Detecting Violent Explosive Neurologic Trauma in American Service Members

by
Jeffrey R. Crowell

A THESIS

submitted to

Oregon State University

Honors College

in partial fulfillment of
the requirements for the
degree of

Honors Baccalaureate of Science in Public Health
(Honors Scholar)

Presented July 1, 2020
Commencement June 2021

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Jeffrey R. Crowell for the degree of Honors Baccalaureate of Science in Public Health presented on July 1, 2020. Title: Detecting Violent Explosive Neurologic Trauma in American Service Members.

Abstract approved:_____

Karl Mundorff

The emergence of high explosives on the battlefield can be defined both literally and figuratively as a seismic event in the landscape of modern warfare. High explosives first came into common use in war in 1914. By the end of World War I just four years later, more than 2.5 billion tons artillery and mortar shells had been detonated. Many veterans were repatriated home physically unscathed but were nonetheless left with an assortment of inexplicable psychological and cognitive complications. Originally referred to as “shell shock”, Post-Traumatic Stress Disorder (PTSD) is the current nomenclature used to explain the myriad of psychological complications arising from combat exposure. Recent studies have shown that these symptoms may be more physiological than they are psychological. Proximal exposure to explosive atmospheric overpressure waves have been shown to cause astroglial scarring in vital regions of the brain often associated with symptoms of PTSD. With the highest ratio of American casualties from explosives since WWI, violent explosive neurologic trauma has once again emerged as the signature injury of an armed conflict in the Global War on Terrorism. Modern preliminary diagnostic protocols have insufficient specificity to accurately distinguish patients exposed to blast shockwaves with traumatic brain injury versus those solely with psychological trauma. Consequently, tens of thousands of American veterans have received misdiagnosis or insufficient treatment of permanent post-concussive syndrome following explosive blast wave exposure.

Key Words: traumatic brain injury, diffuse axonal injury, primary blast injury, explosive neurologic trauma, Global War on Terrorism, counterinsurgency, post-traumatic stress disorder, post-concussion syndrome

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I understand that my project will become part of the permanent collection of Oregon State University, Honors College. My signature below authorizes release of my project to any reader upon request.

_____

Jeffrey R. Crowell, Author

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Acknowledgements

"*Although no sculptured marble should rise to their memory, nor engraved stone bear record of their deeds, yet will their remembrance be as lasting as the land that they honored*."

-Daniel Webster

Dedicated to SPC John Pelham, Beaverton, OR
2nd Battalion, 3rd SFG (Airborne), United States Army
Killed in Action 12 Feb 2014, Kapisa Province, Afghanistan
And to all who gave their lives in the pursuit of peace in the Global War on Terrorism

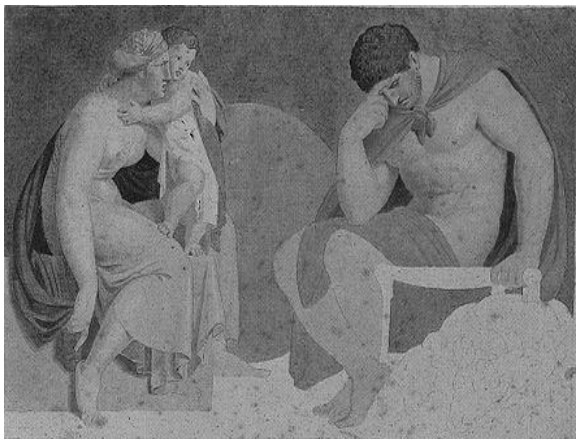
Part I:
The Problem

Background

Traumatic brain injuries have been a signature wound of combat since the earliest accounts of man waging war against one another. While the mechanism of injury has evolved with military technologies of the time periods, head injuries have always been and always will be an unavoidable outcome of war. One of the first casualties in Homer's mythical epic *The Iliad* (written in 762 B.C.) was due to a traumatic brain injury [1]:

> Antilochus, throwing first, struck the horn of the horse head helmet. And the bronze spear point fixed in his forehead and drove inward through the bone, and a mist of darkness clouded his eyes; headlong as a tower he fell...

The Iliad not only describes the nature of ancient warfare, but also offers a detailed perspective on the outcomes of combat injuries even after the Trojan War ended. The epic describes Greek warrior Ajax the Great's plights with the long-term effects of war after returning home. He suffered from hallucinations and his erratic behavior terrorized his family. He slaughtered a flock of sheep, believing they were Greek soldiers. Ultimately, he took his own life by falling on his own sword. Almost all brain injuries in war up until the 20th century were the result of blunt force trauma to the head from impact weapons such as spears, clubs, and swords. However, since the creation of dynamite by Alfred Nobel and the large-scale implementation of trinitrotoluene (TNT) in the First World War, the primary source of head trauma is not from impact but from atmosphere.



Sorrows of Ajax The Greater (Asmus Jacob, 1791) depicting the PTSD of Ajax the Great following the War of Troy.[2]

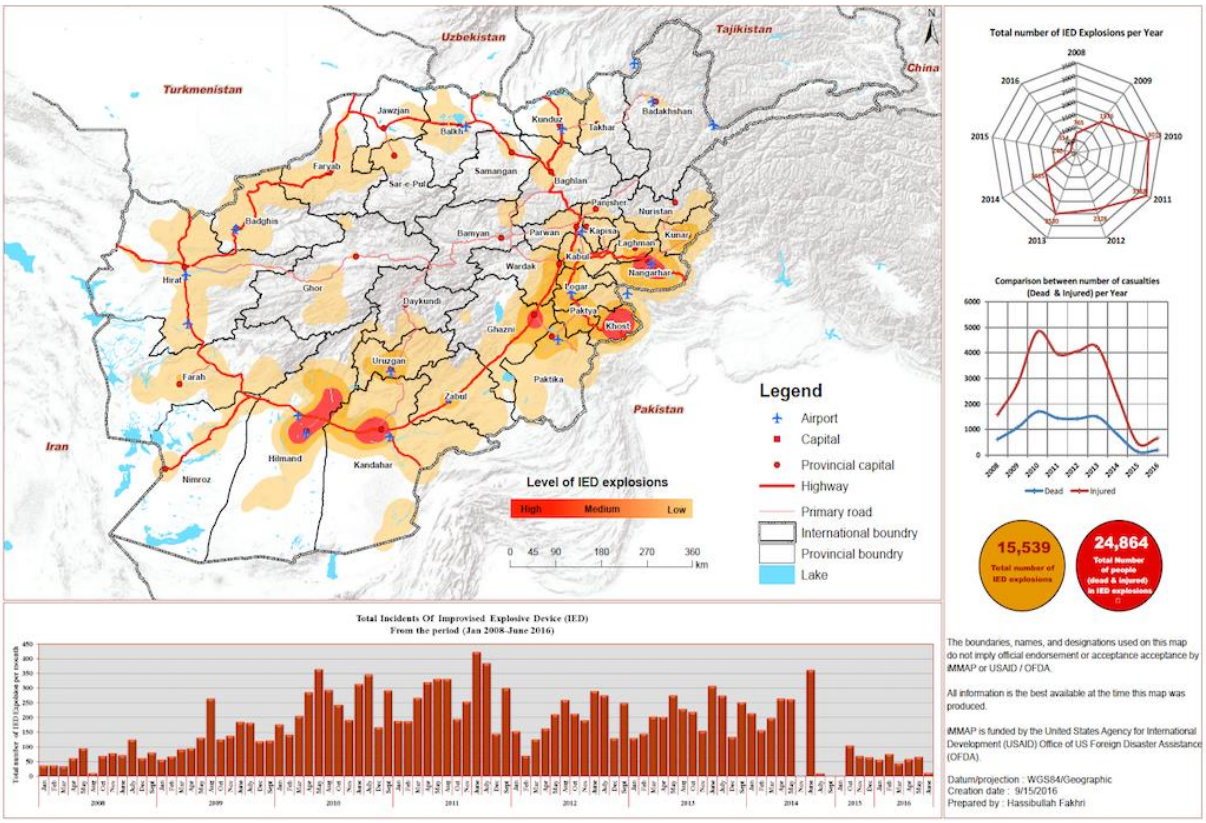
A Senegalese Tirailluer with the French Army charges into a German artillery barrage at the Battle of the Somme, 1916. [3]

High explosive ordnance quickly became the weapon of choice for opposing forces during WWI, and by the end of the war in 1918, over 2,500,000,000 metric tons of TNT were expended.[4] Of the 20,000,000+ soldiers who were killed or wounded, nearly 60% resulted from some type of explosive detonation.[5] Millions of veterans were repatriated home physically unharmed but were nonetheless afflicted with an assortment of inexplicable psychological and cognitive abnormalities. The phrase "shell-shock" was originally used to describe these symptoms in absence of any visible physical trauma. Like many of the timeless lessons of war early civilizations may offer, suicide is a fate all too similar that many veterans still face today. Current figures provided by

A British soldier stands amid thousands of spent artillery shells after the Battle of the Somme, 1916. [6]

the Department of Veterans Affairs estimate that approximately twenty veterans and one active service member will kill themselves every day. The annual loss to veteran suicide is higher than the combined number of U.S. combat-related fatalities in every military engagement since September 11th, 2001.[7][8]

The use of explosives by enemy forces has accounted for approximately 65% of all American service members killed in action or wounded in action in the last twenty years.[9] The Department of Defense estimates that traumatic brain injuries (TBIs) make up approximately 25% of all combat wounds during the Global War on Terror, nearly double the rate during the Vietnam War.[10][11] The United States military has been actively engaged in combat operations in at least nine countries since October 2001 (Afghanistan, Pakistan, Iraq, Syria, Yemen, Kenya, Somalia, Niger, and Libya).[12] During this period, more than 2.7 million American service



Incidence of IED Attacks in Afghanistan, 2008-2016. [13]

members have been deployed to combat theaters; many of these personnel deployed several times to multiple theaters.[14] 5,464 Americans have been killed in action and over 53,000 have been combat wounded.[15] The signature weapon of choice overwhelmingly used by insurgent forces in the Middle East has been improvised explosive devices (IEDs). IEDs are extremely popular with insurgent fighters because they are outmanned and outgunned by American and coalition forces. IEDs can be made from inexpensive and commonplace items, are extremely difficult to detect, and are capable of defeating even the most modern up-armored personnel carriers. The near endless supply of synthesizable ordnance left behind by the Soviet Union following their withdrawal from Afghanistan in 1988 meant that groups like Al-Qaeda and the Taliban possessed considerable caches of conventional and explosive ordnance. These terrorist groups have used them to wage civil war and genocide on ethnic Pashto minority groups since the 1990s. [17] Saddam Hussein's Iraqi military cached explosive munitions throughout major cities shortly after the April 2003 invasion, which were subsequently utilized by paramilitary insurgents throughout Operation Iraqi Freedom (2003-2011) and Operation Inherent Resolve (2014-Present).

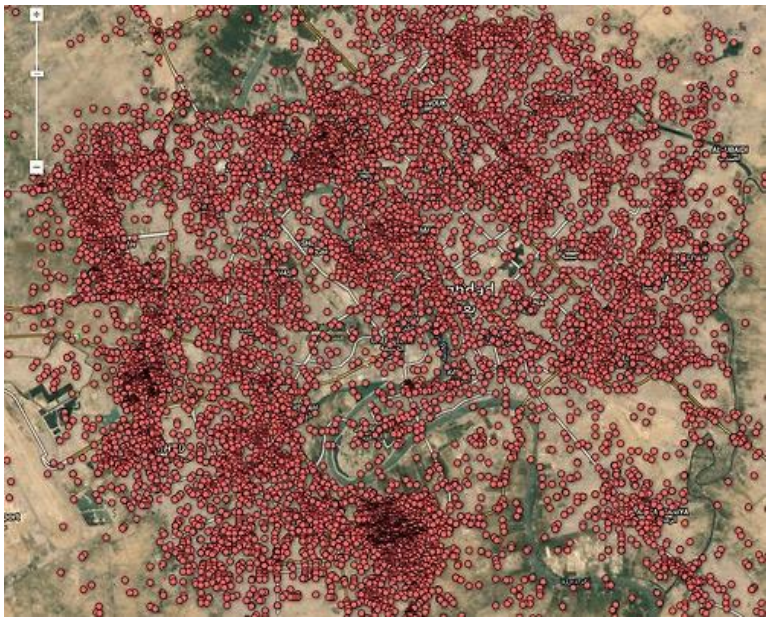
An American EOD specialist waves to a hidden observing insurgent who detonated an IED by remote control. [16]

This epidemic stems from a variety of geopolitical factors dating back several decades. The ultimate causation for the current casualty statistics can be attributed to the Islamic sociopolitical revolutions which occurred during the late 1970s. The final catalyst involved the

Iranian Revolution in early 1979 and Afghanistan's Saur Revolution in the following months.[18][19] The Saur Revolution directly spurred the Soviet Union's invasion of Afghanistan, followed by the invasion of Iran by Saddam Hussein's Iraq less than a year later. [20][21] The Iran-Iraq war would last almost a decade and would serve as a precursor to Iraq's subsequent invasion of the micro-nation of Kuwait and Operation Desert Storm in 1991.[22] The Soviet invasion of Afghanistan led to an occupation that would span a similar timeframe as the Iran-Iraq conflict, creating the Mujahedeen insurgency partially led by Osama Bin Laden.[23] Ironically, the Mujahedeen, who would later on metastasize into the Taliban and Al-Qaeda, received financial and materiel support from the U.S. government. They also perfected the guerilla tactics used in fighting against the Soviets that are currently being used against American soldiers today.[24][25][26] The economy of Afghanistan is largely agricultural, leading to a plethora of available fertilizers synthesizable into inexpensive improvised munitions.[27][28] Since the insurgents are outmanned and ill-equipped for direct contact, they cannot fight against American troops in open combat. Constant conflict for decades in a region rife with readily accessible ordnance and an enemy forced to fight asymmetrically has created the conditions which have led to the resurgence of TBI as the signature injury of conflict.

