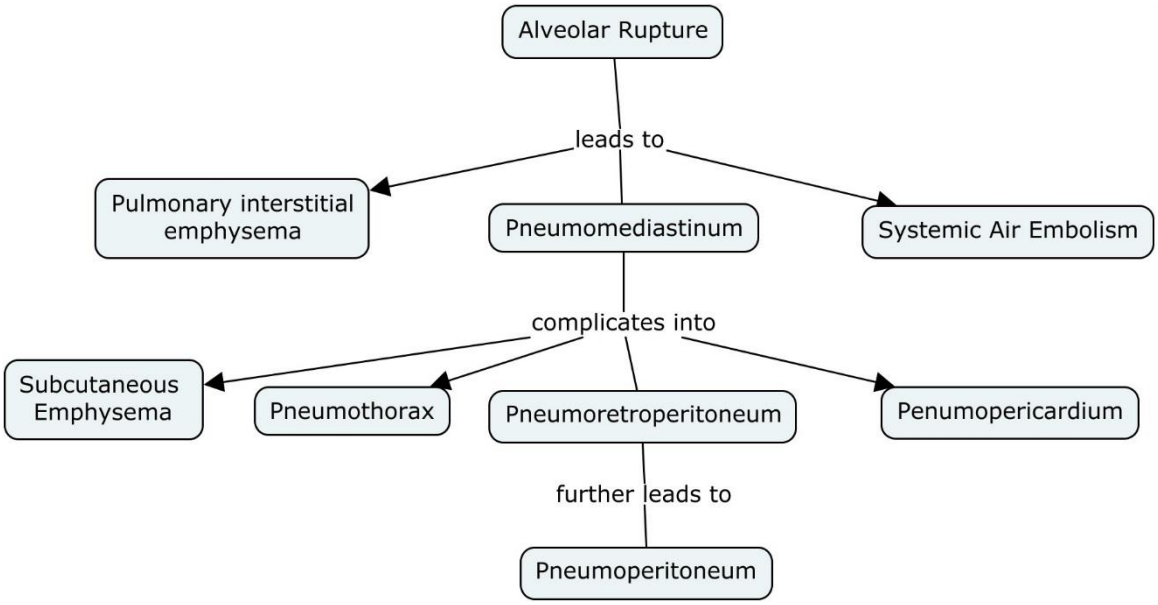

Figure 6. The possible complications and pathologies possible from alveolar rupture [2].

The resulting pathologies shown in figure 5 all stem from the fact there is gas in a space in the lungs where it is not meant to be, either in the thorax, pericardium, retroperitoneal space, or peritoneal cavity.

There is current research into how to rate underwater blast injuries as seen in figure 5. The injury severities were split into seven categories of increasing severity from rank 0 to 6. The symptomatic scale is compared with the Abbreviated Injury Scale (AIS) for comparison. The symptomatic scale was derived by looking at many case studies [5].

Pulmonary Injury Scale			
Rating	**Severity**	**Symptomatic Scale**	**AIS Pulmonary Scale**
0	None		
1	Minor	Some x-ray evidence by asymptomatic	Contusion (unilateral < 1 lobe)
2	Moderate	Coughing; or shallow breathing	Contusion (unilateral whole lob); or laceration (simple pneumothorax)
3	Serious	Mild hemoptysis	Contusion (unilateral > 1 lobe); or laceration (persistent > 72 hours,

			airleak from distal airway); or hematoma
4	Severe	Severe symptoms, treatable by modern medical practice, possible recovery or fatality	Laceration (major airway leak); or hematoma; or vascular
5	Critical	Severe cyanosis; or severe hemoptysis; likely untreatable by modern medical practice; typically fatal	Vascular (hilar vessel disruption); or multilob lung laceration with tension pneumothorax
6	Maximum	Fatality within 30 minutes of exposure	Maximal (currently untreatable)

Table 7. An injury rating scale for pulmonary primary injuries from underwater blasts [5].

