WEAPONS EFFECTS

Chapter 2

Contributing Authors
Sidney B. Brevard, MD, MPH, FACS, COL, US Air Force
Howard Champion, MD, FRCS, FACS
Dan Katz, MD

Disclaimer

The opinions and views expressed herein belong solely to those of the authors. They are not nor should they be implied as being endorsed by the United States Uniformed Services University of the Health Sciences, Department of the Army, Department of the Navy, Department of the Air Force, Department of Defense, or any other branch of the federal government.

Table of Contents

Introduction

Understanding modern warfare, including the types of weapons employed and the mechanisms and patterns of injury they cause, is critical to providing optimal combat casualty care (CCC). Certain types of weapons (e.g., improvised explosive devices) inflict patterns of injury that are repeatedly encountered by military careproviders. By recognizing these patterns and understanding the pathophysiology behind resultant injuries, CCC providers will be better prepared to treat the injured.

The Joint Theater Trauma Registry (JTTR) is a database used to track medical treatment information on troops injured in Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF). Data are collected at various points as injured troops receive medical treatment in-theater and at each medical facility overseas and in the United States (US). The information recorded is extensive and includes patient demographics, mechanism of injury, type of personal protective equipment (e.g., body armor, goggles, helmet) used, body regions injured, and more.[1]

A query of the JTTR database for wounds sustained between October 2001 and January 2005 revealed the following distribution of injuries: extremities (54 percent), head and neck (29 percent), abdomen (11 percent), and chest (6 percent).[2] This injury pattern differs from that of previous conflicts, which had a higher proportion of thoracic injuries and fewer head and neck injuries.[2,3,4,5,6] This shift is likely due to enhanced body armor that protects the chest and reduces mortality.[2] Enhancements in personal protective equipment (PPE) and the shift from conventional warfare to "a complex mix of conventional, set-piece battles, and campaigns against shadowy insurgents and terrorists" contribute to current wounding patterns, which differ from those of previous conflicts (i.e., World War II, Korea, and Vietnam).[7]

The JTTR database for wounds sustained in OEF and OIF between October 2001 and January 2005 reveals the following distribution of injuries: extremities (54 percent), head and neck (29 percent), abdomen (11 percent), and chest (6 percent).

The increase in explosion-related injuries and concomitant decrease in gunshot-related injuries in the past century and a half of US conflicts is summarized in Figure 1. This trend has accelerated substantially during recent years. This is illustrated by increases in explosion-related OEF and OIF casualties from 56 percent in 2003 to 2004 to 76 percent in 2006 and in the number of surgeries for fragment wounds from 48 percent in OIF I (2003) to 62 percent in OIF II (2004 to 2005) performed by US Navy/Marine Corps Forward Surgical Teams in OIF.[8,9]

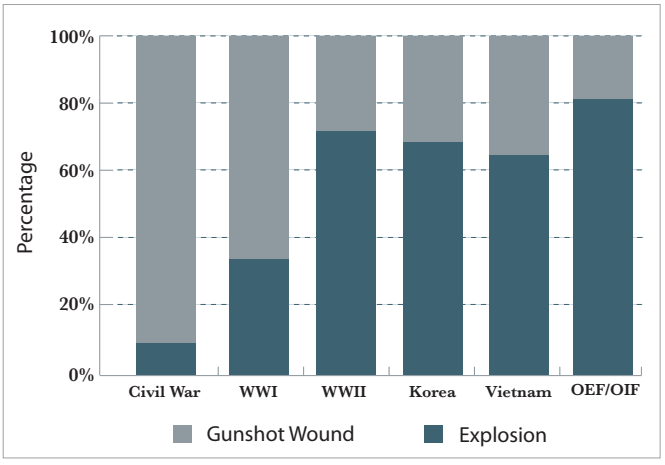


Figure 1. *Primary mechanisms of injury in United States wars.[2] Data sources: Civil War,[10] WWI and WWII,[11] Korea,[5] Vietnam,[6] OEF/OIF.[2]*

Weapons

The primary mechanisms of combat injury in OEF and OIF are small arms (pistols, shotguns, rifles, machine guns) and explosives (mortars, landmines, rocket-propelled grenades [RPGs], and improvised explosive devices [IEDs]). As of 2009, combat casualty statistics for hostile actions indicate that explosive devices are responsible for 80 percent of injuries and 81 percent of deaths in OEF, and for 86 percent of injuries and 90 percent of deaths in OIF.[12] These mechanisms and their effects are discussed below, followed by an overview of blast injury.

Small Arms

Current combat casualty statistics for hostile actions indicate that gunshot wounds are responsible for 22 percent of injuries and 27 percent of deaths in OEF, and for 8 percent of injuries and 19 percent of deaths in OIF.[12] Small arms are easily available in Iraq, which has an estimated combined military and civilian arsenal of seven to eight million firearms containing machine guns, submachine guns, sniper and assault rifles (including AK-47s and AK-47-style models such as the AKM), shotguns, pistols, and carbines.[13]

The degree of tissue damage resulting from small arms fire in OEF and OIF is highly variable. Combat casualty careproviders need to treat each patient's wound(s) individually. Wide surgical exploration of all bullet wounds is no longer routinely recommended.[14] Minimal tissue debridement is typically required for wounds resulting from small arms fire. As a bullet travels through tissue, a temporary cavity is created. Tissue damage in this temporary cavity is usually limited and may heal on its own without debridement.[15] Inelastic tissues, such as the brain and liver, will exhibit the most damage resulting from temporary cavitation. Elastic soft-tissue, such as lung, skeletal muscle, nerves, and blood vessels, may show minimal damage.[15] There may be cases when a bullet strikes bone or another structure and is deflected. In these cases, the damage could be more extensive and require larger debridement. Therefore, each case should be carefully evaluated and managed individually.[14]

The degree of tissue damage resulting from small arms fire is highly variable. Wide surgical exploration of all bullet wounds is no longer routinely recommended.

Explosives

Physics

With the prevalence of explosive weapons in use in Iraq and Afghanistan, it is important that CCC providers have a basic working knowledge of the physics behind explosions. Explosions are the result of chemical conversion of a liquid or solid into a gas with generation of energy. Explosives are classified as low- or high-order based on velocity of detonation (i.e., the interval between activation and release of the explosive energy). Knowing the type of explosive that caused a casualty's injuries is important because low- and high-

Low-Order Explosives	High-Order Explosives
• Dynamite • Gunpowder	• Ammonium nitrate • Nitroglycerin • 2,4,6-trinitrotoluene (TNT) • Pentaerythritol tetranitrate (PETN) • Cyclotrimethylene trinitramine (RDX) • Cyclotetramethylene tetranitramine (HMX) • Nitrocellulose

Table 1. *Examples of low- and high-order explosives.*

order explosives exhibit different patterns of injury and thus warrant different treatment considerations (Table 1).[16,17]

Low-Order

Low-order explosives, which include gunpowder and dynamite, produce their effect through a relatively slow burning process called conflagration.[18] The readily combustible substances in low-order explosives are used primarily for propelling projectiles, but also take the form of pipe bombs and petroleum-based bombs (e.g., Molotov cocktails). The blast wave generated by a low-order explosive typically has a speed of less than 2,000 meters-per-second (m/sec). Low-order explosives have secondary, tertiary, quaternary, and sometimes quinary effects (see classifications described later). Importantly, they do not have the primary blast effects characteristic of high-order explosives.

High-Order

Single-compound high-order explosives include ammonium nitrate, nitroglycerin, 2,4,6-trinitrotoluene (TNT), pentaerythritol tetranitrate (PETN), cyclotrimethylene trinitramine (RDX), cyclotetramethylene tetranitramine (HMX), and nitrocellulose. These compounds may be combined to form mixed-compound explosives, such as dynamite, composition C4, ammonium nitrate/fuel oil (ANFO), and sheet explosives.[19] Commonly-used polymer-bonded high explosives (Gelignite, Semtex) have one and one-half times the power of TNT.

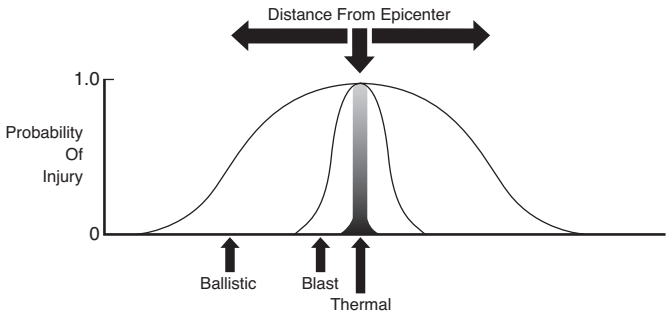


Figure 2. *As a blast wave travels away from the site of detonation, it rapidly loses both pressure and velocity. Combat blast injuries patterns often depend on the proximity of the individual to the site of detonation. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.*

High-order explosives react very quickly and generate heat and energy almost instantaneously. Products of the explosive reaction occupy a greater volume than that filled by the original reactants. This results in a supersonic, superheated rise in pressure called a blast wave, which moves at speeds of 3,000 to 8,000 m/sec.[20] The blast front is the leading edge of the blast wave and has a shattering effect known as brisance. As the blast wave travels away from the site of detonation, it rapidly loses both pressure and velocity.[18,21] The duration and magnitude of the blast wave's peak depend on a host of factors, including

the type of explosive used and the conducting medium.

The blast wave propels fragments with enormous force, generates environmental debris, and often causes intense thermal radiation. Its effects vary with distance from the detonation site (Figs. 2 and 3). High-order explosives are often used in military ordnance and their characteristic brisance can crush soft-tissue and bone and propel debris at ballistic speeds (fragmentation). Unlike low-order explosives, high-order explosives create blast overpressure injuries (barotrauma). As the blast wave passes, a temporary relative vacuum is created as gases continue to expand from their point of origin, and a transient blast wind may travel immediately behind the blast front. In the vicinity adjacent to an explosion, this force can cause traumatic amputation, evisceration, or total disintegration of a body. The blast wind may also cause injury by accelerating the speed of debris and fragments that subsequently strike the victim, or by displacing the victim against a stationary object.[19] These types of injuries are discussed in detail below.

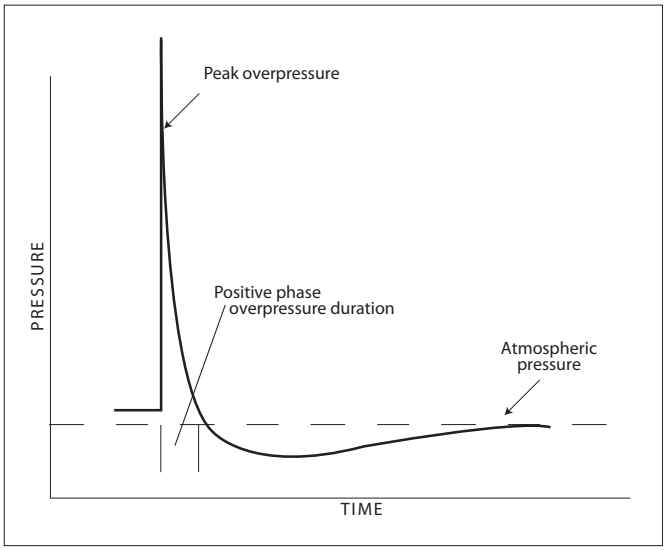


Figure 3. *Idealized blast overpressure waveform. An explosion creates a nearly instantaneous expansion of gas that compresses the surrounding medium (air or water) generating a blast wave. As it travels away from the site of detonation, the blast wave rapidly loses its pressure and velocity with distance and time.*

Low-order explosives have secondary, tertiary, quaternary, and sometimes quinary effects. Importantly, they do not have the primary blast effects characteristic of high-order explosives. High-order explosives can create significant overpressure injuries, especially at close range.

Devices

Explosive devices, including artillery, mortars, rockets, grenades, and RPGs are responsible for more than 3,600 deaths and almost 31,000 injuries of US troops in the current conflicts in Afghanistan and Iraq.[12] Explosive devices are the weapon of choice of terrorists and insurgents, and are becoming ubiquitous in combat theaters and civilian venues alike. The major categories of explosives are landmines and unexploded ordnance, RPGs, and, most commonly, IEDs.

Antipersonnel Landmines and Unexploded Ordnances

Landmines and unexploded ordnances (UXOs) are often discussed together because it can sometimes be difficult to separate the injuries clinically. Landmines are a form of ordnance that are placed on or under ground and explode when triggered, generally by electromagnetic waves or direct pressure (e.g., being stepped upon).[22] Unexploded ordnances include bombs, grenades, missiles, rockets, and mortar and artillery shells that were fired or dropped and did not explode.[23]

Injuries from landmines and UXOs are a risk for civilian and military personnel alike and are a worldwide problem. Landmines and UXOs are common in both Iraq and Afghanistan. Because it has been involved in intense conflict for decades, Iraq is considered one of the most heavily landmine and UXO-contaminated

countries in the world. Landmines and UXOs are particularly prevalent in the north along Iraq's border with Iran and in the central and southern regions as well.[24] In Afghanistan, the International Committee of the Red Cross reports that there are 10 million landmines and more than 50 different types of landmines, and that the most heavily mined areas are along the border with Pakistan and around the cities of Kabul and Kandahar.[25] There are sections of Bagram Air Base, Afghanistan that are still not clear of landmines and are cordoned off to prevent troops from accidentally entering that area. Many of the landmine and UXO victims treated at US military medical facilities are civilians.

A recent report from the US Centers for Disease Control and Prevention (CDC) compiled data from several sources to evaluate landmine and UXO injuries in Afghanistan over a six-year period.[26] Major findings included the following: (1) almost all of the injuries were sustained by men; (2) more than half of the injured were under the age of 18 (one-third were between the ages of 10 and 14); (3) children were twice as likely to be injured by UXOs as adults, although the case-fatality rate (7 percent) was the same for both; and (4) adult males were more likely to be injured by landmines as they traveled for work or military activity, whereas children were more likely to be injured while playing with a newly found object that turned out to be an UXO.[27] These trends were confirmed in later studies.[23]

Landmines and UXOs cause injury through the blast effects described below (i.e., primary blast effect, secondary fragments, tertiary [whole-body propulsion], and quaternary [burns]).[28] The three main types of conventional antipersonnel landmines are blast or static, bounding fragmentation, and directional fragmentation; each has an associated pattern of injury (Table 2).