Kevlar Vests Taped to HMMWV Doors, Invasion of Iraq, 2003 (*Left*). Up-armored HMMWV next to a Mine-Resistant Ambush Protected (MRAP) Transport, Afghanistan 2009 (*Right).*[29][30]

Beginning in the late 2000s, as the war in Afghanistan deteriorated and the Iraqi insurgency peaked, the Department of Defense began to take note of the sudden increase in the incidence of blast TBI in returning service members and began allocating funding towards researching the problem. At the same time, the Department was scrambling to procure enough armor supplements and vehicle upgrades to protect its troops. Before the Global War on Terrorism, the United States military had never fought a dichotomy of asymmetry of this magnitude; even in Vietnam, the vast majority of combat casualties resulted from direct enemy contact .[31] The logistical challenges posed by outfitting an entire mechanized force in theater was estimated to take years to complete; it was an upgrade that would come too late for hundreds of U.S. and allied personnel. From 2007 to 2012, the United States averaged over 600 service members per year killed in action with the vast majority dying from IEDs.[32] The subsequent defensive acquisitions amounted to a complete overhaul to the transportation and engineering branches of the armed forces. The expansion of explosive ordnance disposal (EOD) technician units is one example.[34] At the same time the Defense Advanced Research Projects Agency (DARPA) and the National Institute of Health (NIH) co-founded the program for the Prevention of Neurologic Trauma (PREVENT).[35] The PREVENT program is the Department of Defense's main procurement



Map of Baghdad, Iraq (2003-2011). 1 Dot = 1 IED Attack [33]

asset for developing technology for the detection and diagnosis of underlying blast TBI and has been continuously funded since 2011. While the program has received nearly $100 million dollars in federal funding during the last decade, the only viable solution so far to emerge from the prototype stage is an electronic sensor that utilizes a microphone and Bluetooth capabilities to record and analyze blast waveforms to estimate the sustained force.[36] The device requires service members to wear multiple sensors at a time Each device must be continuously charged and repaired. The device was piloted with troops in Afghanistan in 2014; it was determined that the device could not provide reliable data to determine sustained shock force in a combat environment.[37] The device remains in development by the military.[38]

Mechanism of Injury

In order to understand how blast waves cause traumatic brain injury, it is important to have an understanding of the physical mechanics of the shockwave itself. At the instantaneous

A still image of an explosion allows for visualization of the shockwave, which travels at velocities too fast for the naked eye. At high enough levels (seen above), the wave is dense enough to cause light to refract through it slower than the ambient surroundings. [39]

moment of detonation, the rapid synthesis of gases created by the chemical reaction rapidly expand outward, displacing and compressing the surrounding atmosphere. The sudden compression of air creates a high-pressure atmospheric wave which propagates hemi-spherically away from the epicenter at velocities which exceed the sound barrier. This overpressure wave is referred to as the explosion's shockwave or blast wave. While typically not even noticeable at low conventional detonations, the power of an explosive shockwave is the single-most destructive force ever utilized by man.

The main factor in determining the force experienced by an individual observer from a shockwave is the wave's peak pressure. Peak pressure is correlated to the brisance, or shattering effect, of the explosion's shockwave. Severity of primary blast-related neurologic trauma is directly correlated to the detonation velocity of a given high explosive device. Low velocity explosives such as black powder and petroleum byproducts are typically not capable of creating the atmospheric-shattering effects that are produced by high explosives. Large portions of Afghanistan and Iraq are agricultural with common access to ammonium nitrate, which when combined with petroleum fuel can be used to synthesize inexpensive IEDS. Weapons caches

Chemical Explosive Name	Abbreviation	Detonation Velocity (m/s)
Cyclotrimethylenetrinitramine	RDX	8,650
Pentaerythritol tetranitrate	PETN	8,400
Trinitrotoluene	TNT	6,900
Triacetone triperoxide	TATP	5,300
Ammonium Nitrate-Fuel Oil	AN/FO (ANFO)	4,200
Ammonium Nitrate	NH_4NO_3	2,700

Comparative Detonation Velocities Between Different Common Military and Commercial High Explosives [40]

prepared by the Iraqi Republican Guard Corps in anticipation of the U.S. invasion of 2003 meant

insurgent groups had easy access to military-grade munitions depots after the collapse of Saddam Hussein's regime. Cache interdiction raids were initially subordinated to searches for weapons of mass destruction (WMDs), which were ultimately never found.

In most instances, the munitions used against American and coalition forces in the Global War on Terror are of lesser grades of high explosives. The more serious threat to American and coalition forces are from captured unexploded ordnance, such as undetonated 155mm howitzer rounds, as well as materiel support from outside factions (namely Iran and Pakistan). It is further estimated that thousands of tons of unexploded Soviet ordnance still remain in Afghanistan; increasing age poses just as much of a risk to those trying to utilize them for nefarious purposes as they do to coalition forces. As these unspent munitions age they become increasingly dangerous to handle, presenting insurgents and explosive ordnance disposal technicians alike with the risk of inadvertent detonation. Counterterrorism measures should have targeted manufacturers of ammonium nitrate earlier in Operation Iraqi Freedom and Operation Enduring Freedom in order to transition agricultural industries to alternative fertilizers which cannot be synthesized into high explosives.

While certain factors such as distance or sealed enclosure can protect from the blast wave, there is little protection offered for an unshielded observer. The law of Kinetic Energy (K.E.) states that:[41]

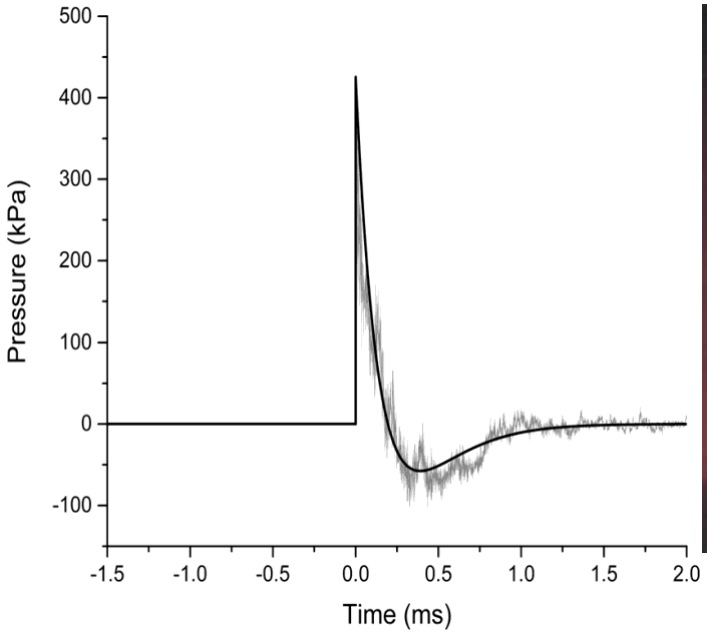
$$(\text{K.E.}) = (\tfrac{1}{2}\ \text{Mass})(\text{Velocity}^2)$$

This means that if an object moving through a 3-dimensional space has its mass increased by a linear factor, its kinetic energy will increase at an equivalent linear rate. When velocity is increased at a linear rate, however, kinetic energy will increase exponentially. By definition, shockwaves are traveling at extremely high velocities exceeding Mach 1 (343m/sec at 20 degrees

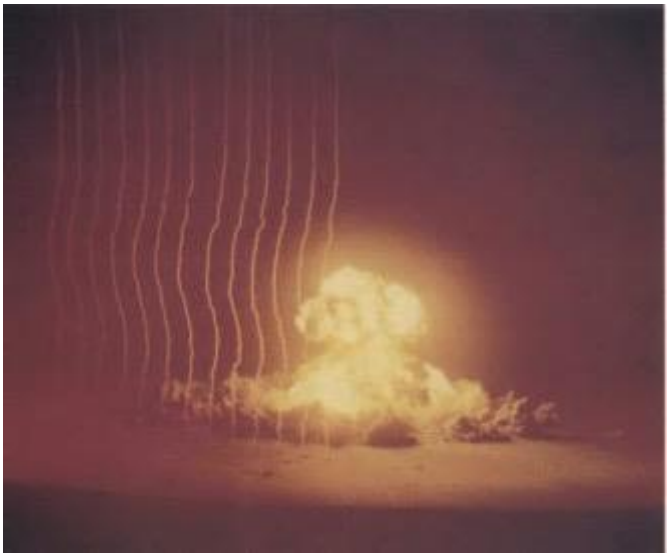
Celsius at sea level).[42] What shockwaves lack in mass, they make up for in speed. The impact of a shockwave conforms across the surface area of an unprotected individual, exposing them to large amounts of absorbed energy.

There are typically two phases involved with a blast experienced by an observer at a single location. The first phase is the shockwave itself, also known as the blast impulse. It is associated with the destructive force of an explosion. The second phase, known as the negative wave, is the result of air returning to the atmospheric vacuum created by the positive phase. Immediately following the negative wave phase are a serious of subsonic winds as the atmosphere re-equalizes. The equation for a shockwave's overpressure level with respect to time "P(t)" for an individual observer is represented as a Friedlander Waveform: [43]

$$P(t)=P_s\, e^{-t/t}(1-t/t^*)$$



An example of a shockwave's Friedlander Waveform displays the pressure with respect to time for an observer at a singular location proximal to the detonation's epicenter.[44]



Smoke trails visualize the positive (outward bend) and negative (inward bend) phases of the explosion's shockwave, 1946. [45]

Where:

$\mathbf{P_s}$ = Peak Pressure; t = time in seconds; t^* = time when wave crosses horizontal axis

