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AHC Media

Blast Injuries

Introduction

Explosions occur in a variety of settings and have multiple causes. As demonstrated by recent tragic events, detonations are occurring increasingly in civilian settings as well as the more typical military zones. Blast injuries may result from explosives that deliver minimal force or massive explosions, resulting in significant injuries, loss of life, and destruction of property. Blasts can cause penetrating and blunt trauma, as well as characteristic injuries associated with the wave of overpressure. Blasts may result in chemical, biologic, and radiation exposures. All emergency healthcare providers need to be aware of and prepared for blast injury patterns and the hazards that can be associated with blast incidents.

Epidemiology

When considering blast incidents, high-profile terrorist bombings tend to come to mind first. Worldwide, there were 13,463 terrorist attacks in 2014, resulting in 32,700 deaths and 34,700 injuries. Bombings accounted for 42% of attacks that caused at least one fatality. Terrorist attacks were concentrated in a small number of countries: 60% occurred in Iraq, Pakistan, Afghanistan, India, and Nigeria.[1]

In the United States, blast injuries are relatively uncommon and sporadic among civilians. The incidents range from small-scale detonation of fireworks to industrial disasters that cause massive destruction of infrastructure with significant loss of life and many critical injuries. The United States Consumer Product Safety Commission estimates that 10,500 people with fireworks-related injuries were treated at emergency departments in 2014 (67% were treated between June 20 and July 20). Eleven people died in non-occupational incidents caused by fireworks explosions, four of them in house fires ignited by fireworks.[2]

The most significant modern industrial blast occurred at the West Fertilizer Company in Waco, Texas, on April 17, 2013. This explosion caused 15 deaths, injured 160 people, and damaged or destroyed dozens of homes and an elementary school. Intentional bomb incidents in the United States are reported every year to the Bureau of Alcohol, Tobacco, Firearms, and Explosives and the Federal Bureau of Investigation (FBI). Between 1987 and 1997, the FBI reported 18,283 "illegal detonations or ignition of explosive or incendiary devices." Those incidents caused 448 deaths and 4170 injuries.[3] The majority of reported incidents involved low-order explosives in crudely built devices, which likely accounts for the relatively high injury-to-fatality ratio of 10:1.[4] Residential bombings are the most commonly reported and result in the most injuries and deaths.[5] These dramatic figures should remind the acute care provider in the United States that, although encountered infrequently, intentional blast incidents are not just a distant problem in other countries.

EXECUTIVE SUMMARY

- Kinetic energy released from the point of detonation displaces outward through the surrounding medium (typically air or water), creating a pressure differential called a blast wave (also known as a shock wave).
- Amplitude and duration of the peak overpressure and environmental factors such as proximity to structures, victims' locations in relation to the blast, and the medium in which the blast occurs are important determinants of injury severity.
- Explosions inside buildings and buses used the most hospital resources and their victims have poorer outcomes.
- High-order explosives consist of chemicals that convert to gas virtually instantaneously in a process known as detonation, resulting in a substantial release of kinetic energy that creates blast overpressure and the resultant blast wave. In contrast, low-order explosives burn slower in a progressive fashion called deflagration.
- Abdominal primary blast injuries may present with pain of varying character and intensity, nausea, vomiting, diarrhea, and tenesmus. The stool or emesis might be bloody. Bowel sounds might be absent. The patient could have varying degrees of tenderness with presence of guarding or rebound.
- Traumatic brain injury and post-traumatic stress disorder have been called the "signature" injuries of military operations in Iraq and Afghanistan.
- Among the initial survivors of an explosion, blast lung injury is the most common primary blast injury that ultimately is fatal.
- The absence of tympanic membrane rupture does not prove absence of other injury. The presence of tympanic membrane rupture, however, is associated with other blast injuries and places a patient in a high-risk group.

Throughout the world, the use of explosive devices in terrorist activities continues to rise. The Terrorism Knowledge Base, created by the Oklahoma City National Memorial Institute for the Prevention of Terrorism and now maintained by the RAND Corporation, demonstrates a four-fold increase in the rates of military and civilian explosive incidents and an eight-fold increase in the injuries caused by them.[6]

Blast incidents account for a large proportion of the injuries and deaths of military personnel deployed in Afghanistan since 2001 and in Iraq since 2003. Improvised explosive devices (IEDs) accounted for 2200 American soldier fatalities and 22,000 injuries from March 2003 to November 2011.[7] The 10:1 injury-to-fatality ratio seen in combat is likely related to the use of protective equipment and advances in troop transport rather than a decrease in the lethal potential of explosive devices.

Blast injuries sustained by military combatants and civilians differ significantly. Injured military personnel tend to be young, healthy, and male. Civilian incidents cause more casualties at the extremes of age, among females, and among people with chronic health problems. Military personnel usually wear some level of personal protective equipment (PPE) at the time of the incident, whereas civilians do not. The military PPE provides protection for the thoracoabdominal area and the head. Explosions that occur as a result of terrorist activity targeting civilians are more likely to occur in enclosed spaces than blasts encountered in military incidents.

The effects of blasts on military personnel have been studied much more thoroughly than those in the civilian population. Ritenour and colleagues retrospectively reviewed the Joint Theater Trauma Registry for Overseas Contingency Operations in Iraq and Afghanistan. During the study period (March 2003 through October 2006), 6687 military personnel were injured in combat and survived to be treated in medical treatment facilities. Among those survivors, explosions were the cause of injury in 4765 (71%).[8]

Blast Physics

A conventional explosion results from the rapid chemical conversion of a solid or liquid into a gas. Kinetic energy released from the point of detonation displaces outward through the surrounding medium (typically air or water), creating a pressure differential called a blast wave (also known as a shock wave). The Friedlander waveform (*see Figure 1*) is an idealized version of this pressure change over time for an open-space explosion. The pressure attains its maximum peak overpressure (P_{max}) rapidly, then dissipates over time and distance until it reaches the reference pressure (P_0), which in most cases is the normal atmospheric pressure. The blast wave can travel as fast as 8000 m/s and reach pressures approaching 30,000 times that of atmospheric pressure.[9] As the blast wave propagates outward, it displaces the air, generating blast winds that can produce flying debris. People located near the center of the blast can be thrown outward. The void left by the displaced air (or water in underwater explosions) causes the pressure to drop below the reference pressure until the maximum negative pressure (P_{min}) is reached and then there is a return to the reference pressure. The negative phase duration (t_n) is much longer than the positive phase duration (t_p).

