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Bombs, Mines, Blast, Fragmentation, and Thermobaric Mechanisms of Injury

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Introduction

Once confined to the battlefield and the occasional industrial accident, the sequela of explosive force has now become all too commonplace and continues to increase as industry expands and explosive weaponry proliferates. The chaos and "fog of war" no longer can be considered the sole province of the battlefield. The ubiquitous threat of terrorism places responsibility for the care of victims not only upon the military surgeon, but upon civilian counterparts as well. The medical system, military, and civilians must understand the pathophysiology of injury induced from explosive devices, be they letter bombs, shaped warheads from a rocket propelled grenade (RPG), antipersonnel land mines, aerial-delivered cluster bombs, or enhanced blast weapons.

Urban warfare is becoming more widespread, providing both a rich environment for the bomber to strike and the ideal medium for enhanced blast weapons. The terrorist may employ pipe bombs, large high-energy car bombs, or the suicide bomber wearing several kilograms of explosive.

In the United States alone from 1990 to 1995, the FBI reported 15 700 bombings, with 3176 injuries and 355 deaths, and these numbers only continue to increase.

Primary blast injury, secondary to conventional high explosives, is uncommon in surviving casualties. This is because they would have been close to the epicenter of the explosion and are likely to have suffered lethal fragment and heat injury. With the advent of enhanced blast weapons (already populating the arms market), primary blast injury will increase in frequency, placing extreme clinical and logistical stress on the medical system.

Although antipersonnel mines were banned by the Ottawa Convention in 1997, civilian mine injuries have become even more common than military mine injuries that occur during combat, with farmers, women, and children 10 times more likely to encounter these abandoned weapons of war. The immense number of antipersonnel mines scattered throughout many parts of the world continues to plague civilization with horrible disabling

injuries that, according to the International Committee of the Red Cross (ICRC), number 24,000 per year.[1]

In this chapter we will address the myriad types of explosive weapons, how they work, and the resulting patterns of injury that threaten to present as mass casualties and to severely impact the health-care system clinically, logistically, and psychologically.

Explosions

An explosive is a chemical compound or mixture that, when subjected to heat, shock, friction, or other impulse, leads to a rapid chemical reaction or combustion and an equally rapid generation of heat and gases. The consequent combined volume is much larger than the original substance.

Explosives are classified as high or low, depending upon the rate at which this reaction takes place. Gunpowder, the first explosive used in military ordnance, is an example of a low explosive. Low explosives change relatively slowly from a solid to a gaseous state, generally less than 2000 meters per second.

By comparison, high explosives (HE) react almost instantaneously, causing sudden increases in pressures and a detonation wave that moves at supersonic speed (1400–9000 meters per second). Common examples are 2,4,6-trinitrotoluene (TNT) and the more recent polymer-bonded explosives, such as Semtex and Gelignite, which are 1.5 times the power of TNT. High explosives are used commonly in military ordnance.

A detonator is a type of explosive that reacts very rapidly and is used to set off other more inert explosives. Fulminate of Mercury mixed with potassium chlorate is the most commonly used detonator. Detonators also can be equipment, which by flame, spark, percussion, friction, or pressure are used to set off a chemical detonator. Detonation refers to the chemical and exothermic reaction that creates a pressure wave propagating throughout the explosive, creating rapid production of heat and gases, resulting in a "runaway" process and producing the resulting explosion.

The rapid release of enormous amounts of energy in a high explosion results in a primary blast wave, propulsion of fragments and environmental material or debris, and often generates intense thermal radiation. The initial explosion creates an instantaneous rise in pressure, resulting in a shock wave that travels outward at supersonic speed. The shock wave is the leading front and an integral component of the blast wave. The generation and propagation of blast waves are governed by nonlinear physics.

The response of structures, including human tissues, also may be nonlinear, as evidenced by the pathophysiology of blast injuries.

This sudden variation in air pressure creates a mass movement of air known as the dynamic overpressure or blast wave**.** The Friedlander Relationship (Figure 3-1) illustrates the physical properties of an ideal blast

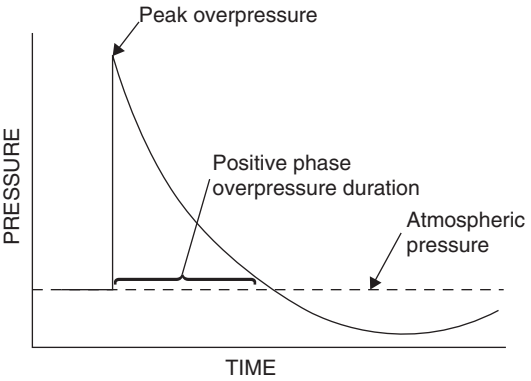

FIGURE 3-1. Friedlander relationship.

wave in open air. With the arrival of the shock wave, the pressure instantly increases to a peak overpressure, then rapidly falls and creates subatmospheric pressures before returning to normal ambient atmospheric pressure.

In reality, the reflection of the blast wave as it encounters environmental structures creates a very complex pattern of overpressures. These overpressures and an actual reversal of the blast wind in the negative phase may cause significant damage. For an in-depth discussion of the creation and propagation of blast waves and how they interact with various structures, the reader is referred to an article by I.G. Cullis.[2]

Enhanced-Blast Weapons

Ongoing research in the ordnance industry and recent technological advances in explosives and material has propagated the development of enhanced-blast weapons (EBW). There are four known types of enhanced-blast explosives:

1. metallized explosives,
2. reactive surround,
3. fuel-air,
4. thermobaric.

Once confined to fuel-air explosive (FAE) bombs, EBWs, recently proliferating the arms market, span the range of weapon systems from small grenades and hand-held weapons to large-caliber rockets (Table 3-1).