The blast propagates through space as a longitudinal pressure wave, compressing the media that it travels through (air, ground, flesh, etc.) in the same direction as the wave's motion of travel. In a perfectly homogenized medium of space, a blast wave's wavelength and frequency (potential energy) can be geometrically modeled with respect to a location along a flat ground at a single point in time by the equation: [46]

$$y(x,t) = y_0 \cos(kx - \omega t + \varphi)$$

Where:

x=location along an axis of shockwave propagation

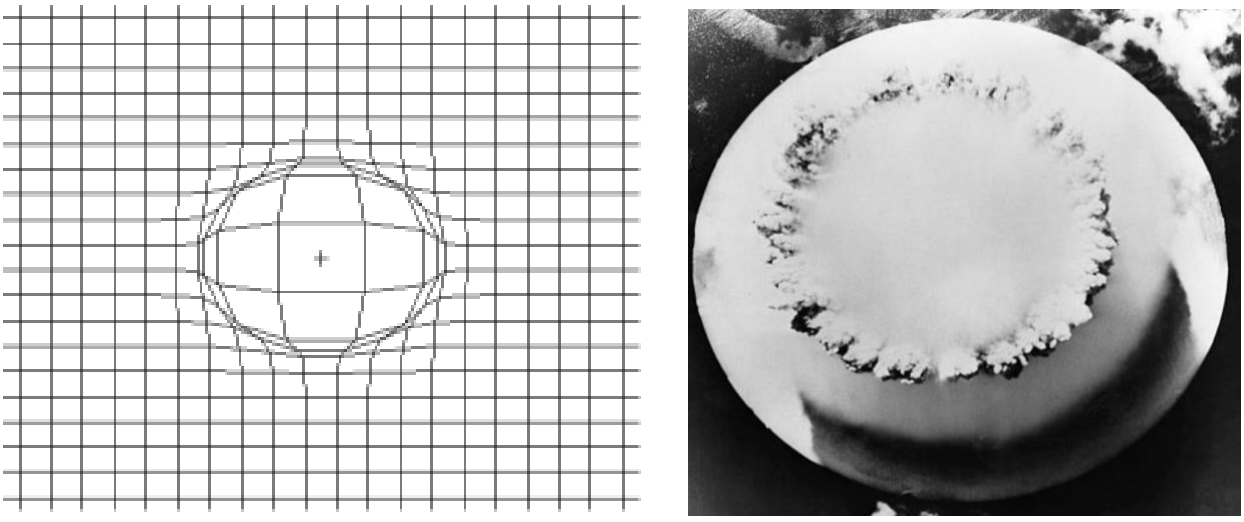
t=time in seconds since detonation

y_0=amplitude of air displacement

k=angular wavenumber

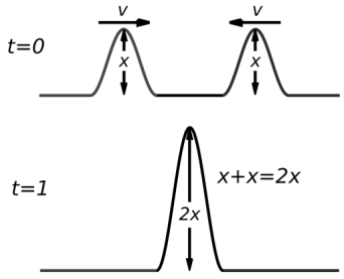
ω=frequency

Φ=phase difference, which is when (in sec) medium to its original state

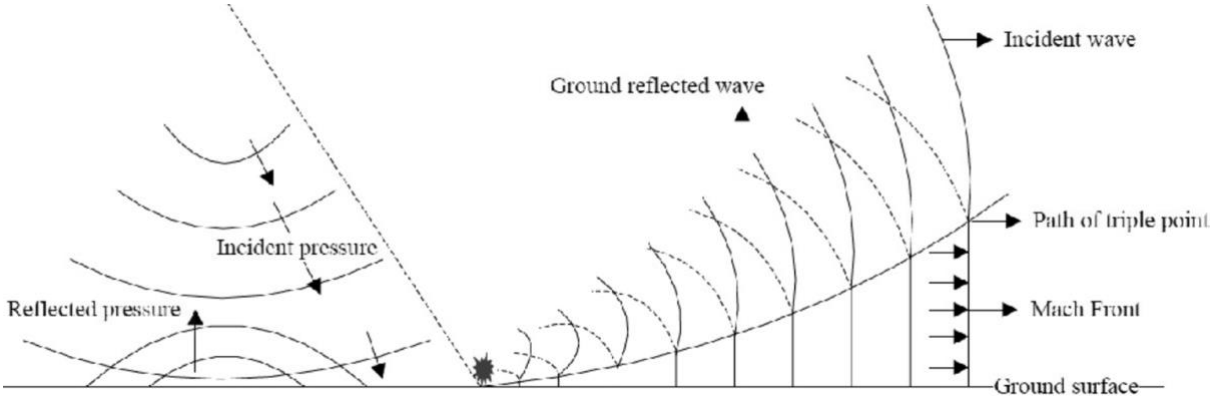


Visualization of an omnidirectional shockwave's spacial distortion across a two-dimensional plane (*left)*. Overhead view of the shockwave from Shot Baker, Operation Crossroads, 1946 (*right)*. [47]

Atmospheric overpressure poses particular risk from explosive detonations occurring above ground-level ("air-burst detonations") due to the concept of constructive interference. Constructive interference is when the meeting of two waves act to increase the amplitude of the resulting wave form. For blast waves, constructive interference results in a wave with much more energy than either of the waves preceding it. When an explosive is detonated above the ground, the resulting overpressure waves radiate outwards with portions of the shock wave reflecting off of the ground surface. Subsequently, these reflected blast waves constructively amplify the destructive potential of the blast wave through the creation of a Mach Front. The Mach Front begins at the point where the reflected waves are at the same distance from the epicenter with respect to time as the initial blast front. This area within the Mach Front extends upward until the two waves diverge. The point of divergence along the constructive interference is known as the path of triple point and is the point of highest overpressure along the interfered waveform. Constructive interference occurs any time a blast wave is reflected off of a surface, including buildings, vehicles, and persons. Anyone or anything in this area of constructive interference will be subjected to peak overpressure levels that can be multiple times higher than the original overpressure front.



Constructive interference.[48]



Depiction of constructive interference along a blast's Mach Front. [49]

Pathophysiology

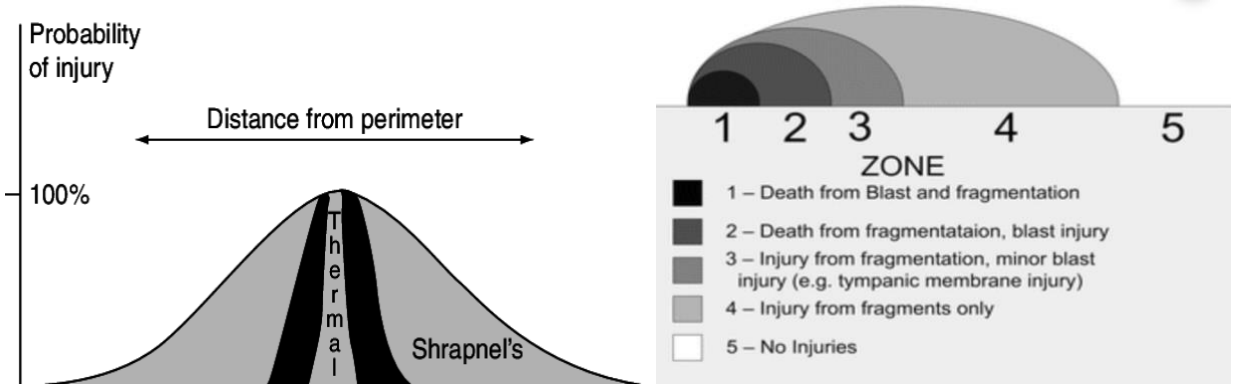
Early in the 20th century, medical knowledge was not advanced enough to understand nor treat the complex nature of neurologic trauma plaguing veterans of World War I. One century later, the scientific community is beginning to visualize the signature injury of modern war. The

Category	Organs affected
Primary	Gas filled structures
Unique to high explosives, results from the impact of the over-pressurization wave with body surfaces	Lungs
	Pulmonary contusion
	Hemoptysis
	Hemothorax
	Pneumothorax
	Pulmonary pseudocyst
	Age
	Gastrointestinal tract: Abdominal hemorrhage and perforation
	Middle ear: TM rupture and middle ear damage
	Globe (eye) rupture
	Concussion (traumatic brain injury without physical signs of head injury)
Secondary	Any body part may be affected
Results from flying debris and bomb fragments	Trauma to the head, neck, chest, abdomen, and extremities in the form of penetrating and blunt trauma
	Fractures
	Traumatic amputations
	Soft tissue injuries
Tertiary	Any body part may be affected
Results from individuals being thrown by the blast wind	Fracture and traumatic amputation
	Closed and open brain injury

Various categories and injuries associated with unprotected proximal exposure to an explosion. [50]

mechanisms of injury from an explosion are comprised of several complex factors. Injuries resulting from the explosion's shockwave itself, including ruptured eardrums and TBIs, are designated "primary blast" injuries. Injuries resulting from flying debris like shrapnel or glass are known as "secondary blast" injuries. If an individual is close enough to a sizable explosion, the shockwave can be strong enough to forcibly propel them through the air. Blunt-force injuries resulting from being launched into other objects, commonly presented as muskuloskeletal fractures, are considered "tertiary blast" injuries. While tertiary injuries can include head trauma,

these are not classified as primary blast TBIs because the injury is caused by sudden acceleration-deceleration, not atmospheric overpressure.

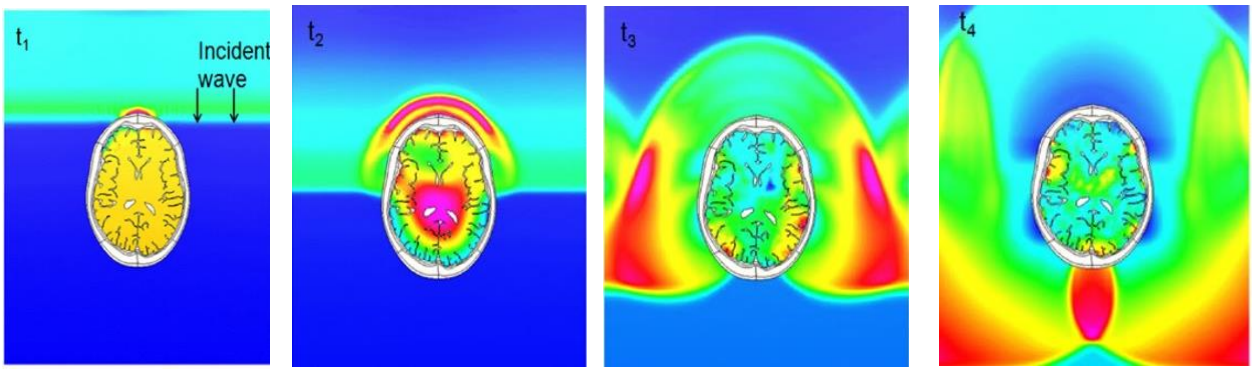


Likelihood of sustaining a particular traumatic injury with respect to distance from the epicenter. [51]

The likelihood of suffering a certain category of blast injury is directly correlated to an individual's proximity to the explosion's epicenter. When an explosive device is detonated, its fuel is catalyzed into a gaseous state in ~.000001 seconds, releasing massive amounts of energy.[52] For individuals within the radius of the thermobaric fireball itself, death is all but a certainty, resulting from a combination of multi-system primary, secondary, and tertiary blast trauma. For those who are further from the epicenter, the likelihood of death from the primary blast alone rapidly decreases; however, even if an individual is far enough away to survive the shockwave, they still may be within the kill radius for secondary blast injuries. In addition, certain external environmental factors, such as whether or not the explosive is buried, or whether there is debris intentionally/coincidentally near the explosive that may act as a shrapnel, can affect the lethality of secondary injuries. It is important to distinguish that certain factors, such as the proper use of body armor and ballistic personal protective equipment (PPE), can reduce the risk of death or injury resulting from the blast. Though this PPE may protect against ballistic projectiles, it cannot protect from overpressure waves as the waves conform to the shape of each surface they collide with. For American service members, the primary blast is capable of causing

injury at further distances than the secondary blast (assuming PPE is worn), but secondary blast injury poses a risk of death at further distances than the shockwave. While secondary blast injuries can be easily identified and treated on the battlefield, much about the exact mechanisms and thresholds of primary blast injury remains unknown, making treatment extremely difficult.

While it may not be readily apparent, the body is highly sensitive to changes in barometric pressure. A simple example would be the sensation of one's ears "popping" when rapidly ascending in elevation. Humans are not biologically capable of withstanding atmospheric pressures not replicable in nature. The velocity of a shockwave is affected by the material it radiates through; areas in the human body where regions of different densities interact are especially at risk for primary blast injury. At lower levels, visceral organs such as the gastrointestinal tract and the tympanic membrane can be damaged. At higher levels, organs such as the spleen, bladder, and lungs are also effected. Unfortunately, the most debilitating injury from shockwaves is one that is current the least understood. Explosions are by far the leading cause of traumatic brain injury in combat. Many cases are on the milder end of severity, meaning the short window for detection is often missed. It has been known for decades that severe exposure to explosive blast waves can cause permanent and debilitating brain trauma, but only recently has evidence begun to emerge showing that violent explosive neurologic trauma is capable of causing mTBIs where onset of symptoms is either delayed or absent.



Computer-generated image (top view) of a simulated shock wave traveling through the skull.[53]

For an individual observer who is exposed to a blast wave strong enough to cause neurologic trauma but mild enough to present only mild symptoms, the experienced pressure is the objective equivalent of being subjected to the equivalent pressure felt at 30 meters below sea level for only a couple of milliseconds; the entire event occurs in just a fraction that it takes neurotransmitters to process what has happened. Subjectively, as describes by those who experienced it firsthand, “it feels like being kicked by a horse, a horse with a hoof the size of your entire body”.[54] Unlike impact TBIs typically seen in athletes which are caused the brain slamming against the sides of the skull, blast TBIs cause direct systemic trauma to the neural axons without coup contrecoup. The kinetic energy of the shockwave causes cellular compression as it passes through the cranium. This compressing force is the unique mechanism for primary blast TBIs.