Characteristics of the Blast Environment

The capacity of a blast to cause injury and death depends on a number of factors. As the amplitude and duration of the peak overpressure increase, so does the potential for harm. Other important determinants include environmental factors such as proximity to structures, victims' locations in relation to the blast, and the medium in which the blast occurs. Confinement of a blast in a closed or semi-closed space, such as a subway tunnel or city bus, can drastically increase its potential to cause harm. In a confined-space explosion, the Friedlander waveform no longer represents the atmospheric changes that occur in the area around the blast. A blast wave will reflect, possibly several times, when it strikes solid surfaces,

Figure 1. Friedlander Waveform

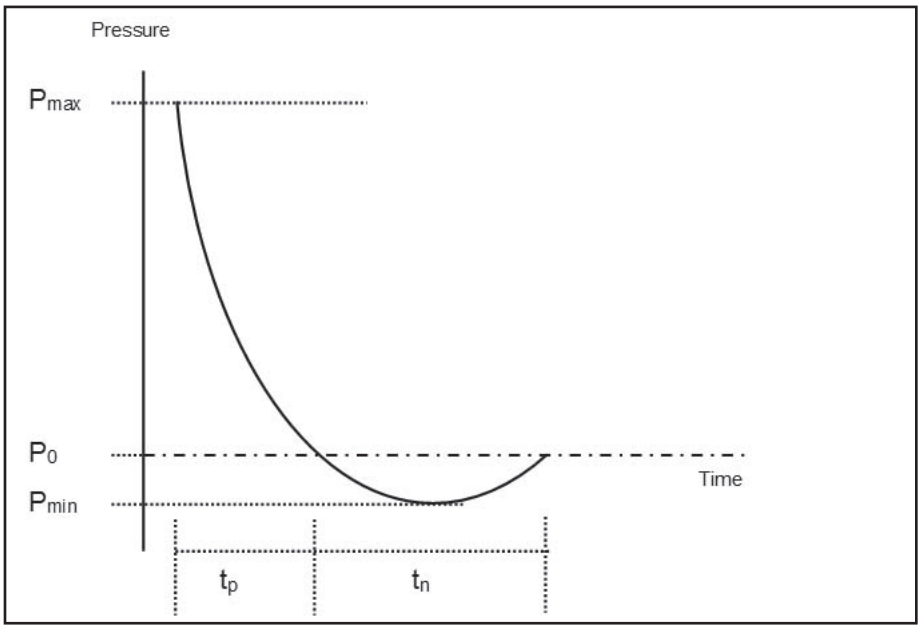
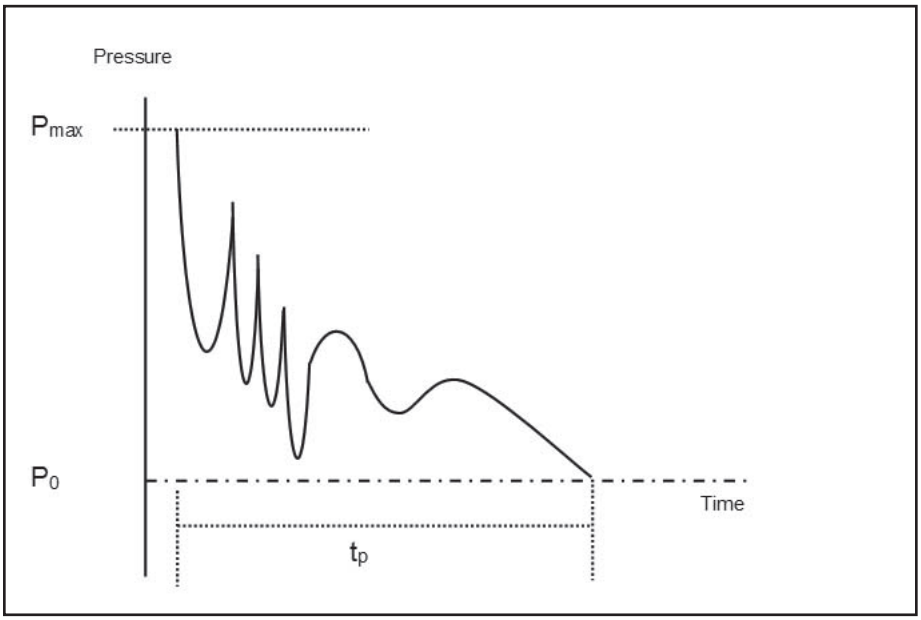


Figure 2. Enclosed Waveform



resulting in complex and erratic pressure changes (*see Figure 2*). The positive-pressure phase is prolonged, with an increased peak overpressure and several subsequent peaks as a result of the pressure wave reflecting off surrounding solid structures. A negative pressure phase might not occur in such situations. Leibovici et al reviewed the medical records of approximately 300 terrorist bombing victims in Israel and calculated a mortality rate of 7.8% for open-air blasts, compared with 49% for blasts confined to a bus.[10] Rozenfeld et al characterized an additional 65 terrorist bombings in Israel, involving approximately 800 victims, and concluded that explosions inside buildings and buses used the most hospital resources and their victims had poorer outcomes.[11] This investigative group also raised concern about blast wave reflection, as evidenced by the high number of injuries sustained by victims located near buildings at the time of explosion. A person located between an explosion and a solid structure capable of reflecting the blast wave can suffer two or three times the degree of injury as a person caught in a similar incident in an open environment.[6]

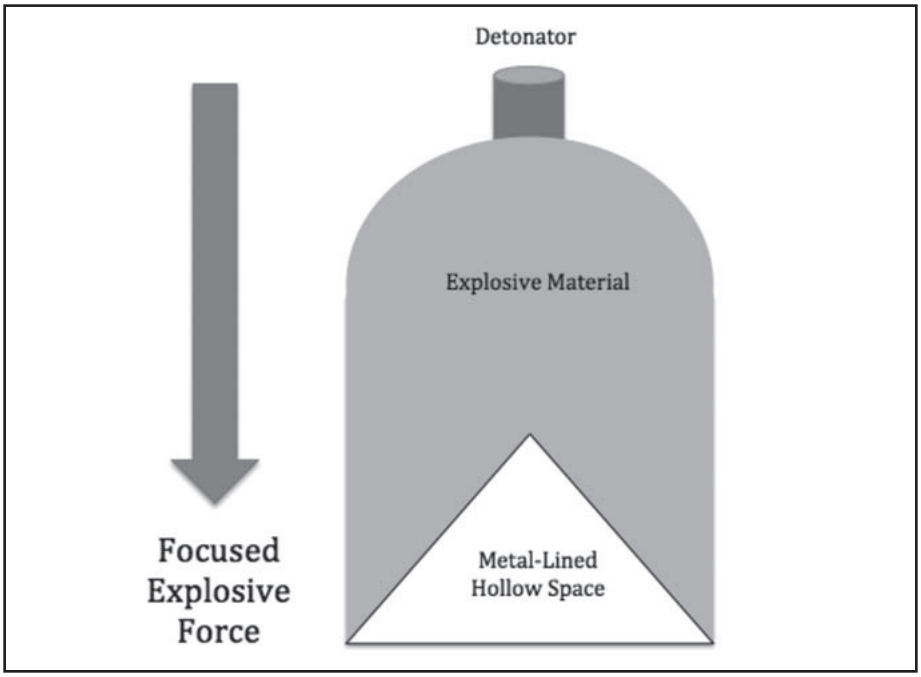
The proximity of victims to the explosion will affect their likelihood for injury. The energy of the blast wave in air dissipates rapidly and is governed by Hopkinson's scaling law, which states that as the distance from the explosion doubles, the peak overpressure experienced by the victim decreases by one-eighth.[12] Based on this ratio, a distance of more than 16 meters is required to protect from the blast overpressure produced by up to 25 kg of 2,4,6-trinitrotoluene (TNT).[9]