Relatively few primary blast injuries have been seen, as there are few survivors with primary blast injury from conventional HEs. Most die immediately, but this may change with the increase of EBW usage. With

TABLE 3-1. EBWs currently in use

	Fuel-air explosives	Thermobaric
United States	Fuel-air warheads • BLU-64/B (200 kg fuel) • BLUE-72B Pave Pat 2 (1202 kg ethylene oxide)	
Former Soviet Union	ODAB-500 PM (193 kg) high-speed low-level attack, can be launched from vehicle KAB-500 Kr S8-DM—3.6 kg multiple-barrel rocket launcher • SPLAV 220 mm (Uragan) BM 9P140 • SPLAV 300 mm (SMerch) BM 9A52	Guided missiles • AT-6-SPIRAL (helicopter launch)* • AT-9 (vehicle launch)* • AT-14 • METIS-M (crew-served weapon) • Khrizantema (BMP)* • TOS-1 (Burantino—mobile rocket launder) S8 unguided air-launched rocket TOS-1 Buratino—multiple-barrel rocket launcher Flame thrower RPO-A (2.1 kg) Grenade launcher GM-94 RShG-1 Multipurpose assault weapon RPG (range 900 m)*
China	250 kg bomb with 2 bomblets 500 kg bomb with 3 bomblets (800 square m)	

Note. * Denotes FAE ability as well.

conventional HEs, the blast wave decays very rapidly and is affected significantly by the environment. Enhanced-blast weapons produce a lower overpressure than conventional HE, but the period of overpressure lasts longer and reaches farther, thereby increasing the lethality zone and producing blast casualties farther from the epicenter of the detonation (Figure 3-2).

The classic EBW mechanism is illustrated best in the FAE, which has an initial small explosion that disperses a vapor cloud of ethylene oxide or other fuel. At a critical time and distance, the dispersed fuel is ignited by a second detonation, producing a uniform dynamic overpressure through the covered area. This may produce lethal overpressures as high as 2 Mpa, whereas a conventional HE would produce only 200 Kpa at a similar distance from the initiating explosion. The EBW also produces a longer-lasting fireball and may produce more energy [area beneath the curve (Figure 3-2)], resulting in more casualties with primary blast injuries combined with burns, crush, and penetrating-fragment injuries.

The blast wave from an EBW can diffract around corners, rapidly expanding and filling a structure, and is enhanced by reflection in enclosed spaces, making this an ideal weapon to defeat field defenses, soft unreinforced buildings, communication equipment, and low-flying aircraft. As an antipersonnel weapon, an EBW can be expected to rapidly produce large numbers of casualties with burns, blast injuries, fragment, translational injuries, and crush injuries from demolished buildings, placing a sudden and intense clinical and logistical strain on medical resources.

Several foreign studies have suggested that body armor enhances the effects of primary blast, creating a "behind armor blunt trauma" (BABT). Although studies by the US Army Soldier Systems Command, Natick, MA, indicated that the Interceptor Body Armor in use by US forces does not enhance the blast effect—and may actually reduce effects—when the ceramic plates are included in the armor, it is safe to say that most body armor currently in use will protect only marginally against primary blast injury and offer little, if any, protection against an accompanying thermal injury. Armor-employing decouplers or layers of material with different acoustic and mechanical properties specifically designed to maximally attenuate the shock wave needs to be designed.

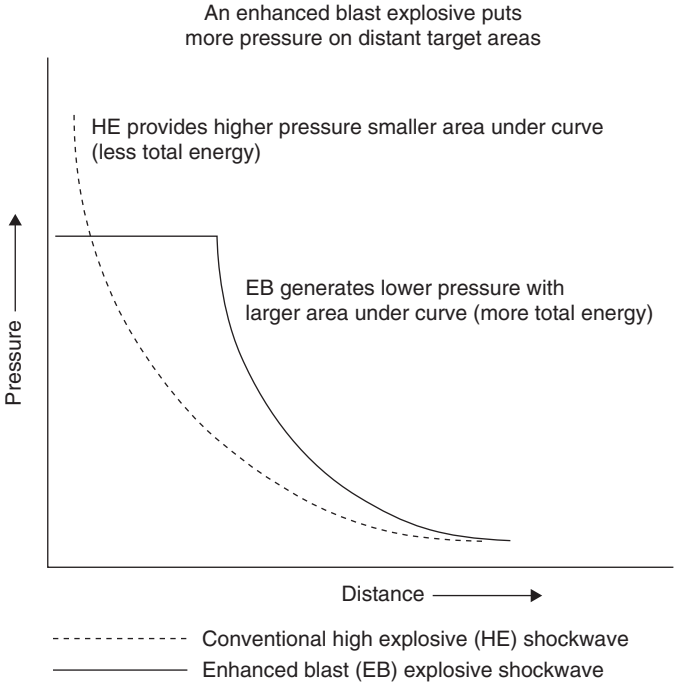

FIGURE 3-2. Enhanced blast wave.

Table 3-2. Classification of injury by mechanism

Class of injury	Mechanism
Primary	Interaction of blast wave (overpressure) with body, gas-filled structures being most at risk, complex stress and shear waves produce organ injury.
Secondary	Wounds produced by fragments from weapon, environmental projectiles, and debris, with penetrating injury predominating
Tertiary	Displacement of body (translational) and structural collapse with acceleration–deceleration, crush, and blunt injuries
Quaternary	All other mechanisms producing injury, burns, toxidromes from fuel, metals, septic syndromes from soil and environmental contamination (*septic meliodosis*)

It is the combination of the shock wave or leading edge, the dynamic overpressure, the secondary and tertiary effects, and the associated thermal energy that result in the characteristic injuries seen following detonation of an explosive device. These injuries may be classified according to the mechanism by which they are produced (Table 3-2).

Both conventional and terrorist weapons are designed to produce multiple wounds with the maximum number of casualties. Indeed, on today's battlefield, up to 90% of casualties are secondary to fragmentation wounds, with wounding from small arms or bullets producing generally less than 15 to 20% of battlefield casualties. Modern fragmentation weapons have a high casing-to-explosive ratio designed to produce preformed fragments, which significantly enhances the wounding radius and casualty probability. The major classes of available weapons are categorized in Table 3-3.

Table 3-3. Major weapon classification

Conventional	Grenades, aerial bombs, artillery, RPG. See all types of injuries with fragment wounds from preformed fragments predominating along with environmental debris
Antipersonnel mines	Point detonating mine (5 kg), traumatic amputation of foot or lower extremity, dirty, contaminated with debris, clothing, footwear, body parts, and soil Triggered mine (Claymore, Bouncing Betty), upper extremity, chest, face, and ocular injury
Enhanced blast munition	Designed to injure by blast wave, dispersed by fuel vapor, pulmonary injury, may have delayed onset, destroy and damage "soft" targets and personnel
Terrorist bombs	Letter bomb to several hundred kg, low mortality, fragment wounds, debris and crushing injury, some primary blast injury, suicide bomber with human-tissue fragments as wounding agent

Pathophysiology of Primary Blast Injury

The biological effects of the blast wave depend on the peak pressure and the duration of the dynamic overpressure and the effects of the secondary, tertiary, and quaternary mechanisms of injury.