The brain is made up of two main types of soft tissue: grey matter and white matter. Grey matter is comprised of the main bodies of the neuron cells which contain vital organelle structures such as the cell nucleus and the mitochondrion.[55] The white matter section of the brain is made up of the neural axons which act as bridges connecting to other neurons. It is these

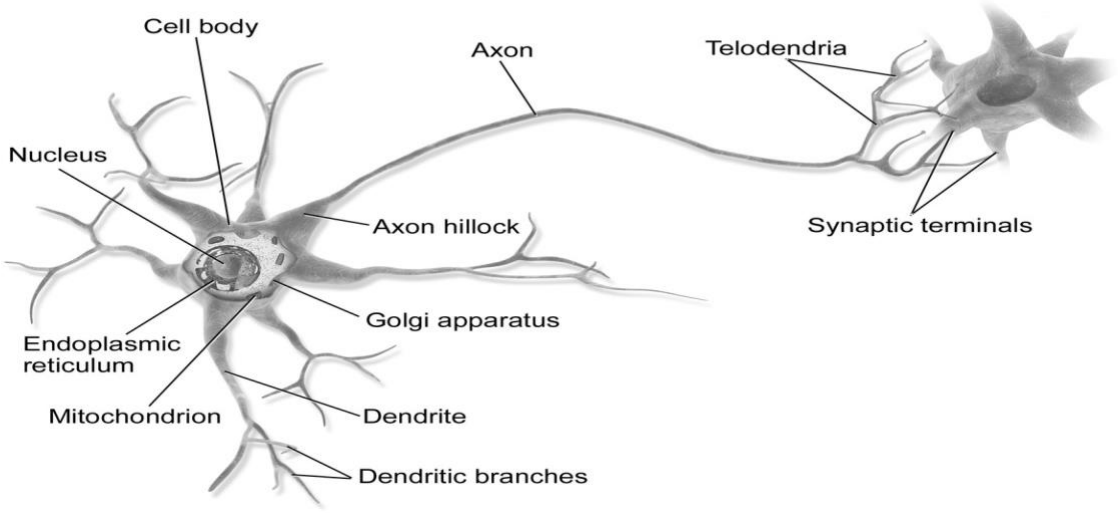
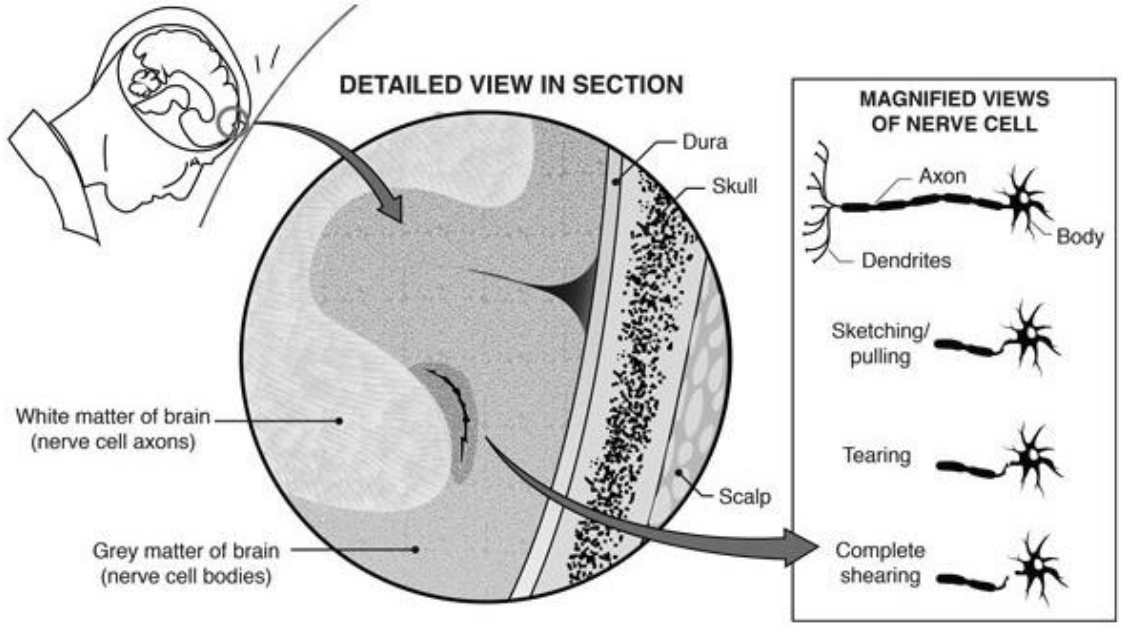


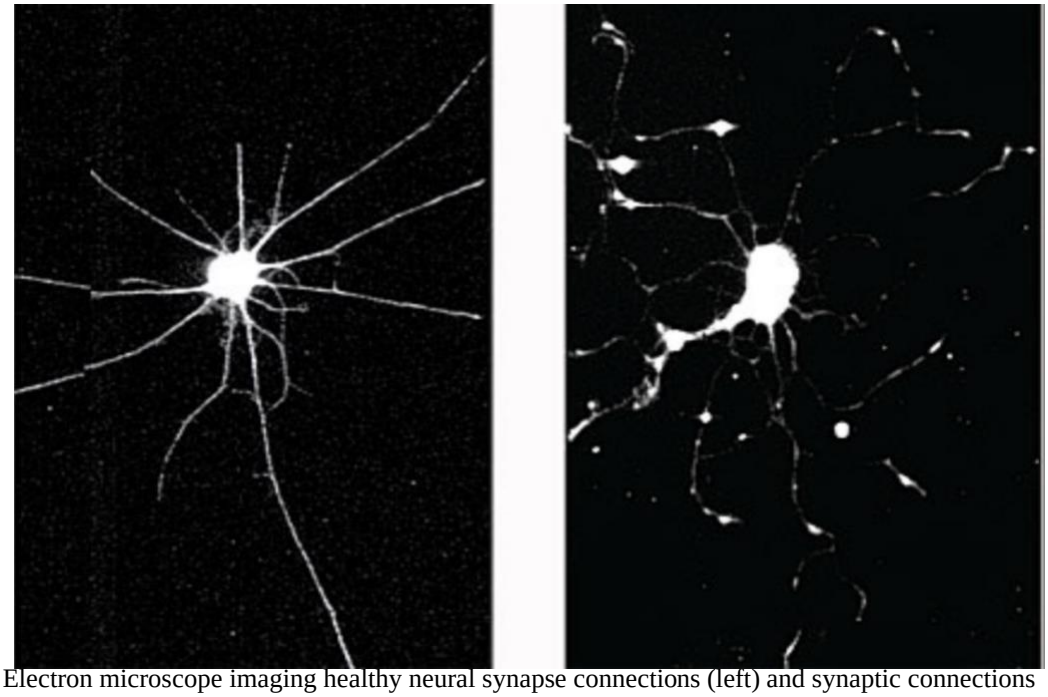
Diagram of a human multipolar neuron cell. The connections made by axons between synaptic terminals comprise the entirety of every individual human experience. [56]

connection of axons with one another which makes up every conscious and unconscious aspect of sentience, including memory, motor function, behavior, and homeostasis. Humans have biologically evolved to maximize the number of axonal connections between neurons in a confined space by manifesting folds. This folding is also what gives the brain it's signature wrinkled appearance. The brain is surrounded by a cerebrospinal fluid (CSF) which acts as a protective barrier between the soft tissue of the brain and the hard calcium of the interior cranium.

The medical and scientific communities understand that, at significant overpressure levels to an unshielded observer, violent explosive neurologic trauma occurs by mechanism of diffuse axonal injury (DAI). DAI describes the mechanism where sudden shear forces cause the axonal synapses in the brain to undergo sudden stress, which manifest as partial or complete tearing of the axon from the neuron body. Typical traumatic causes of DAI in modernized



Concept of Diffuse Axonal Injury (DAI) which while often found in mechanisms of sudden acceleration/deceleration, can also be used to accurate represent the damage to synaptic pathways in explosive neurologic trauma.[57]



Electron microscope imaging healthy neural synapse connections (left) and synaptic connections damaged by diffuse axonal injury from an explosion, postmortem (right).[58]

nations almost exclusively result from motor vehicle accidents or falls. Symptoms of DAI will range in severity from a moderate concussion (Level II or higher) to fatal. In conditions found almost exclusively in war, however, DAI can be caused by atmospheric pressure. Unlike other causes, DIA from a shockwave is not regionalized but rather distributed throughout the entirety of the brain. As the blast wave travels through the brain, the white matter, grey matter, and CSF will compress and absorb the wave's kinetic energy and differing rates. While this rate is relatively equal in homogenous matter, massive shear forces are generated at surfaces of differing densities. The astroglial surfaces are the most vital regions of the brain and are at most risk of damage from primary blast exposure above certain thresholds. In addition to the neuron axons destroyed by DIA, neurons may become damaged through the collapse of protein channels along the cell membrane. Neural membranes are typically durable enough to withstand the compression forces generated by explosive shockwaves, but the collapse of microscopic air bubbles occasionally accumulating along protein channels can cause the channel passageways to

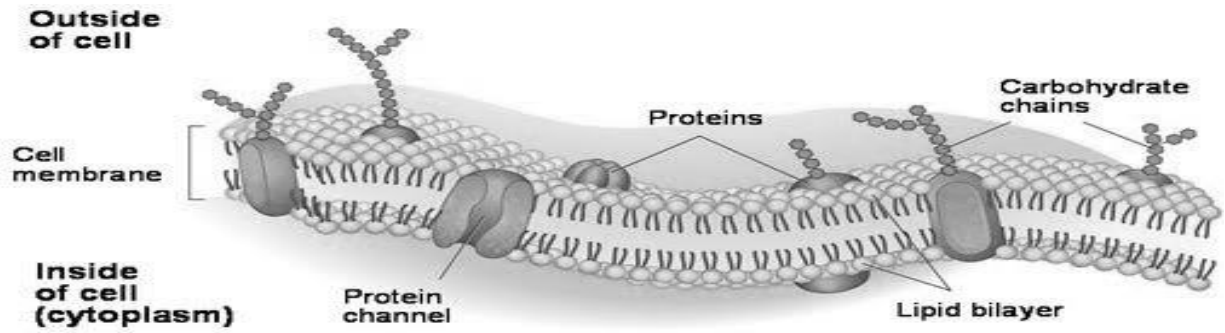
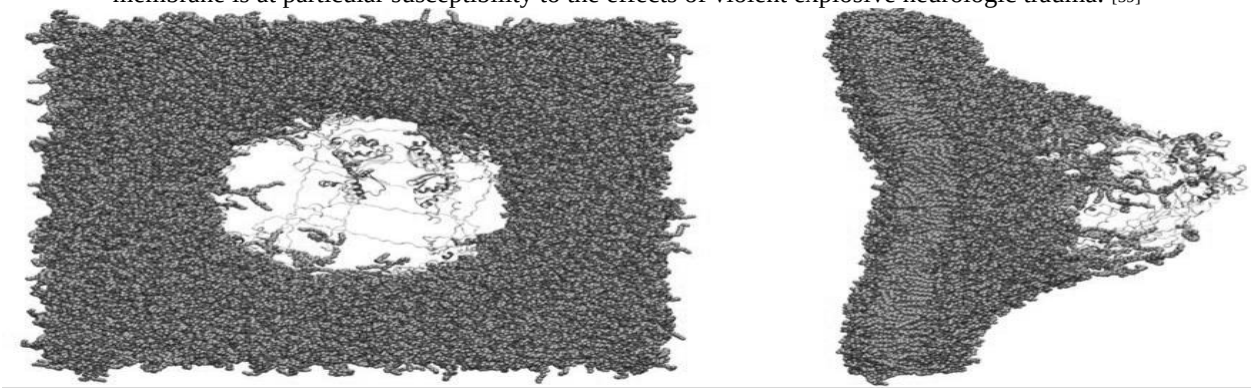
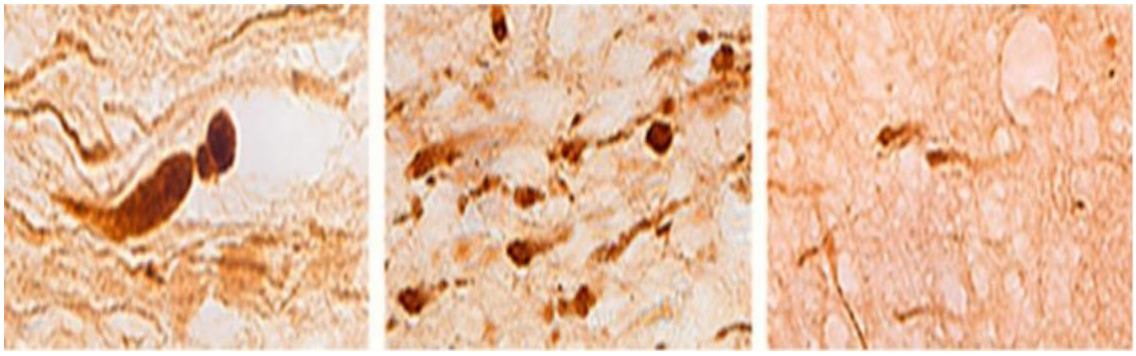