5.4 Protection

It has been mentioned that the TM is the most vulnerable to BOP injury but is also the most easily protected thanks to ear plugs or muffs. The same does not translate to wear a life vest or jacket to protect the chest. Wearing a protective vest will offer a false sense of security because it will served as a reflective surface, which will exacerbate the BOP waves going into the body [2] [9]. The reason why the vest is reflective is due to the difference in impedance between the water and vest. Water and human tissue both have relatively the same impedance, but when the impedance changes against the vest, the wave is reflected back. For instance, a Kevlar vest was used, but it did not confer any protective advantages and instead increased injury [9]. However, materials are being research which reduces the intensity of the BOP before it reaches the wearer. A foamy, porous nickel media was used as a protective material because it reduced the peak pressure, extended the rise time, and shortened the duration of the positive pressure [9]. See figure 2 for the pressure vs. time waveform.

6. Diagnosis

The most reliable method of detecting blast lung is with an x-ray, which will show fluid accumulation, distention of the lungs, or gas accumulation in any cavity of the chest [18]. In addition, taking arterial blood to check oxygen concentration will give a good indicator as the survivor's condition progresses over the 24-48 hour time window. In an x-ray, the blast lung will present itself as looking like a "butterfly" pattern [2] [16]. See figure 7 below. The pattern emerges hours after the explosive event and will subside after a week [2].

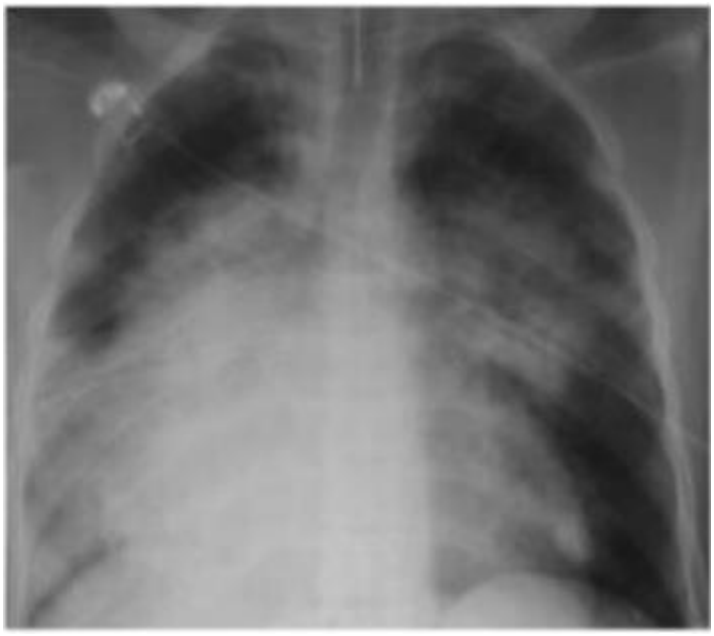

Figure 7. The chest x-ray of a blast lung. Note the opacity on the left [2].

In addition, a CT scan should be done of the head, chest, and abdomen. A CT scan will shows the blood vessels and the soft tissues, can show the distension due to air in the chest cavity and hemorrhaging from damage to blood vessels.

Pulse oximetry can show the concentration of oxygen in the blood and should be used as an adjunct to the other diagnostic studies. Because the survivor will likely progress as over a period of 24-48 hours, it will be valuable to the clinician to see the changes in blood oxygen concentration, and could lead to the etiology of the pathology of concern. Furthermore, multiple hemoglobin concentration determinations will need to be made because it is an indicator of blood loss as well as to determine the mechanism of injury [2]. As one mechanism involves the free radical-mediated breakdown of hemoglobin and its constituents [3]. Finally, because BOP victims have similar responses to trauma victims, the survivors should have baseline blood counts, hematocrit, hemoglobin, and crossmatching if blood transfusion is needed [2].

