



Review Article

Wartime spine injuries: understanding the improvised explosive device and biophysics of blast trauma

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Abstract

The improvised explosive device (IED) has been the most significant threat by terrorists worldwide. Blast trauma has produced a wide pattern of combat spinal column injuries not commonly experienced in the civilian community. Unfortunately, explosion-related injuries have also become a widespread reality of civilian life throughout the world, and civilian medical providers who are involved in emergency trauma care must be prepared to manage casualties from terrorist attacks using high-energy explosive devices. Treatment decisions for complex spine injuries after blast trauma require special planning, taking into consideration many different factors and the complicated multiple organ system injuries not normally experienced at most civilian trauma centers. Therefore, an understanding about the effects of blast trauma by spine surgeons in the community has become imperative, as the battlefield has been brought closer to home in many countries through domestic terrorism and mass casualty situations, with the lines blurred between military and civilian trauma. We set out to provide the spine surgeon with a brief overview on the use of IEDs for terrorism and the current conflicts in Iraq and Afghanistan and also a perspective on the biophysics of blast trauma. Published by Elsevier Inc.

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Introduction

The improvised explosive device (IED) has been the most significant threat by terrorists worldwide and is the leading cause of injury and death for servicemembers operating in Afghanistan and Iraq. To date, there have been more than 50,000 coalition forces injured or killed by explosive devices [1,2]. There has also been a 19% increase in IED-related coalition force casualties in Afghanistan during 2010, which has been the result of increased operational tempo, increased volume of IED placement by insurgent forces, and evolving insurgent doctrine with specific targeting of dismounted forces and increased use of suicide bombs [1,3]. Improvised explosive devices often require limited skill and technology and allow devastating attacks for a relatively small investment.

Exposure of servicemembers to high-energy blast trauma has brought unique challenges in the treatment of wartime spine injuries. Special considerations are necessary after high-energy blast trauma, and a multidisciplinary team is required to care for combat casualties with

complicated multiple organ system injuries. These injuries often involve a combination of highly contaminated extremity injuries and amputations, traumatic brain injury, and thoracic and visceral injuries [2,4–10] (Fig. 1). Combat casualties have also sustained an increased incidence of uncommon spine injury patterns, including chance fractures [11], low lumbar burst fractures, and lumbosacral dissociation injuries [4], (Fig. 2) as well as spine injuries associated with large degloving (Morel-Lavallee) or highly contaminated soft-tissue wounds. Although spine injuries are commonly encountered in civilian trauma, there have been few civilian trauma centers and spine surgeons experienced with the treatment of complex spine injuries after blast trauma. Therefore, treatment decisions for spine injuries after blast trauma require special planning, taking into consideration many different factors not normally encountered at most civilian trauma centers.

The treatment of high-energy wartime spine trauma has been extrapolated from the civilian trauma literature, with regard to their experience treating patients after motor vehicle collisions or falls from a height [12–15]. However, the tremendous energy imparted by an IED explosion is considerably different from civilian injury mechanisms and even from other injuries incurred during combat such

as gunshot wounds or vehicular accidents. Improvised explosive devices were responsible for more than 60% of US combat casualties during the Iraq conflict; however, nearly nine of 10 casualties survived [7,16,17]. This 10.1% case fatality rate is the lowest in history, compared with ground forces during World War II and Vietnam, which were 19.1% and 15.7%, respectively [16,17]. The decline in case fatality is likely multifactorial and may be a result of improvements in modern body armor, frontline battlefield care, far-forward placement of surgical teams, advances in intensive care and surgical capabilities, and significantly decreased medical evacuation times [16,18].

Although most IED attacks occur as a result of military conflict, surprisingly a land mine casualty occurs every 20 minutes and an average of 260 IED incidents per month outside the areas of conflict in Afghanistan and Iraq [3,19]. In addition, just within the United States, there was an average of 205 casualties per year between 2004 and 2006 from criminal bombing incidents [1]. Despite these statistics, most civilian medical providers have limited experience with blast-related injuries, and it is conceivable that they may be called on to manage casualties after an IED attack [16,20,21]. Therefore, we set out to provide the spine surgeon with a brief overview on the use of IEDs for terrorism and the current conflicts in Iraq and Afghanistan and also a perspective on the biophysics of blast trauma.