Primary blast injury results from the interaction of the blast wave with the body or tissue, producing two types of energy: stress waves and shear waves. Stress waves produced by the interaction of the blast wave and the body surface are supersonic longitudinal pressure waves that create high local forces with small, but very rapid distortions, producing microvascular injury; they are reinforced and reflected at tissue interfaces, thus enhancing the injury potential.

Organs with heightened differences in physical properties such as the lungs, auditory system, and the gas-filled intestine are most susceptible.

Injuries from the stress waves are caused in several ways.

Pressure differentials across delicate structures such as alveoli;
Rapid compression of and subsequent re-expansion of gas-filled structures;
Reflection of a component of the compressive stress wave known as a tension wave at the interface of tissue and gas.

These myriad mechanisms result in damage originating in the mucosa and submucosa, but also reflect outward. Therefore, evidence of serosal injury may well represent full thickness damage. The interaction of these forces at the tissue interface is also known as spalling, characterized by the "boiling" effect seen at the air–water interface following an underwater explosion. A similar phenomenon most likely occurs at tissue interfaces, with resultant microvascular damage.

Shear waves are transverse waves with a lower velocity and longer duration that cause asynchronous movement of tissues. Actual damage depends on the degree to which the asynchronous motions overcome the inherent tissue elasticity, resulting in tearing of tissue and possible disruption of attachments. However, muscle, bone, and solid-organ injury is far more likely to result from the tertiary and quaternary effects of the blast than from the blast wave alone.

Thoracic

Blasts producing overpressure of less than 40 pounds per square inch (psi) generally will not cause pulmonary injury, (40 psi being produced by 20 Kg TNT exploding 6 meters away). Approximately 50% or more of casualties will sustain pulmonary damage with pressures of 80 psi or more, with overpressures of 200 psi being uniformly fatal in open-air blasts.

Blast injury to the lungs is the cause of the greatest morbidity and mortality from the blast effect alone. In the lungs, reflection of stress waves at more rigid interfaces account for the predilection of paramediastinal, peri-

bronchial, and subpleural tissue disruption and hemorrhage. Propagation of the stress wave results in pulmonary contusions distant from the site of impact in air-filled tissues, with damage to alveolar septae. Type I and some Type II pneumocytes are disrupted structurally and dysfunctionally, with loss of surfactant production, which when combined with capillary endothelial damage and release of ecosinoids and TXA2 may lead to progressive hypoxic respiratory failure and a clinical picture resembling acute respiratory distress syndrome (ARDS).

Pathologically, when the alveola septae are disrupted, hemorrhage occurs in three distinct patterns

1. pleural and subpleural,
2. multifocal and diffuse parenchymal and alveolar hemorrhage,
3. peribronchial and perivascular hemorrhage.

These patterns of injury may range from isolated scattered petechiae to confluent, consolidated areas of hemorrhage. Subpleural cysts and lacerations of pleura may lead to hemo-pneumothorax, pneumomediastinum, or tension pneumothorax. A lethal primary blast injury potentially could present with no outward signs of trauma. In severe blast injury, immediate death may be attributed to a characteristic triad of physiologic responses of primary thoracic blast of bradycardia, apnea, and hypotension that is unrelated to hemorrhage. Immediate death also has been attributed to massive air embolism resulting from the disruption of the alveolar wall and adjacent pulmonary capillaries, with the air emboli primarily affecting cerebral and coronary vessels.

Multiple animal studies have demonstrated large alveolar–venous and broncho–venous fistulae following blast injuries. This occurs in both air and underwater blasts and is commonly found in both cerebral and coronary circulation at autopsy and in experimental animal studies. Dysrythmias, signs of neurologic injury, and retinal artery air emboli may be seen in immediate survivors. The Trendelenberg position is not advisable in these patients, as now it is thought to predispose patients to coronary air embolus. The immediate therapy is supplemental oxygen, with Hyperbaric Oxygen being the definitive treatment of systemic air embolus, although not usually available or clinically practical. Alveolar–venous fistulae are thought to resolve in 24 hours, but must be considered a continuing risk in casualties that require positive pressure ventilation, especially with application of positive end expiratory pressure (PEEP), which is commonly used for hypoxic pathophysiology.

In survivors, clinical manifestations of primary blast may be present immediately or may have a delayed onset of 24 to 48 hours.

Intrapulmonary hemorrhage and focal alveolar edema leading to ventilation perfusion (V/Q) mismatch, increased intrapulmonary shunting, and decreased compliance results in hypoxia and increased work of breathing that is pathophysiologically similar to pulmonary contusions induced by

other mechanisms of nonpenetrating thoracic trauma. Chest X-rays have revealed diffuse patchy infiltrates that present in a butterfly pattern. These become more extensive over the first 48 hours, but are usually nearly resolved in seven days. Continued progression of the infiltrates after 48 hours should lead one to consider ARDS or superimposed pneumonia. Clinically, one also may see pneumothorax, hemothorax, subcutaneous and mediastinal emphysema, and even pneumoperitoneum or tension pneumoperitoneum, which may or may not be secondary to ruptured hollow viscous injury. Rib fractures should always alert one to tertiary or quaternary injury to the thorax.

Blast Lung casualties are more susceptible to pulmonary barotrauma (pneumothorax, air embolism) than other pulmonary injuries, and although early positive-pressure mechanical ventilation with the application of positive-end expiratory pressure to maintain adequate oxygenation may be required, the risk of barotrauma may be enhanced by such therapeutic requirements. Various ventilatory strategies have been proposed to lessen such risk, including Continuous Positive Airway Pressure (CPAP), Intermittent Mandatory Ventilation (IMV), and volume-controlled ventilation with low tidal volumes and permissive hypercapnia. Prophylactic tube thoracostomy should be considered if casualties must be evacuated by air or when close observation is impractical.

Fluid resuscitation should be managed judiciously, and early monitoring of hemodynamic parameters should be considered. The ideal fluid for resuscitation in blast injury is not known; however, pre-load should be optimized without overload using crystalloid with or without colloid. Patients probably should not be resuscitated to a mean arterial pressure (MAP) of greater than 60 millimeters mercury. In the absence of hemorrhage, many patients with thoracic blast manifest a prolonged hypotension for several days, with MAPs typically in the range of 50 to 60 millimeters Hg, with systolic pressure of 80 to 90 millimeters Hg and diastolic pressure of 40–50 millimeters Hg. The mechanism of this hypotension is poorly understood and may complicate management in the face of ongoing blood loss.