7. Treatment

The management given to an underwater BOP survivor will be equal to a patient with total body trauma [18]. The treatment will depend on the severity of blast lung injury. Gas exchange should be supported by correcting the effects of BOP or barotrauma. The clinician can make pneumothorax worse by providing positive ventilation because often an acute gas embolism can arise [2]. If the survivor has mild respiratory distress then additional oxygen can be given through a nasal cannula; however, if there is severe distress then an endotracheal tube needs to be placed [2]. If the patient is found to have an air embolism, then the survivor should be given hyperbaric oxygenation.

8. Conclusion

The effects of BOP on a human body is devastating as the effects are analogous to trauma because of the physics of the wave, and the effects are even more dangerous underwater because of the properties of water differ to that of air. The difficulty in treating those with “blast lung” is due to the many different pathologies and symptoms that may be presented, and the condition of the survivor may change over the course of 24-48 hours. The treatments require a proactive approach where the survivor needs to receive a chest x-ray as soon as possible and arterial blood measurements periodically to determine the extent of injury and the progression.

9. Future Work

The research going forward needs to provide a method of evaluating the injury criteria from various explosives. Further, the mechanism of injury from primary blast needs to be better elucidated to provide a better idea of the physiological changes to the body after exposure. A protective vest that has the right impedance properties to diminish the strength of the incoming BOP and reduce its impact on the wearer.

10. References

[1] J. J. Stuhmiler, "Blast Injury: Translating Research into Operational Medicine," Borden Institute.

[2] C. Stewart, "Blast Injuries: "True Weapons of Mass Destruction"," University of Oklahoma, Tulsa, 2010.

[3] N. M. Elsayed, "Toxicology of blast overpressure," *Toxicology,* vol. 121, pp. 1-15, 1997.

[4] T. Josey, "Investigation of Blast Load Characteristics on Lung Injury," University of Waterloo, 2010.

[5] e. a. Rachel M. Lance, "Human Injury Criteria for Underwater Blasts," University of California, San Diego, 2015.

[6] M. H. e. al., "Blast Injury of the Chest," *Clinical Radiology,* vol. 20, pp. 362-370, 1969.

[7] e. a. Edward Cudhay, "The Effects of Underwater Blast on Divers," Naval Submarine Medical Research Laboratory, Groton, 2001.

[8] D. D. Sharpnack, "The Pathology of Primary Blast Injury".

[9] Y.-G. Z. Yan Zhao, "The past and present of blast injury research in China," *Chinese Journal of Traumatology,* vol. 18, pp. 194-200, 2015.

[10] D. C. C. e. al., "Blast Injuries: Mechanics and Wounding Patterns," *Journal of Surgical Orthopaedic Advances,* 2010.

[11] R. M. L. e. al., "Association of causation: evaluating links between "environment and disease"," The Royal Society of Medicine Press.

[12] "How do Epidemiologists Determine Causality?," University of Southern Alabama.

[13] D. R. e. a. Richmond, "Far-Field in Underwater-Blast Injuries Produced by Small Charges," Lovelace Foundation for MEdical Education and Research, 1973.

[14] Z. e. a. Yang, "Biological Effects of Weak Blast Waves and Safety Limits for Internal Organ Injury in the Human Body," *The Journal of Trauma and Acute Care Surgery,* vol. 40, no. 3S, pp. 81-84, 1996.

[15] C. f. D. Control, "Explosions and Blast Injuries".

[16] U. S. D. o. Defense, "Clinical Presentation of Primary Blast Injury," in *Emergency War Surgery NATO Handbook: Part I: Types of Wounds and Injuries*.

[17] "MedlinePlus," U.S. National Library of Medicine, [Online]. Available: https://www.nlm.nih.gov/medlineplus/mplusdictionary.html.

[18] J. T. e. a. Yelverton, "A Review of the Treatment of Underwater Blast Injuries," Lovelace Foundation for Medical Education and Research, Albuquerque, 1976.