Type of Mine	How Concealed	How Detonated	Primary Areas of Wounding
Blast or static	Buried just below ground surface	Pressure (e.g., being stepped upon)	Foot, upper leg, lower leg
Fragmentation			
• *Bounding*	Buried just below surface with fuse protruding, or laid on surface	Fuse or tripwire	All
• *Directional*	Laid on surface	Electrical charge, timed fuse, or tripwire	All

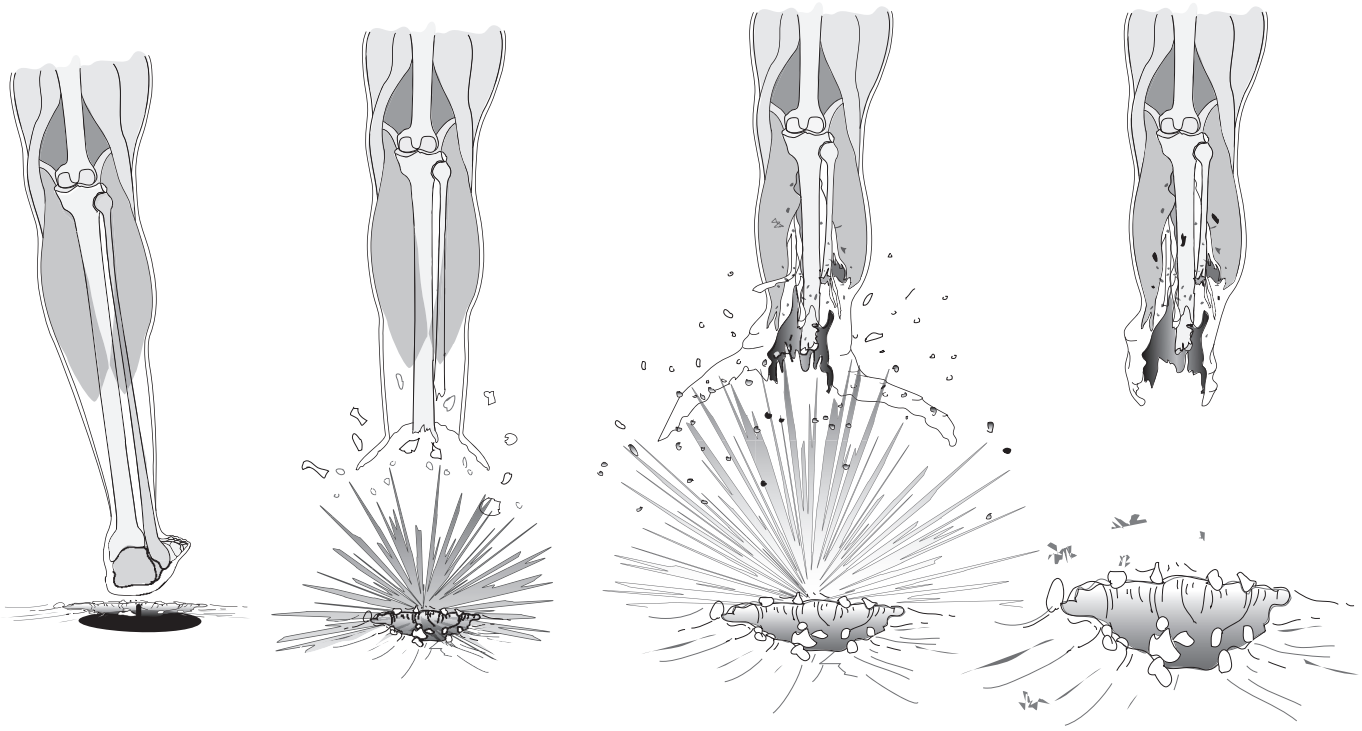
Table 2. *Categories of Antipersonnel Landmines. Adapted from Bellamy, 1991[10] and the International Committee of the Red Cross.[29]*

Blast (Static) Landmines

Static landmines are small mines planted and designed to activate when a person steps on them (Fig. 4). Many of these devices are designed to injure but not kill an individual.[30] However many are lethal, either due to the immediate injury or to subsequent uncontrolled hemorrhage. There are classically two patterns of

Figure 4. (Right) *Example of static landmine. Image courtesy of the United Nations Mine Action Service.*

Figure 5. (Below) *Static landmines detonate when stepped on, resulting in partial or complete lower limb amputation, most commonly at the midfoot or distal tibia. Debris may be driven up along fascial planes with tissue stripped from the bone. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC. Illustrator: Bruce Maston.*



injury: (1) complete or near-complete amputation of the foot (Fig. 5); and (2) random penetrating fragment injuries along the tissue and fascial planes of the lower leg (Fig. 6).[31] When these types of mines explode, particles of the dirt in which they were buried, debris, clothing, bone, and mine fragments can be driven by the blast up the leg into the upper or mid-calf causing gross contamination.[10,15]

Fragmentation Landmines

The two types of fragmentation landmines are bounding and directional fragmentation landmines (Fig. 7). The bounding type of antipersonnel mine is so named because it bounds upward and then explodes mid-air at approximately torso level. Upon detonation, this type of mine propels hundreds of fragments in all directions (as far as hundreds of meters), inflicts injuries higher in the body (e.g., torso, upper extremities, neck, or head) compared to static mines, and has the highest mortality of any landmine type.[15] Perhaps the

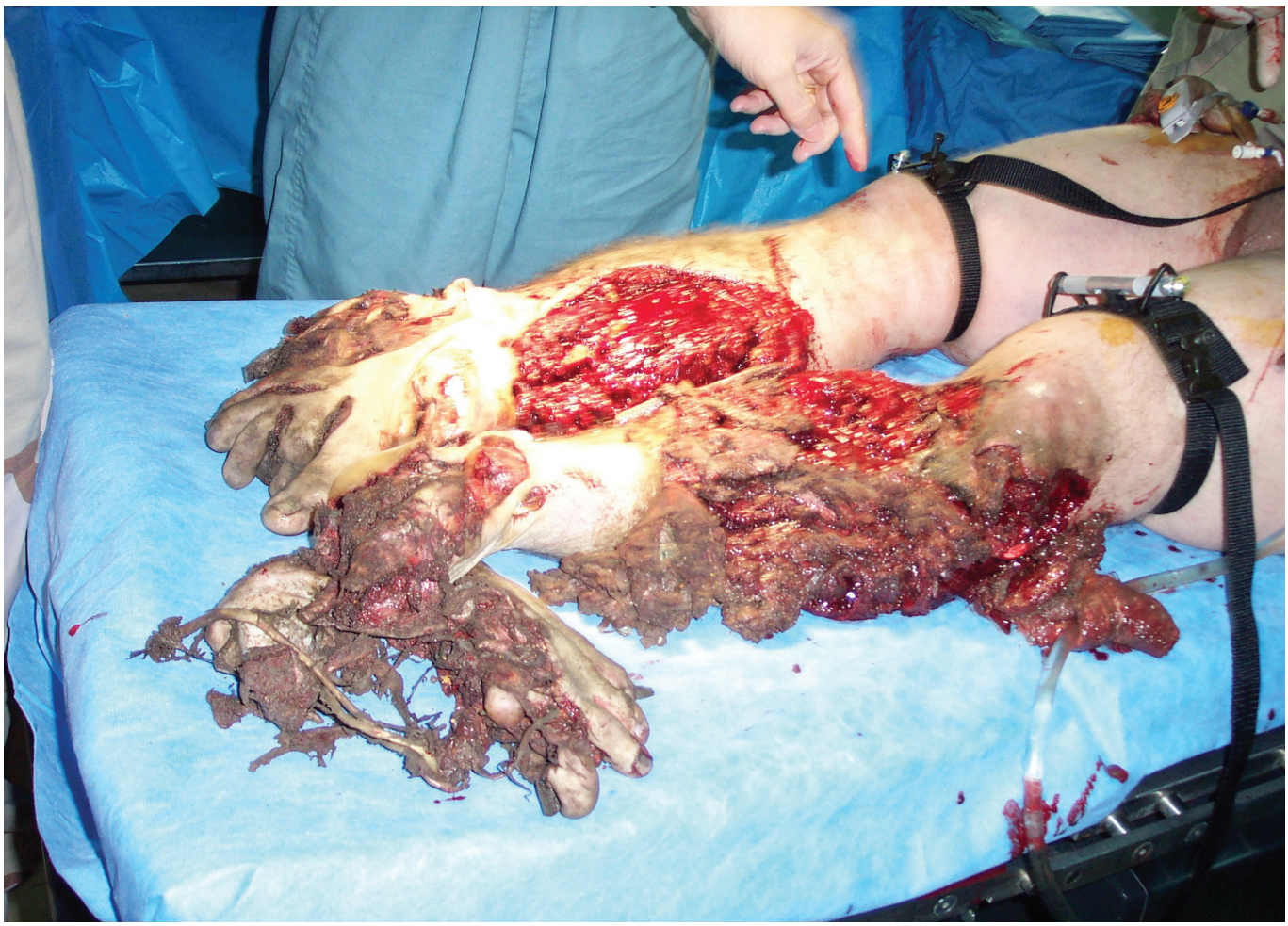


Figure 6. (Above) *A landmine blast leads to an umbrella effect in which the soft tissues, vessels, and nerves are stripped from the bone. This results in a more proximal injury than may be clinically apparent. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.*

Figure 7. (Right) *When triggered, fragmentation mines project a lethal shower of metallic fragments in all directions. The bounding types are projected upwards, prior to exploding mid-air at approximately torso level. Image courtesy of the United Nations Mine Action Service.*

best-known type of bounding mine is the M16A2 or "Bouncing Betty," which was developed in the 1930s and widely used during World War II.[10,32]

Directional Fragmentation Landmines

Upon detonation, directional fragmentation landmines project fragments in a single direction to cause multiple wounds both high and low on the body.[15] A commonly used directional fragmentation mine is the Claymore mine, which is placed above-ground and can spray 700 circular pellets over an arc

Figure 8. *Directional fragmentation landmines project fragments in a single direction to cause multiple wounds, both high and low on the body. Image courtesy of the United Nations Mine Action Service.*

Figure 9. *A rocket-propelled grenade (RPG). The high-explosive warhead is affixed to a rocket motor and stabilized in flight by fins. Image courtesy of the United Nations Mine Action Service.*

of 60 degrees (Fig. 8).[15] Lethal injuries occur within 50 meters from the point of detonation, and nonlethal fragmentation injuries can occur as far as 300 meters away.[10]

Rocket-Propelled Grenades

Rocket-propelled grenades (RPGs) are muzzle-loaded, shoulder-fired weapons that are primarily used against armored vehicles and ground personnel (Fig. 9). The various types of RPGs can fire fragmentation and high-explosive (e.g., high-explosive antitank [HEAT]) rounds and have a lethal blast radius of four meters.[33] Ground troops are sometimes injured when anti-vehicle rounds are aimed at adjacent structures, resulting in structural collapse and generation of multiple fragments. Because they are inexpensive and easy to transport and operate, RPGs are the weapon of choice for insurgents in many former Soviet-supported countries, including Iraq and Afghanistan. They can be found in almost 40 countries throughout the world.[34] Although RPG effects vary case-by-case, they frequently cause devastating injuries.[33]

Improvised Explosive Devices

Improvised explosive device (IED) attacks have become a mainstay in the current conflicts. IED attacks are most often used in insurgency and terrorist operations. They have been responsible for 40 to 60 percent of military casualties (wounded and killed) in Iraq between 2006 and mid-2009, and 50 to 75 percent in Afghanistan.[35,36] The incidence of IED-related injuries will vary depending on the phase of military operations. The decline in IED-related casualties in Iraq has been partly attributed to the increase in mine-resistant ambush protected (MRAP) vehicles sent to Iraq.[36] The sharp increase in IED-related casualties in Afghanistan has been attributed to "expanded military operations, a near-doubling of the number of troops since the beginning of the year and a Taliban offensive that has included a proliferation of roadside bombings."[37] Pentagon sources indicate that the number of IEDs in Afghanistan has increased 350 percent since 2007, with a subsequent increase in the number of IED-related combat injuries and deaths of more than 700 and 400 percent, respectively.[38]

IEDs are defined as devices that are placed or fabricated in an improvised manner incorporating destructive, lethal, noxious, pyrotechnic, or incendiary chemicals and are designed to destroy, incapacitate, harass, or distract (Fig. 10).[39] They may incorporate military weapons, such as artillery shells or antitank mines, but

are usually devised from non-military components.

IEDs vary in size, shape, form, and explosive power. They are easy to make and use, can be housed in almost any type of container, and can be hidden almost anywhere. The various types of IEDs use a range of explosive materials and are concealed, deployed, and detonated in different ways:

Figure 10. *IEDs are defined as devices that are placed or fabricated in an improvised manner incorporating destructive, lethal, noxious, pyrotechnic, or incendiary chemicals and are designed to destroy, incapacitate, harass, or distract. Image courtesy of the United Nations Mine Action Service.*

- Casings, ranging in size from a cigarette pack to a large vehicle, are used to hide the IED and possibly provide fragmentation. Small or large packages, including 120-mm and larger artillery or mortar projectiles with armor-piercing capability, are often placed in potholes covered with dirt, behind cinder blocks or sand piles to direct the blast, hidden in garbage bags or animal carcasses, or thrown in front of vehicles.
- Common hardware such as ball bearings, bolts, nuts, or nails can be used to enhance the fragmentation. Propane tanks, fuel cans, and battery acid have been added to IEDs to increase their blast and thermal effects. The damaging effects of IEDs can be maximized via coupling (linking one munition to another), boosting (stacking one munition upon another), and daisy-chaining (many munitions physically and temporally linked together length-wise).
- Triggers can be command-detonated by a remote device such as a cell phone, car alarm, toy car remote, or garage door opener, or with a time-delay device to allow the bomber to escape or to target military forces operating in a pattern. The initiator almost always includes a blasting cap and batteries as a power source for the detonator.
- Person-borne or victim-actuated devices (suicide bombs), typically using a powerful explosive with enhanced fragmentary effects, are employed to kill or maim as many people as possible. These are concealed in clothing worn by the assailant and hand-detonated.
- Vehicle-borne devices can vary in size from 100 to 1,000 pounds, depending on the size of the vehicle. The explosive charge can include mortar and artillery rounds, rocket mortars, warheads, and PE4 explosives. These can be concealed in vehicles of all types (cars, trucks, donkey carts). They can be deployed singly or in multiple vehicles. A lead vehicle is used to slow traffic and is followed by the main explosive device to maximize casualties. Detonation is by a command firing system.
- IEDs can be engineered to overcome IED detection measures through rolling (i.e., a target vehicle rolls over an initial unfused munition and then triggers a second trailing munition, which in turn detonates the initial munition). This sequencing positions the second (and most damaging) explosion directly under the target vehicle.

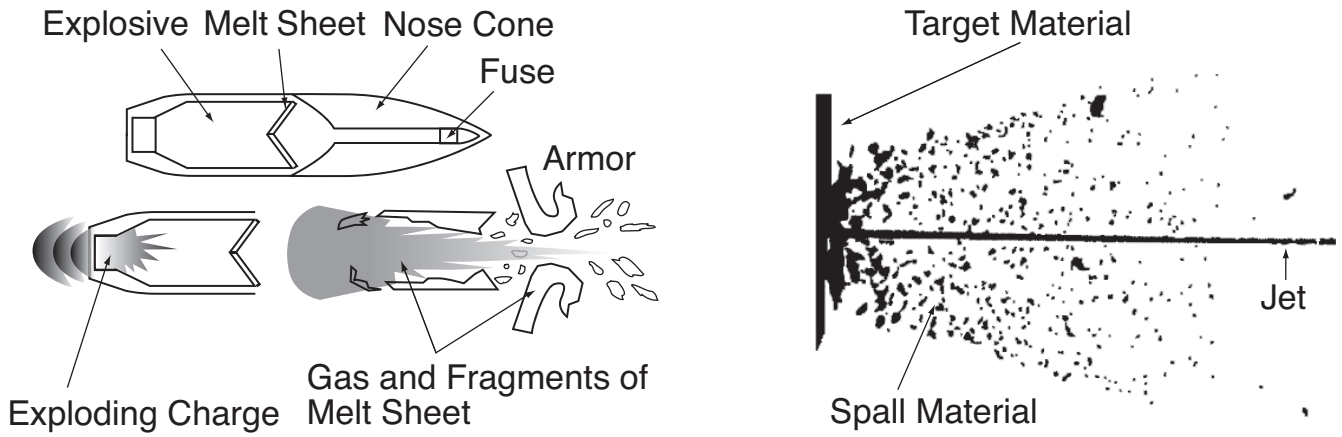


Figure 11. *Shaped-charge round:* (Left) *Disruptive mechanisms of the shaped-charge warhead include the jet of the charge itself and the debris knocked off from the inside face of the armored plate.* (Right) *Diagram taken from photograph of an actual detonation of a shaped-charged warhead against the armor plate caused by antitank land mines.*
Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.
Illustrator: Bruce Maston.

Improvised explosive device (IED) attacks have become a mainstay in OEF and OIF. They have been responsible for 40 to 60 percent of military casualties (wounded and killed) in Iraq between 2006 and mid-2009, and 50 to 75 percent in Afghanistan.

Antitank Munitions

In Iraq, there has been a trend away from small bombs (e.g., concealed in containers such as soft drink cans) to large rocket propellant or shaped-charges with armor-piercing capability.[40] Heavily armored vehicles are less susceptible to smaller, home-made roadside IEDs, and newer vehicle designs such as the MRAP provide enhanced protection to occupants from even larger IEDs. Antitank munitions are categorized as: (1) shaped-charges; (2) kinetic energy rounds; and (3) antitank landmines.[15]

Shaped-Charge

Shaped-charges have various degrees of armor-piercing capability (Fig. 11). High-explosive antitank (HEAT) rounds are composed of explosive charges packed around a reverse cone (this is the concept behind the anti-armor warhead of an RPG) (Fig. 12). If the charge is able to defeat the armor of the vehicle, injury to the occupants occurs via two methods. The initial potentially catastrophic injuries (including burns) are caused by the jet of the shaped-charge after it penetrates the vehicle's armor. Next, as the weapon strikes the armor, small pieces of irregularly shaped debris (spall) break away from the interior of the vehicle and are propelled into the occupants.