Understanding the IED

The US Department of Defense has broadly defined an IED as a “device placed or fabricated in an improvised manner incorporating destructive, lethal, noxious pyrotechnic, or incendiary chemicals, designed to destroy, disfigure, or harass ...” [22]. Improvised explosive devices not only inflict devastating injuries but are also used to intimidate local populations, challenge legitimate government authority, and restrict or slow coalition force freedom of movement [3]. Terrorist groups have easy access to commercial technologies, training via the Internet, the ability to manufacture or procure explosive materials, improvement in coordination/communication, and expansive financial support networks to continue their IED campaigns [3]. However, a common misconception is that IEDs are limited to crude homemade explosives placed on roadsides to target military convoys. The term “IED” encompasses a wide spectrum of explosive weapons, ranging from rudimentary homemade explosives or unused artillery rounds to sophisticated weapon systems and triggering devices containing high-grade explosives [1,20].

The various forms of IEDs have become increasingly lethal, while providing the enemy with standoff, precision lethality and near total anonymity [1]. However, all explosive devices, regardless of their use, are characterized by three elements: a fusing mechanism, explosive mixture, and casing [19]. The fusing element allows controlled



Fig. 1. Combat casualty after improvised explosive device attack, sustained bilateral lower extremity amputation (Top), open lumbar and sacral spine fractures, and severe soft-tissue wounds to gluteal and presacral regions (Bottom).

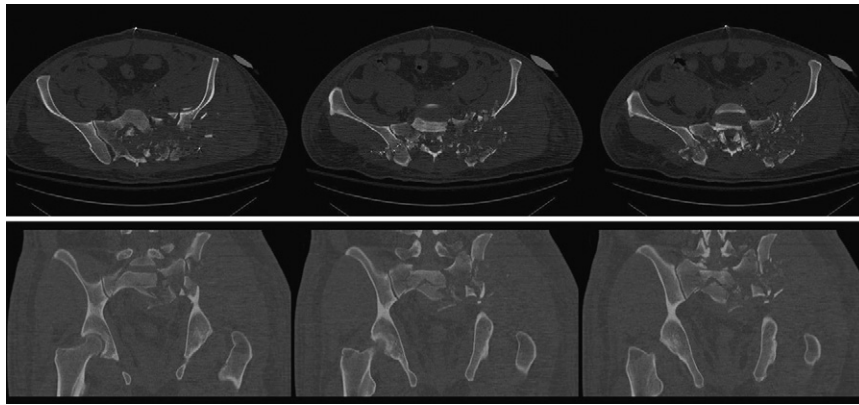


Fig. 2. Computed tomography demonstrating lumbosacral dissociation injury. Axial (Top) and coronal reconstruction images (Bottom).

initiation for the blast, and insurgents have used techniques such as boosting, coupling, and daisy chaining to increase the lethality of IEDs. Boosting involves fusing multiple buried IEDs, stacked on top of one another to increase the upward force of a blast. Coupling is a method used to link the initiation of multiple IED explosions to increase the maximum effective radius, a term used to describe the distance at which casualties can be expected [22]. Daisy chaining describes sequential initiation of multiple IED explosions and allows a vehicle with a mine roller device to first pass over multiple sequentially fused explosive devices without causing detonation (Fig. 3). Once a trigger IED is reached and detonated by the mine roller device, a “daisy chain” of explosions of overpassed IEDs underneath the vehicle or convoy of vehicles is initiated [23].

Second, the explosive mixture provides the energy for the blast, and the type and amount of explosive mixture incorporated into the IED principally determines its destructive power [19,24]. The power of an explosive is often rated in terms of its explosive equivalent to trinitrotoluene (TNT) [19]. There is a large spectrum of explosive mixtures, including solids and liquids, and differences in explosive mixtures are largely regional and based on supply and technology (Table 1) [1,20,22,25,26]. Previously, ammonium nitrate/fuel oil was a readily available explosive mixture in Afghanistan; however, recent countermeasures by coalition and local security forces have banned its production [3].