Underwater explosions pose an increased risk of injury given the virtually incompressible nature of water molecules compared with air. Fortunately, exposure to this type of explosion is rare outside combat scenarios. Nguyen and associates described the injuries sustained by a young man when a firework failed to launch into the air and instead detonated underwater approximately 1.5 meters away from where he was submerged to his neck. The incident led to the development of severe multilobar hemorrhagic pulmonary contusions requiring intubation. In this scenario, the overpressure created by underwater detonation of a typical consumer firework containing 3-10 grams of explosive powder comparable to TNT exceeded the threshold for lung injury by 10-fold.[13] Had this been an open-air rather than an underwater explosion, the peak overpressure would not have reached the threshold for causing lung injury. For swimmers treading water in a vertical position when a blast occurs, bowel injury is the most likely form of trauma, possibly related to the fact that blast loading increases with depth. Measurements of peak overpressures taken at depths of 1 and 2 feet demonstrated a 2- to 10-fold increase with depth.[12]

Characteristics of the Explosive

Explosives can be categorized as either high or low order, based on the velocity of decomposition. High-order explosives consist of chemicals that

Figure 3. Shaped Charge



convert to gas virtually instantaneously in a process known as detonation, resulting in a substantial release of kinetic energy that creates the aforementioned blast overpressure and resultant blast wave. High-order explosives can be pure compounds, such as nitroglycerin, TNT, acetone peroxide, cyclonite, or pentaerythritol tetranitrate (PETN). They may also be compounds of different materials, as found in dynamite and ammonium nitrate-fuel oil. Plastic explosives use a plasticizer, usually phthalate esters, to increase flexibility and durability. C-4 is an explosive composite that contains the explosive RDX (research department explosive), a nitroamine, a binder, and a plasticizer.[14]

Compared with high-order explosives, low-order explosives burn slower in a progressive fashion called deflagration. Because of this characteristic, they can be purposed as mining tools, propellants, fuses or detonators for certain high-order explosives, or pyrotechnics when manufactured for light, heat, and sound. Low-order explosives typically do not generate a blast overpressure but are still capable of producing blast winds, projectiles, and burns. Common examples are black powder, gunpowder, and petroleum-based explosives. When low-order explosives are intentionally confined within a casing, as in a pipe bomb, the speed of detonation increases drastically, though not to the extent of high-order explosives.[14]

Shaped and projectile charges are explosives engineered to create directionality to blast overpressures. A shaped charge has a lined hollow space that focuses explosive force, improving control over the area of damage — this characteristic can increase the likelihood of inflicting damage on a desired target while limiting collateral damage (*see Figure 3*). An explosively formed projectile is a special type of shaped charge that uses a metal plate designed to deform to an aerodynamic shape when struck by a blast wave, resulting in its propulsion toward the intended target at high velocity and with narrower focus of force. These are often used to circumvent military armor.[15]

Enhanced-blast weapons have been developed to increase the lethality of explosive weapons by increasing the energy released from an explosion. Thermobaric and fuel-air explosives are currently in use by the United States, China, and Russia. These weapons have a lower peak overpressure, but duration of the overpressure is greatly increased and the blast wave propagates farther. Fuel-air explosives create a small initial explosion that disperses a vapor cloud of a fuel such as ethylene oxide. A subsequent detonation ignites this fuel cloud, resulting in a uniform dynamic overpressure.[16] Thermobaric explosives use a reactive metal to augment the blast.[17]

Some civilian situations have the potential for blasts, for example, dust/air mixtures in grain silos or coal mines and slowly leaking flammable gases. Boiling liquid-expanding vapor explosions take place when liquefied gas stored above its boiling point vaporizes and ignites as a result of container failure.[18] A situation in which this can occur is after a fuel tanker collision when the tanker is resting on its side and the relief valves on the top of the truck, designed to ventilate vapor and reduce pressure, fail to function properly. As previously discussed, confinement of a blast, such as in a building or tunnel, results in faster detonation and higher overpressure.[19] Asphyxia can result if the reaction consumes all available atmospheric oxygen.

Classification of Blast Injuries

Traditionally, injuries resulting from blasts have been categorized by the mechanism through which they are sustained. The system described by Zuckerman in 1941, classifying these injuries as primary, secondary, tertiary, and quaternary, is still widely used today.[20] These injuries may occur in isolation or in any combination. A fifth class, quinary blast injury, was proposed recently. (*See Table 1.*) The lethality of a blast and the severity of injuries that a blast can cause depend on numerous factors: magnitude of the explosion, proximity of the casualty to the explosion, occurrence in an open vs enclosed space, and the presence of other structures or objects that can cause additional injuries.

Primary Blast Injuries

Primary blast injuries are caused predominantly by high-order explosives, which create a blast wave of overpressure also known as a shock wave. The blast wave travels outward from the explosion at 3-9 km/sec. Low-order explosives form a lower energy blast wave that travels at subsonic speed. The overpressure formed by a blast wave

Table 1. Blast Injury Mechanism Categories

Category	Mechanism	Typical Injuries
Primary	Caused by blast wave	Tympanic membrane rupture, blast lung, intestinal hemorrhage and rupture
Secondary	Caused by flying debris and shrapnel	Blunt and penetrating traumatic injuries
Tertiary	Due to individual being thrown by blast	Blunt and penetrating traumatic injuries
Quaternary	Thermal, toxic, and asphyxiant effects	Thermal burns, chemical burns, exposure to toxins, asphyxiation
Quinary	Delayed hyperinflammatory response	Tachycardia, low central venous pressure not responsive to fluid, fever

characteristically affects areas at or near junctions of tissues of different density, for example, the interface in air-containing structures such as the eardrums, lungs, and bowel.

The mechanisms by which primary blast injuries occur are described by the concepts of spalling, implosion, and inertia, which were first proposed by Schardin in 1950.[21] In spalling, the blast wave propagates from a more dense to a less dense medium, resulting in fragmentation and displacement of the dense medium into the less dense medium. An underwater detonation demonstrates this effect with the splash of fragmented water into the air. Another example is the propagation of the blast wave through the armor of a tank without physical penetration. When the blast wave approaches the interior surface of the tank's armor, it causes fragmentation of metal, which can injure the tank's occupants.

In implosion, the blast wave overpressure compresses gases and gas-filled structures. When the wave passes, the gas expands, releasing significant kinetic energy to the surrounding tissue. At the level of the lung alveoli, air can enter the pulmonary vasculature and cause systemic air embolism.[22]

Inertial effects occur when a force acts on two structures of different densities simultaneously. The less dense material will have greater acceleration than the more dense material, which causes significant stress at the boundary of the two structures.[23] The resulting disruption is similar to an acceleration-deceleration injury.[6]

Secondary Blast Injuries

Secondary blast injuries are caused by the acceleration of objects, especially bomb fragments or shrapnel, outward from the blast center. These projectiles can cause significant penetrating and blunt injuries. The "shimmy" caused by irregularly shaped shrapnel causes fragments to tumble within the tissue, increasing the amount of damage.[24]

Tertiary Blast Injuries

Tertiary blast injuries result from the victim being physically displaced by the blast wave. Commonly found in this category are fractures, crush injuries, compartment syndrome, internal organ injury, and traumatic brain injury. Injuries sustained in a structural collapse after an explosion are also classified as tertiary blast injuries.[25]