A commonly used shaped-charge variant is the explosively formed projectile (EFP) (Figs. 13 and 14). This IED variant consists of a cylindrical casing, such as a metal pipe. The side facing the target is closed with a concave-shaped metal plate facing inward, and the explosive charge is placed behind the metal plate.[41] On detonation, the concave plate is propelled out of the casing, becoming a high-speed aerodynamic penetrator (velocity can exceed 1,500 m/sec). This bullet or rod-shaped projectile easily pierces vehicle armor, causing catastrophic damage to vehicle occupants and other personnel in its path.[42]

The increased use and effects of EFPs are illustrated in a review of IED injuries seen in a British field hospital in 2006.[42] All casualties had injuries from roadside bombs directed at Coalition vehicles. Almost all (91 percent) of the explosions were caused by an EFP, and EFPs were responsible for all deaths. Main findings included the following:

- Most casualties (87 percent in survivors and nonsurvivors) had extremity injuries
- Most casualties had injuries to several regions of the body (e.g., 2.6 mean areas injured in survivors and 4.7 in nonsurvivors)
- All casualties had open wounds
- More than half of casualties (53 percent) had fractures
- There was little primary blast injury; only two casualties were thought to have died directly from a primary blast mechanism (blast lung)
- Only 15 percent of casualties had burns; no burns covered more than five percent total body surface area (TBSA)
- Approximately half of the survivors required immediate operative intervention at the field hospital

Figure 12. *Cross-section image of a high-explosive antitank (HEAT) round. Note the reverse cone of metal liner in the mid-section and the exploding charge at the base of the round. Image courtesy of Wikimedia Commons.*

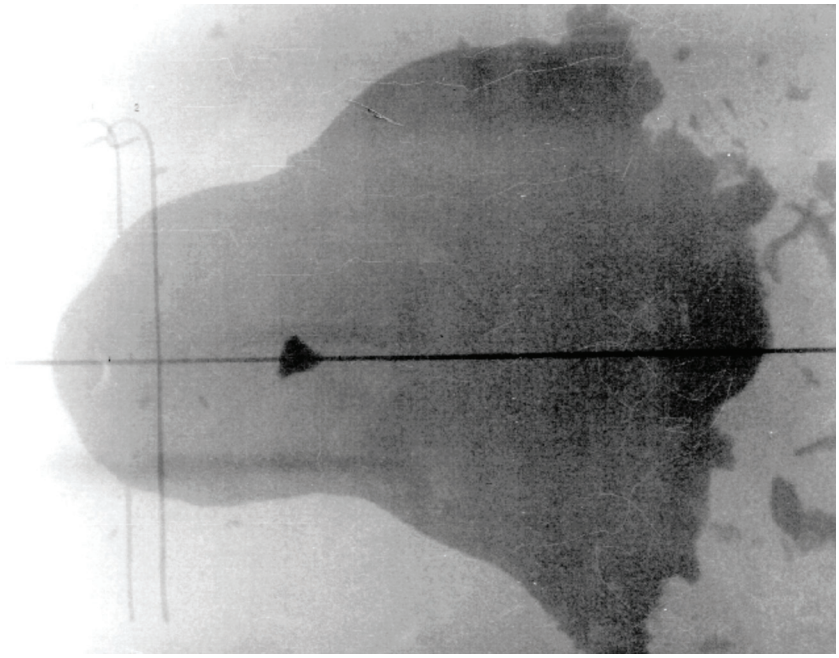


Figure 13. (Left) *An explosively formed projectile is an IED variant consisting of a cylindrical casing, closed with a concave-shaped metal plate facing inward, and an internal explosive charge. On detonation, the concave plate is propelled out of the casing and can inflict catastrophic injury. Image courtesy of Defense-Update.com.*

Figure 14. (Right) *X-ray of explosively formed projectile (EFP) detonation. Image courtesy of Applied Research Associates, Inc.*

Explosively formed projectiles (EFPs) generate "all or nothing" wounding patterns whereby casualties experience either catastrophic injuries or relatively minor wounds.[42] Significant EFP attacks cause multiple injuries in each survivor, including a high incidence of open wounds, extremity injuries, and fractures.

Kinetic Energy Rounds

Kinetic energy rounds are shaped like darts and are made from hard metals such as depleted uranium. Like shaped-charges, these weapons inflict damage by direct penetration of the vehicle or by generating spall. Warfighters with wounds caused by depleted uranium fragments should undergo standard wound care. Although there is a potential long-term risk from chronic exposure to depleted uranium, it does not justify extensive procedures to remove the fragments.[43]

Antitank Landmines

Antitank landmines are being modified and used as buried IEDs in OEF and OIF. Often, as described previously, more than one mine will be linked together to enhance the level of destruction.[14]

Explosion-Related Injury

Patterns

Explosive devices produce the ultimate polytrauma (i.e., a wide range of injury types to many body regions caused by the full range of injury mechanisms).[44] Explosions produce patterns of injury that are distinct from those of other mechanisms.[45] The simultaneous combination of different injury mechanisms (below) produces a complex array of injuries that must be understood to produce the best patient outcomes. In comparison with trauma patients whose injuries were not caused by explosions, bombing victims have lower states of consciousness as well as increased hypotension, injury severity, presence of multiple injuries, need for surgery, use of critical care services, length of hospital stay (LOS), and mortality.[45]

Explosive devices produce a complex array of injuries that must be understood to produce the best patient outcomes.

Military Casualties

A report that examined victims of close-proximity IED blasts of a variety of types (antipersonnel and antitank, including 105 to 120 mm mortars, 155 mm artillery-round IEDs, and a VS-1.6 antitank mine) revealed complex injuries in all cases and a 50 percent mortality rate despite the fact that all had been wearing Kevlar helmets, ballistic eye protection, and full body armor.[46] Some were injured on foot patrol, and some were in vehicles. The aforementioned report demonstrates the complexity of IED-related injuries.[42] The types of injuries produced by antitank weapons are shown in Figure 15 and include:

A. Translational blast injury (tertiary blast injury) can occur as the vehicle and its occupants are suddenly propelled upward causing blunt injury to occupants.
B. Toxic gases (a form of quaternary blast injury) can cause significant inhalation injury.
C. Primary blast injury can cause injury to the ears, lung, bowel, brain, and other organs.
D. Ballistic injury from the weapon and resultant debris fragments as the vehicle armor is defeated also occurs (secondary blast injury), as do thermal injuries resulting from flammable materials within the vehicle (quaternary blast injury).

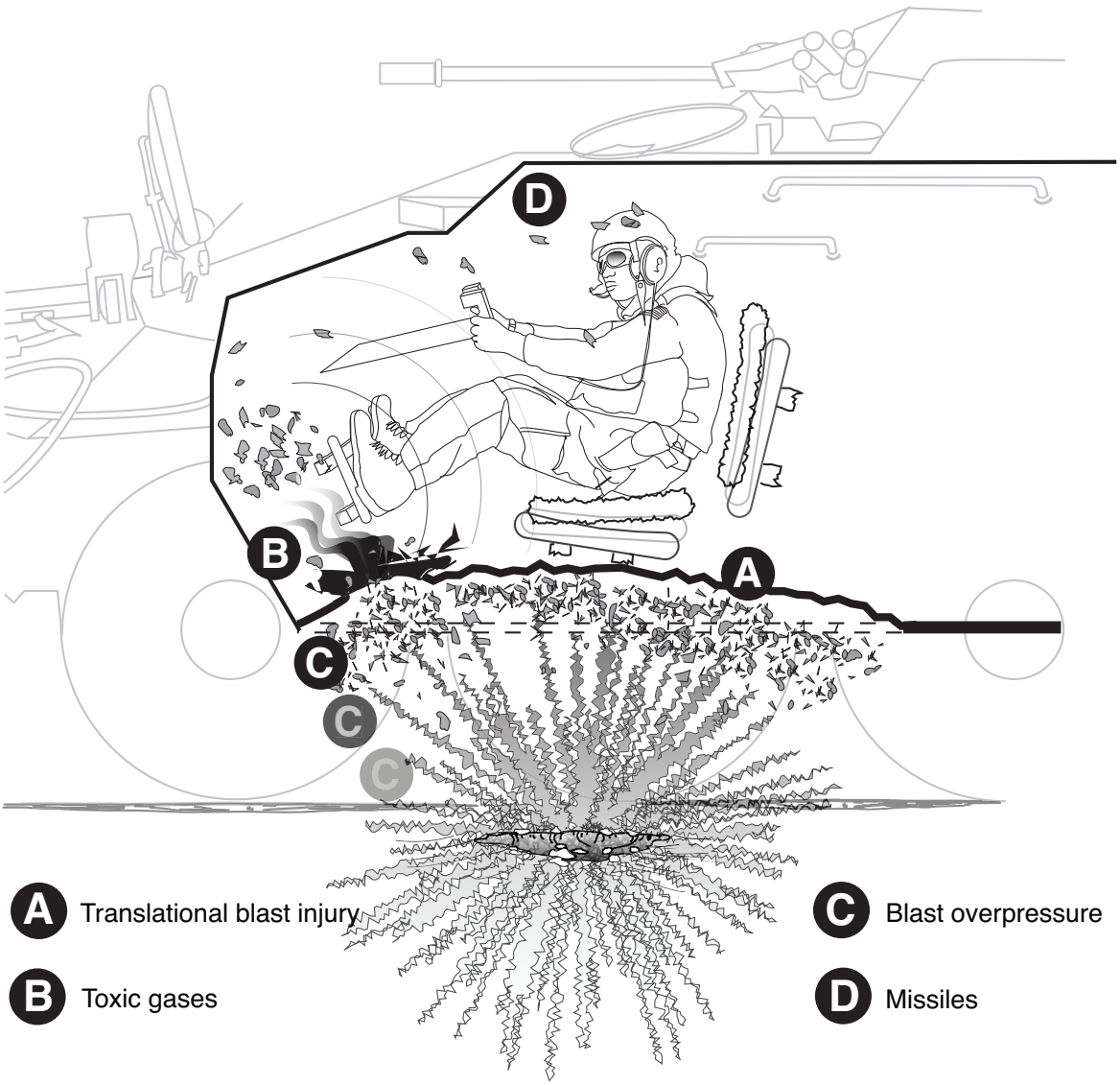


Figure 15. *Injuries sustained as a result of defeated armor: (A) translational blast injury, (B) toxic gases, (C) blast overpressure, and (D) penetrating missile wounds.*
Adapted image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.
Illustrator: Bruce Maston.

Civilian Casualties

Following an explosion in the civilian sector (e.g., open market bombing), most patients with lethal injuries will die immediately. Although the majority of survivors do not have life-threatening injuries, approximately 10 to 15 percent of casualties will have critical injuries and may be saved with appropriate management.[47,48,49]

Morbidity and mortality are generally dictated by the size of the explosive charge, whether the explosion occurs within a confined space, and whether it causes structural collapse.[50] Patterns of injury unique to blast include the following:[51]

- Most injuries are noncritical soft-tissue or skeletal injuries
- Head injury predominates as a cause of death (50 to 70 percent)
- The incidence of head injuries is disproportionate to exposed total body surface area (TBSA)
- Most blast lung injury kills immediately

In Israeli reviews, victims of terrorist bomb attacks, when compared to victims of non-terrorist trauma, have been shown to: (1) sustain more severe injuries, as measured by Injury Severity Score (ISS) (ISS greater than 16 in 74 percent versus 10 percent) and median intensive care unit (ICU) LOS (5 days versus 3 days);[52] (2) commonly have a combination of blunt and penetrating injuries (85 percent versus 15 percent)[53] and injuries to several areas of the body (three or more body regions injured in 28 percent versus 6 percent patients);[52] and (3) have injuries that are more likely to be fatal (mortality 6 percent versus 2 to 3 percent).[52,53] As demonstrated repeatedly among civilian populations that have been dealing with terrorism for years, terrorist bomb attacks produce injuries that are more complex, more severe, more lethal, and occur in a greater number of body regions than non-bomb-associated injuries.[45,54]

In civilian sector explosions, most patients with lethal injuries die immediately. Although the majority of survivors do not have life-threatening injuries, some 10 to15 percent of casualties with critical injuries may be saved with appropriate treatment.

Potentiators

A variety of strategies are used to increase the wounding and killing potential of explosives. These include: (1) increasing the size of the charge and amount of explosive; (2) increasing the number and type of secondary fragments (e.g., packing the devices with metal objects or pieces of concrete); (3) adding harmful substances such as chemicals, animal feces, or bacterial contaminants to produce infection; (4) planting explosives under vehicles to generate secondary fragments; and (5) adding incendiary substances such as petroleum products. Secondary explosions are often initiated by fuel-air explosives that disperse and ignite a spray of aerosol fuel, or by cluster bombs that distribute bomblets over a wide area.

The damage of the initial explosion is compounded by deploying snipers, subsequent bombs, or a remotely-detonated explosion to damage rescuers and first responders and vastly enhance the chaos. These tactics were used in Northern Ireland and are common in Iraq and Israel. Precise timing and location are also used to maximize the numbers of injured and dead.[47] Responders at the scene must be aware of these tactics and their effects, especially as recent data show increased coordination of terrorist attacks, including secondary attacks on first responders at the scene of an explosion, and increased variability in IEDs, including the introduction of chemical IEDs.[55]

Perhaps one of the most effective potentiators is the planting of explosives in confined spaces. Explosions that take place in confined spaces (e.g., buses and buildings) have patterns of injury that differ from those in open spaces (e.g., markets). Confined-space (closed-space) explosions generally produce more primary blast injury (discussed below) and penetrating injuries than explosions in open areas (open-space). The pressure

	Open-Space	Closed-Space
Deaths	8 percent	49 percent
Injuries		
• Primary blast injury	34 percent	77 percent
• Burns, TBSA	18 percent	31 percent
• Injury severity: median Injury Severity Score (ISS)	4 (minor)	18 (moderate/severe)

Table 5. *Comparison of open- and closed-space bombing deaths and injuries. Adapted from Leibovici, 1996.*[58]

wave associated with high-order explosive detonation reflects off doors, ceilings, and walls in confined spaces, lasts longer, and comprises what is termed a "quasi-static" exposure to overpressure effects.[56]

In OEF and OIF, most explosions are open-space bombings, and most injuries and deaths are from explosive fragments (secondary blast injury).

Israeli studies show significantly increased morbidity and mortality among those in confined-space bombings compared to those in open-space attacks.[57,58,59,60] In a 1996 study, an 8 percent mortality rate was observed among open-air (open-space) bombings versus 49 percent in bus bombings (Table 5).[58] An earlier study showed high percentages of primary blast injuries in bus bombings. In this study, 76 percent of victims had tympanic membrane perforation, 38 percent had blast lung, and 14 percent had abdominal blast injury.[57]

Blast Injury Effects	Mechanism of Injury
Primary	Injury caused by the effect of the blast wave on the body. Primary blast injury occurs principally in the gas-filled organs and results from extreme pressure differentials developed at body surfaces. Organs most susceptible include the middle ear, lung, brain, and bowel.
Secondary	Injury caused by flying debris and fragments, propelled mostly by the blast winds generated by an explosion. Most commonly produces penetrating injury to the body. At very close distance to the explosion, debris and fragments may cause limb amputation or total body disruption. This is the most common mechanism of injury from blast.
Tertiary	Injury results from victim being propelled through space by the blast wind and impacting a stationary object.
Quaternary	Injury suffered as a result of other effects of bomb blasts, including crush injury from a collapsed structure, inhalation of toxic gases and debris, thermal burns, and exacerbation of prior medical illnesses.
Quinary	Injury resulting from contamination via biological and chemical agents, radioactive materials, or contaminated tissue from attacker or other person at the scene.

Table 6. *Categories of blast injury effects with corresponding mechanisms of injury.*

When the confined space is a building, the force of the blast may break windows, producing thousands of glass shards, and buckle the walls, floor, and ceiling, resulting in partial or complete building collapse and subsequent crush injuries.[61] Studies contrasting open-space bombings with bombings involving buildings (closed-space) show a much higher mortality rate in the latter. For example, all deaths and almost all (96 percent) injuries in the 1996 Khobar Towers bombing in Saudi Arabia occurred inside the buildings; and in the 1995 Oklahoma City bombing, 87 percent of those in the collapsed section of the Murrah Building died, compared with 5 percent of those in the uncollapsed section.[48,62]

In OEF and OIF, most explosions are open-space bombings and most injuries and deaths are from fragments.[44,63]

Categories

Blast injuries are categorized as having primary, secondary, tertiary, quaternary, and quinary effects, each with its own mechanism of injury (Table 6).