Diagram of a cross-sectional view of a neuron cell membrane. The protein channels seen along the membrane is at particular susceptibility to the effects of violent explosive neurologic trauma. [59]



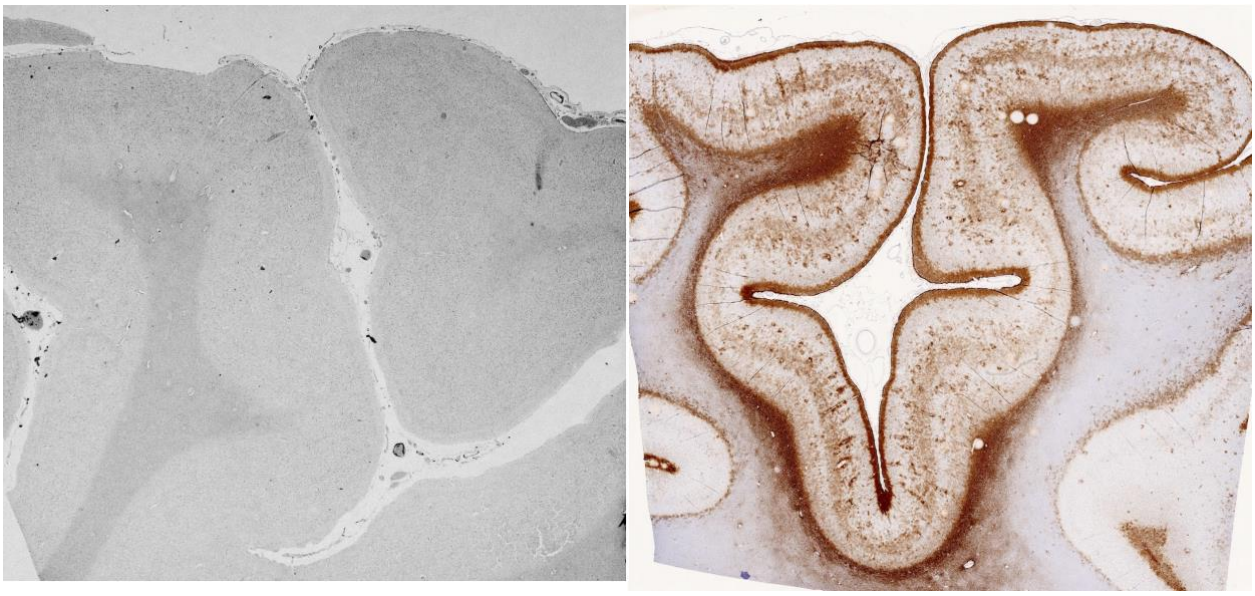
Front (left) and cross-sectional (right) simulation of neuron protein channel damaged by violent explosive neurologic trauma.[60]

rupture. Neuron protein channels damaged by shockwaves are unable to regulate the exchange of molecular ions between the inside and outside of the cell, resulting in the uncontrolled release of a variety of neurochemicals in the hours to days following exposure. This uncontrolled diffusion of chemicals is cytotoxic in high concentrations to the immediately surrounding cells resulting in secondary cellular damage and death.



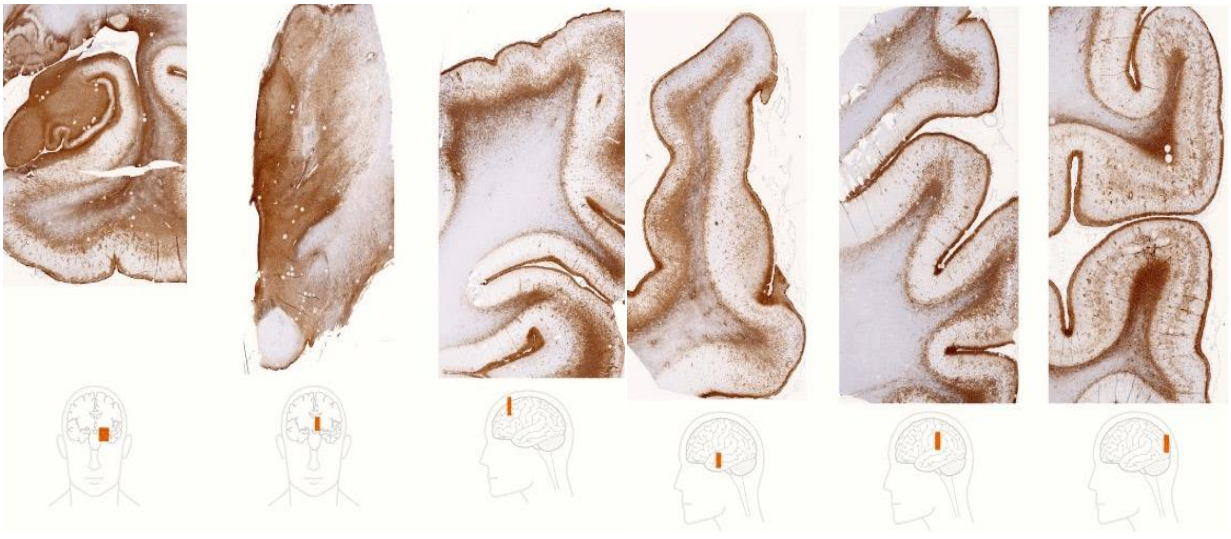
Postmortem cross-sections showing unique brain lesioning from a motor vehicle crash (*left*), explosive shockwave exposure (*center*), and drug overdose (*right*).[61]

At regions in the brain where grey matter contacts cerebrospinal fluid or white matter, the resulting kinetic energies are enough to cause permanent synaptic shearing and/or lesioning of



Samples showing astroglial intersections in a healthy adult brain (*left*) and a brain that has confirmed blast TBI (*right*). Discolorations in areas where CSF meets grey matter and grey matter meets white matter represent irreversible lesioning of synaptic pathways.[62]

the axon, resulting in irreparable scarring to the astroglial surfaces. The resultant lesions are distinct in appearance from lesions seen in postmortem samples from DAI patients in automobile accidents, as well as non-TBI causes such as opioid overdoses. This is important to distinguish as



Post-mortem samples of astroglial junctions in veterans with diagnosed blast exposure. Samples taken show abnormalities in a wide variety of neural sections impacting a wide range of each case's life.[62]

reckless behaviors can be long-term manifestations of TBI, making postmortem confirmation of violent explosive neurologic trauma more arduous. Unfortunately, no known method exists for determining whether an individual has sustained these lesions to the brain from exposure to explosions other than autopsy. Since the majority of service members who have sustained primary blast TBIs are likely to survive their injuries, they will continue to live with the long-term consequences of the synaptic damage. Damage is not limited to specific regions of the brain; the long-term afflictions of systemic axonal shearing afflicts a variety of critical cognitive and behavioral functions. Impacted neural centers include areas responsible for activities such as sleep regulation, vision, memory, hormone regulation, and decision-making.

Impacts

The exact pressure thresholds corresponding to different severities of primary blast TBIs have yet to be definitively established due to the unique environment of warfare. By extrapolating on historical casualty statistics from contemporary American conflicts and military studies, general safety ranges can be inferred with a likelihood of sustained injury for blast overpressure exposures:

Threshold (PSI)	Corresponding Injury
3	GI (repeated exposure)
5	Tympanic membrane rupture
10-15	**mTBI, asymptomatic**
20-25	Symptomatic mTBI
30-40	Moderate TBI
50	Pneumothorax, serious neurologic disruption
80-100	Fatal multisystem trauma (threshold)
100+	Death

Approximate Primary Injury Overpressure Thresholds [63][64][65]

A U.S. Army UH-60 Blackhawk prepares to MEDEVAC casualties from a roadside IED, Afghanistan.[66]

Primary blast injury is universally understood to cause TBI at moderate overpressure thresholds while also presenting other comorbid traumas as these levels also pose the risk of secondary and tertiary injuries. Aerial superiority during the War on Terror has allowed for drastic improvements in medical evacuations with over 90% of all battlefield casualties surviving their injuries. An explosion may cause many distracting injuries, such as traumatic extremity amputation, which while immediately life threatening can easily be stabilized under primary field care. Many mTBIs from shockwaves were not diagnosed simply because patients often had distracting injuries which required a higher level of immediate attention. It is typically patients on the lower range of overpressure exposure who suffer the worst outcomes over the long term. In fact, overall long-term outcomes are worse for soldiers with asymptomatic head trauma

compared to those with a limb amputation. Patients in the range of mTBI who were protected from secondary injury by their body armor and tympanic membrane rupture by their hearing protection are unlikely to display any symptoms other than momentary discomfort. Most cases of mTBI will heal over a period of months but it is estimated that approximately 10-20% of primary blast mTBIs develop into permanent post-concussion syndrome (PPCS).[67] DOD studies have shown that soldiers with histories of symptomatic blast TBI had abnormalities found in the white matter of their brains which directly correlated with with physical symptoms such as headaches, nausea, and dizziness.[68]

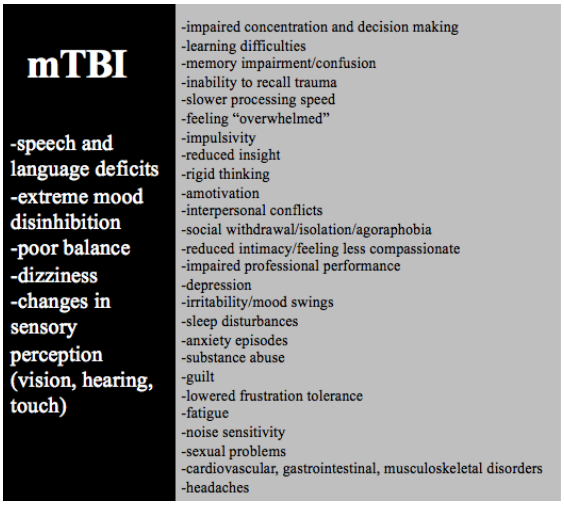
EOD technicians search for secondary IEDs around the wreckage of a destroyed vehicle, Afghanistan. [69]

The temporary symptoms of mTBI may not be apparent to either patients nor medical providers in a combat setting. The austere environment of warfare may create further circumstances that could be used to rationalize the decrease in neurological performance, such as sleep deprivation, dehydration, and general fatigue. Thus, mildly symptomatic patients may subject themselves to repeated exposure believing their TBI symptoms are caused by combat-related stress. Exposure to more immediately serious secondary injuries like shrapnel likely distracts line and Role II medical providers from accurately detecting symptomatic mTBI in patients. It is often not until the end of the combat deployment and the rotation to garrison that service members and their loved ones begin to notice behavioral changes. These behavioral changes are quickly rationalized as some other factor, such as the difficulty adjusting to peacetime life, or

mischaracterized as solely a psychological deviation related to the subjective experiences of each soldier during war. While a sizable percentage or veterans returning from the War on Terror will have diagnosed PTSD without exposure to explosive overpressure, the presentation of virtually indistinguishable symptoms for those with known exposure makes differentiating mTBI from PTSD all but impossible. Without imaging technologies or other biometric measures/tests to distinguish blast mTBI from PTSD, diagnosis is solely based on subjective accounts of exposure to the explosion. These accounts, which extrapolate off of rough estimates of the explosion's size are known to be unreliable. The combination of the myriad of coexisting factors makes the accurate and timely diagnosis of mTBI in the combat setting extremely difficult for medical personnel. The vast majority of all blast TBIs during the Global War on Terrorism went undiagnosed. It is estimated that 80% of all combat TBIs between October 2001 and December 2006 were never diagnosed nor recorded.[70][71] While preliminary screening methods have been improved in recent years to assess for delayed onset of symptoms, the current lack of a method for definitive diagnosis is a serious obstacle to measuring the true prevalence of injury. As a result, there are tens of thousands of GWOT veterans who have returned from war, been redeployed, or are currently serving serving, that are unaware they had suffered neurological trauma from explosive shockwave exposure. By the end of Operation Iraqi Freedom in 2011, 239,200 GWOT veterans had received an ICD-10 coded diagnosis of PTSD, with 9.5% of them also having a coded diagnosis of blast TBI.[73] Incidence of PTSD in GWOT veterans increased

A roadside IED killed 11 out of a 12-Marine squad in Iraq in August 2005. [72]

from 170:100,000 in 2000 (comparable to U.S. civilian rate) to 1,110:100,000 (5x civilian average) in 2011, reflecting an 11-year increase of roughly 650% above pre-9/11 numbers.[74] A total of 30,521 veterans had received an ICD-10 coded diagnosis of TBI from blast exposure by 2012, with 74% of them also having a coded diagnosis of PTSD. Many patients never received medical treatment for neurologic trauma. Instead, most GWOT veterans were only treated for psychological issues that may be secondary to the TBI itself. A 2017 study funded by the Department of Veterans Affairs found that treatment with the UCH-L1 protein, which is found in high quantities in healthy brains, had potential to the neurological function of TBI victims but only if administered within weeks of the exposure.[75]



Similarities between blast mTBI and PTSD symptoms.