Quaternary Blast Injuries

Quaternary blast injuries occur as a result of the thermal, asphyxiant, and toxic properties of the explosive substance. Exacerbations of chronic conditions, such as asthma in the setting of smoke exposure, are in this category.[19] These effects are greatly increased when the explosion and the blast victim are in an enclosed space. Bones and teeth from suicide bombers create an intentional form of secondary blast injury that has the potential to become a quaternary injury due to transmission of infectious agents. Braverman et al published a case report of bone fragments from a suicide bomber being recovered from within a victim. These fragments tested positive for hepatitis B surface antigen. As a result of this case, Israel's Ministry of Health ordered active immunization against hepatitis B for all patients injured in such attacks.[26]

Quinary Blast Injuries

The term quinary blast injury is used to describe delayed effects of explosions, such as infections, radiation exposure, and other toxic exposures.[8,27,28] A group in Israel applied this term to a hyperinflammatory state seen in four patients with tachycardia, low central venous pressure not responsive to fluid, and fever. It was presumed to have been caused by exposure to the high-order explosive, PETN, which also functions as a potent vasodilator.[29]

Prehospital Care

Prehospital management of blast injuries must focus on situational aspects prior to individualized patient care. First, the responders must consider secondary hazards such as structural instability or delayed devices that could cause harm to rescuers. Consideration also must be given to contaminants that might be present in the environment and on the victims. These may be chemical, biological, or radioactive. Proper steps must be undertaken to decontaminate patients and provide protection to rescue personnel. The number of casualties also must be assessed. Obviously, a single casualty from a fireworks explosion requires significantly fewer resources than a large group of people injured by an IED detonated within a crowded enclosed space. A mass casualty incident is an event that exceeds the healthcare capacity of the response. Large-scale blast injuries follow a typical pattern of presentation. Those closest to the blast have a very high initial mortality rate. Survivors have injury severity indirectly proportional to their distance from the explosion. Many of the minimally injured come to the closest healthcare facilities as "walking wounded" prior to arrival of the more severely injured patients who undergo on-scene triage.

Triage

Although numerous triage methodologies have been created, there is no national standard for the prehospital management of mass casualty incidents. These triage methods strive to provide the maximum amount of benefit to the maximum number of patients in an environment with limited resources.[30] There is a tendency of those performing triage to up-triage casualties, thus directing them to receive more immediate care than is warranted. In some cases, overtriaging increases the overall case fatality rate by redirecting critical care resources away from the patients most in need.[31] The negative effect of overtriage is magnified as the number of patients increases. At some point, every healthcare system will reach a critical juncture where it is unable to cope with needs of the injured.

The START (Simple Triage and Rapid Treatment) system was developed by the Newport Beach Fire and Marine Department and Hoag Hospital in Newport Beach, California, in 1983, and has been adopted by numerous medical systems around the world. This system rapidly places a casualty into one of four categories based on a simple clinical assessment. The areas assessed are ability to walk, the presence of spontaneous breathing, respiratory rate, the presence of a pulse, and alertness. The categories are green for minor injury or "walking wounded," yellow for delayed treatment, red for immediate treatment, and black for deceased.[32,33] Multiple companies have developed and sell materials to assist with the performance of triage at mass casualty incidents. (*See Figure 4.*)

Use of Tourniquets

Secondary and tertiary blast injury patterns often result in extremity amputations and penetrating injuries that place the patient at risk for exsanguination. The use of tourniquets to control bleeding in extremity injuries has been credited with reducing mortality in both military and civilian settings. At the start of military operations in Afghanistan and Iraq, medical support personnel were not routinely supplied with manufactured tourniquet devices. However, they started to become available to troop medics around 2005 and were standard issue by 2007. Eastridge et al retrospectively reviewed the battlefield fatalities that occurred between October 2001 and June 2011. They found that the death rate associated with extremity hemorrhage decreased from 23.3 fatalities per year before widespread tourniquet use to 3.5 deaths per year after implementation of the practice.[34] It is likely that the lower mortality rate was the result of tourniquet use as well as improved training of emergency care providers.

The Boston Marathon bombing in 2013 involved two improvised explosive devices that were detonated in close succession near the finish line, causing three deaths and 264 injuries. Twenty-nine victims, including 15 with lower extremity traumatic amputations, had life-threatening exsanguination. Tourniquets were applied to 27 of the 29 people during prehospital care. All of the tourniquets were improvised and applied by both emergency medical services (EMS) and non-EMS responders.[35] Because of these experiences and other military studies,[36-38] recent guidelines have promoted the use of tourniquets in the prehospital setting.[39]

Organ-Specific Injury Patterns

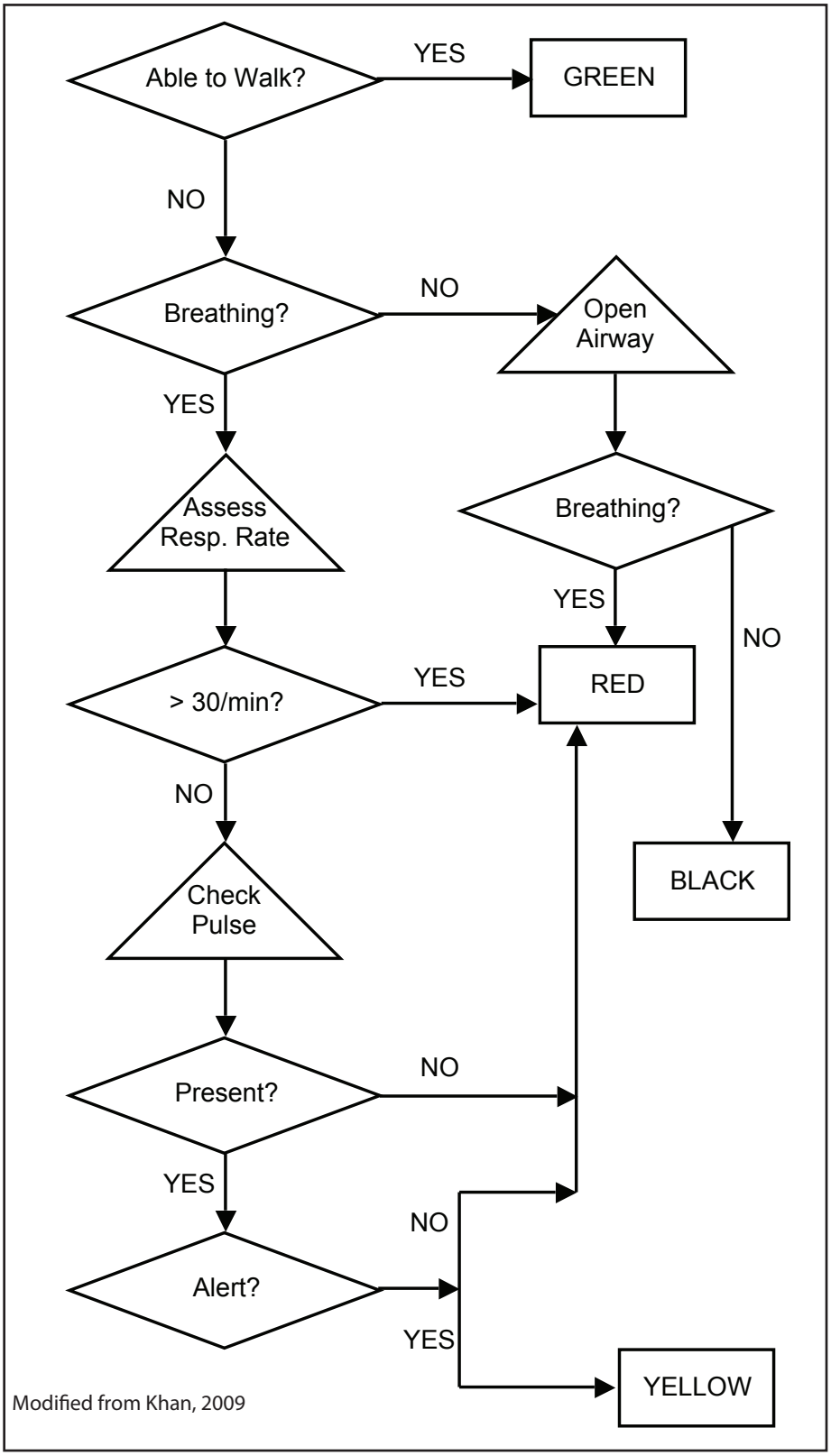
Central Nervous System. Traumatic brain injury (TBI) and post-traumatic stress disorder (PTSD) have been called the "signature" injuries of military operations in Iraq and Afghanistan. According to the Defense and Veterans Brain Injury Center, more than 333,000 service members have been diagnosed with TBI since 2000.[40] In 2010, Wojcik and colleagues reported that explosions were the cause of 46.7% of brain injuries sustained by American troops in Iraq and 63.9% of such injuries in Afghanistan.[41] Six months after the Oklahoma City bombing of the Alfred P. Murrah Federal Building, 34% of the survivors who could be assessed had symptoms of PTSD.[42] The clinical symptoms of post-concussion syndrome and PTSD have significant overlap, making it difficult to determine a blast's contribution to this entity beyond the psychological trauma suffered by the victim.