Auditory

The auditory system is very susceptible to blast and is the most commonly observed blast injury. Perforation in the anteroinferior part of the pars tensa is the most common manifestation of injury. Perforation occurs at 5 to 15 psi, and 33% of injuries are associated with ossicular injury, which does not occur in the absence of tympanic disruption. Cholesteatoma from embedded squamous debris is a long-term complication occurring in up to 12% of blast-perforated ears, dictating long-term follow up. Associated ossicular injury is a feature of more severe blast injury in as many as a third of reported cases. Sensorineural hearing loss associated with a high-pitched tinnitus frequently occurs immediately following a blast. Hearing loss may

resolve in hours or may become permanent in greater than 50% of patients, as has been reported in some series. Although not a priority for treatment, auditory injury should be addressed in 24 hours and auditory canal cleaned of all debris. Fifty to 80% of ruptured tympanic membranes will heal spontaneously without further treatment.

Although studies have not shown perforated tympanic membranes to be a marker for blast injury, traumatic loss of an ear or ear lobe secondary to primary blast is a marker of severe primary blast injury and associated morbidity and increased mortality.

Ophthalmic

The eyes are markedly resistant to primary blast injury and more often tend to suffer to secondary and tertiary mechanisms with resultant penetrating trauma.

Intestinal

While primary blast injury to the intestine may be overshadowed by the more immediate life-threatening pulmonary and cardiovascular manifestations, a review of US Army collective animal data indicates that gastrointestinal primary blast injury may be far more prevalent and occur with equal frequency in free-field blasts. In the case of immersion blast or in enclosed spaces, primary blast injury may occur even more frequently than pulmonary injury and at less intense exposure to dynamic overpressures.

The lower gastrointestinal (GI) tract more often tends to be air filled, with the ileo-cecal area being the most susceptible to primary blast injury and the small intestine generally spared. The mechanism of injury, as discussed earlier, is varying degrees of rapid compression/decompression resulting in wall damage and immediate rupture leading to peritonitis and hemorrhage. Displacement and tearing of mesenteric and peritoneal attachments with bleeding and devascularizing injury and the reflection of stress waves and spalling at the mucosal–gas interface, resulting in submucosal to transmural injury. The characteristic injury seen is a multifocal intramural hematoma beginning in the submucosa, extending with increasing severity to large transmural confluent hematoma and may involve the mesentery and vascular supply. Serosal injury always should be considered indicative of transmural injury. Cripps identified those lesions at greater risk of perforation in experimental studies in pigs, suggesting that serosal lesions greater than 15 millimeters in the small intestine and greater than 20 millimeters in the large bowel are at higher risk of perforation and should be resected. Delayed perforation up to 14 days post injury can occur and most likely is related to progressive ischemia and necrosis with transmural injury or adjacent mesenteric injury.

Clinically, patients present with nausea, vomiting, abdominal pain, rectal and testicular pain, tenesmus, and rarely hemetemesis. Treatment is guided best by clinical judgment, with selective use of diagnostic peritoneal lavage (DPL) being the most sensitive diagnostic test for early diagnosis of GI injury in the obtunded or intubated patient. Diagnostic peritoneal lavage fluid should be examined for blood, fecal material, bile, food particles, Alkaline phosphatase >10 international units (IU), Amylase >175 IU, elevated lactate dehydrogeanse (LDH), aspartate aminotransferase (AST), and Phosphate all being suspicious for possible GI injury. Computed tomography (CT), which has not proven to be sensitive for intestinal injury, should only be used in selective cases (suspected solid organ hemorrhage) and should probably not be considered in the situation with multiple casualties. Free air and excess free fluid not characteristic of blood, when seen on CT, are considered indications for laparotomy in blunt trauma patients; however, in primary blast with pulmonary injury, free-air and even tension pneumoperitoneum without intestinal injury has been reported and should be kept in mind.

Solid Organ Injury

Liver, spleen, adrenal, kidney, and testicle injuries have all been reported in underwater blasts; solid organ injury is less common in air blasts. Such injuries are most likely the result of shear forces and present similarly to solid organ injury resulting from blunt trauma. Gallbladder, renal pelvis, and bladder injury secondary to primary blast rarely have been recorded.

Central Nervous System

Traumatic brain injury (TBI) remains a major cause of death in bombing, accounting for 71% of early and 52% of later deaths. However, TBI and death usually is due to secondary and tertiary effects, not the primary blast. Recent studies have shown that significant histologic damage and CNS dysfunction does occur with primary blast. Patients may present with prolonged periods of loss of consciousness, agitation, excitability, and irrational behavior. Long-term sequelae such as posttraumatic stress syndrome also have been related to TBI from primary blast mechanisms.

Musculoskeletal

Traumatic amputation as a result of a primary blast is a marker of injury severity that has few survivors (1.5%), but relatively more common in nonsurvivors. Traumatic amputation is seen in 11% of immediate fatalities in suicide bombings. Patients with traumatic amputation caused from the blast effect of a conventional bomb usually are within one meter of the detonated ordnance. Traumatic amputations of the ear lobe also should be con-

sidered a marker of injury severity and mortality. Stein, reporting in 1999 on a series of suicide bombings in Israel, noted only one survivor among traumatic amputations of the ear lobe.[9]

Thermal Blast

The flash (fireball) produced by the detonation of an explosion can reach temperatures greater than 3000 degrees centigrade**.** There is some controversy regarding the incidence of burns in surviving casualties, although Stein reported an incidence as high as 31%. With the increasing prevalence of FAEs and thermobaric EBWs that have a larger and longer lasting fireball, the incidence of burns may increase. Flash burns, flame burns from secondary fires, and inhalation injury from toxic substances all may be seen, complicating an already severe mechanism of injury.

Fragmentation Injury

There are a myriad of weapon systems and missiles, ranging from grenades to aerial delivered bombs weighing several tons that depending on the size and design of the weapon may deliver several thousand fragments ranging in weight from a few milligrams to many grams with an initial velocity of greater than 1500 meters per second. These fragments decline in velocity rapidly generally producing multiple low velocity incapacitating wounds. Modern fragmentation weapons are designed with preformed fragments to optimize velocity, distance, and probability of hit producing multiple casualties with multiple wounds (Figure 3-3). Body armor has altered the pattern of distribution of fragmentation injury so that the most common casualty seen on today's battlefield will have multiple extremity, head, and facial wounds (Table 3-4).