The third component of an IED device is the casing. The stochastic injury pattern of each individual IED is largely because of the bomb casing and modifications made by insurgents and terrorists to maximize fragmentation [19]. Conventional military ordinance propels fragments created by the breakup of the metal casing surrounding the explosive. In contrast, IEDs have been purposely designed to increase destructive power through secondary projectiles and fragments by incorporating metal objects, such as nails, nuts, bolts, or ball bearings, packed inside or around the explosive device [1,19]. Terrorists and insurgents have also used vehicle-borne IEDs as another method to increase fragmentation injury because of the breakup of the vehicle during the explosion and also because larger amounts of explosive material can be placed into the vehicle versus other casings or containers.

Another specially designed IED that deserves mention is the explosively formed penetrator, which has been a prominent feature of military operations during the Iraq conflict. Also termed a “shape charge,” an explosively formed penetrator involves the use of an explosive device to propel a metal projectile at a significant velocity allowing penetration into an armored vehicle [1,22,27,28]. A typical explosively formed penetrator involves a cylindrical casing filled with explosive material, capped with either a saucer-shaped or conical piece of metal [29]. Detonation of the explosive material causes the metal cap to become a molten

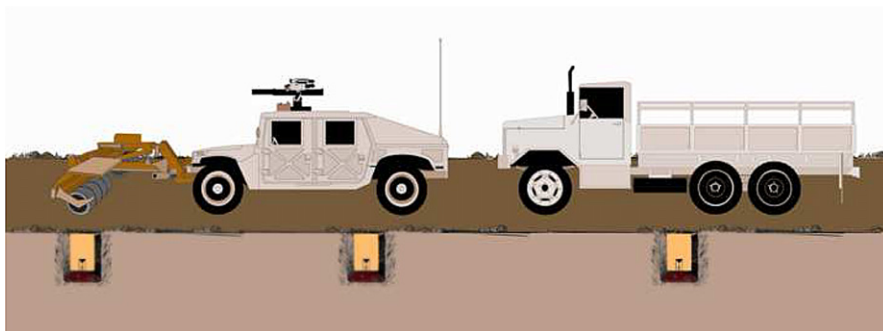


Fig. 3. Illustration of daisy chain method of improvised explosive device detonation to counteract mine roller device.

Table 1
Common explosive types and characteristics [1,20,21,23–25]

Explosive type	TNT equivalent	Consistency	Characteristic
ANFO	0.82	Powder	Used in Oklahoma City attack in 1995 (4,800 lbs TNT equivalent)
NG	N/A	Liquid	Highly volatile; used to make dynamite
TNT	1.00	Solid	Pipe bomb (5 lbs TNT) 155-mm artillery round (220 lbs TNT) Car bomb (2,000 lbs TNT)
Composition B	1.11	Solid	40% TNT+60% RDX
RDX	1.19	Solid	
PETN	1.27	Solid	
Composition C4	1.37	Plastic	91% RDX+(rubber, oil, plasticizer); triggered by mechanical shock wave resulting in rapid detonation (“brisanse”)

TNT, trinitrotoluene; ANFO, ammonium nitrate/fuel oil; NG, nitroglycerin; RDX, cyclotrimethylenetrinitramine; PETN, pentaethyltrinitride; N/A, not applicable.

projectile, propelled by a high-pressure wave (up to 30 million psi) along the axis of symmetry of the cylindrical casing, accelerating it up to velocities of 6,600 mph [22]. On impact with an armored vehicle, the projectile “penetrates” the target and dissipates a significant amount of kinetic energy [22].

Another tactic used by insurgents and terrorists to maximize casualties involves complex multistaged ambushes and explosions, which were devised after studying coalition force combat doctrine. An example includes the use of secondary IEDs in which a military convoy or patrol is brought to a halt with an initial IED blast, and after troops dismount from the protection of their armored vehicles, a larger and more powerful IED is detonated. Insurgents and terrorists have also attacked targets of opportunity, such as medical personnel and evacuation helicopters, by identifying potential casualty collection areas and landing zones and triggering preplaced IEDs or ambushing these areas during medical evacuation [1].

And finally, IED attacks have increasingly been in the form of suicide bombings in which the insurgent or terrorist is indistinguishable from the civilian population. This allows the explosive device to be mobilized through public spaces and detonated at the optimum opportunity to cause maximal indiscriminate injury [22]. The detonation of a suicide bomb is meant to generate fear, chaos, and dramatize the effect of the attack through targeting of busy restaurants or nightclubs, crowded public transportation, military recruiting stations, or open public spaces [30–33]. These suicide bombs force change in everyday behavior and cause an additional level of psychological and emotional damage on the local population and coalition forces [22]. Suicide bombs have increased in lethality with the use of high-grade military explosives, and as previously described, with the addition of projectile material surrounding the casing to intensify fragmentation injury.