Primary Blast Injury

Primary blast injuries result when the pressure wave interacts with the body, especially the gas-containing organs, via spalling, implosion, acceleration-deceleration, or initiation of pressure differentials.[31]

- "Spalling, or spallation, occurs when particles from a more dense substance are thrown into a less dense substance at their interface."[31] Spall is a flake or small particles that are broken off a larger solid body and can be produced by a variety of mechanisms, including projectile impact (Fig. 16).
- Implosion is the momentary contraction of gas pockets that occurs when the blast wave moves through the tissue. The pressure differential may force blood and fluid into the previously air-filled spaces, as seen with pulmonary contusion and hemorrhage in blast lung injury.[31]
- Acceleration-deceleration, or shear injury, occurs when movement of the body wall in the direction of the blast wave displaces the internal structures. Because the structures accelerate at different rates, shearing or disruption may occur.
- The pressure differential between the inside and outside of the body induced by the blast wave produces injuries.[31]

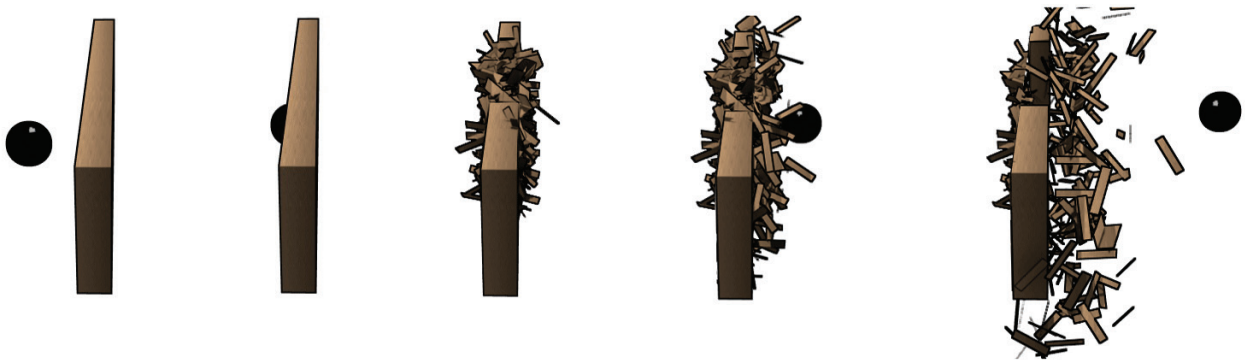


Figure 16. *Spall is debris generated when particles from a more dense substance are thrown into a less dense substance at their interface. In this illustration, the ball impacts a metallic plate and knocks off material from the inside surface into the air. Adapted image courtesy of Wikimedia Commons.*

Survival after a primary blast injury is dependent on the energy of the blast, whether it occurred in an open or enclosed (closed) space, and the distance of the individual from the point of detonation (standoff distance).[20] The main sites of primary blast injury are the ears, lungs, intestinal tract, and brain.[64,65]

Ears

A powerful blast wave can overwhelm the extremely delicate structures within the ear, causing tympanic membrane rupture, fracture or dislocation of the ossicles, and permanent inner ear damage. Rupture of the tympanic membrane is a common injury following an explosive blast.[66] Further, the tympanic membranes are the structures that are injured at the lowest pressure, and thus have been used as a sentinel for other, more serious injuries.[64] Recent reports have disputed the reliability of tympanic membrane rupture as a sensitive screening tool for primary blast injury detection.[21,66] The absence of tympanic membrane rupture does not

exclude other types of blast injury. An increase in pressure of as little as five pounds per square inch (psi) may cause eardrum rupture, 15 psi carries a 50 percent chance, and 30 to 40 psi will almost certainly rupture the eardrum.[67] Recent data from OEF and OIF with explosion-related injuries indicated an approximate 15 to 16 percent incidence of tympanic membrane rupture.[21,66] The most common symptoms reported by the patients experiencing an audiovestibular injury are hearing loss (60 percent), tinnitus (49 percent), otalgia (26 percent), and dizziness (15 percent).[68,69]

Rupture of the tympanic membrane is a common injury following an explosive blast. Its absence may not be adequate to rule out primary blast injury and does not exclude other types of blast injury.

During the secondary survey in the initial evaluation of a blast victim, the tympanic membranes should be evaluated. Improvised explosive device detonations typically propel debris into the external auditory canal. The debris should be carefully removed to allow full visualization of the ear canal. The external auditory canal should not be blindly irrigated because this can result in pain and vertigo in patients with perforated tympanic membranes.[70] If debris is noted in the external auditory canal or behind the ruptured tympanic membrane, topical antibiotic eardrops, such as a fluoroquinolone, are recommended to prevent infection.[64,70] The presence of cerebrospinal fluid or blood in the external auditory canal or hemotympanum is suggestive of a basilar skull fracture.

Most (80 to 90 percent) tympanic membrane perforations heal spontaneously. The larger the perforation, however, the lower the probability that it will heal spontaneously.[21] Perforations involving more than 30 percent of the surface area of the tympanic membrane are significantly less likely to heal spontaneously than smaller perforations (Fig. 17).[70] Spontaneous healing also varies with the location of the rupture. Central tympanic membrane ruptures have the least likelihood of healing spontaneously, whereas inferior perforations are the most likely.[21]

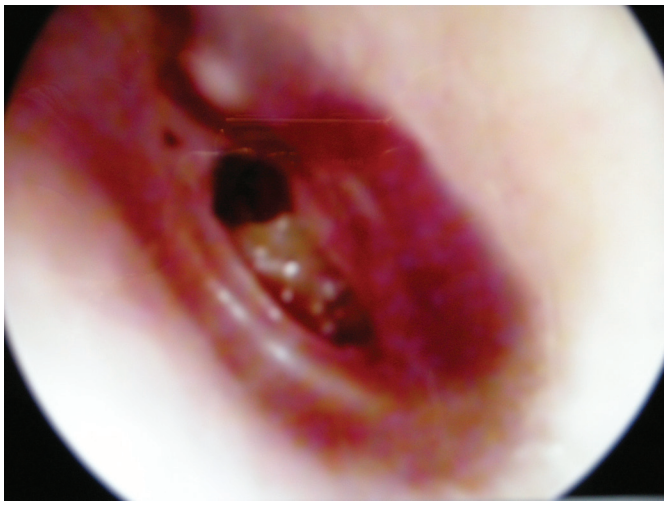


Figure 17. *Tympanic membrane perforation. Image courtesy of Gene Liu, MD, Cedars-Sinai Medical Center.*

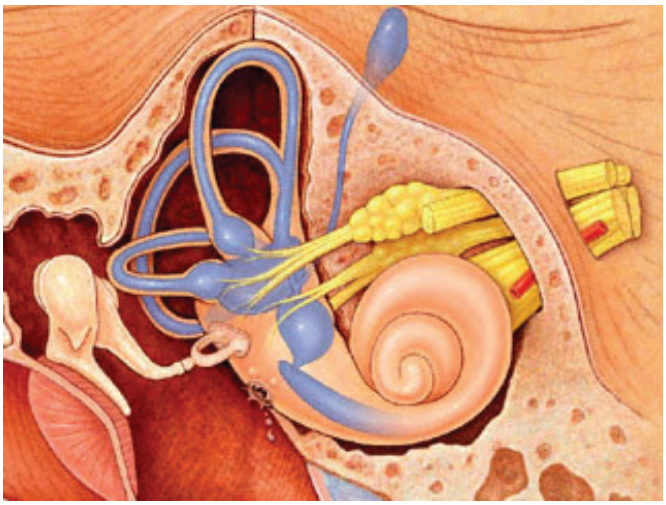


Figure 18. *Blast effect can cause inner ear injuries, such as the perilymphatic fistula shown here, and ruptures of the saccule, utricle, and basilar membrane. In the middle ear, the ossicles may fracture or disarticulate, independent of a tympanic membrane perforation. Image courtesy of Timothy Hain, MD, Northwestern University.*

Besides rupturing the tympanic membrane, the blast can also cause middle ear damage, such as fracture of the ossicles or disarticulation of the ossicular chain.[68] Although these usually occur in conjunction with tympanic membrane perforation, they can occur independently. Injury to the inner ear, such as perilymphatic fistulae in the oval window and ruptures of the saccule, utricle, and basilar membrane, may also occur (Fig. 18). Sensorineural hearing loss may be seen with loss of hair cell integrity. Similarly, damage to the vestibular apparatus may occur and manifest as vertigo.[19]

Consultation with the otolaryngology service should be performed when greater than 50 percent tympanic membrane perforation occurs or if other audiovestibular symptoms are noted. All blast injury patients requiring inpatient care should have audiometric testing when possible.[21,71] The management guidelines used at Balad Air Base in Iraq are presented in Table 7. Hearing protection has been shown to significantly reduce the incidence of tympanic membrane rupture, and its use should be encouraged in combatants who are deployed in high-risk environments.[72]

Lungs

The lungs are also vulnerable to primary blast effects. Explosions can cause a variety of thoracic injuries including pulmonary contusion, pneumothorax, pneumomediastinum, air emboli, hemothorax, and subcutaneous emphysema (Fig. 19).[64] An external force acting on the chest wall may compress the lungs slowly enough to allow air contained in the alveoli to be expelled through the trachea. However, when a significant blast wave impacts the chest wall, there is little time for pressure equilibration. The pressure

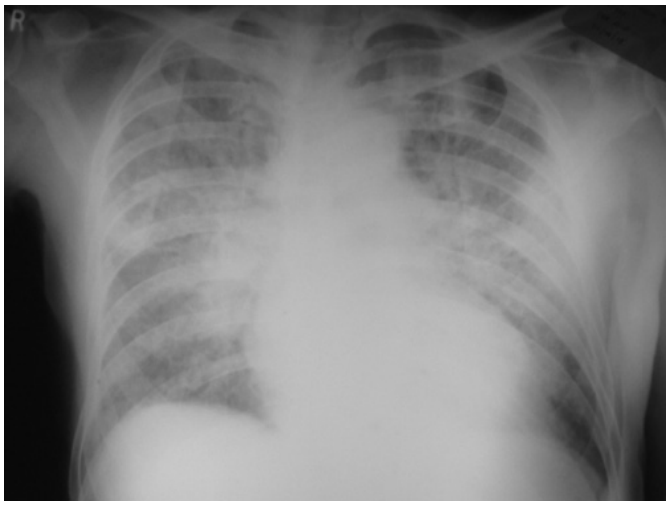
Consultation	Indications	
	Absolute	Relative
Otolaryngology	Vertigo lasting greater than three days	Tympanic membrane rupture greater than 50 percent
	Presence of clear otorrhea	Debris in the external auditory canal that does not resolve with topical antibiotics
	Discolored otorrhea that persists despite seven days of topical antibiotic therapy	Inability to visualize the tympanic membrane despite removal of debris from the external auditory canal
Audiology	An average hearing threshold greater than 30dB at frequencies of 500, 1000, and 2000Hz	Significant communication problems
	A hearing threshold greater than 35dB at frequencies of 500, 1000, or 2000Hz	Tinnitus significantly affecting quality of life
	A hearing threshold greater than 55dB at frequencies of 3000 or 4000Hz	
	New-onset asymmetrical hearing loss	

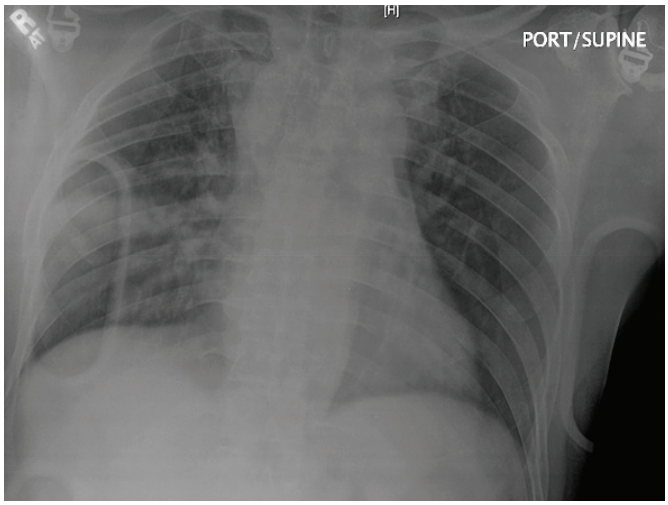
Table 7. *Management guidelines for otolaryngology and audiology consultations used at Balad Air Base, Iraq. Adapted from Depenbrock, 2008.*[70]

differentials that develop at the interface between media of different densities tear the alveolar walls, disrupt the alveolar–capillary interface, and cause the emphysematous spaces to fill with blood, resulting in primary blast injury to the lung (blast lung).[19] Pressures of 30 to 40 psi are associated with possible lung injury, and at 80 psi, a 50 percent chance of lung injury exists.[67] As a point of reference, pressures in the 100 to 200 psi range may be lethal, and when psi exceeds 200 to 250, death is almost certain.[67]

Lungs are vulnerable to primary blast effects. Explosions can cause a variety of intrathoracic injuries including pulmonary contusion, pneumothorax, pneumomediastinum, air emboli, hemothorax, and subcutaneous emphysema.

Pulmonary manifestations vary greatly depending on the size of the blast wave. The mildest form of this tissue disruption was noted to be pleural and subpleural petechiae in animal studies.[73,74] The classic chest radiograph demonstrates bilateral central infiltrates and has been described as a butterfly or batwing pattern (Fig. 20). This pattern is probably caused by reflection of the blast wave off of the mediastinal structures within the thoracic cavity. Additionally, the central location of the infiltrates helps differentiate this from the more classic lateral infiltrates seen with pulmonary contusion (Fig. 21).[75]





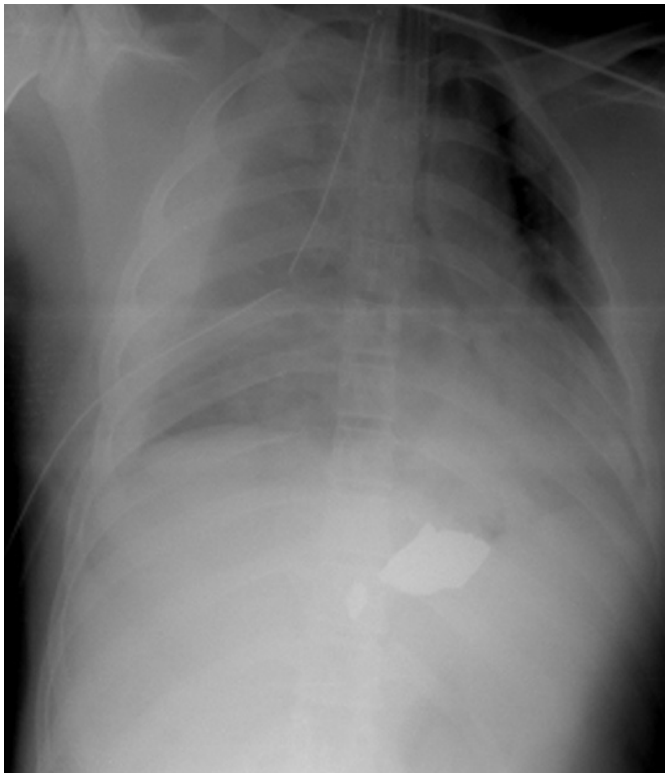


Figure 19. (Top Right) *Chest radiograph demonstrating pneumothorax, hemothorax, and a penetrating fragment, following an IED explosion.*

Figure 20. (Top Left) *The classic chest radiograph seen with primary blast injury to the lung demonstrates a butterfly or batwing pattern.*

Figure 21 (Bottom Left) *Chest radiograph demonstrating a peripherally located pulmonary contusion resulting from blast injury.*

Indications & Requirements	Blast Lung Injury Categories		
	Mild	Moderate	Severe
Indications			
Radiographic infiltrates	Unilateral	Asymmetrical and bilateral	Diffuse
PaO_2 to FiO_2 Ratio (mm Hg)	>200	60 to 200	<60
Bronchopleural fistula	No	Yes	Yes
Requirements			
Positive pressure ventiliation (PPV) requirement	Unlikely for respiratory problem	Highly likely but usually conventional methods	Universal and unconventional methods common
Positive end-expiratory pressure (PEEP) requirement (cm H_2O)	<5 if PPV needed	5 to 10 usually needed	>10 commonly needed

Table 8. *Severity categories for blast lung injury based upon radiographic appearance, oxygen requirement, and the presence of bronchopleural fistula. Adapted from Pizov, 1999.*[80]