Central nervous system (CNS) injuries following explosions are caused predominantly by secondary and tertiary blast mechanisms. These injuries are consistent with blunt and penetrating mechanisms from non-blast causes. Quaternary CNS injuries can be caused by toxic exposures, burns, or asphyxiation. The contribution of a primary blast to CNS injury has been a matter of debate since World War I.[43] Some blast-exposed patients have evidence of CNS injury in the absence of secondary or tertiary blunt or penetrating trauma. A proposed mechanism for this counterintuitive scenario is blast wave propagation directly through the skull or sinus openings.[44] Another proposed mechanism is thoracoabdominal compression that engorges the cerebral vascular and cerebrospinal fluid systems. Increased pressure in the cerebral vasculature damages small cerebral vessels and causes a loss of integrity of the blood–brain barrier.[45] Blast-wave-induced damage to air-filled structures, such as the lung, can lead to formation of air emboli through the process of spallation. The emboli then travel to the brain or spinal cord and cause ischemia or infarction. Structural findings of primary blast CNS injuries include skull fracture, diffuse axonal injury,[46] contusion, hemorrhage, and edema. Immediately after the blast, survivors with CNS blast injury may have loss of consciousness, memory loss, headache, confusion, nausea, and focal neurologic deficits. Patients with PTSD report the persistent re-experiencing of their symptoms (through flashbacks, dreams, and intrusive thoughts), avoidance symptoms, memory loss, depression, and hyperarousal.

Pulmonary. Blast lung injury (BLI) is the term for disruption of the alveolar architecture from supersonic pressure, with resultant pulmonary contusions (with or without laceration), other pulmonary barotrauma (pneumothorax, pulmonary interstitial emphysema, pneumomediastinum, subcutaneous emphysema), and acute gas embolism (AGE). Among the initial survivors of an explosion, BLI is the most common primary blast injury that ultimately is fatal.[8]

Originally, it was thought that a blast

Figure 4. START Triage Algorithm



Modified from Khan, 2009

wave travels down a patient's nasal and lung passages, resulting in alveolar disruption. Subsequent evidence suggests that the blast wave acts on the chest wall, causing compression of the chest and creating an associated transient intrathoracic pressure wave, which travels through the lung parenchyma and can even reflect off the mediastinum, creating complex shearing forces.[47]

Tsokos et al characterized the damage of such forces from a histopathological perspective, noting a nearly uniform injury pattern in a series of human autopsy cases. Close-range blast victims of chemical explosives were compared with non-blast controls. In addition to the expected alveolar rupture, pulmonary hemorrhage, and edema, the authors noted air, bone marrow, and fat emboli, postulating that the latter could be a major determinant in the development of acute respiratory distress syndrome (ARDS).[48]

Clinically, the initial assessment of a patient at risk for BLI should focus on the fundamentals of airway and breathing assessment and management. Signs and symptoms of the injury are usually present on initial exam. They include chest pain (often retrosternal), coughing with or without hemoptysis, dyspnea, and tachypnea. On exam, the patient may have dullness to percussion, palpable crepitus, and a retrosternal crunch suggestive of pneumomediastinum.[49] Other diagnostic clues include hypopharyngeal petechia, hypoxia, cyanosis, diminished breath sounds, and hemodynamic instability.[19]

A proposed "triad" — respiratory distress, hypoxia, and the classic "butterfly" or "bat wing" pattern of bilateral hilar pulmonary infiltrate seen on chest radiograph — underscores the absolute need for pulse oximetry evaluation and chest radiograph in all suspected cases of BLI. The chest radiograph should be evaluated for subcutaneous emphysema, rib fractures, hemopneumothorax, pneumomediastinum, as well as the presence of foreign bodies such as shrapnel. Thoracic computed tomography (CT) may serve a role in the diagnosis of small pneumothoraces and pulmonary lacerations as well as the quantification of interstitial and alveolar fluid burden,[12] and the identification of foreign bodies not seen on plain imaging.[51] Interestingly, Avidan wrote that there is no good pathophysiological explanation for why BLI infiltrates are central, in contrast to the peripheral infiltrates characteristic of blunt trauma.[50] The infiltrates may progress over days. Although such progression can be the

Table 2. Severity Categories for Primary Blast Lung Injury

	Mild	Moderate	Severe
Radiographic infiltrates	Unilateral	Asymmetrical bilateral	Diffuse bilateral
PaO_2/FiO_2 ratio (PFR) (mmHg)	> 200	60-200	< 60
Bronchopleural fistula	No		Yes
Positive pressure ventilation (PPV) requirement	Unlikely to require	Likely to require conventional methods	Universal, may require unconventional methods
Positive end-expiratory pressure (PEEP) requirement (cm H_2O)	If needed, < 5	5-10 usually needed	> 10 commonly needed

Severity categories for primary blast lung injury, which may help to predict necessity for use of PPV and PEEP, as originally proposed by Pizov et al[55] and expanded by Wightman et al[12]

result of aggressive fluid resuscitation, practitioners should retain a high clinical suspicion for pneumonia.