The use of antipersonnel bomblets or submunitions effectively has increased the probability of a hit and increased the lethality and wounding area of the munition. In the Israeli–Egyptian October War of 1973, each antipersonnel canister released 600 Guava bomblets (named from an Egyptian fruit with large numbers of seeds), with each bomblet containing 300 pellets. Each bomblet is released at one-meter intervals and can travel 150 meters, with an explosive lethal radius of 5 to 8 meters. Each pellet acts as a small missile of moderate velocity, striking from different angles within the lethal zone. This raised the incidence of multiple system injuries, with penetrating wounds of the extremities constituting 56% of injuries. There also was a 15% increase in head and neck injuries, with 14% of injuries to the chest and abdomen. Pellet paraplegia was a characteristic injury seen with the Guava bomblet in the Israeli–Egyptian conflict. Penetrating abdominal wounds with visceral injury proved difficult, with frequently missed visceral injury due to the small pellet size (Figure 3-4).

Improved grenade launchers with preformed fragments, laser-sighted accuracy, and precision fusing such as the US Objective Infantry Combat

FIGURE 3-3. Preformed fragments from cluster bomblet. (Maj Scott Gering, Operation Iraqi Freedom).

TABLE 3-4. Anatomical distribution of penetrating wounds as a percent (80% fragment)

Conflict	Head & Neck	Thorax	Abdomen	Limbs
World War I	17	4	2	70
World War II	4	8	4	75
Korea	17	7	7	67
Vietnam	14	7	5	74
Northern Ireland	20	15	15	50
Israel 1975	13	5	7	40
Israel 1982	14	4	5	41
Falkland Island	16	15	10	59
Gulf War (UK)	6	12	11	71
Gulf War (US)	11	8	7	56
Afghanistan (US)	16	12	11	61
Chechnya (Russia)	24	9	4	63
Somolia	20	8	5	65
Average	15	9.5	7.4	64.6

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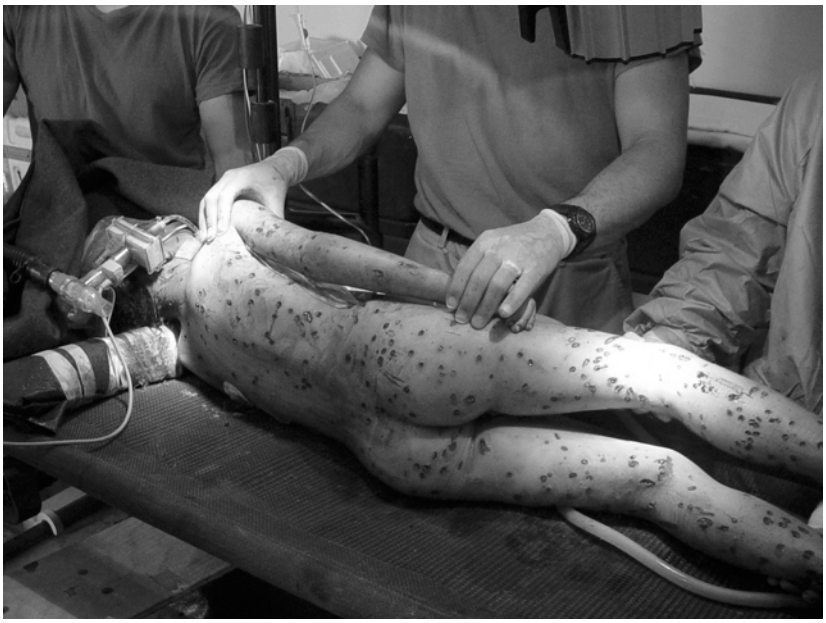

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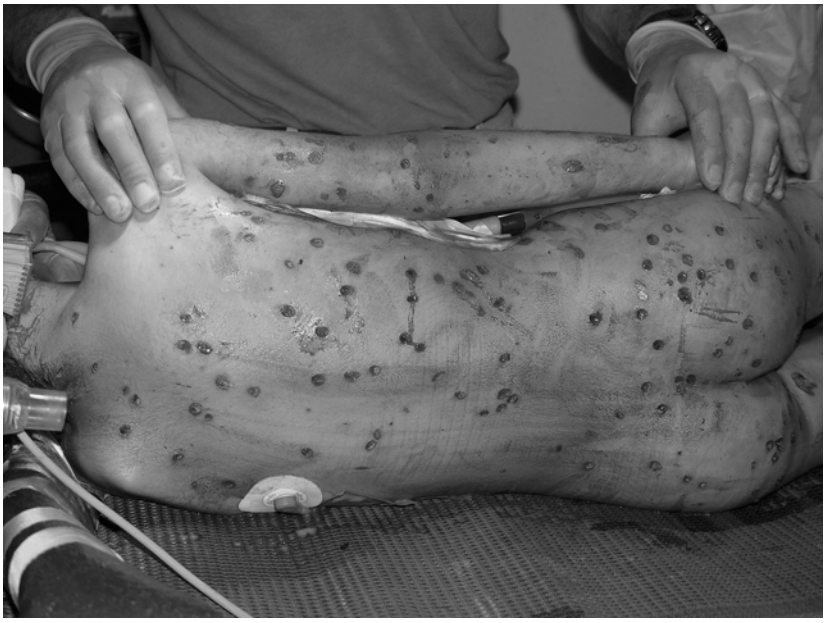

FIGURE 3-4. (a, b) Multiple fragment wounds from cluster bomblet (US) (Maj Scott Gering, Operation Iraqi Freedom). (c) Multiple fragment wounds from cluster bomblet with environmental fragments. (Maj Scott Gering, Operation Iraqi Freedom).