The incidence of blast lung in OEF and OIF has been low because open-space explosions predominate. When blast lung occurs in patients, it has high associated morbidity and its treatment is resource-intensive.[76,77,78] Primary blast injury to the lung may not be immediately obvious upon external examination.[79] Symptoms of blast lung can manifest within the first few minutes following a blast or can develop and evolve over a period of hours to days.[21,57,75,80,81] Blast lung has been shown to have the following characteristics:

- Symptoms include dyspnea, chest pain, hemoptysis, and cough[19]
- Clinical signs include cyanosis, tachypnea, rapid or shallow breathing, crackles, diminished breath sounds, dullness to percussion, increased resonance, retrosternal crunch, subcutaneous crepitus, and tracheal deviation[19]
- Hypoxemia and hypercarbia[81]
- Rapid respiratory deterioration with progressive hypoxia[58]
- Progressive need for ventilation with high FiO_2[58]
- Progressive haziness in serial chest radiographs[58]
- Hemodynamic instability[58]
- Pulmonary edema with frothing at the mouth, frequently lethal[64]

Enclosed-space (closed-space) bombings should raise the index of suspicion for blast lung and other primary blast injuries.[53] Patients with blast lung require supportive care with special emphasis on ensuring adequate oxygenation and ventilation. Standard ventilator management with initial use of positive end-expiratory pressure of 10 centimeters (cm) water is acceptable.[18] However, advanced ventilatory methods, such as independent lung ventilation, high-frequency jet ventilation, nitric oxide inhalation, and extracorporeal membrane oxygenation, may also be of value.[47,80,82] Intravenous fluids should be administered judiciously to minimize capillary leak and pulmonary edema. Patients should be monitored closely for development of pneumothorax. The clinical efficacy of prophylactic antibiotics and steroids in blast lung injury is undetermined.[64] Published blast lung injury severity categories, based on radiographic appearance, oxygen requirement, and the presence of bronchopleural fistula, may be helpful in determining which patients

require positive pressure mechanical ventilation and positive end-expiratory pressure (Table 8).[80] While ear protection has been shown to offer some protection of the tympanic membrane against primary blast injury, thoracic body armor may not have the same protective effect on the lungs.[64,83]

Solid and Hollow Organs

A blast wave can cause rapid compression and expansion of air in gas-filled organs, which often results in contusions, perforations, or intramural hemorrhage. When air emboli fill the pulmonary and coronary vessels, early death often occurs.[54] Delayed rupture of the intestinal tract can occur secondary to significant ischemia and infarction within the mesentery.[64] While the gastrointestinal tract is particularly susceptible to primary blast injury, especially the colon, primary blast injury of hollow organs in OEF and OIF is rarely encountered.[84,85,86,87]

Solid organs, principally the liver, spleen, and kidney, have a relatively uniform liquid density. When a blast wave impacts these organs, little compression occurs, and significant injury to the tissue is less likely to occur.[19,88] Solid intraabdominal organs are more likely to be injured through secondary or tertiary mechanisms. However, blast waves can cause shear forces to develop at points of attachments of organs or at the surfaces of the organs. In the former case, an organ may tear off of its point of attachment, while in the latter case, subcapsular petechiae, contusions, lacerations, or rupture may occur.[19]

Patients may present with a variety of abdominal signs and symptoms including pain, nausea, vomiting, hematemesis, melena, and signs of peritoneal irritation.[21] Patients with overt hemodynamic instability should undergo immediate exploratory laparotomy for presumed active hemorrhage from the intestinal mesentery or a solid organ injury.[89] More stable patients can be evaluated using computed tomography (CT) imaging. Ritenour noted that "CT evidence of blast injury includes pneumoperitoneum, free intraperitoneal fluid not consistent with blood, and a sentinel clot seen on bowel wall or mesentery."[21] Intestinal contusion, submucosal hematoma, and mesenteric hematoma can also be seen on CT imaging following blast injury.[89]

> The gastrointestinal tract is particularly susceptible to primary blast injury, especially the colon. Significant ischemia and infarction within the mesentery following primary blast injury can lead to delayed rupture of the intestinal tract.

Brain

The prevalence of traumatic brain injury (TBI) among combat casualties is higher in the current conflicts than in previous wars. This is primarily because many patients with previously lethal injuries are now surviving, largely due to enhanced helmets that prevent or reduce penetrating head trauma, advances in battlefield medicine, and rapid evacuation to a well-honed system of care.[90] Thus, TBI has become the current signature injury of combat, much as shell shock was the signature injury of World War I.[65,91] Traumatic brain injury potentially affects up to one-third of OEF and OIF combatants and approximately 320,000 reported experiencing symptoms that may be related to TBI during deployment.[92] Of patients admitted to Walter Reed Army Medical Center (WRAMC) between 2003 and 2005 who had been exposed to explosive blasts, 59 percent were found to have symptoms that may relate to TBI. Of these, 56 percent had moderate/severe TBI and 44 percent had mild TBI.[93,94] In contrast, only 20 percent of civilian TBIs are moderate/severe.[95] It is difficult to determine which explosion-related TBIs can be attributed to primary blast effects alone, even in cases where no fragment injuries are present.[96] In a recent study of 2003 to 2008 OIF casualties with head trauma, 48 percent had closed head injury that was attributed to primary and/or tertiary blast

injury mechanisms.[97] Kinetic energy following blasts causes shearing in the central nervous system, resulting in both focal and diffuse axonal injury, air embolism, and cranial fractures with associated sinus cavity involvement.[64,89,98] Cognitive and biochemical changes occur in animals exposed to blasts (oxidative stress in the hippocampus), and electroencephalographic changes, punctuate hemorrhages, and chromatolysis have been seen in the brains of human blast victims.[21,88,99,100] The authors of the aforementioned studies could not reliably differentiate between injury mechanisms due to lack of specifics about the individual explosions and/or coexistence of blunt trauma mechanisms (e.g., vehicle incidents). The exact mechanism(s) of brain injury from blast overpressure remains unclear.[44,101,102,103,104,105]

Traumatic brain injury has become the current signature injury of combat. It is difficult to determine which explosion-related TBI-type symptoms can be attributed to primary blast alone, as opposed to other blunt trauma-related TBI. The exact mechanism(s) of brain injury from blast overpressure remains unclear.

Patients can present with a variety of signs and symptoms ranging from a headache to coma. Clinical findings may include fatigue, headache, back or generalized pain, vertigo, paralysis (transient or persistent), and altered mental status.[21,99] Psychological symptoms include excitability, irrationality, amnesia, apathy, lethargy, poor concentration, insomnia, psychomotor agitation, depression, or anxiety.[21,104]

Cumulative and long-term effects of mild TBI on US troops are beginning to be a cause for concern. In one study, 44 percent of soldiers suffering mild TBI with loss of consciousness (LOC) met the criteria for post-traumatic stress disorder (PTSD) on evaluation three to four months after returning home.[65] Twenty-seven percent of soldiers who were simply dazed after a blast subsequently reported PTSD symptoms. Many soldiers reported significant problems with their general health, poor work habits, and a variety of symptoms. A study by Hoge "concluded that PTSD and depression were mediators of the relationship between mild TBI and physical health problems."[65] The Defense and Veterans Brain Injury Center (DVBIC), the lead agency in investigating TBI in the military, publishes updated data on military TBI. Their recommendations have included pre-deployment neurocognitive testing and the use of the Brief TBI Screen (BTBIS) in the post-deployment process.

Clinical Practice Guidelines (CPGs) published by the Joint Theater Trauma System (JTTS) provide algorithms for TBI evaluation at Level I (medic at point of wounding), Level II (Forward Surgical Team [FST]), and Level III (Combat Support Hospital [CSH]).[106,107] For mild TBI (GCS score of 13 to 15), Level I and II facility careproviders should perform the standard physical examination and use the Military Acute Concussion Evaluation (MACE) for assessment (MACE form available through the Defense and Veterans Brain Injury Center). Level III facility evaluation is often more comprehensive and may involve further neurocognitive testing following MACE performance.[108] Patients with a head injury and a GCS score of nine to 12 are classified as having moderate head injuries, and patients with GCS scores lower than nine are considered to have severe TBI. The lower the GCS score within this range, the higher the chance of death and the lower the chance that the patient will return to independent living (Fig. 22).

Blast effects to the brain can result in neurocognitive changes that may not manifest as obvious physical symptoms requiring treatment. Possible injury to the brain may be manifested in other ways, which can be assessed using the MACE scale. Individuals who have "seen stars" or are "just not themselves" may

have suffered a mild TBI. In an effort to decrease the possibility of exposure to sequential concussive brain injury, warfighters who have been exposed to explosive blasts should be tested using MACE. Scores lower than 25 warrant further evaluation and treatment. As a preventive measure, it is recommended that these individuals only return to light duty in an effort to decrease the possibility of a subsequent exposure to a blast or vehicle crash while their brains recover.[106]

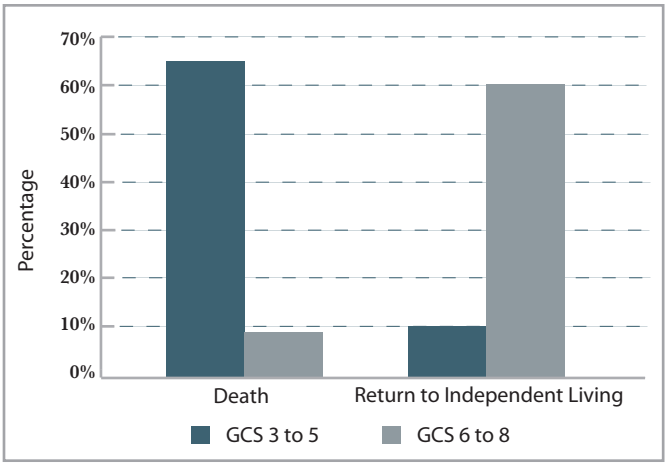


Figure 22. *Prognosis for OEF/OIF combatants with severe TBI (GCS score less than 9). Data source: Joint Theater Trauma Registry (JTTR).*

Eyes

Although primary blast injury to the eye is rare because of the uniform density of the eye, it occasionally occurs in the form of globe rupture, retinitis, and hyphema.[64] The most common sign of primary blast injury to the eye is subconjunctival hemorrhage.[74] Injuries to the eye are more commonly caused by secondary blast fragments (e.g., splinters of glass and other debris), many of which are preventable with simple eye protection equipment.

Extremities

Primary blast injury resulting in amputation is rare and often part of a pattern of lethal injuries.[109] As the blast wave impacts an extremity, tremendous pressure differentials may shatter the bone, and the near-simultaneous blast wind may subsequently avulse the extremity. On the whole, avulsions are observed mainly along the shaft of long bones and are most common among dead or dying victims. In one study, traumatic amputations due to primary blast primarily occurred in the upper third of the tibia.[110] These amputation injuries have a high risk for exsanguination, and the limbs are rarely reattachable.[19]

Secondary Blast Injury

The overpressurization wave created by the primary blast is followed by a negative-pressure phase. This generates a blast wind that propels debris and objects with ballistic speed and force to create multiple penetrating injuries.[31] Although they are termed secondary blast injuries, these are the predominant explosion-related injuries in survivors.[63,111]

> The greatest diagnostic challenges for clinicians at all levels of care in the aftermath of explosions are the large numbers of casualties and multiple penetrating injuries.[44]

Primary and Secondary Fragments

Flying fragments and debris from the explosive and its surrounding environment are differentiated as primary and secondary fragments. In conventional military ordnance, primary fragments typically consist of bits of the exploding weapon. In IEDs, primary fragments include the shell casing as well as items packed into the explosive to increase wounding potential, such as nails, bolts, ball bearings, or other small, sharp items (Fig. 23). The effectiveness of this technique has been demonstrated. For example, following a suicide bomb attack in Israel, the bodies of all those who died immediately after the blast and all with severe injury (ISS greater than 16) were "saturated with steel spheres."[112]

All explosives generate secondary fragments that consist of debris from manufactured (e.g., metal from vehicle interiors, shattered furniture, splinters of window glass) and natural environments (e.g., rocks, dirt).[113] Dust and tiny grains of dirt can become embedded in the skin, causing a characteristic, dusky, tattooed effect.[114] Among all fragment types, glass causes a disproportionate amount of secondary injury. Of the 95 percent of survivors of the 1996 Khobar Towers bombing with fragment injuries, 88 percent were injured by glass (primarily from windows).[62]

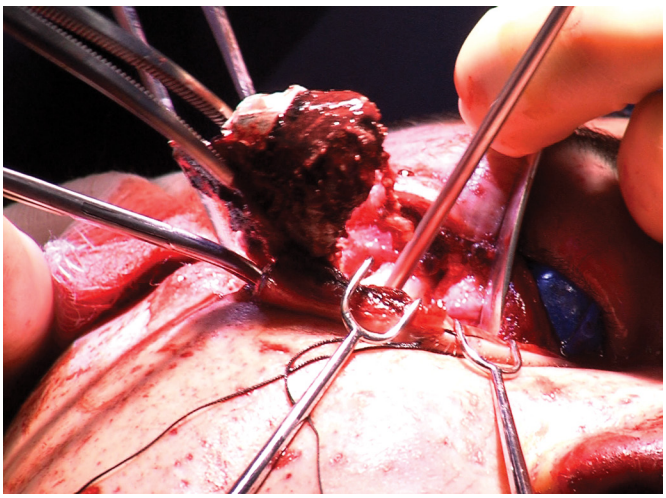


Figure 23. *A combat casualty undergoing removal of metallic shrapnel embedded in his right periorbital region following an IED blast.*

Fragment Physics

Fragment projectiles differ from bullet projectiles in that they are scattered (not channeled through a barrel), are irregularly shaped, and have different velocities upon impact.[31] After detonation, aerodynamic drag is exerted on the fragments, which then strike the body as both high- and low-velocity projectiles.[31] Initial velocities of primary fragments can be as high as 1800 m/sec, but under 600 m/sec appears to be the upper limit of survivability.[15,115] Low-velocity fragments may tumble or shimmy, crush large areas of tissue, and fragment further to exacerbate the injuries.[116,117,118] This is counter to the previously held notion that the higher the velocity of a missile, the more tissue damage there will be.[15] In addition, fragments contaminate wounds with environmental debris. All of these factors likely account for the differences in fragment and bullet injuries, even though both are caused by small missiles propelled at great speeds.[31]

Fragment Wounds

The distinguishing feature of most explosion-related injuries is the presence of multiple penetrating fragment injuries to several regions of the body (Fig. 24).[44,119] Because fragment wounds can be so numerous (e.g., 30 to 40 in a single patient), CCC providers can find it difficult to determine which wound(s) requires high-priority evaluation. The body region involved and associated clinical findings determine clinical impact and treatment priorities.[119]

Because of the protection offered by body armor, military personnel have a high incidence of fragment injuries to the head, extremities, and the junctions between the torso, arms, neck, and legs. These should be managed in the same way as other penetrating injuries. Meticulous wound inspection and debridement are important in the management of such injuries. Secondary blast injury also frequently results in facial and ocular injuries.[21,64] The eyes are particularly vulnerable to secondary blast injuries largely caused by minute bits of shattered glass or metal. As many as 10 percent of all blast injury survivors have significant eye injuries from projectiles, with signs and symptoms that include pain, irritation, sensation of a foreign body, changes in visual acuity, swelling, and contusions.[17] Most such eye injuries are preventable with appropriate eye protection. Among survivors of the September 11, 2001 attacks on the World Trade Center, 26 percent had ocular injuries.[120]

The distinguishing feature of most explosion-related injuries is the presence of multiple penetrating fragment injuries, or fragment wounds, to several regions of the body. Injuries and deaths from fragments occur much further from the point of detonation than do those associated with the primary blast.