Other external factors such as readjusting to life at home, work, family, and other aspects of daily life can exacerbate the progression of psychological symptoms and make determining physiological roots more challenging. TBIs left untreated over time ultimately wreak havoc on the personal lives of service members and their loved ones, as they directly reduce quality-adjusted life years (QALY) in a myriad of ways. According to recent studies, GWOT combat veterans are 65% more likely to get divorced than a comparable civilian cohort.[76] In addition, TBIs have been associated with increased risk of substance abuse or alcoholism, as well as higher rates of unemployment. [77][78] 53% of the homeless population in the United States have

previously sustained TBIs.[79] In addition, research suggests that scarring of tau proteins in the brain from blast exposure leads to an increase in incidence of early onset Alzheimer's and Parkinson's Disease.[80] Of the more than 400,000 veterans diagnosed with Parkinson's or dementia, more than 25% had previously been diagnosed with a TBI or PTSD.[81] Blast exposure could also explain a variety of other neurological afflictions impacting the central nervous system; combat veterans have the highest rates of Multiple Sclerosis (MS) and Amyotrophic Lateral Sclerosis (Lou Gehrig's Disease) of any American demographic.[83][84] For service members who have been trained to suppress emotion or stigmatized by modern society because of their mental health, the prognosis of living with the permanent effects of a TBI can seem like a fate worse than death itself. Studies have shown considerable causation between decreased QALY associated with TBIs and increased likelihood of suicidal ideation and attempt.[85] Every day, 20 veterans and 1 active service member will commit suicide.[86] It is

U.S. soldiers from the 10th Mountain Div. treat a casualty after a vehicle-borne IED attack in Kabul, Afghanistan. [80]

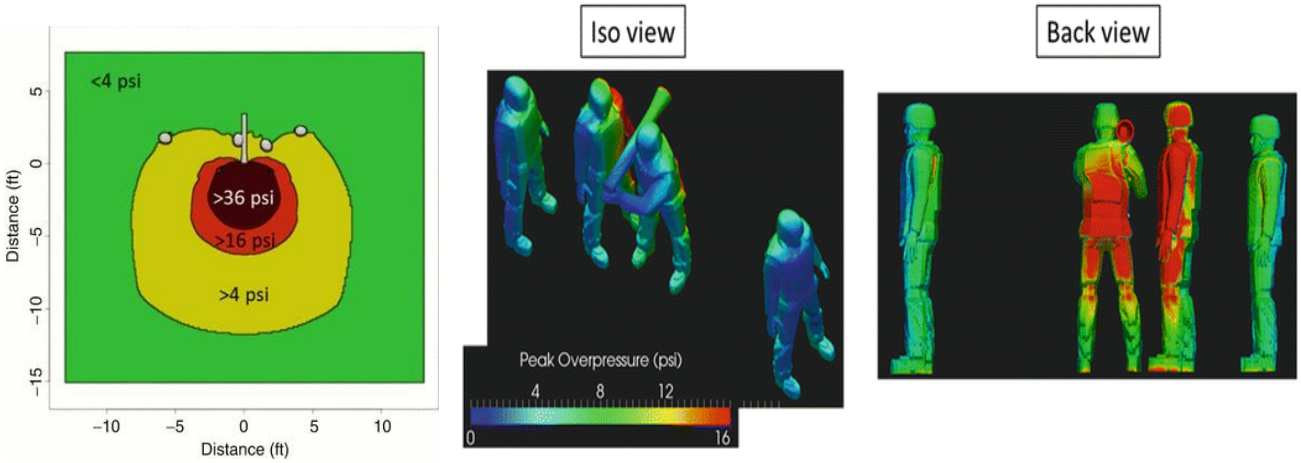


Diagram displaying blast overpressure impulse levels from an M-48 Carl Gustav Recoilless Rifle.[87]

highly probable that the number of GWOT veterans with undiagnosed TBIs who later killed themselves eclipses the total of all U.S. veterans killed in combat since 9/11.[88]

One of the leading doctrines of the United States military is the mentality to "*train as you fight*". For maneuver forces, this typically may involve the use of live ammunition and ordnance during training operations, referred to as "live fire exercises" or "live fire maneuvers."[89] It was discovered that some of these types of weapons, such as artillery, mortars, and recoilless rifles, pose particular risk for blast overpressure to the user and for those in the immediate vicinity.[90] Little is known about the long-term effects on the brain from repeated exposure.

What is understood is mostly inferred from the realm of conventional neurological trauma research, which itself still relatively young. It is understood that deterioration of neurologic pathways progresses at an exponentially increasing rate for cases with repeated injury. In instances where trauma is sustained repeatedly at moderate severity, the summative effects are worse than the extent of each injury on its own. Repetitive head trauma is most commonly seen in professional boxing and football athletes, who may sustain thousands of blunt force impacts to the head over the course of their careers. The long-term summation of these compounding

(*Left*) An anti-tank infantryman from the Swedish Army with epistaxis after a day practicing with the M48 recoilless rifle. The M48, made in Sweden, is named after reigning king Carl Gustav. (*Right*) U.S. Army Rangers practice firing the Carl Gustav. In Australia, Rangers commonly refer to the Carl Gustav as the "Charlie Gutsake[1]".[91][92]

[1] Gutsake: ("Guts aches"), slang used to describe discomfort felt from gastrointestinal injury from repetitive blast exposure.

injuries manifests into a condition far worse: Chronic Traumatic Encephalopathy (CTE). While much is yet unknown about the underlying mechanisms of CTE since its recent discovery in 2002, what is known is that the disease progressively deteriorates and that there are no known treatments nor cures. It is also known that since shockwaves pass through the brain at much faster velocities than blunt-force impacts, the neurologic trauma from blasts is more extensive than conventional mechanisms. While a direct causation between repeated blast exposure and CTE has not been conclusively identified in a clinical study, a postmortem case study was conducted in 2014 of the brains of 8 service members with multiple blast TBIs who had subsequently committed suicide. Of the 8 service members studied, 7 identified positive for CTE.[93]

Further Considerations

The Global War on Terrorism has cost the American taxpayers more than $2.4 trillion dollars since the September 11th Attacks; the true price of the wars in Afghanistan and Iraq is significantly higher as the indirect and immeasurable tolls will never be truly known. [94] While the military has made several medical advancements from the wars in the Middle East, the lessons learned regarding blast TBI bear a high price. The current lifetime expenses for medical treatment of a TBI range from $85,000 to $3,000,000. Despite knowing thousands of veterans with blast TBI and PPCS were misdiagnosed, the VA continues to deny disability claims for TBI for GWOT veterans. Instead, they approve claims for PTSD, arguing it is impossible to clinically differentiate TBI from PTSD. [95][96] In addition, the VA revised the official statistic for the number of veterans who commit suicide daily from 22 to 17.[97] This revision is misleading, as this does not mean fewer veterans committed suicide; in fact, more veterans killed themselves that year than the year prior. The number is lower because the VA began excluding reservists

and guardsmen who did not serve in a designated combat theater. Apparently, the VA appears more concerned with garnering public support than being truthful about the scope of the damage done by blast TBIs. The VA has made some effort to combat the epidemic such as expanding the number of veteran crisis hotline centers from one to three.[98] In addition, the United State Congress ordered the DOD to begin appropriating funding to include blast exposure in service members' permanent medical records in December 2019.[99]

The synthesis of trinitroluene (TNT) and the discovery of high explosives by Alfred Nobel unwittingly unlocked Pandora's Box. Sometimes it's better not to open the box. There are times when its simply best not to know what's inside. The problem with people is that sometimes they wait until the box is opened before asking if they should understand what is inside. Yet the first artillery salvo over 100 years ago signaled to the world that the lid has been forever opened, and the true extent of its wickedness has patiently waited through time to be discovered.

"Prometheus begged her not to open it. She opened it. Every evil to which human flesh is heir came out of it. The last thing to come out of the box was hope." [100]

Counterterrorism tactics in the Global War on Terror have widely seen the use of military working dogs (MWDs) to detect explosives and insurgents. These dogs are such adept predators that it is not a matter of if they will find their target, but when. For America's military adversaries, it is analogous to going against one of these dogs. If the handler at the end of the dog's leash has decided someone is a threat, the outcome of the fracas is a foregone conclusion. The United States military will win in

MWD "Conan" with the U.S. Army was responsible for cornering ISIS Leader Abu Bakr Al Baghdadi who committed suicide with an IED in Syria in 2019. [101]

any direct face-to-face engagement. The problem is the enemy is adapting to fight the handler instead of the dog. America's adversaries are learning that if they can make it appear to the handler as if the dog is just wandering aimlessly with no clear objective, the handler will give up. In the mid 2010s, an article written by a veteran-owned satirical website commented on a fictional army private who had the honor of patrolling the same supply routes his father once did in Afghanistan. Ironically, 2020 marks the first year where U.S. soldiers are deployed in Afghanistan who were not born when the war began. By disregarding civilian populaces and forcing American soldiers into lose-lose situations, the enemy has created conditions for a situation in Afghanistan which appears hopeless. The Global War on Terror will draw to a conclusion one day, but to what end and at what cost?

Part II:
The Solution

While the physiological damage from primary blast injury may be extremely subtle at low levels, atmospheric overpressure is the single greatest destructive force ever harnessed by mankind. On July 16th, 1945, the United States conducted the Trinity Test. Less than a decade later on March 1, 1954, Castle Bravo was detonated at Bikini Atoll in the southern Pacific Ocean.[102] It was the largest man-made explosion ever created at the time with an explosive power of 15 Megatons (~30 billion pounds of TNT).[103] With a yield 2.6x larger than anticipated, the atmospheric shockwave narrowly avoided killing the military scientists on the island. At the detonation's epicenter, the peak atmospheric overpressure was greater than that which exists at Challenger Deep in the Marianas Trench 11,034 meters below sea level.[104] The test confirmed America's blueprints for a hydrogen bomb and also heavily irradiated a majority of the Marshall Islands with radioactive fallout creating an international incident. As described by U.S. Naval personnel based on the islands nearby to observe meteorological equipment, the magnitude of the bomb's overpressure was like as if experiencing Armageddon: [106]

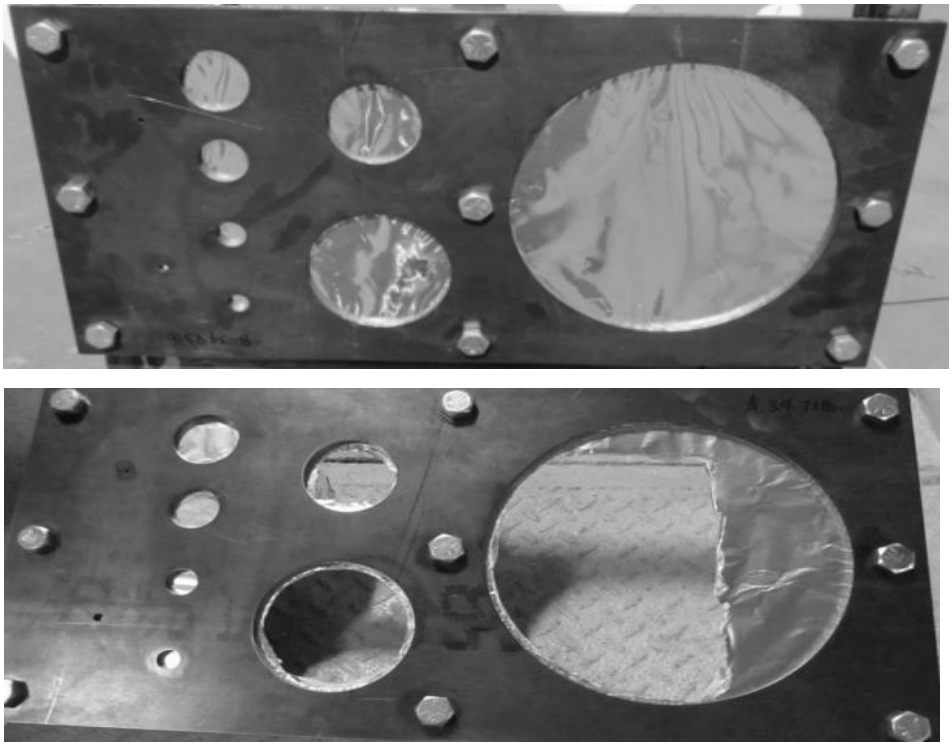
Atmospheric condensation disks visible around Castle Bravo fireball. Photo taken T+15 seconds, 60 miles from ground zero.[105]

> First was the overpressure, then the sucking out by the underpressure. The concrete building creaked, but stayed firm. A few yards away, as we found out later, frame buildings had been blown down by the hurricane winds from the blast. Immediately after the air blast, we noticed some water coming in through the conduits behind the control panel. And about the same time water in the lavatory started shooting up to the ceiling. [Tsunami] effects were not expected for six more minutes. We later found out that this

water had been forced up from the lagoon by the overpressure created from the air blast, and had come in through pipes and conduits.