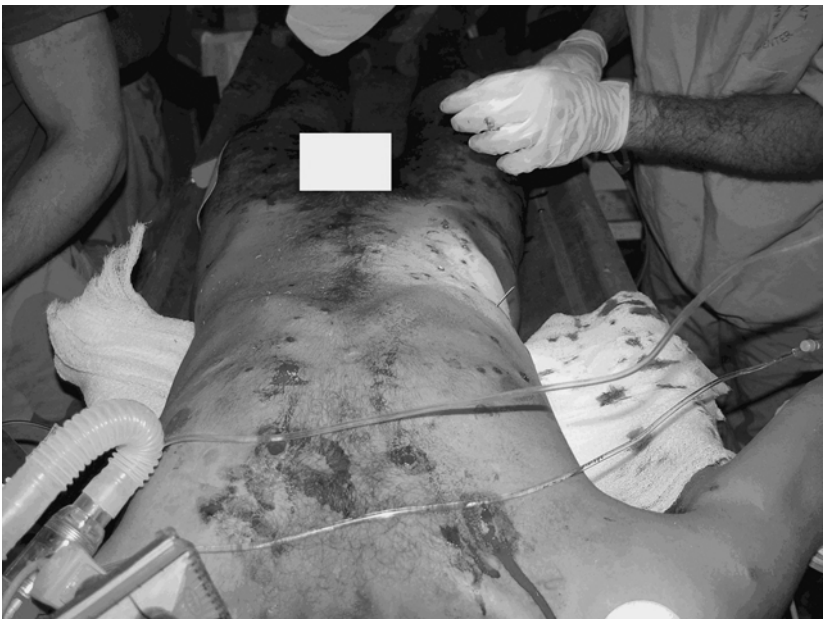

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FIGURE 3-4. *Continued*

Weapon, with a 5.56 barrel combined with a 20-millimeter grenade launcher, will increase firepower and extend the killing range to 1000 meters. The use of flechettes, depleted uranium, and tungsten missiles capable of penetrating body armor and conventional cover may further compound the complexity of wounding with toxicities that have yet to be defined, thus increasing the impact on the medical support system.

Many of the more modern fragment munitions are designed to produce multiple preformed fragments that weigh 100 to 200 milligrams and are 2 to 3 millimeters in diameter, whereas others may weigh as much as 20 grams. Both have initial velocities of 1500 meters per second, which falls off rapidly, especially with the large, more irregular-shaped fragments. The mechanism of injury is related as much to energy transfer as to the velocity of the projectile, and the magnitude of injury is thought to depend more upon the inherent tissue characteristics of the organ involved than upon the projectile itself. The clinical impact and priority of treatment depends on the tissue or organ involved. Extremity wounds that may be innumerable usually are not life threatening and perhaps may not be immediately disabling. In contrast, wounds of the eyes or thorax are far more likely to be immediately disabling or life threatening, respectively.

Environmental debris such as glass, splinters, soil, and various structural particles are propelled with similar velocities by the blast wind and may well be the major cause of fragment wounding. The advent of the human

suicide bomber brings a new dimension to fragment wounding, with human body parts acting as missile fragments and projectiles that may carry with them the specter of human immunodeficiency virus (HIV), hepatitis, and other serious and yet to be identified threats of unknown clinical consequence, thus presenting a rather complex therapeutic dilemma for the clinician.

Penetrating fragment wounds of the abdomen and thorax are no different than other penetrating wounds except that the number of pellets and the small size of visceral injuries demand meticulous attention to detail. Almost all penetrating fragment wounds of the abdomen can be closed primarily and 85% of penetrating thoracic wounds can be managed successfully by tube thoracostomy. Animal studies examining multiple colonic injuries found that colotomies closed by either one-layer interrupted absorbable suture or stainless steel skin staples were equivalent, except that the stapled anastomosis histologically healed more quickly than the sutured anastomosis, supporting definitive repair of intestinal low-velocity wounds.

All war wounds are contaminated by soil, clothing, and skin. High-velocity missiles have been shown to widely contaminate a wound track,[3] whereas low-velocity fragmentation wounds are minimally contaminated with debris. Bacterial contamination is ubiquitous in fragmentation wounds, with soil and skin organisms, Clostridia, Streptococcus, Staphylococcus, Proteus, E Coli, and Enterococcus,[4] although infection is uncommon in small low-velocity wounds of the extremity.

Although somewhat controversial, some reports in the literature support early antibiotics and nonoperative treatment of extremity wounds less than a centimeter in size in patients who show no evidence of neurovascular injury or compartment syndrome and also have a stable fracture pattern.[5] Operative debridement of these numerous wounds can lead to increased morbidity and, in general, is unnecessary.[6] However, in the authors' experience, small low-velocity wounds involving a major joint resulted in a higher incidence of infection when treated with early antibiotics and delayed operative treatment of more than 6 hours (Operation Just Cause).

Small (less than one centimeter) low-velocity wounds with no evidence of contamination that can be cleaned and dressed with early administration of appropriate broad-spectrum antibiotics may be treated nonoperatively. However, when there is question, delay in treatment greater than 6 hours, or evidence of cavitation and contamination in wounds greater than one centimeter, operative debridement should be the standard.

Land Mine

Land mines currently are deployed in 64 countries around the world and number between 84 to 100 million. Two thousand victims a month fall prey

to this indiscriminate forgotten remnant of war that are ten times more likely to injure a noncombatant than a soldier. Although banned by the Ottawa Convention of 1997 and prohibited by International Humanitarian Law, mines continue to be laid across the world. It is estimated that in countries with existing mine fields such as Cambodia, Angola, and Somalia, one in every 450 persons undergoes traumatic amputation compared to United States, where amputations only number one per 22 000. It is estimated that only half of these noncombatant victims even live to reach a hospital and undergo treatment for these devastating injuries.[7]

Mines can cost no more than $3.00 apiece and can be distributed by a plethora of weapon systems to include aerial delivery and Multiple Launch Rocket Systems (MLRS) that can deliver 8000 bomblets and hundreds of mines in a matter of minutes. The American Gator mines (72 antitank and 22 antipersonnel) are delivered aerially in containers with one fighter aircraft able to deliver 600 mines in a single sortie. There is no reason to expect that the use of antipersonnel mines will cease or that the incidence of landmine injuries will decline. Mines with increased blast radius and lethality, and with fuel-air–enhanced blast technology already are in development.[8]

There are essentially three classes of conventional antipersonnel land mines based on mechanism of action—static, bounding, and horizontal-spray mines.

Static mines are implanted in the ground and vary from 5 to 15 centimeters in diameter, contain 20 to 200 grams of explosive, and most commonly are detonated by direct contact, although newer mines that detonate on motion and proximity motion are being developed.

Bounding mines, known as "Bouncing Betty," have the highest mortality. These mines propel a small explosive device 1 to 2 meters above ground then explode, dispersing multiple small preformed fragments.

Horizontal-spray or directional fragmentation mines, of which the US M18A1 Claymore AP munition mine is the best known, can be command detonated or victim detonated by means of trip wires. The Claymore fires 700 steel spheres, each weighing 0.75 grams in a 60 degree arc, resulting in multiple penetrating wounds dispersed throughout the body, creating multiple system injuries and multiple casualties. The horizontal spray and bounding mines essentially produce multiple penetrating injuries of both high and low velocity, depending on the range of the target, with a very high mortality. Thus, the mechanism of injury is no different than any other penetrating wound and surviving casualties are treated as such.