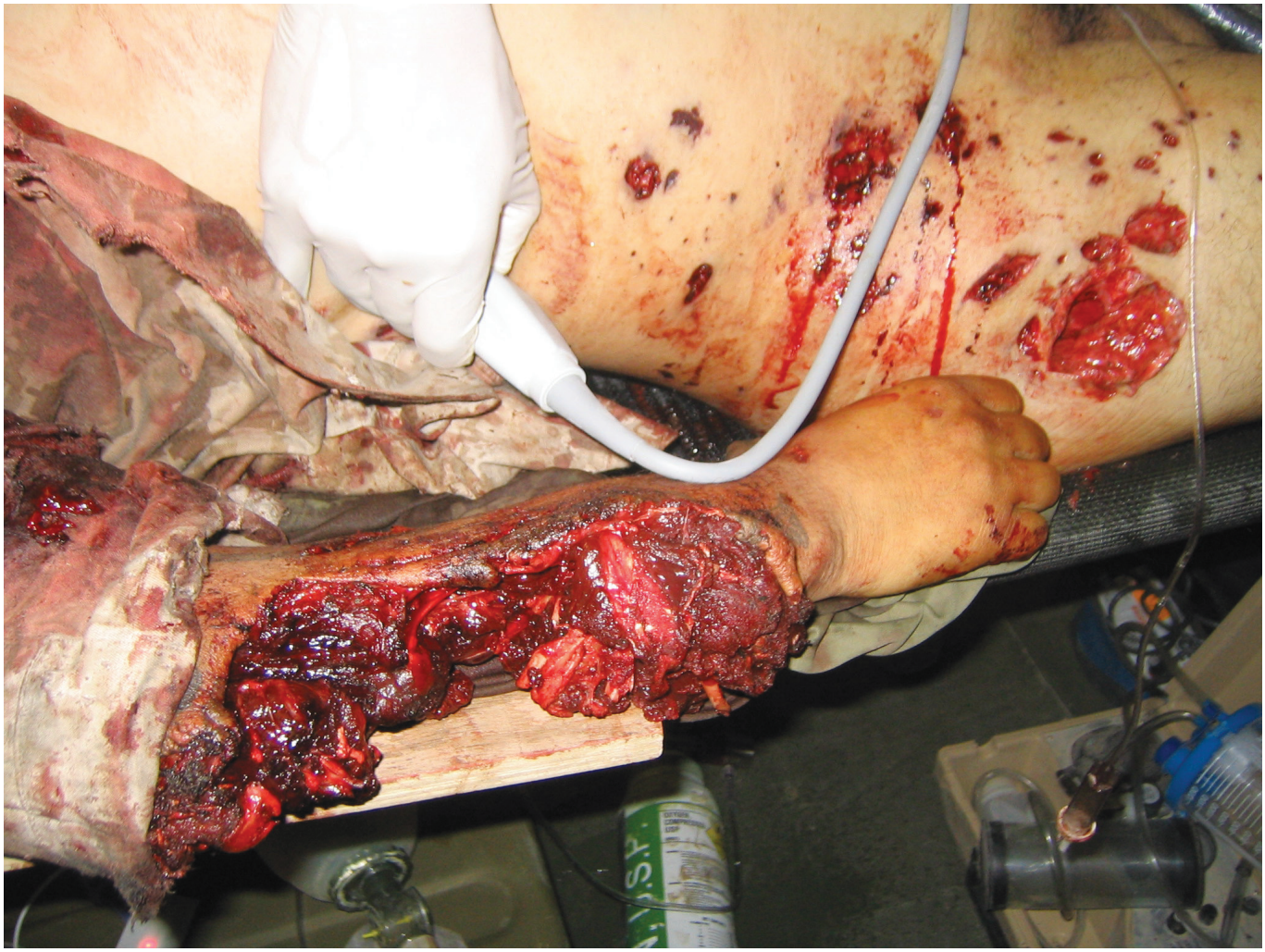


Figure 24. *A casualty seen at Camp Taqaddum, Iraq in 2004, with fragmentation wounds from an IED blast. The distinguishing feature of most explosion-related injuries is the presence of multiple individual injuries in several regions of the body. Image courtesy of Harold Bohman, MD, CAPT, MC, US Navy.*

Although prior literature advocated extensive debridement of fragment wound tracts, recent experience shows that this is no longer required. This is because: (1) high-velocity projectiles often do not cause temporary cavitation; (2) elastic soft-tissue generally heals without excision if the blood supply is intact; and (3) antibiotics play a larger role in mitigating infection.[121] In cases involving multiple fragments, it is not recommended to attempt to extract every fragment, but instead to remove those that pose a threat to life or health. The potential damage that could be caused by removing a fragment or through extensive wound exploration or debridement must be weighed against the damage that might result from not removing it. For example, in casualties with low-velocity penetrating head injury, debridement was limited to minimize risk of causing additional neurologic injury, with no apparent adverse affects on outcome.[122]

Fragment Range

Risk of fragment injury occurs over a much wider radius than blast overpressure. Thus, in an open-space explosion, the primary mechanism of injury is fragment penetration.[119] The safe standoff distance for fragments has been noted to exceed that for blast overpressure by a factor of 100. Injuries and deaths from fragments occur much further from the point of detonation than do those associated with the primary blast

	Morbidity and mortality	
Distance From Blast	Primary Blast Injury	Secondary Blast Injury
0 to 50 feet	Death, eardrum rupture	Death
50 to 80 feet	Eardrum rupture	Death
80 to 130 feet	Temporary hearing threshold shift	Injury
130 to 1800 feet	None	Injury

Table 9. *Blast injury effects based on distance from open-space blast explosion (155-mm shell). Adapted from Champion, 2009.*[44]

(Table 9).[47,119] Following the 1998 terrorist bombing of the US Embassy in Nairobi, fragment injuries were sustained by people as far as two kilometers from the point of detonation.[18] Secondary injury is largely penetrating, but victims can experience nonpenetrating injuries as well. For example, the low-velocity fragments responsible for all Khobar Towers bombing injuries caused penetrating, blunt, and crush injuries.[62] A large proportion of blunt injuries, however, are caused by tertiary blast effects.

In an open-space explosion, the primary mechanism of injury is fragment penetration. Injuries and deaths from fragments occur much further from the point of detonation than do those associated with the primary blast.[119]

Tertiary Blast Injury

Tertiary blast injuries are caused by propulsion and displacement of the blast victim, of large fragments, or of surrounding structures such as a building or vehicle. The subsequent impact of victims upon structures or structures upon victims causes blunt and penetrating injuries that include crush, impalement, and other injuries whose severities vary with the degree of fragmentation and structural collapse.[64]

Although most tertiary blast injuries comprise soft-tissue wounds or fractures that are not immediately life-threatening, complete structural collapse is rarely survivable.[123] This was illustrated in the examples of the Khobar Towers and Oklahoma City bombings.[16] Individuals inside vehicles sustaining an IED blast can also experience tertiary blast injuries as the vehicle is propelled upward against the occupants or as the occupants are projected within the vehicle. In blast injury tests on vehicles, the vast majority of the injuries were tertiary. For undercarriage blasts, lower limbs were crushed, and in roadside blasts, occupants sustained severe head and side-thoracic impacts. These results are not dissimilar from those observed in data from OEF/OIF.

Crush syndrome, or traumatic rhabdomyolysis, often follows structural collapse and entrapment causing crush injury. Severe muscle damage, prolonged ischemia, and cell death can result in release of myoglobin, urates, and potassium. Myoglobinuria produces dark amber urine that will test positive for hemoglobin on urine dipstick analysis. Significant rhabdomyolysis can cause hypovolemia, metabolic acidosis, hyperkalemia, hypocalcemia, and coagulopathy.[124] Early and aggressive fluid resuscitation to ensure adequate renal perfusion and urinary output is vital in preventing renal failure.[124,125]

Crush syndrome, or traumatic rhabdomyolysis, often follows structural collapse and body entrapment.

Myoglobinuria produces dark amber urine that will test positive for hemoglobin on urine dipstick analysis. Significant rhabdomyolysis can cause the following:

- hypovolemia,
- metabolic acidosis,
- hyperkalemia,
- hypocalcemia, and
- coagulopathy.

Osmotic diuretics (mannitol) and intravenous sodium bicarbonate are commonly advocated as adjuncts to prevent renal failure.[124] Alkalinization of the urine with intravenous sodium bicarbonate is thought to decrease intratubular precipitation of myoglobin in the kidneys.[124] Mannitol has been suggested to minimize intratubular pigment deposition, act as a renal vasodilator, and act as a free-radical scavenger.[124,126,127,128] It is worth noting that some authors feel that there is no clear clinical data showing benefit with either of these agents over simple fluid resuscitation.[124,129] Compartment syndromes can also develop in association with a crush injury or over-resuscitation and are discussed in later chapters.

Alkalinization of the urine with intravenous sodium bicarbonate is thought to decrease intratubular precipitation of myoglobin in the kidneys. Mannitol has been suggested to

- minimize intratubular pigment deposition,
- act as a renal vasodilator, and
- act as a free-radical scavenger.

Quaternary Blast Injury

Quaternary blast injury encompasses blast sequelae that include, but are not limited to, burns, inhalation injury, and asphyxiation.[119] Burns are a form of quaternary blast injury in OEF and OIF and more frequently occur when victims are trapped in a burning vehicle or building than because of the blast fireball (which lasts for milliseconds). Burns that immediately follow an explosion result from exposure to the intense heat of the blast and indicate close proximity to the point of detonation.[123]

An analysis of OEF and OIF casualties with significant burns treated at the US Army Institute of Surgical Research (USAISR) between 2003 and 2005 revealed increases in burn frequency, extent, and severity.[130] Findings included:

- Burns caused by explosions increased from 18 percent to 69 percent
- Total body surface area burned increased from 15 percent (± 12 percent) to 21 percent (± 23 percent)
- Injury severity scores (ISS) increased from minor (8 ±11) to moderate/severe (17 ±18)[130]

Inhalation injury is especially prevalent with building collapse.

As illustrated in the 1993 World Trade Center bombing, 93 percent of victims suffered acute and chronic inhalation injuries

Burns were caused primarily by IEDs (55 percent), car bombs (16 percent), and RPGs (15 percent) and were largely sustained in unprotected areas of the body. The hands and face were the most frequently burned areas, and only one-third (36 percent) of burned patients resumed full military duty. The study also revealed an increase in the frequency of inhalation injury in the current conflicts from 5 percent to 26 percent.[130]

Inhalation injury is especially prevalent with building collapse, as illustrated in the 1993 World Trade Center bombing, in which 93 percent of victims suffered acute and chronic inhalation injuries.[113]

Burns are a form of quaternary blast injury and occur more frequently when victims are trapped in a burning vehicle or building, rather than due to a blast fireball.

Quinary Blast Injury

Quinary effects largely refer to contamination of tissues resulting from the release of chemical, biological agents, or radioactive materials upon detonation of an explosive device. A unique type of quinary injury encountered in OEF and OIF is that inflicted by human-remains-shrapnel, or pieces of bone from suicide bombers or other victims that cause penetrating injuries and increase the risk of transmission of blood-borne diseases such as hepatitis or human immunodeficiency virus (HIV).[87,131,132,133]

These agents are classified as

- nerve
- blister (vesicant), and
- choking agents.

Indications of nerve agent exposure include a variety of autonomic and neuromuscular signs and symptoms, for example.

- pinpoint pupils,
- muscular twitching,
- unexplained nasal secretion,
- hypersalivation,
- tightness of the chest,
- shortness of breath,
- nausea,
- abdominal cramps,
- seizures,
- paralysis, and
- respiratory failure.

Chemical agents may be inhaled or absorbed through the skin, and can induce coughing, itching, skin, and eye inflammation.[119] These agents are classified as nerve, blister (vesicant), and choking agents. Indications of nerve agent exposure include a variety of autonomic and neuromuscular signs and symptoms (e.g., pinpoint pupils, muscular twitching, unexplained nasal secretion, hypersalivation, tightness of the chest, shortness of breath, nausea, abdominal cramps, seizures, paralysis, and respiratory failure). Immediate intramuscular injection of atropine, combined if possible with pralidoxime chloride (2-PAM), is recommended.[134] Blister agents cause a spectrum of injury to exposed surfaces (e.g., skin, eyes, and mucous membranes) and result in symptoms over varying timeframes (minutes to several hours). Immediate decontamination by removal of contaminated clothing and irrigation of exposed surfaces with large amounts of water is first-line therapy.[134,135,136] Choking agents cause coughing, tightness in the chest, vomiting, headache, and lacrimation.[134] Treatment consists of removing the patient from the offending agent and providing supportive care. All of these effects can exacerbate preexisting conditions.[64] Exposure to radiation released in an explosion will result in a variety of effects that are largely determined by the size and type of explosion, which radioactive elements are involved, length of exposure, and other factors.[137] While discussion of chemical and biological

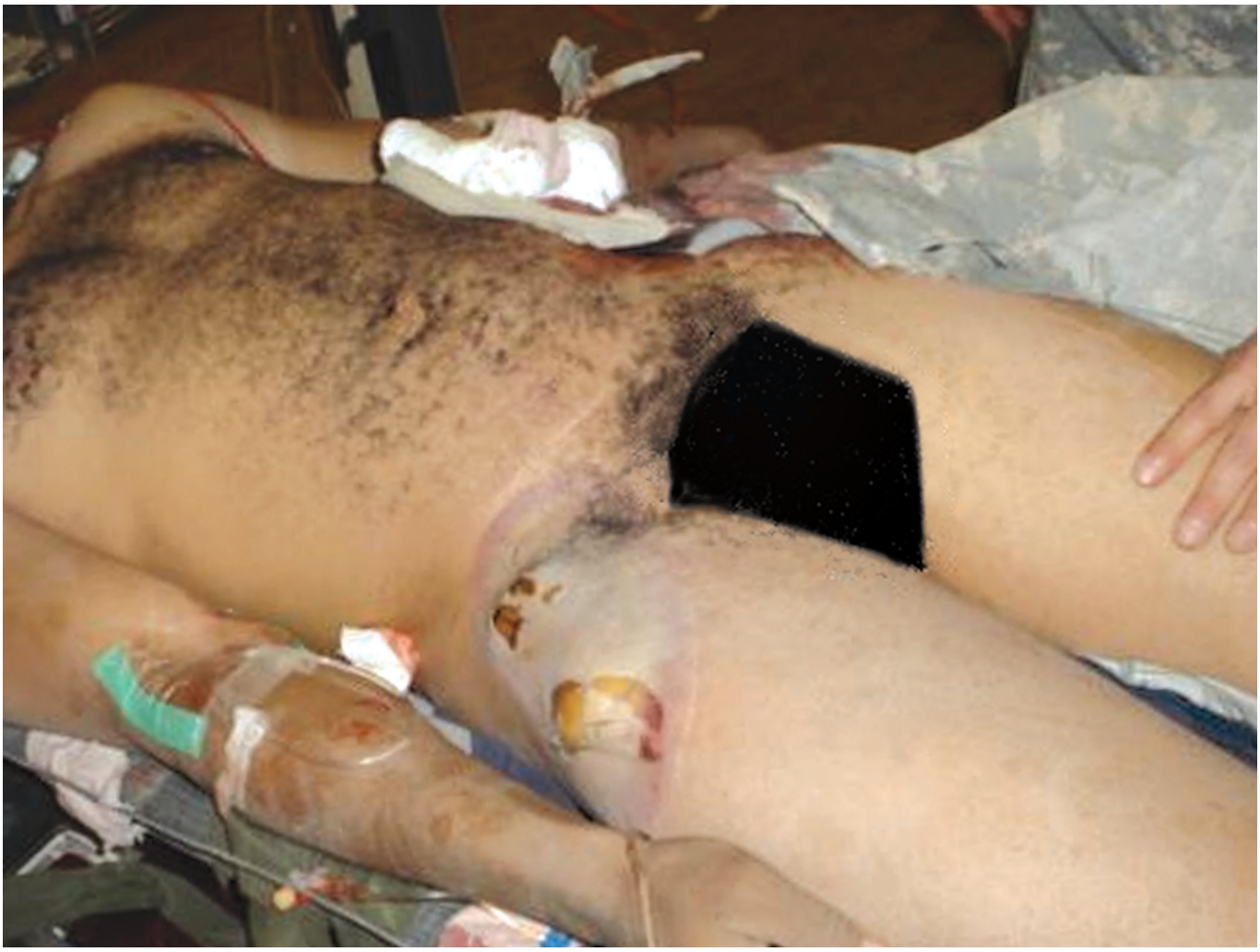


Figure 25. (Above) *Unexploded ordnance tenting the subcutaneous tissue of the right thigh, having traversed the pelvis in a left-to-right trajectory. The extruding tail of the rocket is demarcated by the arrow. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.*

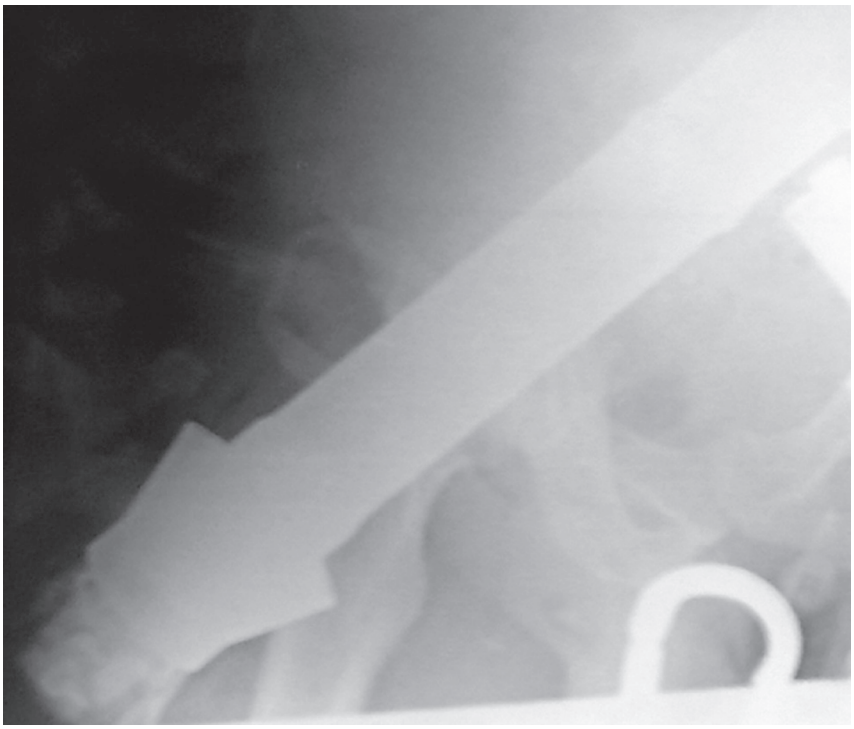


Figure 26. (Right) *A radiograph of the UXO embedded in the pelvis and femur confirms the warhead is not attached to the rocket. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.*

agents and radiation threats is beyond the scope of this chapter, CCC providers should have a decontamination plan in place to avoid secondary contamination of their combat care facility and themselves.