Biophysics of blast trauma

Explosions produce complex and astonishing injury patterns, with multisystem involvement including pulmonary

injury, traumatic brain injury, burns, amputations, crush syndromes, and blunt or penetrating injuries to the viscera, axial skeleton, and extremities [1]. The behavior and characteristics of these blast events have been difficult to predict and generalize because the variable severity and spectrum of injury with each attack is largely related to the explosive weight and material, as well as the preparation and detonation technique of the IED device. However, an increasing understanding of the basic blast biophysics and pathophysiological effects will allow better planning and treatment of the consequences after IED attacks.

Explosive devices cause injury by multiple mechanisms, some of which are exceedingly complex. The wide spectrum of explosion-related injuries are often collectively referred to as “blast injuries” and has disguised the fact that most debilitating and lethal wounds are predominately caused by penetrating fragments, not by blast overpressure [1]. Therefore, when discussing explosion-related injuries, there must be a clear understanding of the four types of blast injuries, which include: primary, secondary, tertiary, and quaternary (Table 2) [1,19,22,27].

When an explosive device is detonated, the explosive material undergoes a rapid exothermic chemical reaction. This releases a significant amount of stored potential energy by transforming the explosive material from a liquid or solid to a gas [22,24,34]. This highly compressed superheated gas, termed the detonation product, then rapidly expands with a local pressure typically reaching 1.4 to 3 million psi and temperatures ranging from 2,000°C to 6,000°C [22]. This gas expansion then instantaneously compresses the surrounding ambient air, forming a blast wave that propagates supersonically and radially from the detonation site. The air is highly compressed at its leading edge, termed the blast front, and is the principle factor causing primary blast injury or what is commonly referred to as “overpressure” injuries [35,36]. The blast front interacts with the human body by causing a rapid change in pressure at the moment of impact and transfers an impulse of energy from the transmitting medium (air or water) to the body surface [24,25]. As the blast wave and blast front quickly dissipate, they are followed by the blast wind. The

Table 2
Blast injury classification

Classification	Description	Injury pattern
Primary [1,20,21,24,27]	<ul style="list-style-type: none"> • “Overpressure” injury • “Implosion” occurs at time of contact with body surface, blast front rapidly compresses gas-filled organs and then near instantaneously reexpands as blast front passes • “Spalling” occurs as blast front propagates through body, significant shear and stress forces because of differences in tissue density of adjacent organs and tissue at air-fluid interfaces; causes forcible explosive movement of fluid from more dense to less dense tissues 	<p>Implosion injuries</p> <ul style="list-style-type: none"> • Auditory shift (2 psi) • Tympanic membrane rupture (5 to 15 psi) • Lung injury; pneumothorax; pneumomediastinum; air embolism; intestinal emphysema (30 to 80 psi) • 50% chance of death (130 to 180 psi) • Probable death (200 to 250 psi) <p>Spalling injuries</p> <ul style="list-style-type: none"> • TBI • Gastrointestinal tract injury • Tearing of organ pedicle • Eye injury
Secondary [1,24,38,40–42]	<ul style="list-style-type: none"> • Ballistic injury from primary bomb casing fragments; also from secondary fragments (ie, environmental material, metallic debris, glass); become projectile after energized by explosion • Fragments strike the body and cause penetrating injuries; can also cause traumatic amputations • Variable velocity depending on size/shape of fragment and distance from explosion epicenter; rapid deceleration because of aerodynamic drag • “Shimmy” effect from irregularly shaped fragment contacts body and exhibits tumbling; increases amount of local tissue damage 	<ul style="list-style-type: none"> • Penetrating injury • Traumatic amputation • Laceration • TBI
Tertiary [1,23,26]	<ul style="list-style-type: none"> • Whole body translocation • Blast wave energizes and propels individual to tumble along the ground or thrown through air to strike hard surface • Large object may become projectile and impact individual causing significant blunt or crushing injuries • Crush injuries caused by structural damage and building collapse 	<ul style="list-style-type: none"> • Blunt injury • Crush injury • Compartment syndrome • TBI
Quaternary [1]	<ul style="list-style-type: none"> • All other explosion-related injuries 	<ul style="list-style-type: none"> • Burn injury • Toxic gas or smoke inhalation injury • Asphyxiation