The static mine is most common throughout the world, and its mechanism of injury is unique to this weapon system. Upon contact and detonation, an instantaneous rise in pressure or shock wave is produced, which along with the products and heated air produce a blast wave or dynamic overpressure. Contact with the body produces stress waves that propagate proximally along with shear waves produced by the blast effect. These stress waves can propagate as far as the middle thigh with demyelination of nerves

occurring 30 centimeters above the most proximal area of tissue injury. This, combined with fragments from the device, soil, and footwear, produces the classic land-mine injury of complete tissue destruction, distally associated with traumatic amputation at the midfoot or distal tibia (Figures 3-5 and 3-6). Proximal to the variable level of amputation there is complete stripping of tissue from the bony structures and separation of fascial planes contaminated with soil debris, microorganisms, pieces of the device, footwear, and clothing. Associated penetrating injury to contralateral limb and perineum are common.

Injuries occur in three distinct patterns. Pattern 1 injuries occur with contact of a buried mine that produces severe lower-extremity, perineal, and genital injury. Pattern 2 injuries occur with a proximity device explosion that produces less severe lower-extremity injury with less traumatic amputation. Head, thoracic, and abdominal injuries are common. Pattern 3 injuries occur with handling or clearing that produce severe head, face, and upper-extremity injury.

Ocular injuries are not uncommon with all categories of mines. The products of detonation and environmental fragments and debris producing penetrating ocular wounds are the primary mechanism and were seen in 4.5% of all antipersonnel mine injuries in Afghanistan.

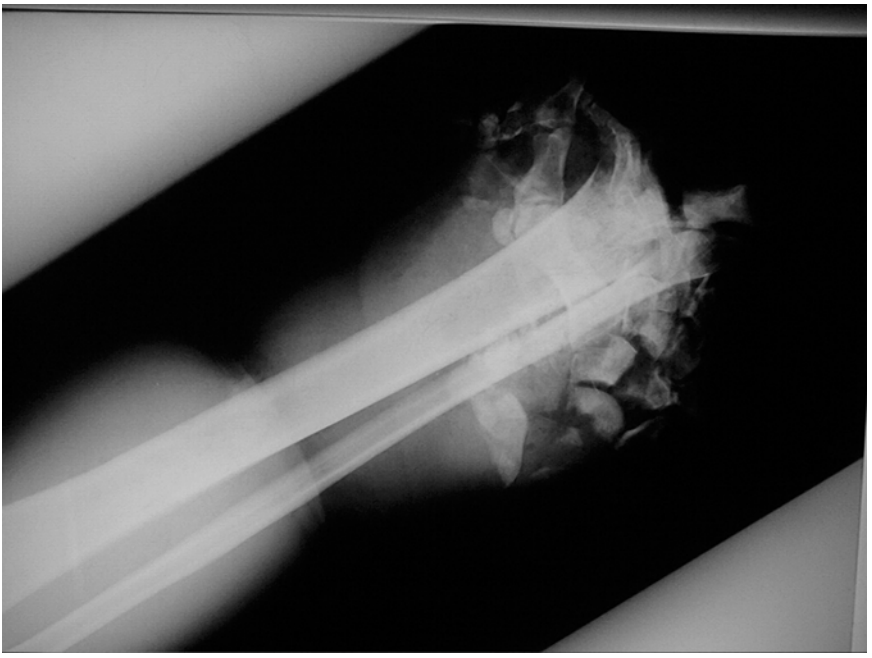
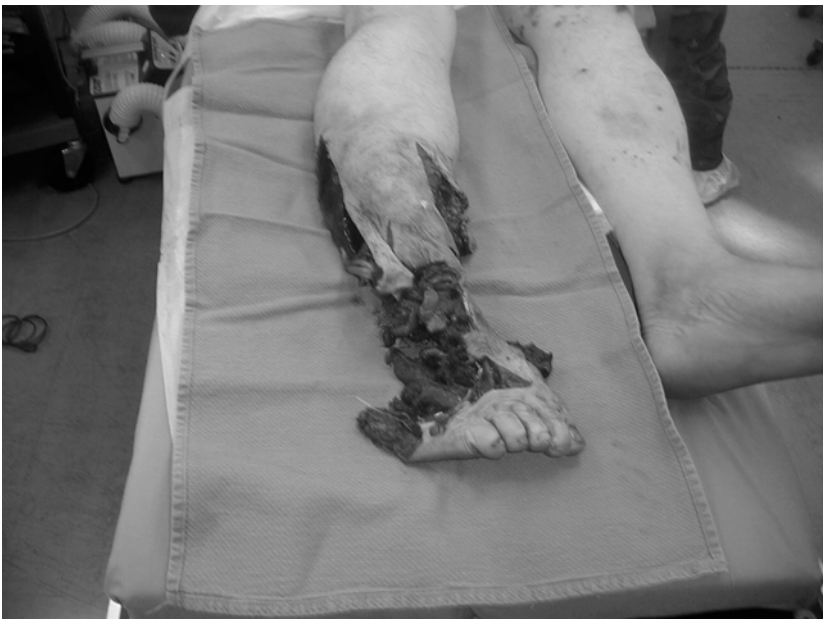
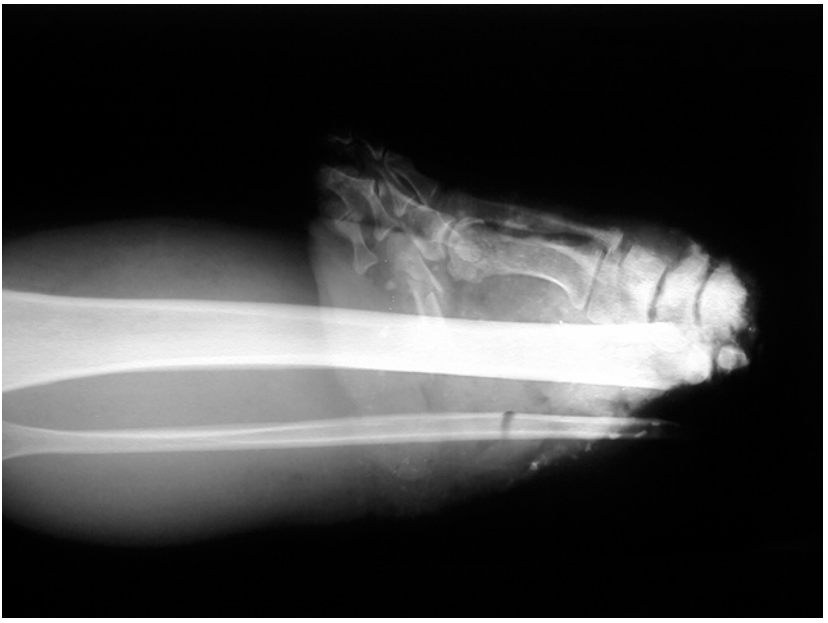


FIGURE 3-5. Boot and clothing debris from small antipersonnel land mine. (Major Scott Gering, Operation Iraqi Freedom).



a



b

FIGURE 3-6. Injury from antipersonnel mine. (Major Darryl Scales, Kosovo, 2000).