Initial stabilization measures should be based on the clinical findings at presentation. Hypoxia is caused by impaired oxygen diffusion and should be managed aggressively with supplemental oxygen, likely via non-rebreather mask.[19] The use of non-invasive positive-pressure devices, such as bilevel positive airway pressure (BiPAP) and continuous positive airway pressure (CPAP), is not recommended. A patient with diminished or asymmetric breath sounds in the setting of possible pneumothorax should undergo immediate needle decompression, followed by tube thoracostomy. Shorter, larger bore tubes are recommended, given the possibility of large air leaks associated with bronchopleural fistulas.[12]

Most critically ill patients with BLI require intubation with mechanical ventilation. The positive pressure used in mechanical ventilation may worsen the findings of BLI. Care should be taken to minimize the risk of barotrauma, tension pneumothorax, and AGE. A pressure-control or pressure-limited mode should be used, with the goal of maintaining an end-inspiratory plateau pressure of less than 30 cm H_2O using about 6 mL/kg of tidal volume.[52] Permissive hypercapnia has been studied in BLI; no evidence of organ dysfunction related to respiratory acidosis was found,[53] but some authors caution against its use with coexisting neurologic injury.[54] Elevated positive end-expiratory pressure (PEEP) higher than 10 cm H_2O might be required to overcome initial hypoxia but should be titrated down as soon as possible.[12]

Pizov and colleagues proposed a system to stratify BLI based on severity and to prognosticate the need for mechanical ventilation strategies. This classification scheme was based on a series of victims of Israeli bus bombings. They used radiographic findings and the PaO_2/FiO_2 (PF) ratio to determine the severity of the BLI.[55] A PF ratio > 200 mmHg with only unilateral infiltrates was deemed mild, not requiring mechanical ventilation. Patients with a moderate PF ratio (between 60 and 200 mmHg) with bilateral infiltrates were found to require intubation but were supported successfully with conventional ventilator techniques similar to those described above. Patients with severe BLI, described as a PF ratio < 60 mmHg and diffuse radiographic infiltrates, often had pneumothoraces or bronchopulmonary fistulas and required significantly higher PEEP. The determinants used to classify severity were expanded by Wightman and Gladish to include requirement of positive pressure ventilation and use of PEEP.[12] (*See Table 2.*) Some unconventional ventilator methods have been used in patients with severe BLI, including independent lung ventilation, high-frequency jet ventilation, and nitric oxide.[55] The actual benefit of these techniques is unclear. Extracorporeal membrane oxygenation also has been used in patients with BLI,[56] but some clinicians advise caution in its use given the risk of catastrophic pulmonary hemorrhage.[55]

AGE can occur with the initial blast wave through the mechanism of spalling, or it can occur later, when pulmonary tissue disruptions cause air to move into the arterial or venous system. When a lung injured by a blast experiences high alveolar pressure, such as that seen with positive pressure ventilation, the risk of AGE increases. Emboli have been noted to travel to the eye, brain, spine, and coronary arteries, resulting in sudden blindness, neurologic deficit, loss of consciousness, and chest pain.

Providers should assess the patient for retinal arterial gas bubbles on fundoscopy, focal neurologic deficits, or findings of ischemic changes on an electrocardiogram (ECG).[19] Initial treatment is high-flow oxygen. Definitive treatment is hyperbaric oxygenation, which reduces the size of the gas bubble through the principle of Boyle's law. Placement of the patient in the left lateral decubitus position with as much pronation as possible elevates the left atrium, making it less likely for air bubbles to pass to the ventricles and to the systemic circulation. This position also allows the coronary artery ostia to sit in the lowest possible position, minimizing the risk of embolic myocardial infarction. Conversely, if there is high suspicion for isolated right lung injury, place the patient in the right lateral decubitus position — a dependent injury has a higher capillary pressure and thus less risk of AGE.[12]

Historically, BLI was thought to have the potential for delayed presentation, up to 48 hours after an explosion. In more recent case series, all clinically significant pulmonary decompensation requiring mechanical ventilation occurred within the first 2 to 6 hours.[50,55] However, these studies were based on predominantly closed-space

explosions, limiting the generalizability of the findings. It is possible that victims of closed-space explosions, with exposure to prolonged overpressures, have a shorter latency period.[57] Approximately 70% of critically injured patients with BLI who survive to admission will also survive to discharge, most with normal or near-normal lung function by 1 year after the injury.[19]

Cardiac. A primary blast is one of several mechanisms that can cause blunt injury to the heart. In the literature, causes of blunt cardiac injury (BCI) are typically grouped together. BCI can be rapidly fatal, causing death at the scene; it can be relatively apparent through clinical clues upon presentation at the emergency department; or it can have only subtle signs and symptoms, with delayed development. It is unlikely that a primary blast will cause a cardiac contusion in isolation. If a blast's overpressure is significant enough to cause cardiac contusion, the patient will likely have multisystem effects.

Important acute injuries to consider in patients with BCI are free wall or septal rupture, pericardial tamponade, coronary artery injury, papillary muscle rupture, aortic or mitral regurgitation, and arrhythmia. Subtle or delayed injuries can include rupture of a low-pressure chamber or coronary vein. Asymptomatic hemopericardium can take months to organize into constrictive pericarditis. Pulmonary and tricuspid valve injuries might remain asymptomatic for years.[58]

Physical examination findings suggestive of significant BCI include tachycardia (secondary to stress, acute blood loss, hypoxia, exertion, or dehydration), bradycardia, hypotension, delayed capillary refill, and presence of an S3 gallop, rub, or new murmur.[59] Several reviews of blast injury discuss animal studies in which bradycardia and hypotension resulted from a primary blast.[60,61] A bimodal distribution of vital sign instability occurs immediately and again 2-3 hours after the blast (which could have been caused by vagus nerve-mediated bradycardia without compensatory vasoconstriction, resulting in cardiogenic shock).[62]

All patients with suspected BCI should have a 12-lead ECG. There are no electrocardiographic findings specific for BCI.[59] Non-specific abnormalities, including sinus tachycardia, are present in up to 80% of patients with BCI.[58] Evaluate the ECG for signs of ischemia, infarction, and arrhythmia. A patient's sympathetic stress response to a blast event could precipitate acute coronary syndrome in the setting of baseline coronary artery disease. Address dysrhythmias in accordance with standard practice. Electrical alternans, the electrocardiographic finding of alternating amplitude and vector of the QRS complex, suggests the presence a large pericardial effusion.

Perform bedside echocardiography in any patient suspected of having BCI. Findings may include the presence of pericardial effusion with or without evidence of tamponade, aortic rupture, and intracardiac thrombus or gas.[58] It is also useful for assessing cardiac contractility and helps in the evaluation of volume status. Right ventricular enlargement might be seen in patients with tricuspid valve injury, left ventricular failure, pulmonary embolism, or ARDS.[63]

The use of a cardiac biomarker, most frequently troponin, as a screening tool for BCI is a controversial topic. Troponin can be elevated in the setting of catecholamine release, reperfusion injury after hypovolemic shock, microcirculatory dysfunction, and oxidative injury. Conversely, troponin can be negative in patients with cardiac dysrhythmias, a finding that requires further monitoring and possible intervention.[64] Previous guidelines for evaluation of patients for BCI did not require biomarker evaluation in the setting of an initial normal ECG.[65] More recent guidelines call for screening for BCI with both electrocardiography and measurement of troponin.[66] The timing of troponin evaluation and the use of serial testing have not been established.