Know your environment, and have a decontamination plan in place to avoid secondary contamination of yourself and your combat care facility.

Management Considerations

While damage-control practices will need to be applied to explosion-related injury management by CCC providers, the polytrauma that ensues in bomb explosions creates management challenges.[79] Patients with concurrent brain and hemorrhaging solid organ injuries often need to undergo immediate damage control surgery prior to delineation of a brain injury via CT imaging.[79] Advanced ventilatory strategies (e.g., permissive hypercapnia, high-frequency oscillatory ventilation) may often be required to manage lung overpressure injuries.[79] The coagulopathy that often accompanies blast injury will need to be rapidly recognized and appropriately managed.[79] Finally, the crystalloid and blood product requirements in patients with multiple injuries that include burns, head, and pulmonary injuries must be balanced against the risks (among others) of dilutional coagulopathy and compartment syndromes.[45,138]

Embedded Unexploded Ordnance

The management of intracorporeal unexploded ordnances (UXOs) represents a unique challenge for CCC providers. Mortars, rockets, and grenades that fail to trigger may become embedded in a casualty without exploding (Figs. 25 and 26). Due to the extensive time and resources needed to appropriately manage these casualties and the potential for collateral damage from premature detonation, military recommendations include initially triaging such patients as nonemergent, isolating them from others, and operating on them last.[15,139]

Military recommendations include:
- initially triaging such patients as nonemergent,
- isolating them from others, and
- operating on them last.

According to Lien, "the fuse is the key to understanding unexploded ordnance."[139] A fuse serves as a trigger for an explosive device and may be set off by impact, electromagnetically, or as a function of time or distance traveled. Care should be taken to minimize manipulation or movement of the UXO and casualty. If helicopter transport is necessary, the patient should be flown independent of other patients, and the flight crew should be kept to a minimum and protected with body armor.[140] Diagnostic and therapeutic

Electrical equipment, such as:
- electrocautery,
- surgical saws or drills,
- blood warmers,
- monitors,
- defibrillators,
- ultrasound, or
- computed tomography imaging,

should be avoided until the unexploded ordinance is removed.

medical equipment can trigger a fuse and inadvertently cause an explosion. Electrical equipment, such as electrocautery, surgical saws or drills, blood warmers, monitors, defibrillators, ultrasound, or computed tomography imaging should be avoided until the UXO is removed.[15] Some of these diagnostic and treatment adjuncts may radiate electrical fields, cause severe vibration, or result in elevated temperatures that may arm the fusing mechanism.[139]

Plain radiography is considered safe and is used to identify the type of munition and fuse, as well as to define the surgical approach to embedded UXOs.[139] As part of preoperative planning, the explosive ordnance disposal (EOD) team should be notified and present to assist in the proper handling and disposal of the UXO.

Traditional recommendations for removal of UXOs include the use of regional or spinal anesthesia and departure of operating room personnel except for the operating surgeon.[15] Recent case reports from OEF and OIF have suggested that general anesthesia allows for a more controlled environment, and that having the appropriate, rather than minimal, number of assistants in the operating room can lead to the most successful outcomes.[140] Operating room staff should wear protective gear, including body armor, ballistic eye protection, and a helmet. Sandbags should be positioned around the patient. Gentle technique and en-bloc resection of the UXO minimizes manipulation and the inherent risk of detonating the device. If embedded in an extremity, amputation should be considered.[15,140]

Conclusions

Understanding modern warfare, including the types of weapons employed and the mechanisms and patterns of injury they cause, is critical to providing optimal CCC. The primary mechanisms of combat injury in OEF and OIF are small arms and explosives. Explosion-related injuries account for a majority of the injuries and deaths in OEF and OIF. Improvised explosive device attacks have become a mainstay in the current conflicts. Explosive devices produce the ultimate polytrauma (i.e., a wide range of injury types to many body regions caused by the full range of injury mechanisms). Explosions produce patterns of injury

Understanding modern warfare, including the types of weapons employed and the mechanisms and patterns of injury they cause, is critical to providing optimal combat casualty care.

that are distinct from those of other mechanisms. In an open-space explosion, the primary mechanism of injury is fragment penetration. Injuries and deaths from fragments occur much further from the point of detonation than do those associated with the primary blast. The simultaneous combination of different blast injury mechanisms produces a complex array of injuries. Combat casualty care providers must fully understand these complex injuries and their management to ensure optimal patient outcomes.

References

1. Eastridge BJ, Jenkins D, Flaherty S, et al. Trauma system development in a theater of war: experiences from Operation Iraqi Freedom and Operation Enduring Freedom. J Trauma 2006;61(6):1366-1372; discussion 1372-1373.

2. Owens BB, Kragh JF Jr, Wenke JC, et al. Combat wounds in Operation Iraqi Freedom and Operation Enduring Freedom. J Trauma 2008;64(2):295-299.

3. Burns BD, Zuckerman S. The wounding power of small bomb and shell fragments. London, England: British Ministry of Supply, Advisory Council on Scientific Research and Technical Development; 1942.

4. Beebe GW, DeBakey ME. Location of hits and wounds. In: Battle casualties: incidence, mortality, and logistic considerations. Springfield, IL: Charles C Thomas; 1952. p. 165-205.

5. Reister FA. Battle casualties and medical statistics: U.S. Army experience in the Korean War. Washington, DC: The Surgeon General, Department of the Army; 1973.

6. Hardaway RM. Viet Nam wound analysis. J Trauma 1978;18(9):635-643.

7. Shanker T. Pentagon to outline shift in war planning strategy. The New York Times, 2009 June 22.

8. Kelly JF, Ritenour AE, McLaughlin DF, et al. Injury severity and causes of death from Operation Iraqi Freedom and Operation Enduring Freedom: 2003-2004 versus 2006. J Trauma 2008;64(2 Suppl):S21-26; discussion S26-27.

9. Brethauer SA, Choo A, Chambers LW, et al. Invasion vs insurgency: US Navy/Marine Corps forward surgical care during Operation Iraqi Freedom. Arch Surg 2008;143(6):564-569.

10. Bellamy RF, Zajtchuk R. The weapons of conventional land warfare. In: Bellamy RF, Zajtchuk R, editors. Conventional warfare: ballistic, blast, and burn injuries. Washington, DC: The Borden Institute; 1991. p. 1-52.

11. Beebe GW, DeBakey ME. Death from wounding. In: Battle casualties: incidence, mortality, and logistic considerations. Springfield, IL: Charles C Thomas; 1952. p. 74-147.

12. US Department of Defense (US DoD). DoD personnel and military casualty statistics. Global War on Terrorism by Reason: October 7, 2001 through October 31, 2009. DoD, Statistical Information Analysis Division (SIAD), Defense Manpower Data Center (DMDC) [cited 2009 Nov 4]. Available from: URL: http://siadapp.dmdc.osd.mil/personnel/CASUALTY/gwot_reason.pdf.

13. Amnesty International. Blood at the crossroads: making the case for global arms trade treaty. London, UK: Amnesty International Publications 2008 [cited 2009 Oct 28]. Available from: URL: http://www.amnesty.org/en/library/asset/ACT30/011/2008/en/19ea0e74-8329-11dd-8e5e-3ea85d15a69/act300112008en.pdf.

14. Santucci RA, Chang YJ. Ballistics for physicians: Myths about wound ballistics and gunshot injuries. J Urol 2004;171(4):1408-1414.

15. US Department of Defense (US DoD). Weapons Effects and Parachute Injuries. In: Emergency War Surgery, Third United States Revision. Washington, DC: Department of the Army, Office of the Surgeon General, Borden Institute; 2004. p. 1.1-1.14.

16. Arnold JL, Halpern P, Tsai MC, et al. Mass casualty terrorist bombings: a comparison of outcomes by bombing type. Ann Emerg Med 2004;43(2):263-273.

17. Centers for Disease Control and Prevention (CDC). Explosions and blast injuries: A primer for clinicians. 2006 [cited 2009 Nov 5]. Available from: URL: http://www.bt.cdc.gov/masscasualties/explosions.asp.

18. Wightman JM, Gladish SL. Explosions and blast injuries. Ann Emerg Med 2001;37(6):664-678.

19. Votey SR, UCLA Emergency Medicine Multimedia Education Working Group. Chemical, Biological, Radiological, Nuclear, Explosives: Emergency Preparedness for Medical Care Providers. Multimedia CD-ROM. California Hospital Bioterrorism Preparedness Committee and California Emergency Medical Services Authority, 2004.

20. Eastridge BJ. Things that go boom: injuries from explosives. J Trauma 2007;62(6 Suppl):S38.

21. Ritenour AE, Baskin TW. Primary blast injury: Update on diagnosis and treatment. Crit Care Med 2008;36(7 Suppl):S311-317.

22. Butler DK. Landmines and UXO. The Leading Edge 1997;16(10):1460-1461.

23. Surrency AB, Graitcer PL, Henderson AK. Key factors for civilian injuries and death from exploding landmines and ordnance. Inj Prev 2007;13(3):197-201.

24. Human Rights Watch. Landmines in Iraq: Questions and Answers. Human Rights News, Arms Division Homepage. 2002 Dec [cited 2009 Oct 30]. Available from: URL: http://www.hrw.org/legacy/campaigns/iraq/iraqmines1212.htm.

25. Afghanistan ICRC Mine Data Collection Programme – Details [cited 2001 May 25]. Available from: URL: http://www.icrc.org.

26. Centers for Disease Control and Prevention (CDC). Injuries associated with landmines and unexploded ordnance—Afghanistan, 1997-2002. JAMA 2003;52(36):859-862.

27. Injuries associated with landmines and unexploded ordnance—Afghanistan, 1997-2002. MMWR 2003;52:859-862.

28. Hamdan TA. Missile injuries of the limbs: an Iraqi perspective. J Am Acad Orthop Surg 2006;14(10 Spec No.):S32-36.

29. International Committee of the Red Cross (ICRC). Book I: Weapon Contamination. Geneva: ICRC; 2007 Aug [cited 2009 Nov 2]. Available from: URL: http://www.scribd.com/doc/22010755/Book-I-Weapon-contamination-environment.

30. Office of the Secretary of Defense (OSD). Landmines. Force Health Protection and Readiness. 2009 [cited 2009 Nov 1]. Available from: URL: http://fhp.osd.mil/factsheetDetail.jsp?fact=14.

31. Covey DC. Blast and fragment injuries of the musculoskeletal system. J Bone Joint Surg Am 2002;84-A(7):1221-1234.

32. German Mine 35/44 Bouncing Betty. European Center of Military History 2009 Feb 22 [cited 2009 Nov 1]. Available from: URL: http://www.eucmh.com/2009/02/22/schrapnellmine-3544-bouncing-betty/.

33. Woebkenberg BJ, Devine J, Rush R, et al. Nonconventional uses of the rocket-propelled grenade and its consequences. Mil Med 2007;172(6):622-624.

34. RPG-7: The famous RPG-7 is in use with over 40 countries. Military Factory 2009 Jul 7 [cited 2009 Nov 2]. Available from: URL: http://www.militaryfactory.com/smallarms/detail.asp?smallarms_id=10.

35. Wilson C. CRS Report for Congress – Improvised Explosive Devices (IEDs) in Iraq and Afghanistan: Effects and Countermeasures. Order Code RS22330, 2008.

36. Vanden Brook T. IEDs now cause 75% of Afghanistan casualties. USA Today 2009 Apr 4.

37. Tyson AS. U.S. combat injuries rise sharply. The Washington Post 2009 Oct 31;Sect A1, A7.

38. Shaunessy L, Hornick E, Starr B. Detecting IEDs: a daunting challenge for U.S. military. Joint IED Defeat Organization (JIEDDO) 2009 Oct 27 [cited 2009 Nov 4]. Available from: URL: https://www.jieddo.dod.mil/article.aspx?ID=685.

39. US Marine Corps. Field Medical Service Technician Student Manual—2008 Web Edition. Camp LeJeune, NC; 2008: FMST 1206, Improvised Explosive Devices. Available at: http://www.operationalmedicine.org/TextbookFiles/FMST_20008/FMST_1206.htm. Accessed May 14, 2012.

40. Anderson JW. Bigger, stronger homemade bombs now to blame for half of U.S. deaths. The Washington Post 2005 Oct 21.

41. Morrison JJ, Mahoney PF, Hodgetts T. Shaped charges and explosively formed penetrators: Background for clinicians. J R Army Med Corps 2007;153(3):184-187.

42. Ramasamy A, Harrisson SE, Clasper JC, et al. Injuries from roadside improvised explosive devices. J Trauma 2008;65(4):910-914.

43. McDiarmid MA, Engelhardt SM, Oliver M, et al. Health surveillance of Gulf War I veterans exposed to depleted uranium: updating the cohort. Health Phys 2007;93(1):60-73.

44. Champion HR, Holcomb JB, Young LA. Injuries from explosions: Physics, biophysics, pathology, and required research focus. J Trauma 2009;66(5):1468-1477.

45. Kluger Y, Peleg K, Daniel-Aharonson L, et al. The special injury pattern in terrorist bombings. J Am Coll Surg 2004;199(6):875-879.

46. Nelson TJ, Clark T, Stedje-Larsen ET, et al. Close proximity blast injury patterns from improvised explosive devices in Iraq: A report of 18 cases. J Trauma 2008;65(1):212-217.

47. Stein M, Hirshberg A. Medical consequences of terrorism: The conventional weapon threat. Surg Clinic North Am 1999;79(6):1537-1552.

48. Mallonee S, Shariat S, Stennies G, et al. Physical injuries and fatalities resulting from the Oklahoma City bombing. JAMA 1996;276(5):382-387.

49. Quenemoen LE, Davis YM, Malilay J, et al. The World Trade Center bombing: injury prevention strategies for high-rise building fires. Disasters 1996;20(2):125-132.

50. Phillips YY, Richmond DR. Primary blast injury and basic research: a brief history. In Bellamy RF, Zajtchuk R, editors. Conventional warfare: ballistic, blast, and burn injuries. Washington, DC, Office of the Surgeon General, Department of the Army; 1989. p. 221-240.

51. Frykberg ER, Tepas JJ 3rd. Terrorist bombings. Lessons learned from Belfast to Beirut. Ann Surg 1988;208(5):569-576.

52. Almogy G, Rivkind AI. Surgical lessons learned from suicide bombing attacks. J Am Coll Surg 2006;202(2):313-319.

53. Aschkenasy-Steuer G, Shamir M, Rivkind A, et al. Clinical review: The Israeli experience: conventional terrorism and critical care. Crit Care 2005;9(5):490-499.