Despite the success of the test, the dusting of a large area of the island chains with nuclear-contaminated fallout led to the U.S. to reconsider the need for such firepower in its' arsenal; the majority of modern strategic nuclear warheads have yields in the kilotons rather than megatons.[107] Bikini Atoll and the surrounding Marshall Islands remain considerably irradiated to this day. [108]

During the years of America's above-ground thermonuclear weapons tests, military and civilian scientists were extremely interested in gathering data associated with nuclear blast waves. To do so, scientist and engineers alike constructed a plethora of gauges which could be used to approximate the atmospheric overpressure from a known detonative yield. Among these devices was a passive sensor called a Bikini Gauge, with its namesake originating from the



Cold War-era shockwave gauges, named "Bikini Gauges" after the Bikini Atoll, intact (*top*) and after blast exposure (*bottom*). [109]

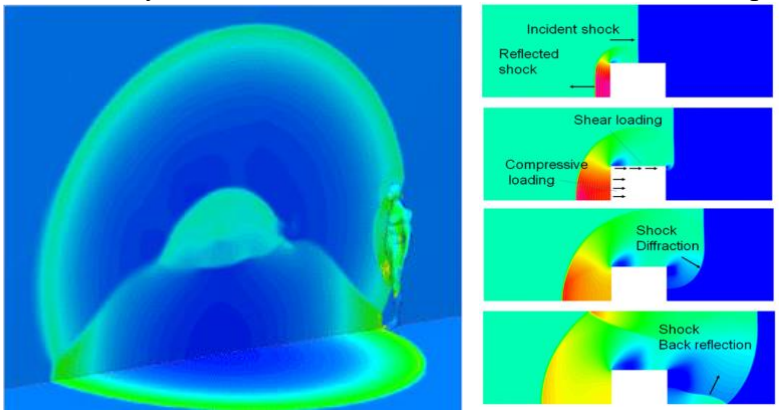
Bikini Gauges used to estimate blast force from scaled recreation of a meteor impact with TNT, illustrating scalability of the gauge's application. [110]

island chain where it was first utilized. The gauge, simplistic in application, consistent of a sheet of metal foil pinned between two metal plates with holes of varying radii. By measuring the amount of loading force it would take to shear a membrane of a given size, scientists could calculate the shockwave's pressure at a given distance. Multiple devices could be placed at varying sites from a test epicenter to approximate the explosion's Friedlander Waveform. Although all atmospheric nuclear testing was banned on August 5th, 1963[110], and since then technological advancement has created more precise methods of estimating blast force, the Bikini Gauges is still an inexpensive and accurate passive instrument still utilized by scientists today.

Spurred by the need for a rugged wearable pressure sensor, the Department of Defense has spent millions of dollars in research and development contracts, but so far a workable solution has not been found. The closest resemblance to a viable option is the Blast Gauge

(*Left) Blast Gauge Field Testing-Afghanistan, 2014.* [112] *(Right) Blast Gauge System.* [113]

created by B3, Inc. in 2012. [112] This sensor system is comprised of three individual sensors worn on the head, shoulder, and chest, respectively. The gauge utilizes a complex system of electronics to record blast waves so the data can be transferred onto a computer for further analysis. In concept, this seems like a perfect solution; however, following field testing of the sensors in Afghanistan, the Pentagon concluded that "the DoD's current inventory of blast gauges does not provide consistent and reliable data in the combat environment".[37] Despite this, the military has continued to invest in the device's development. For example, in August 2019 I witnessed this sensor being worn by cadre from the U.S. Army's 4th Infantry Division at Fort Knox, Kentucky. Over the course of the summer, the soldiers selected to instruct basic hand grenade training would be collectively subjected to more than 14,000 overpressure exposure events. As I crouched behind a concrete wall, I asked one of the cadre wearing the device how well it worked. "Beats me, man. We're just the guinea pigs". In the nearly ten years since the Pentagon began searching for a solution, a significant number of American service member have sustained traumatic brain injuries from explosions. A solution must be found before the next major armed conflict or everything learned will be in vain. It is vital to identify all of these men and women who are neurologically injured so that they can receive prompt triage and treatment, as the alternative option is another veteran suicide epidemic.



(*Left)* Blast wave impulse from M67 fragmentary grenade. [114]
(Right) Complication of blast wave interpretation with denser medium. [115]

There is an old maxim which states “close enough is the enemy of perfect”. Oddly enough, there is a saying in the military that goes “‘close enough’ only matters for horseshoes and hand grenades”. So far, investing countless amounts of resources into developing sophisticated equipment has failed to produce a viable solution. Too often is an approach to a problem mired not by the approach itself but rather by the overcomplicating of the problem. In order to address this, the U.S. Navy began using the KISS (“Keep It Simple Stupid”) acronym in 1960.[116] The concept of KISS is that most systems work best if kept simplistic--any unneeded complexity interferes with purpose and should be avoided. If simplistic blast sensors worked well enough for scientists in the Cold War, why could the same concept not be inventively applied to the current issue? Inspired by this rationale, I have designed and prototyped a single-use, passive shockwave exposure indicator designed to be worn on a service member’s PPE. It is similar in concept to the Bikini Gauges of the 1950s except instead of using the same membrane and differing apertures, the apertures remain constant while the membrane varies. The membranes are contained in in a high-density impact polymer housing and can contain multiple passive thresholds in one singular device. Membranes can be calibrated based on material and thickness to rupture at given thresholds corresponding to certain primary blast injuries (see pg. 23 for specific injuries), allowing for the initial triaging of non-symptomatic patients for further medical evaluation which otherwise would not occur. The sensor is single use, allowing the device to accompany the wearer to Role I and II medical facilities in the event of an exposure. They are inexpensive to

Shockwave Exposure Indicator prototype affixed to hearing protection.

produce and thus easily replaceable. Most importantly, the simplicity of design means that the costs associated with outfitting an entire military are a mere fragment of the current alternatives.

Proof of concept of the design was validated by exposing prototypes to low levels of atmospheric overpressure emitted by an ALS Technologies less-lethal concussion grenade producing a known overpressure exposure of 1.2 PSI (172 dB) at 5 feet from detonation [117],

(Left) ALS TRMR concussion grenade.[117][118] (*Right)* Setup of prototype devices concentrically around epicenter.

used as a scaled recreation of a violent explosive event. For this detonation, the value of peak pressure is approximately inverse to the cube distance from the epicenter. Pressure values correlated to injury thresholds were extrapolated from the known impulse with a prototype device being affixed at each radii. The concussion grenade was dropped through a PVC tube from a height of approximately 5 feet.

Results from initial testing demonstrated feasibility at a scaled pressure range. However, the 3D-printed polylactic acid (PLA) plastic housing which is currently being used in the prototype showed poor performance at higher impulse ranges. The next developmental steps needing to occur are to refine membrane thicknesses to a higher level of sensitivity as well as researching stronger polymer housing alternatives. In addition, a method of equalizing pressure on both sides of the membrane needs to be created in order to reduce the number of false positive

readings caused by variance in operational altitude. A provisional patent was filed on the design on May 14, 2020. I expect to have a fully functioning device by the end of 2021.

When the first bombs were dropped more than a century ago, no one knew about the invisible dangers they posed to the brain. Likewise, when the United States began experimenting with nuclear weapons, no one understood the gravity of the environmental health risks involved with exposure to another invisible foe—ionizing radiation. Exposure to radioactive material can injure tissue resulting in burns and acute radiation sickness in the immediate aftermath and can prove to be fatal in cases of higher exposure. At lower exposure ranges, radiation can cause aplastic anemia and cancer, often without presenting symptoms for months or years. When Trinity was detonated in July 1945, the safe exposure limits to radiation were nowhere near yet identified. Rather, it was through the implementation of hazard monitoring on a large scale in the form of radiation dosimetry which led to the developed safety thresholds now recognized globally. Today, dosimetry is seen as the Gold Standard of occupational hazard monitoring. It is my hopes that, through the implementation of my device, this model can be replicated so that every victim of violent explosive neurologic trauma can be identified promptly and appropriately treated.

Shockwave Exposure Indicator, latest version, June 2020 (*left)* and Blast Gauge (*right).*

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Appendix A

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SYSTEMS AND METHODS OF DETERMINING PRESSURE WAVE EXPOSURE

FIELD

[001] The present disclosure pertains to systems and methods of determining a threshold pressure of a pressure wave, such as a blast wave.

BRIEF DESCRIPTION OF THE DRAWINGS

[002] FIGS. 1-4 illustrate various views of a pressure dosimeter, according to one embodiment.

DETAILED DESCRIPTION

[003] The present disclosure pertains to systems that can be worn on a user's body, and which are configured to record at least a threshold pressure of a pressure wave experienced by the user, such as a pressure wave associated with an explosion. FIG. 1 illustrates a representative embodiment of a pressure dosimeter/sensor/meter/gauge 10. The pressure dosimeter 10 can comprise a main body or housing 12 including a plurality of pressure sensors 14 arranged in a spaced apart arrangement (e.g., a grid). With reference to FIG. 2, in certain embodiments the housing can comprise a first or top portion 16 and a second or lower portion 18 together defining a thickness of the housing. In certain embodiments the top portion 16 and the lower portion 18 can be separately formed and secured together to enclose the pressure sensors 14. In other embodiments, the housing 12 can be formed around the pressure sensors 14, such as by three-dimensional printing, injection molding, etc.

[004] In certain embodiments, the pressure sensors 14 can be located in corresponding wells/recesses/bores/openings/windows 20 defined in the housing 12, with one end of the wells 20 being open to the atmosphere and the opposite end being closed by the rear wall of the housing. The pressure sensors 14 can comprise respective membranes/diaphragms 22 extending across the wells 20. In certain embodiments, the membrane 22 of each pressure sensor 14 can be configured to visually indicate exposure to a predetermined pressure threshold, for example, by rupturing, indenting, etc.

[005] The membranes 22 can be directly coupled/secured to the walls of the wells 20, and/or can be integrated into inserts received in the wells 20. For example, FIG. 3 illustrates a

representative example of a non-reclosing pressure sensing device configured as a rupture disk, pressure safety disk, burst disk, or burst diaphragm 24. In the illustrated configuration, the rupture disk 24 can comprise a membrane 22 secured to a circular frame member or support configured as a ring or collar 26.

[006] FIG. 4 illustrates a cross-sectional view of the dosimeter 10. In certain embodiments, the wells 20 can have a first diameter D_1, and can define a chamber 28 inwardly offset from the opening of the well having a second, larger diameter D_2. In the illustrated embodiment, a rupture disk 24 can be positioned in the well 20 with the membrane 22 located in the chamber 28. In certain embodiments, the rupture disk 24 can be retained or secured in the well 20 by the chamber 28. In certain embodiments, each of the wells 20 can have the same, or substantially the same, first diameter D_1.

[007] The membranes of one or more of the pressure sensors 14 can be configured to rupture, indent, etc., upon exposure to a pressure wave of predetermined intensity. In certain embodiments, the pressure values can be correlated with pressure thresholds at or above which identifiable injuries may occur to a wearer. For example, in certain embodiments the dosimeter can comprise one or a plurality of pressure sensors configured to indicate exposure to overpressure events (e.g., events in which local air pressure exceeds normal ambient air pressure) associated with eardrum rupture (e.g., overpressure of 5 psi), asymptomatic neurological injury (e.g., overpressure of 10 psi), symptomatic neurological injury such as symptoms associated with concussion (e.g., overpressure of 20 psi), moderate neurological damage and/or lung damage (e.g., overpressure of 30 psi), critical neurological trauma and/or lung damage (e.g., overpressure of 50 psi), and/or fatal overpressure (e.g., overpressure of 90 psi). For example, in the illustrated embodiment the dosimeter includes a grid of four pressure sensors 14 wherein each pressure sensor is configured to rupture at a different pressure threshold, but the dosimeter may include any number of pressure sensors configured to rupture at any pressure, including multiple pressure sensors configured to rupture at the same pressure.