Abdomen. Most gastrointestinal injuries caused by blasts are the result of secondary and tertiary injury mechanisms. The large and small bowel are gas-containing organs and are thus at risk for primary blast injury. Abdominal primary blast injury was first reported in 1917 in a case series of three sailors who were in the water when a torpedo struck their vessel.[67] Solid organ injuries are rarely found as a result of a primary mechanism alone. When they do occur, they are most commonly subcapsular hematomas of the liver, spleen, and kidneys.[49] The risk of abdominal injury is increased when the blast occurs in a confined space[68] or under water.[69,70]

The blast wave transmits shearing and stress forces on the bowel wall, resulting in submucosal and subserosal hemorrhages. Gastrointestinal hemorrhage results from mucosal rupture. The bowel can be perforated directly from the blast, or a perforation might have a delayed presentation due to necrosis of injured portions of the bowel.[69]

The presentation of abdominal primary blast injury is inconsistent. Symptoms can be masked by more severe acute symptoms associated with primary blast injury of the brain and lung. Clinical symptoms include pain of varying character and intensity, nausea, vomiting, diarrhea, and tenesmus. The stool or emesis might be bloody. Bowel sounds might be absent. The patient could have varying degrees of tenderness with presence of guarding or rebound.

As with other abdominal trauma, screening with ultrasound is appropriate. Stable patients in whom abdominal injury is being considered should undergo CT. It is important to note that CT might have insufficient sensitivity to identify hollow organ injury.[71] The colon and ileocecal region are at greatest risk of injury.[6,72] Indications for surgery in abdominal primary blast injury mirror those for other abdominal injuries. Non-operative management of patients with evidence of injury but stable clinical status should allow frequent serial exams for a minimum of 3-5 days (the risk of perforation persists up to 14 days).[69]

Eye. The ocular surface constitutes only 0.1% of the total body surface area but it is frequently found to be injured in blast survivors.[19,73] Many survivors of recent devastating terrorist attacks had ocular injuries: 8% in the 1995 Oklahoma City bombing, 21% in the 1998 U.S. Embassy bombing in Nairobi,[54] and 18% in the 2004 Madrid train bombings.[51] In the Nairobi bombing, patients as far as 2 km from the site sustained eye injuries.[54]

These significant and potentially devastating injuries largely occur as the result of secondary or tertiary blast mechanisms. Overall, their management is consistent with that of conventional ocular trauma and, therefore, will not be discussed here. However, several circumstances unique to primary blast injury of the eye are described. Emergency enucleation is not advised.[25]

As previously discussed, ocular AGE can occur concomitantly with primary BLI. Retinal artery air bubbles detected on fundoscopic exam should be treated with high-flow oxygen therapy. Consideration of hyperbaric oxygen therapy is based on sound physiologic principles despite lack of clinical trials. Other ocular injuries that can be caused by a primary blast mechanism include globe rupture, serous retinitis, and hyphema.[8]

The globe might be capable of withstanding considerable force during the primary blast, transferring the energy and causing a "blowout" of the orbital floor and lamina papyracea. CT scan of the orbits with coronal views is the most useful radiographic test for assessing such injuries, and ophthalmologic assessment is indicated. Shamir et al described two patients who had been caught up in a terrorist bombing on an Israeli bus, who presented 2 weeks after the incident with complaints of ocular discomfort. The patients had isolated orbital blowout fractures without any other sign of injury. They were found to have upward gaze restriction and slight enophthalmos, though they did not demonstrate other classic findings of diplopia or infraorbital paresthesia.[73]

Ear. The delicate and sensitive structures of the auditory system are the most frequently injured structures during a blast incident. The external ear is subject to trauma from secondary, tertiary, and quaternary blast mechanisms. Debris scattered in the blast may result in secondary blast injuries such as lacerations, amputations, and damage to structures in the middle or inner ear. An example of a quaternary mechanism would be fire and extreme heat causing burns to exposed structures of the auditory system. The middle and inner ear are most frequently injured by the primary blast wave overpressure.

The tympanic membrane (TM) is the structure most sensitive to pressure differentials. A TM exposed to as little as 5 psi (35 kPa) of pressure is at risk of perforation. Individuals exposed to 15-50 psi have a 50% risk of perforation.[9] The risk is affected by the magnitude of the blast, distance from the blast center, and the person's head position relative to blast wave propagation. Another important factor is whether the blast occurred in an open or enclosed space. Researchers evaluating civilian bus blast survivors found that the rate of TM perforation for people on the bus was markedly higher than for those adjacent to the bus (29% vs 1%).[68] Other middle ear injuries that can occur include disarticulations and fracture of the three bones of the ossicular chain — the malleus, incus, and stapes. These structures convert acoustic vibrations from the tympanic membrane to pressure waves within the inner ear. Any disruption along this chain results in conductive hearing loss. Sensorineural hearing loss can occur when the cochlear hair cells of the inner ear are damaged.

After exposure to a blast, patients might complain of hyperacusis, hearing loss, tinnitus, otalgia, or vertigo. Differences in rates of auditory system injury between civilian and military blasts may be related to the use of ear protection. Remenschneider and associates reported on 94 survivors of the Boston Marathon bombing who were evaluated by otolaryngologists. Shrapnel injury to the external ear and external auditory canal was found in eight patients. Perforation of the tympanic membrane was found in 48 patients, with 14 being bilateral.[74] The incidence of ear injuries among survivors of blast injuries during Operation Iraqi Freedom was reported by Dougherty and coworkers in 2013. Of 3981 blast survivors, 1223 (30.7%) had injury to the auditory system and 319 (8.0%) had TM rupture. The most common injury was tinnitus, found in 767 survivors (19.3%).[75]

All survivors of blasts should undergo an otoscopic examination. Physical exam findings may include lacerations, burns, and avulsions of the external ear, hemotympanum, TM rupture, and the presence of foreign bodies in the external auditory canal. The most frequent location of TM rupture is inferiorly at the pars tensa.[76]

An injured external ear is often difficult to manage due to its irregular contour, poor blood supply, and underlying cartilage. Concomitant injuries to vital organs often take precedence over the auditory system. For avulsions of the pinna, clean the wound edges thoroughly and debride non-viable tissue. A postauricular pocket can be created to avoid necrosis and tissue loss until definitive repair can be performed.[77] Treatment of TM perforations is generally expectant, since many heal spontaneously. Remove any debris from the external auditory canal. While a perforation is present, ototoxic eardrops should be avoided. Tympanoplasty is usually performed if spontaneous resolution is not observed after a period of conservative management. This procedure has shown good results in the hands of experienced surgeons, with an 82% closure rate in one study.[78] Healing might be complicated by the formation of cholesteatoma, a mass of keratinizing squamous epithelium that can erode into other structures of the middle ear. Choleastoma formation complicating TM rupture healing has been reported in 4% to 12% of blast injury survivors.[79-81]

Orthopedic/Soft Tissue. Traumatic amputations from blast incidents are considered a marker of exposure to severe overpressure and portend a high likelihood of death.[82] They are typically associated with head trauma, chest wounds, evisceration, and BLI as well as the obvious risk of hemorrhage.[54] In general, it is accepted that a primary blast wave creates axial stresses in bones, leading to fractures.[83] A near-simultaneous high-velocity blast wind flails and separates the soft tissue.