[19] R. K. Wright, "The effects of underwater explosions on shallow water divers submerged in 100 ft of water," 1950.

Appendix A

Charge Wt. (lbs)	**Charge depth (ft)**	**Animal Depth (ft)**	**Range (ft) [Based on slant range]**	**Pressure (psi)**	**Impulse (psi-msec)**	**Body Wt. (kg)**	**Effects**
0.5	10	2	93	103	5.9	40	none
0.5	10	2	93	103	5.9	32	none
0.5	10	2	93	103	5.9	36	petechial lung hemorrhage
0.5	10	10	100	93	12.1	19	None
0.5	10	10	100	93	12.1	18	petechial lung hemorrhage

0.5	10	10	100	93	12.1	20	petechial lung hemorrhage
0.5	10	10	100	111	11.7	34	petechial lung hemorrhage
0.5	10	10	100	111	11.7	37	petechial lung hemorrhage
0.5	10	10	100	111	11.7	36	petechial lung hemorrhage
0.5	10	10	100	92	11.5	38	none
0.5	10	10	100	92	11.5	41	lung discolored
0.5	10	10	100	92	11.5	40	lung discolored
0.5	10	10	100	97	11.2	50	petechial lung hemorrhage
0.5	10	10	100	97	11.2	50	petechial lung hemorrhage
0.5	10	10	100	97	11.2	48	petechial lung hemorrhage
1	10	2	33	436	44.4	37	slight lung hemorrhage
1	10	2	33	436	44.4	41	slight lung hemorrhage
1	10	2	33	436	44.4	34	extensive lung hemorrhaging
1	10	2	54	259	21.9	36	petechial lung hemorrhage
1	10	2	54	259	21.9	34	petechial lung hemorrhage
1	10	2	54	259	21.9	34	petechial lung hemorrhage
1	10	2	83	150	9.7	43	petechial lung hemorrhage
1	10	2	83	150	9.7	41	petechial lung hemorrhage
1	10	2	83	150	9.7	33	none
1	10	10	48	269	45.5	42	slight lung hemorrhage
1	10	10	48	269	45.5	41	slight lung hemorrhage
1	10	10	48	269	45.5	40	slight lung hemorrhage
1	10	10	84	153	22.8	41	petechial lung hemorrhage
1	10	10	84	153	22.8	42	slight lung hemorrhage
1	10	10	84	153	22.8	44	none

1	10	10	84	166	21.7	43	petechial lung hemorrhage
1	10	10	84	166	21.7	41	petechial lung hemorrhage
1	10	10	84	166	21.7	37	petechial lung hemorrhage
0.5	10	1	110	89	3.1	41	None
0.5	10	1	110	89	3.1	47	None
0.5	10	1	110	89	3.1	42	None
0.5	10	1	110	88	3	36	None
0.5	10	1	110	88	3	33	None
0.5	10	1	110	88	3	36	None
0.5	10	1	93	103	5.9	40	None
0.5	10	1	93	103	5.9	32	None
0.5	10	1	93	103	5.9	36	petechial lung hemorrhage
1	10	1	26	478	41.5	20	slight lung hemorrhage
1	10	1	26	478	41.5	6	slight lung hemorrhage
1	10	1	26	563	40.6	41	petechial lung hemorrhage
1	10	1	26	563	40.6	37	None
1	10	1	26	563	40.6	42	None
1	10	1	30	481	34.7	38	None
1	10	1	30	481	34.7	38	None
1	10	1	30	481	34.7	41	None
1	10	1	34	389	29.7	5.6	None
1	10	1	34	389	29.7	21	None
1	10	1	34	407	27.5	41	None
1	10	1	34	407	27.5	36	None
1	10	1	34	407	27.5	37	None
1	10	1	38	381	23.9	38	None
1	10	1	38	381	23.9	39	None
1	10	1	38	381	23.9	37	None