54. Kluger Y. Bomb explosions in acts of terrorism – detonation, wound ballistics, triage and medical concerns. Isr Med Assoc J 2003;5(4):235-240.

55. National Counterterrorism Center (NCTC). 2007 Report on Terrorism. 2008 [cited 2008 Sept 2]. Available from: URL: http://wits.nctc.gov/Reports.do?page=5.

56. Cooper GJ, Maynard RL, Cross NL, et al. Casualties from terrorist bombings. J Trauma 1983;23(11):955-967.

57. Katz E, Ofek B, Adler J, et al. Primary blast injury after a bomb explosion in a civilian bus. Ann Surg 1989;209(4):484-488.

58. Leibovici D, Gofrit ON, Stein M, et al. Blast injuries: Bus versus open-air bombings–a comparative study of injuries in survivors of open-air versus confined-space explosions. J Trauma 1996;41(6):1030-1035.

59. Kluger Y, Kashuk J, Mayo A. Terror bombings–mechanisms, consequences, and implications. Scand J Surg 2004;93(1):11-14.

60. Mayo A, Kluger Y. Terrorist bombing. World J Emerg Surg 2006;1:1-33.

61. Federal Emergency Management Agency (FEMA). Explosive blast. In Primer to Design Safe Schools Projects in Case of Terrorist Attacks. Washington, DC: FEMA; 2003. p. 4-1–4-13.

62. Thompson D, Brown S, Mallonee S, et al. Fatal and non-fatal injuries among U.S. Air Force personnel resulting from the terrorist bombing of the Khobar Towers. J Trauma 2004;57(2):208-215.

63. Wade CE, Ritenour AE, Eastridge BJ, et al. Explosion injuries treated at combat support hospitals in the global war on terrorism. In: Elsayed N, Atkins J, editors. Explosion and blast-related injuries: effects of explosion and blast from military operations and acts of terrorism. Amsterdam; Boston: Elsevier; 2008. p. 41-72.

64. DePalma RG, Burris DG, Champion HR, et al. Blast injuries. N Engl J Med 2005;352(13):1335-1342.

65. Hoge CW, McGurk D, Thomas JL, et al. Mild traumatic brain injury in U.S. soldiers returning from Iraq. N Engl J Med 2008;358(5):453-463.

66. Harrison CD, Bebarta VS, Grant GA. Tympanic Membrane Perforation After Combat Blast Exposure in Iraq: A Poor Biomarker of Primary Blast Injury. J Trauma 2009;67(1):210-211.

67. Owen-Smith M. Bomb blast injuries: In an explosive situation. Nurs Mirror 1979;149(13):35-39.

68. Chait RH, Casler J, Zajtchuk JT. Blast injury to the ear: Historical perspective. Ann Otol Rhinol Laryngol 1989;140(Suppl):9-12.

69. Cave KM, Cornish EM, Chandler DW. Blast injury to the ear: Clinical update from the global war on terror. Mil Med 2007;172(7):726-730.

70. Depenbrock P. Tympanic membrane perforation in IED blasts. J Spec Oper Med 2008;8:51-53.

71. Kronenberg J, Ben-Shoshan J, Modan M, et al. Blast injury and cholesteatoma. Am J Otol 1988;9(2):127-130.

72. Xydakis MS, Bebarta VS, Harrison CD, et al. Tympanic-membrane perforation as a marker of concussive brain injury in Iraq. N Engl J Med 2007;357(8):830-831.

73. Sharpnak D, Johnson A, Philips Y. The pathology of primary blast injury. In: Bellamy R, Zatchuk R, editors. Conventional warfare: ballistic, blast, and burn injuries. Washington, D.C.: Office of the Surgeon General of the US Army, 1991. p. 271-294.

74. Mayorga MA. The pathology of primary blast overpressure injury. Toxicology 1997;121(1):17-28.

75. Avidan V, Hersch M, Armon Y, et al. Blast lung injury: clinical manifestations, treatment, and outcomes. Am J Surg 2005;190(6):927-931.

76. de Ceballos JP, Turegano-Fuentes F, Perez-Diaz D, et al. 11 March 2004: The terrorist bomb explosions in Madrid, Spain—an analysis of the logistics, injuries sustained and clinical management of casualties treated at the closest hospital. Crit Care 2005;9(1):104-111.

77. Aharonson-Daniel L, Klein Y, Peleg K. ITG. Suicide bombers form a new injury profile. Ann Surg 2006;244(6):1018-1023.

78. Goh SH. Bomb blast mass casualty incidents: initial triage and management of injuries. Singapore Med J 2009;50(1):101-106.

79. Kashuk JL, Halperin P, Caspi G, et al. Bomb explosions in acts of terrorism: evil creativity challenges our trauma systems. J Am Coll Surg 2009;209(1):134-140.

80. Pizov R, Oppenheim-Eden A, Matot I, et al. Blast lung injury from an explosion on a civilian bus. Chest 1999;115(1):165-172.

81. Cohn SM. Pulmonary contusion: Review of the clinical entity. J Trauma 1997;42(5):973-979.

82. Halpern P, Tsai MC, Arnold JL, et al. Mass-casualty, terrorist bombings: Implications for emergency department and hospital emergency response (Part II). Prehosp Disaster Med 2003;18(3):235-241.

83. Mellor SG, Cooper GJ. Analysis of 828 servicemen killed or injured by explosion in Northern Ireland 1970-84: The Hostile Action Casualty System. Br J Surg 1989;76(10):1006-1010.

84. Irwin RJ, Lerner MR, Bealer JF, et al. Shock after blast wave injury is caused by a vagally mediated reflex. J Trauma 1999;47(1):105-110.

85. Guzzi LM, Argyros G. The management of blast injury. Eur J Emerg Med 1996;3(4):252-255.

86. Yang Z, Wang Z, Tang C, et al. Biological effects of weak blast waves and safety limits for internal organ injury in the human body. J Trauma 1996;40(3 Suppl):S81-84.

87. Singer P, Cohen JD, Stein M. Conventional terrorism and critical care. Crit Care Med 2005;33(1 Suppl):S61-65.

88. Born CT. Blast trauma: The fourth weapon of mass destruction. Scand J Surg 2005;94(4):279-285.

89. Mellor SG. The pathogenesis of blast injury and its management. Br J Hosp Med 1988;39(6):536-539.

90. Grady D. The wounded: The survivors – Surviving multiple injuries; struggling back from war's once-deadly wounds. New York Times 2006 Jan 22.

91. Henry M. Jackson Foundation for the Advancement of Military Medicine, Inc. The Defense and Veterans Brain Injury Center—Providing care for soldiers with traumatic brain injury. 2006 [cited 2009 Jan 4]. Available from: URL: http://www.hjf.org/research/featureDVBIC.html.

92. Tanielian T, Jaycox LH, editors. Invisible Wounds of War: Psychological and Cognitive Injuries, Their Consequences, and Services to Assist Recovery. Santa Monica, CA: Rand Corporation; 2008.

93. Okie S. Traumatic brain injury in the war zone. N Engl J Med 2005;352(20):2043-2047.

94. Warden DL, Ryan LM, Helmick KM, et al. War neurotrauma: The Defense and Veterans Brain Injury Center (DVBIC) experience at Walter Reed Army Medical Center (WRAMC). J Neurotrauma 2005;22:1178.

95. Brain Injury Association of America (BIAA). Traumatic brain injury in the United States: A call for public/private cooperation. McLean, VA: BIAA; 2007.

96. Taber KH, Warden DL, Hurley RA. Blast-related traumatic brain injury: What is known? J Neuropsychiatry Clin Neurosci 2006;18(2):141-145.

97. Bell RS, Vo AH, Neal CJ, et al. Military traumatic brain and spinal column injury: A 5-year study of the impact blast and other military grade weaponry on the central nervous system. J Trauma 2009;66(4 Suppl):S104-111.

98. Cernak I, Wang Z, Jiang J, et al. Ultrastructural and functional characteristics of blast injury-induced neurotrauma. J Trauma 2001;50(4):695-706.

99. Guy RJ, Glover MA, Cripps NP. Primary blast injury: pathophysiology and implications for treatment. Part III: Injury to the central nervous system and limbs. J R Nav Med Serv 2000;86(1):27-31.

100. Trudeau DL, Anderson J, Hansen LM, et al. Findings of mild traumatic brain injury in combat veterans with PTSD and a history of blast concussion. J Neuropsychiatry Clin Neurosci 1998;10(3):308-313.

101. Belanger HG, Kretzmer T, Yoash-Gantz R, et al. Cognitive sequelae of blast-related versus other mechanisms of brain trauma. J Int Neuropsychol Soc 2009;15(1):1–8.

102. Wilk JE, Thomas JL, McGurk DM, et al. Mild traumatic brain injury (concussion) during combat: lack of association of blast mechanism with persistent postconcussive symptoms. J Head Trauma Rehabil 2010;25(1):9–14.

103. Cernak I, Savic J, Malicevic Z, et al. Involvement of the central nervous system in the general response to pulmonary blast injury. J Trauma 1996;40(3 Suppl):S100-104.

104. Cernak I, Savik J, Ignjatovic D, et al. Blast injury from explosive munitions. J Trauma 1999;47(1):96-103.

105. Ling G, Bandak F, Armonda R, et al. Explosive blast neurotrauma. J Neurotrauma 2009;26(6):815–825.

106. Joint Theater Trauma System (JTTS) Clinical Practice Guideline. Management of mild traumatic brain injury (mTBI)/concussion in the deployed setting. 2008 Nov 21 [cited 2009 Nov 12]. Available from: URL: http://www.usaisr.amedd.army.mil/cpgs.html.

107. Joint Theater Trauma System (JTTS) Clinical Practice Guideline. Management of patients with severe head trauma. 2009 Feb 13 [cited 2009 Nov 12]. Available from: URL: http://www.usaisr.amedd.army.mil/cpgs.html.

108. Defense and Veterans Brain Injury Center (DVBIC). Military Acute Concussion Evaluation (MACE). 2007 Jul [cited 2007 Jul 13]. Available from: URL: http://www.dvbic.org/pdfs/DVBIC_pocket_card.pdf.

109. Centers for Disease Control and Prevention. Blast Injuries: Fact Sheets for Professionals. National Center for Injury Prevention and Control 2008 [cited 2008 Dec 22]. Available from: URL: http://emergency.cdc.gov/masscasualties/blastinjuryfacts.asp.

110. Hull JB, Cooper GJ. Pattern and mechanism of traumatic amputation by explosive blast. J Trauma 1996;40(3 Suppl):S198-205.

111. Linsky R, Miller A. Types of explosions and explosive injuries defined. In: Keyes DC, editor. Medical response to terrorism: preparedness and clinical practice. Philadelphia, PA: Lippincott Williams & Wilkins; 2005. p. 198-211.

112. Kluger Y, Mayo A, Hiss J, et al. Medical consequences of terrorist bombs containing spherical metal pellets: analysis of a suicide terrorism event. Eur J Emerg Med 2005;12(1):19-23.

113. Arnold JL, Tsai MC, Halpern P, et al: Mass-casualty, terrorist bombings: epidemiological outcomes, resource utilization, and time course of emergency needs (Part I). Prehosp Disaster Med 2003;18(3):220-234.

114. Crane J. Explosive injury. In: Payne-James J, Byard RW, Corey TS, et al, editors. Encyclopedia of forensic and legal medicine. London: Elsevier; 2005. p. 98-110.

115. Bowyer GW, Cooper GJ, Rice P. Small fragment wounds: biophysics and pathophysiology. J Trauma 1996; 40(3 Suppl):S159-164.

116. Fasol R, Irvine S, Zilla P. Vascular injuries caused by anti-personnel mines. J Cardiovasc Surg 1989;30(3):467-472.

117. Amato JJ, Billy LJ, Gruber RP, et al. Vascular injuries. An experimental study of high and low velocity missile wounds. Arch Surg 1970;101(2):167-174.

118. Fackler ML. Wound ballistics. A review of common misconceptions. JAMA 1988;259(18):2730-2736.

119. Champion HR, Baskin T, Holcomb JB. Injuries from explosives. In: McSwain NE, Salomone J, editors. Basic and advanced pre-hospital trauma life support, military edition. 2nd ed. St. Louis, MO: Mosby, National Association of Emergency Medical Technicians; 2006.

120. Boodram B, Torian L, Thomas P, et al. Rapid assessment of injuries among survivors of the terrorist attack on the World Trade Center—New York City, September 11, 2001. MMWR 2002 Jan 11 [cited 2009 Nov 13];51(01);1-5. Available from: URL: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5101a1.htm.

121. Wedmore IS, McManus JG Jr, Coakley TA. Penetrating and explosive injury patterns. In: Schwartz RB, McManus JG, Swienton RE, editors. Tactical emergency medicine. Philadelphia, PA: Lippincott, Williams & Wilkins; 2008. p. 63-73.

122. Amirjamshidi A, Abbassioun K, Rahmat H. Minimal debridement or simple wound closure as the only surgical treatment in war victims with low-velocity penetrating head injuries. Indications and management protocol based upon more than 8 years follow-up of 99 cases from Iran-Iraq conflict. Surg Neurol 2003;60(2):105-110.

123. Frykberg ER. Explosions and blast injury. In: Shapira SC, Hammond JS, Cole LA, editors. Essentials of terror medicine. New York: Springer; 2009. p. 171-193.

124. Huerta-Alardin AL, Varon J, Marik PE. Bench-to-bedside review: Rhabdomyolysis – an overview for clinicians. Crit Care 2005;9(2):158-169.

125. Abassi ZA, Hoffman A, Better OS. Acute renal failure complicating muscle crush injury. Semin Nephrol 1998;18(5):558-565.

126. Zager RA. Rhabdomyolysis and myohemoglobinuric acute renal failure. Kidney Int. 1996;49(2):314-326.

127. Shilliday I, Allison ME. Diuretics in acute renal failure. Ren Fail 1994;16(1):3-17.

128. Odeh M. The role of reperfusion-induced injury in the pathogenesis of the crush injury. N Engl J Med 1991;324(20):1417-1422.

129. Homsi E, Barreiro MF, Orlando JM, et al. Prophylaxis of acute renal failure in patients with rhabdomyolysis. Ren Fail 1997;19(2):283-288.

130. Kauvar DS, Wolf SE, Wade CE, et al. Burns sustained in combat explosions in Operations Iraqi and Enduring Freedom (OIF/OEF explosion burns). Burns 2006;32(7):853-857.

131. Braverman I, Wexler D, Oren M. A novel mode of infection with hepatitis B: penetrating bone fragments due to the explosion of a suicide bomber. Isr Med Assoc J 2002;4(7):528-529.

132. Leibner ED, Weil Y, Gross E, et al. A broken bone without a fracture: traumatic foreign bone implantation resulting from a mass casualty bombing. J Trauma 2005;58(2):388-390.

133. Wong JM, Marsh D, Abu-Sitta G, et al. Biological foreign body implantation in victims of the London July 7th suicide bombings. J Trauma 2006;60(2):402-404.

134. Federation of American Scientists (FAS). Types of chemical weapons. 2008 [cited 2008 Dec 30]. Available from: URL: http://www.fas.org/programs/ssp/bio/chemweapons/cwagents.html.

135. Chemical & biological attacks, detection, & response FAQ. KI4U, Inc., 2008 [cited 2008 Dec 30]. Available from: URL: http://www.ki4u.com/chemical_biological_attack_detection_response.htm.

136. Centers for Disease Control and Prevention. Chemical categories: A to Z [cited 2008 Dec 30]. Available from: URL: http://www.bt.cdc.gov/agent/agentlistchem-category.asp.

137. DeGarmo B. Radiological terrorism: the 'dirty bomb.' 2003 [cited 2008 Dec 31]. Available from: URL: bioterrorism.slu.edu/bt/products/ahec_rad/ppt/Dirty%20Bomb.ppt.

138. Kashuk JL, Moore EE, Johnson JL, et al. Postinjury life threatening coagulopathy: is 1:1, fresh frozen plasma: packed red blood cells the answer? J Trauma 2008;65(2):261-270; discussion 270-271.

139. Lein B, Holcomb J, Brill S, et al. Removal of unexploded ordnance from patients: a 50-year military experience and current recommendations. Mil Med 1999;164(3):163-165.

140. Nessen SC, Lounsbury DE, Hetz SP, editors. Removal of an Unexploded Ordnance. In: War Surgery in Afghanistan and Iraq: A Series of Cases, 2003-2007. Washington, DC: Department of the Army, Office of the Surgeon General, Borden Institute; 2008. p. 373-376.