TBI, traumatic brain injury.

blast wind is a region of high pressure, which travels slower than the blast wave, propelling fragmentation, large objects, and humans considerable distances and is the principle factor causing secondary and tertiary blast injuries [24,25,37]. Although termed secondary blast injury, this mechanism continues to be the most common cause of injury and death after an explosion [1,25,27,38]. The severity of injuries and risk of death increase with a larger amount of explosive material and at a closer distance to the explosion epicenter.

There has been an overemphasis on primary blast injury, with only 3% to 5% of injuries after an explosion occurring from primary blast injury, with most being tympanic membrane ruptures [1]. This is because an intense blast is necessary to produce manifestations of primary blast injury other than tympanic membrane rupture, and there is a rapid decrease in peak overpressure with increased distance from the explosion epicenter. Secondary blast injury is much more common because fragments after an explosion can

travel long distances from the explosion epicenter with initial velocities up to 6,000 m/s. [19,32,39–43] At close range, potentially survivable fragmentation injuries are unlikely, and in an open space environment, the maximum effective range for secondary blast injury exceeds primary blast injury by a factor of 100 (Fig. 4) [1,24,44,45]. Although injury patterns after IED explosions are exceedingly complex, in general, a casualty close enough to sustain a significant primary blast injury will likely be killed by fragmentation [1,24].

Blast biophysics of open versus closed space environment

There are many factors that influence blast trauma biophysics; however, the environment in which the explosion takes place has a significant role and must be considered [25]. Although the current conflicts in Iraq and Afghanistan

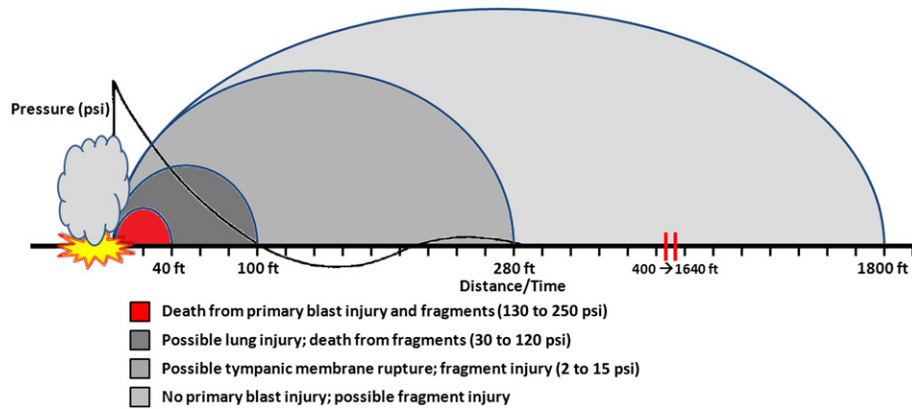


Fig. 4. Illustration of “Friedlander curve,” with maximum effective radius for primary and secondary blast injuries of an open-field 155-mm mortar shell explosion with 200 lbs (100 kg) of trinitrotoluene equivalent; potential injury from fragmentation can exceed 1,800 ft [1,19,24,45]. Adapted from Champion et al. [1].

have predominately experienced open space environment explosions, there are substantial different effects when an explosion occurs within an enclosed space [1]. In an open space environment, the nearly instantaneous peak in ambient air pressure quickly decays as it travels away from the explosion epicenter through a well-defined pressure/time curve called a “Friedlander wave” [24,25] (Fig. 4). In an enclosed space, this typical relationship does not occur, as blast waves deflect, reflect, and coalesce, which can magnify the destructive power eight to nine times and cause significantly greater injury [25,28,40,46,47] (Fig. 5). As a consequence, immediate death from primary blast injury from pulmonary injury occurs more often in an enclosed space explosion [28,46,47]. The increased energy of the complex and reflected waves can also generate a larger number of secondary fragments through destruction of the building or vehicle, with an increased likelihood of tertiary blast injury from structural collapse [22]. During the 1995 Oklahoma City bombing, in the uncollapsed portion of the building, 5% died and 18% of survivors were hospitalized,