1	10	1	46	306	18.2	40	None
1	10	1	46	306	18.2	36	None
1	10	1	46	306	18.2	17	None
1	10	1	46	274	18.2	18	None
1	10	1	46	274	18.2	4	None
1	10	1	56	246	14.2	21	None
1	10	1	56	246	14.2	21	None
1	10	1	56	246	14.2	24	None
1	10	1	56	253	14	20	None
1	10	1	56	253	14	19	None
1	10	1	56	253	14	20	None
1	10	1	56	218	11.7	14	None
1	10	1	56	218	11.7	14	None
1	10	1	56	218	11.7	14	None
1	10	1	56	200	11	21	None
1	10	1	56	200	11	5.9	None
1	10	1	56	207	10.4	46	None
1	10	1	56	207	10.4	46	None
1	10	1	56	207	10.4	43	None
1	10	1	78	160	6.6	20	None
1	10	1	78	160	6.6	15	None
1	10	1	78	160	6.6	19	None
1	10	1	78	156	6.6	36	None
1	10	1	78	156	6.6	37	None
1	10	1	78	156	6.6	39	None
1	10	1	78	157	6.2	21	None
1	10	1	78	157	6.2	4.6	None
1	10	1	78	136	5	19	None
1	10	1	78	136	5	14	None
1	10	1	78	136	5	14	None
1	10	1	110	104	4.2	16	None
1	10	1	110	104	4.2	3.4	None
1	10	1	110	104	4.2	36	None
1	10	1	110	104	4.2	39	None
1	10	1	110	104	4.2	36	None
1	10	1	130	111	3.2	46	None
1	10	1	130	111	3.2	45	None
1	10	1	130	111	3.2	47	None
1	10	1	130	108	3	46	None

1	10	1	130	108	3	45	None
1	10	1	130	108	3	45	None
1	10	1	130	106	2.3	49	None
1	10	1	130	106	2.3	45	None
1	10	1	130	106	2.3	50	None
1	10	1	130	110	1.8	50	None
1	10	1	130	110	1.8	50	None
1	10	1	130	110	1.8	47	None
3	10	1	36	538	40.3	36	slight lung hemorrhage
3	10	1	36	538	40.3	34	slight lung hemorrhage
3	10	1	36	538	40.3	36	petechial lung hemorrhage
3	10	1	61	299	15.9	41	None
3	10	1	61	299	15.9	45	None
3	10	1	61	299	15.9	43	None
3	10	1	72	248	11.8	39	None
3	10	1	72	248	11.8	42	None
3	10	1	72	248	11.8	41	None
3	10	1	97	191	7.4	39	None
3	10	1	97	191	7.4	39	None
3	10	1	97	191	7.4	39	None
8	10	1	52	556	33.2	36	slight lung hemorrhage
8	10	1	52	556	33.2	37	slight lung hemorrhage
8	10	1	52	556	33.2	36	slight lung hemorrhage
8	10	1	60	477	26.4	34	None
8	10	1	60	477	26.4	34	None
8	10	1	60	477	26.4	37	None

1	10	1	16	987	99.6	43	extensive lung hemorrhaging
1	10	1	16	987	99.6	46	slight lung hemorrhage
1	10	1	16	987	99.6	45	extensive lung hemorrhaging
1	10	1	26	588	50.6	36	petechial lung hemorrhage
1	10	1	26	588	50.6	34	slight lung hemorrhage
1	10	1	26	588	50.6	16	slight lung hemorrhage
1	10	0.5	13	1147	132.6	43	extensive lung hemorrhaging
1	10	0.5	13	1147	132.6	49	slight lung hemorrhage
1	10	0.5	13	1147	132.6	43	extensive lung hemorrhaging
1	10	0.5	13	1224	135.4	48	extensive lung hemorrhaging
1	10	0.5	13	1224	135.4	50	extensive lung hemorrhaging
1	10	0.5	13	1224	135.4	49	extensive lung hemorrhaging