All lower-extremity injuries need debridement or completion of amputation and many may require laparotomy, with all wounds of perineum, buttocks, back, and abdomen having a low threshold for laparotomy. Every effort should be made to conserve the contralateral limb. The primary injury is treated with excision, lavage, and exploration and lavage of fascial planes with delayed closure. All casualties should receive broad-spectrum antibiotics to cover indigenous soil spore-forming microorganisms.

Combined Injury

Combined injuries, including primary blast injury, penetrating fragment wounds, crush, burn, and inhalation injuries, are to be expected, especially in urban-warfare environments or urban terrorist bombings. With the advent of EBWs and handheld thermobaric weapons, these combined injuries are likely to become even more common, placing extreme stress on medical support systems, be they military or civilian. Triage and appropriate patient distribution may be the most critical piece of patient management. Combined blast and penetrating injuries are almost always the most life threatening and the basic principles of the ABCs should always be adhered to. Blast lung may not present for 24 to 48 hours; therefore, all patients requiring early mechanical ventilation or those going immediately to the operating room should be managed with low tidal volumes of 5 to 6 milliliters per kilogram of ideal body weight, peak inspiratory pressures of less than 25 centimeters H_2O, allowing for permissive hypercapnia. If associated TBI is present, hypercapnia should be avoided or minimized, if possible, due to the deleterious effects on intracranial hypertension. For a patient undergoing anesthesia with obvious signs of blast lung injury, there is an increased risk of barotrauma with resultant tension pneumothorax. The authors recommend consideration of bilateral prophylactic tube thoracostomy.

Patients without vascular compromise or evidence of compartment syndrome who have penetrating injuries of the extremities of one centimeter or less may be managed conservatively with early antibiotics and cleansing with frequent observation.

Penetrating injuries of the trunk associated with hemodynamic instability should undergo immediate operation. Most thoracic injuries can be managed with tube thoracostomy, and thoracotomy is rarely indicated in combined injury. Emergent thoracotomy for penetrating thoracic injury in the presence of primary blast should be considered futile and be abandoned.

Abdominal wounds with associated hemodynamic instability require immediate laparotomy once the airway is controlled.

Management of truncal penetrating injuries combined with blast should undergo, at minimum, a focused abdominal sonogram for trauma (FAST) exam if available, but the decision to operate frequently must be based on clinical judgment and a high index of suspicion. Patients with blast injury

and associated blunt abdominal trauma from tertiary and quaternary mechanisms who are hemodynamically unstable should undergo immediate laparotomy, as conservative management in this scenario is not indicated at this time. However, the use of Recombinant factor VIIa and other non-operative hemorrhage control methods may alter the clinical management of some categories of blast-injured casualties in the near future.

Retained Ordnance

An injury that is truly unique to the military is the casualty presenting with retained and unexploded ordnance. Since World War II there have been 36 documented cases with only four deaths. The deaths occurred not from the detonation of the missile, but from the moribund condition of the casualty, usually due to hemorrhage. The M79 grenade launcher (40 mm), mortars, and RPG missiles have been the most commonly reported cause of retained ordnance. A fuse or detonator that can be triggered by impact, electromagnetic energy, or time and distance normally detonates this type of ordnance. There is often a safety built into the device requiring a defined number of revolutions or a required distance and time before the missile is armed to explode. The RPG round must travel a proscribed distance before the fuse is armed to trigger on impact. All missiles that have been fired must be considered armed, and a defined and predetermined algorithm for the care of the casualty and the safety of the medical personnel should be followed.

A casualty with unexploded ordnance should be transported in the position found so as not to change the missile orientation and should always be grounded to the airframe if evacuated by air. These patients should be isolated and, in a mass casualty situation, should be treated last as the removal of ordnance is time consuming and the surgeon must attend to other casualties before placing his or herself at risk. Closed chest massage or defibrillation should never be attempted, and during removal, any equipment emanating electrical energy, heat, vibration, or sonic waves, such as the electrocautery, ultrasound, blood warmers, or power instruments, should be avoided. The patient should be placed in a protected area away from the main hospital, and all personnel in the immediate area should employ body armor or explosive ordnance disposal (EOD) equipment. Explosive ordnance disposal personnel should be involved prior to removal to help with identifying the round and fuse; a plain radiograph will help in planning the operative removal and will not cause the round to explode. The minimum anesthesia required should be used and in such a manner that the anesthesia provider need not be present during actual removal. The only personnel required during removal are surgeon and an assistant—ideally, EOD personnel. The round should be removed *en bloc* without touching the missile with metal instruments. Every effort should be employed to maintain the orientation of the missile until removed from the area by EOD. The basic guidelines for removal of ordnance are outlined in Table 3-5.

Table 3-5. Removal Un-exploded Ordnance

1. Notify EOD
2. No CPR or electric shock
3. Isolate to protected area (sandbagged bunker)
4. Protective equipment for medical personnel
5. Do not use cautery, power equipment, blood warmers
6. Avoid vibration, change in temperature, change in missile orientation
7. Do plain radiograph, no CT or Ultrasound
8. Minimal anesthesia, anesthesia provider to leave after induction
9. Surgeon and assistant (EOD) only personnel present during removal
10. Remove without changing orientation and hand over to EOD
11. Move casualty to Operating theater for definitive procedure

Source: Lein B, Holcomb J, Brill S, Hetz S, McCrorey T. Removal of unexploded ordnance from patients: a 50-year military experience and current recommendations. Mil Med 1999;164:163–5.

Triage

Triage of overwhelming numbers of casualties with multisystem injury from terrorist explosive munitions, once the sole province of the battlefield hospital, now threaten the urban hospital, the civilian trauma surgeon, and health systems throughout the populated world. All medical providers must bear the burden of preparing for the eventuality of casualties in overwhelming numbers, sustaining primary blast, penetrating wounds, burns, and crush injuries. We must understand the nature of the weapon and the physiologic consequences of these weapons of war and terror and be prepared to provide care that will save lives and reduce morbidity.

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