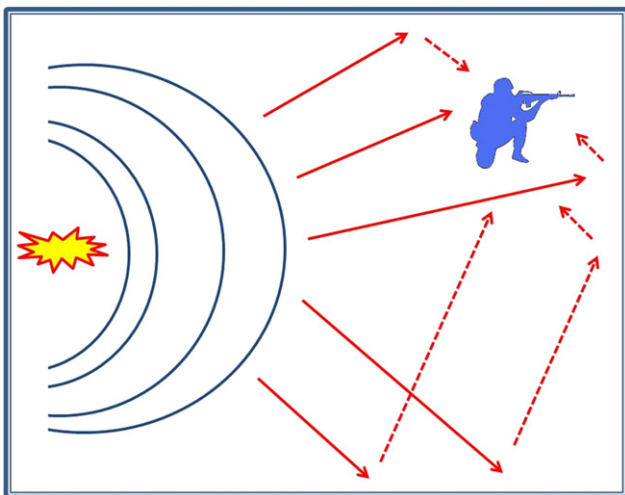


Fig. 5. Illustration of blast wave magnification in an enclosed space.

whereas in the collapsed portion of the building, 87% died and 82% of survivors were hospitalized [1,48]. In another study, closed space bus bombings were found to reach mortality of nearly 50%, whereas open space environment bombings were associated with an 8% mortality [1,28].

Blast interaction with vehicles

There is a particularly complex sequence of injury mechanisms when an explosion occurs outside of an enclosed space such as a building or vehicle. The occupants are protected to some extent from primary blast injury as the blast wave diffracts around and reflects off the exterior of the building or vehicle, with only a small portion transmitted into the interior of the structure [1]. Holcomb et al. [17] demonstrated that the risk of overpressure injuries is substantially reduced when inside a vehicle, with the peak overpressure outside a vehicle approximately 28 times than that inside the vehicle when a 17-kg explosive is detonated 3-m away [1]. For example, after an IED attack, an individual inside a vehicle exposed to an overpressure of 5 psi may result in tympanic membrane rupture, whereas a combatant outside the vehicle and protected from fragment injury would likely experience an overpressure of 150 psi, which can result in a 50% chance of death from primary blast injury [1,37]. Although offering protection from primary blast injury, there can still be significant injury if there is enough energy to cause fragmentation of building or vehicle material, projectile glass from broken windows, or complete destruction or collapse of the building or vehicle. A vehicle may also experience enough momentum to cause acceleration and displacement and is particularly prone to vertical displacement if a flat vehicle floor traps the detonation product and allows for considerable pressure concentration and energy transfer and may result in vertical acceleration and displacement of the vehicle [20]. This can also cause rupture of the vehicle floor and endangers the occupants by exposing them to secondary fragmentation and superheated high-pressure gases leading to quaternary blast injury (Fig. 6, Left). Therefore,

a more slender or V-shaped vehicle floor allows the detonation product to flow and bend along the path of least resistance and is released around the vehicle (Fig. 6, Right) The development of a V-shaped hull sitting high on the vehicle chassis dates back to World War II, with the first implementation in the Swedish Armored Personnel Carrier during the North Africa campaign [20]. The mass of the vehicle also has a significant effect on acceleration and displacement of the vehicle, with the most basic relationship demonstrating acceleration to be inversely proportional to mass (acceleration=force/mass) [20]. Therefore, for the same explosion intensity, a heavier vehicle with a V-shaped hull will experience decreased acceleration, peak velocity, and displacement compared with lighter flat hull vehicle [20].

Tertiary blast injury is the most significant injury mechanisms for occupants in a vehicle after an IED attack and can occur at several different times after the explosion event. First, the forces causing vertical acceleration of the vehicle can be transferred to the seated occupant causing lower extremity, pelvis, and spinal column injuries. The occupant can then incur significant head and spinal column injuries from striking the roof of the vehicle, as they are thrown around inside or ejected from the vehicle; especially with only limited seat belt use and availability and no air bag restraint system available in military vehicles [1]. After reaching the peak of its force-dependent displacement, the vehicle then accelerates back to the ground where the occupants are again subjected to blunt- or crushing-type injuries at impact [20]. Finally, there can also be significant injury if poorly stowed or inadequately restrained equipment becomes projectile and strikes the occupants [20].