[008] In certain embodiments, the membranes 22 can comprise any of various materials including metallic foils or films comprising aluminum, zinc, copper, gold, any of various steel alloys such as carbon steel, stainless steel, etc., nickel alloys such as nickel-molybdenum or nickel-chromium-molybdenum alloys (e.g., HASTELLOY®), polymeric materials such as

polypropylene, polystyrene, polyvinyl chloride (PVC), low density polyethylene (LDPE), high density polyethylene (HDPE), natural membranes such as cellulose or cellophane (e.g., nitrocellulose-lacquered cellophane), dialysis tubing, etc., composite materials, etc.

[009] In certain embodiments, the depth and/or shape of the wells 20, the diameter of the wells 20, the diameter and/or shape of the membranes 22, the material and/or thickness of the membranes 22, etc., can be configured or tuned such that the membranes rupture at pressures exceeding a predetermined threshold, such as any of the pressure thresholds noted above.

[010] Although the pressure sensors 14 are round in the illustrated embodiment, the pressure sensors can have any shape, for example, rectangular, square, etc. The wells may also have any diameter along their length/depth. One or more pressure sensors may be configured differently from the others according to the particular pressure threshold for which they are configured to indicate exposure.

[011] In certain embodiments, the dosimeters described herein can be configured for mounting on a user's body, for example on equipment or clothing worn by the user (e.g., helmets, vests, headsets, etc.), and/or on the interior or exterior of vehicles, and/or on stationary objects such as buildings, or any other location or surface where exposure to overpressure events is desired to be monitored.

[012] In certain embodiments, the pressure sensors, areas of the housing around the pressure sensors, etc., can include visual indicia or markings (e.g., colors, patterns, values, etc.) indicating the minimum pressure threshold at which the associated membrane is configured to rupture. Thus, upon exposure to an overpressure event such as an explosion, the wearer, their team members, bystanders, medical personnel, etc., can examine the dosimeter to determine a minimum pressure threshold experienced by the wearer based on any ruptured membranes and the associated pressure value. This minimum pressure threshold can inform any likely injuries that may have been suffered by the wearer, and can be used to determine appropriate treatment.

[013] In certain embodiments, the dosimeter may include electronic pressure transducer(s), computer-readable storage memory, and/or transmitter, receiver, and/or transceiver capability for determining, recording/storing, and/or transmitting data of overpressure events experienced by the wearer.

Explanation of Terms

[014] For purposes of this description, certain aspects, advantages, and novel features of the embodiments of this disclosure are described herein. The disclosed methods, apparatus, and systems are not limiting in any way. Instead, the present disclosure is directed toward all novel and nonobvious features and aspects of the various disclosed embodiments, alone and in various combinations and sub-combinations with one another. The methods, apparatus, and systems are not limited to any specific aspect or feature or combination thereof, nor do the disclosed embodiments require that any one or more specific advantages be present or problems be solved. The scope of this disclosure includes any features disclosed herein combined with any other features disclosed herein, unless physically impossible.

[015] Although the operations of some of the disclosed embodiments are described in a particular, sequential order for convenient presentation, it should be understood that this manner of description encompasses rearrangement, unless a particular ordering is required by specific language set forth herein. For example, operations described sequentially may in some cases be rearranged or performed concurrently. Moreover, for the sake of simplicity, the attached figures may not show the various ways in which the disclosed components can be used in conjunction with other components.

[016] As used in this disclosure and in the claims, the singular forms “a,” “an,” and “the” include the plural forms unless the context clearly dictates otherwise. Additionally, the term “includes” means “comprises.” Further, the terms “coupled” and “associated” generally mean electrically, electromagnetically, and/or physically (*e.g.*, mechanically or chemically) coupled or linked and does not exclude the presence of intermediate elements between the coupled or associated items absent specific contrary language.

[017] In some examples, values, procedures, or apparatus may be referred to as “lowest,” “best,” “minimum,” or the like. Such descriptions are intended to indicate that a selection among many alternatives can be made, and such selections need not be better, smaller, or otherwise preferable to other selections.

[018] In the description, certain terms may be used such as "up," "down," "upper," "lower," "horizontal," "vertical," "left," "right," and the like. These terms are used, where applicable, to provide some clarity of description when dealing with relative relationships. But, these terms are

not intended to imply absolute relationships, positions, and/or orientations. For example, with respect to an object, an "upper" surface can become a "lower" surface simply by turning the object over. Nevertheless, it is still the same object.

[019] Unless otherwise indicated, all numbers expressing material quantities, angles, pressures, molecular weights, percentages, temperatures, times, and so forth, as used in the specification or claims are to be understood as being modified by the term “about.” Accordingly, unless otherwise indicated, implicitly or explicitly, the numerical parameters set forth are approximations that can depend on the desired properties sought and/or limits of detection under test conditions/methods familiar to those of ordinary skill in the art. When directly and explicitly distinguishing embodiments from discussed prior art, the embodiment numbers are not approximates unless the word “about” is recited. Furthermore, not all alternatives recited herein are equivalents.

[020] Although there are alternatives for various components, parameters, operating conditions, etc., set forth herein, that does not mean that those alternatives are necessarily equivalent and/or perform equally well. Nor does it mean that the alternatives are listed in a preferred order unless stated otherwise.

[021] In view of the many possible embodiments to which the principles of the disclosure may be applied, it should be recognized that the illustrated embodiments are only examples and should not be taken as limiting the scope of the disclosure. Rather, the scope of the disclosure is at least as broad as the following claims. We therefore claim all that comes within the scope and spirit of these claims.

CLAIMS:

1. An apparatus, comprising:
a body-mountable housing; and
a plurality of pressure sensors coupled to the housing, the plurality of pressure sensors being configured to rupture at different pressure thresholds.

2. The apparatus of claim 1, wherein each pressure sensor comprises a well-defined in the housing and a membrane disposed across the well.

3. The apparatus of claim 1, wherein the wells are open to the atmosphere at one end and closed at the opposite end.

4. The apparatus of claim 2, wherein the diameters of the wells are substantially the same.

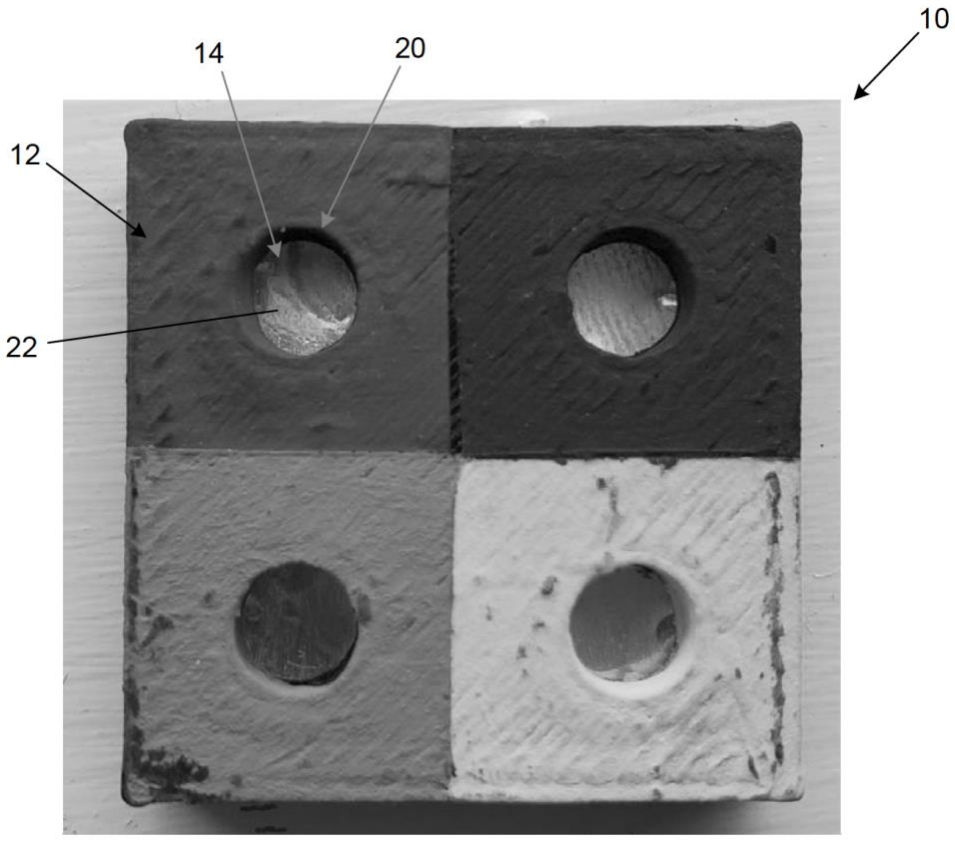


FIG. 1

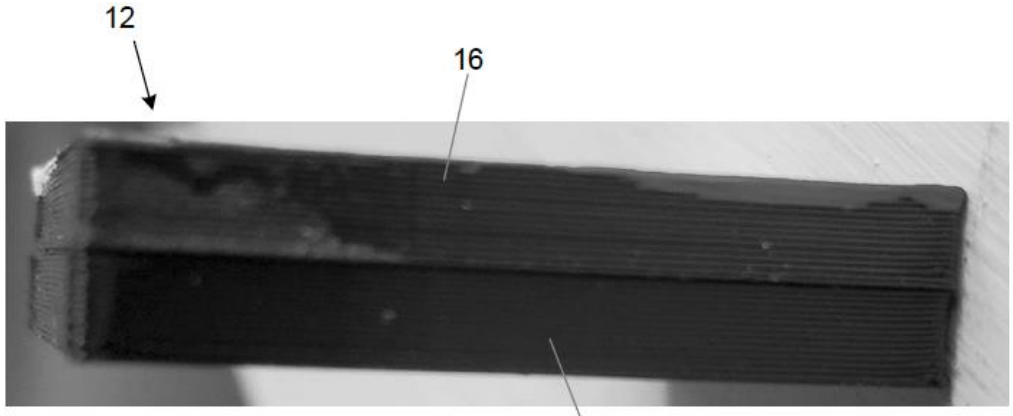
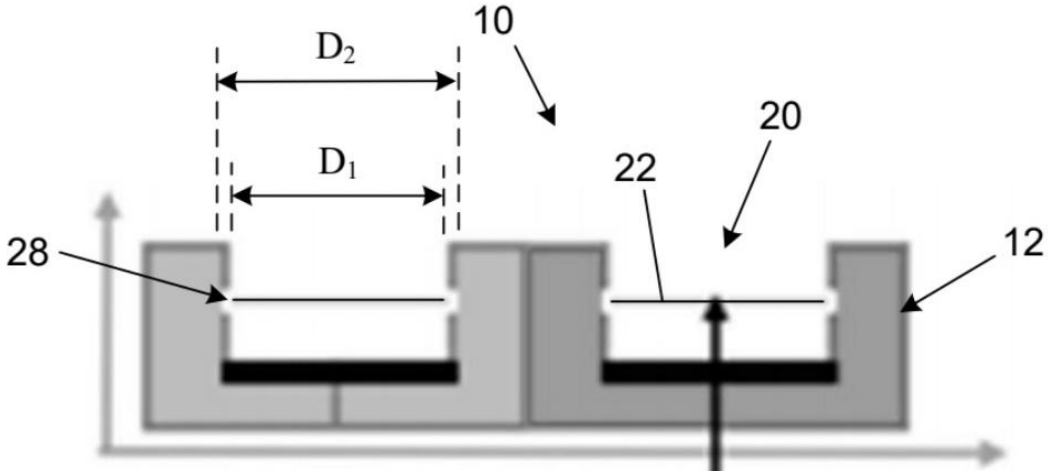


FIG. 2



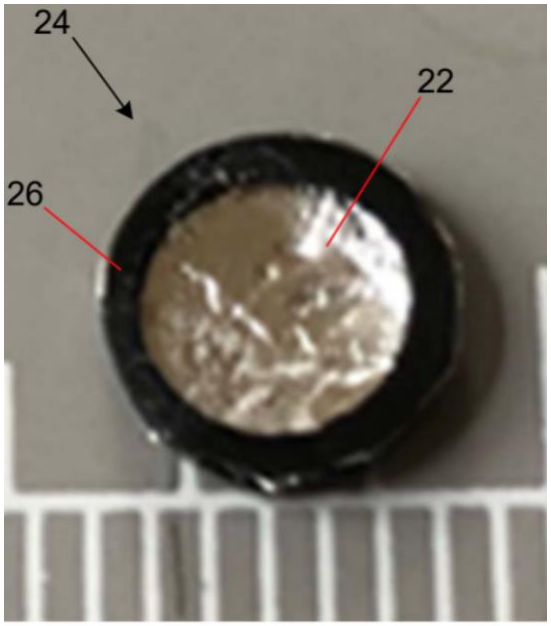


FIG. 3