Many of our combat casualties have occurred by IED attacks on vehicle convoys, and there have been advances in military vehicle designs to improve survivability after an explosion, and most recently with the development of the Mine Resistant Ambush Protected armored vehicle. A number of different strategies have been implemented to reduce the lethality and morbidity of the four different blast injury mechanisms. Increasing the distance between the epicenter of the explosion and the crew compartment can reduce primary

blast effects; either through mine roller devices or through increased height of the crew compartment. Improvements in vehicle armor, blast deflectors, and personal protection to prevent fragment penetration have reduced the secondary blast injury. Tertiary blast injuries have been countered through altering vehicle geometry and structure, increasing vehicle mass, and blast deflectors to reduce energy transfer to the vehicle [20]. Quaternary injury, particularly protection from thermal energy, is through fire-resistant materials in the vehicle structure and in personal clothing, as well as separating the crew compartment from the fuel cell and ammunition storage.

Conclusion

Blast trauma has produced a wide pattern of combat injuries not commonly experienced in the civilian community. Spine injuries after an IED blast are likely multimechanistic, but most are caused by secondary and tertiary blast injuries resulting in blunt- and crushing-type injury patterns. We have experienced an unusual increase in rare injury patterns, such as low lumbar burst fractures and lumbosacral dissociation injuries, and although several different mechanisms have been postulated, there is no convincing evidence to date to further focus our research efforts. Despite the significant impact of explosion-related injuries on our deployed servicemembers, there has only been limited literature concerning the epidemiology, treatment, and outcomes of wartime spinal column and spinal cord injuries [2,5,7,49,50]. In fact, most studies regarding wartime-related spine injuries have focused on epidemiology and wounding patterns, with only limited treatment and outcomes-related data. However, as our understanding of the epidemiology and patterns of injury continues to expand, we hope to further elucidate the optimal clinical treatment algorithm based on patient outcomes and focus our research efforts to prevent injury through personal or vehicle protection design. Also, little is known about the long-term consequences on spine health after exposure to blast

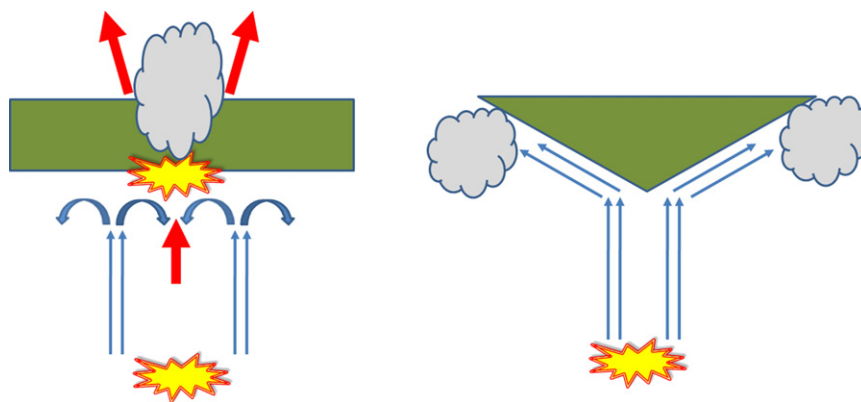


Fig. 6. Illustration of detonation product buildup and increased impulse under flat hull, with rupture of vehicle floor and occupants exposed to fragmentation and superheated high-pressure gases (Left); detonation product flowing around V-shaped vehicle hull (Right) [20].

trauma, particularly after multiple exposures in the absence of obvious spine injury. While the treatment and immediate consequences of the combat casualty with spine injury have been the purview of military orthopaedic spine surgeons and neurosurgeons, spine surgeons throughout the community will be faced with a growing population of war veterans who have been exposed to blast trauma and report disability from back pain or previous spine injury. Also, an understanding about the effects of blast trauma by all spine physicians in the community has become imperative, as the battlefield has been brought closer to home in many countries through domestic terrorism and mass casualty situations, with the lines blurred between military and civilian trauma [30–33,51–53]. We hope to have encouraged further thought and innovation for spine injury prevention, provoked future experiments, or investigations to improve treatment of these complex spine injuries and stimulated awareness of the possible consequences on spine health after exposure to blast trauma.

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