Reported survival rates for patients with traumatic amputations caused by blasts range from 1% to 3%.[18,54,83] However, the 69 patients with traumatic amputations sustained in the 2007 suicide bombing attack on the London Underground had a survival rate of 24.5%. Patel and colleagues postulated that the higher survival rate in this incident was related to the placement of the bomb on the floor in addition to

the positions of the survivors in the subway cars. Many passengers were tightly packed in standing position, so the primary blast wave was probably channeled through a "forest" of legs, which shielded other organs. Other passengers were seated, so their torsos were partially protected from the blast and resultant shrapnel by the seats beneath them. A blast wave strong enough to cause an upper extremity amputation is thought to be more likely to be fatal because it impacts nearby structures — brain, heart, lungs — with similar force.[83]

Shuker documented that for survivors of a blast, the presence of facial fractures was a marker for severe multisystem trauma. Transverse mandibular fractures are unique to blast injury and occur when a significant blast overpressure encounters the juncture of the relatively weaker cancellous body of the mandible and its firm lower border of solid cortical bone. The shearing along this line can result in a horizontal fracture, which runs parallel to the jaw. Because teeth are meant to withstand vertical forces, victims might also experience shearing of the teeth along the cementoenamel junction. Cover the pulp exposed by such fractures with cotton impregnated with zinc oxide and eugenol paste, pending definitive dental intervention.[84]

The nature of blast wounds makes them inherently contaminated.[85] Dirt, shrapnel, and bits of clothing usually contaminate the soft tissue of victims. Bone fragments can act as secondary projectiles. All bony fragments should be removed, because they carry a high risk of infection.[51] Heavy polymicrobial contamination can occur as a result of tissue stripped from bone, disruption along fascial planes, and secondary projectiles with unknown trajectories. Given the risk for infection, treat all wounds with copious irrigation and debridement of non-viable tissue. Some soft-tissue wounds and all open fractures require prophylactic antibiotic treatment. Patients should be given appropriate tetanus prophylaxis. Apply direct pressure to bleeding sites unless threatened exsanguination necessitates the use of a tourniquet or vessel ligation. Fracture management includes reduction and splinting, if immediate operative repair is not indicated, to prevent further soft-tissue, vascular, and nervous damage as well as for pain relief.[86] Cover open wounds with sterile moist gauze in preparation for surgical debridement; early and regular debridement plays a critical role in the management of large wounds.[31] Maintain a high index of suspicion for the development of rhabdomyolysis and compartment syndrome in patients with crush injuries.[54]

Special Populations

The indiscriminate nature of a blast overpressure in the setting of a terrorist attack or other disaster scenario requires consideration of special populations. In pregnant women, fetal injury is uncommon in the absence of maternal injury, but a blast wave can result in placental abruption via the spalling mechanism. The Centers for Disease Control and Prevention (CDC) recommends that all women in the second or third trimester be kept for continuous fetal monitoring and screening for fetal-maternal hemorrhage.[87]

Children are at particular risk for blast injury because of their relative size and increased risk for head and abdominal trauma. As a result of terrorism, the medical community is gaining experience in characterizing blast injuries and fatalities in pediatric patients. The Joint Theatre Trauma Registry provides data on all civilians admitted to U.S. military treatment facilities. Edwards and associates reviewed the registry from 2002 to 2010 for pediatric patients admitted for blast injury. The 1000 Afghan and Iraqi children who were included in their dataset had an increased risk for head and neck injuries compared with pelvic and extremity trauma.[28]

Many children involved in a blast are in a closed or confined space. As discussed above, these areas are associated with higher rates of injury and fatality compared with open-air blasts. As with adults, only a relative few pediatric patients will survive major injuries sustained in the worst areas of blast overpressure. Those who do survive will likely need care at a dedicated pediatric medical center. Only 5% of hospitals in the United States have a dedicated pediatric center and operate at capacity or over-capacity on a regular basis. The ability of these hospitals to absorb additional patients from a major blast incident is limited.[88]

The elderly are also more prone to prolonged and complicated hospitalizations after blast injury. Underlying medical conditions can be exacerbated by the blast or make recovery from new injuries more difficult. Orthopedic injuries are more prevalent in this population. Given the risk for decompensation, blunt chest trauma is of greater significance, and the provider should have a low threshold for prolonged observation and monitoring. The CDC specifically cites the challenge of decontamination for individuals with limited mobility, noting that technical decontamination of wheelchairs, walkers, and other walking aides may be necessary.[87]

Special Considerations: Chemicals and Radiation

Blasts can be associated with exposure to toxic chemicals or radiation, whether intentional in the case of terrorist activity or unintentional in industrial incidents. Providers caring for blast injury survivors should consider these possibilities to help prevent cross-contamination and predict injury patterns in survivors. For example, a patient developed methemoglobinemia after exposure to a blast caused by detonation of TNT.[89] Survivors might need to be decontaminated prior to their entry into the hospital to prevent further absorption and exposure of healthcare workers to the agent. Every hospital should have a plan in place for how to respond to a mass casualty incident and provide decontamination. The most important step to adequately decontaminate a patient is to remove all of his/her clothing and jewelry. This step will reduce the contamination load by 70-85%.[90]

A radiological dispersion device, popularly known as a dirty bomb, presents a challenging scenario for disaster management. This type of device would be an ideal weapon for terrorists because of the injuries that the blast would inflict and the psychological consequences of the presence of radiation.[91] Dirty bombs are not the same as a nuclear weapon, which works by the process of fission or a combination of fission and fusion to release a very large amount of energy. The radiation consequences of a dirty bomb likely would be much less than

the effects of the blast itself. Delays in the treatment of survivors as a result of the need to protect care providers might increase the morbidity and mortality stemming from the initial blast. Industrial incidents involving an explosion at a nuclear power plant or a processing plant for radioactive materials carry a much greater threat for radiation injury. The Fukushima Daiichi nuclear disaster, initiated by an earthquake-induced tidal wave, resulted in the breakdown of pumps used to cool fuel rods. Subsequent explosions led to the prolonged release of radioactive material into the environment. Despite the major damage that occurred at the plant, the exposure to inhabitants of the area is estimated to be below the cumulative background dose.[92] Healthcare personnel who are treating blast survivors contaminated with radioactive material should limit their exposure to radiation by limiting time, increasing distance from the source, and the use of shielding.[54]

Disposition

No evidence-based, definitive guidelines exist regarding the disposition of patients exposed to blasts but without significant injury. The assessment and treatment of obviously injured patients is more straightforward. Initial evaluation should follow standardized Advanced Trauma Life Support protocols. The use of TM perforation as a screening test to determine a patient's potential to develop delayed lung or gastrointestinal injury is no longer recommended. In a study of military personnel in Iraq who were exposed to blasts, TM rupture was seen in only four of nine patients with primary blast injury to the lung or gastrointestinal tract.[93] The sensitivity of < 50% in this patient series demonstrates that the absence of TM rupture does not prove absence of other injury. However, the presence of TM rupture is associated with other blast injuries and places a patient in a high-risk group. One study of military blast survivors showed an almost three-fold relative risk for loss of consciousness in those with TM rupture compared to those without.[94] Patients who are asymptomatic with normal vital signs; normal physical examination findings, including intact TMs; and a normal chest radiograph are likely safe for discharge after 4-6 hours. Patients who are at higher risk for injury should be observed for longer periods with frequent reassessments. These include individuals exposed to large blasts, to blasts in enclosed spaces, or to underwater blasts, and those in close proximity to the blast center. Patients who are discharged home should be given discharge instructions that detail the signs and symptoms of delayed lung and gastrointestinal injury.

Conclusion

The principles of caring for patients with blast injuries follow the principles of disaster and trauma management. At the site of the blast, first responders must initially assess the safety of the scene not only for those who have been injured but also for themselves. If concern exists about contamination with biological, chemical, or radioactive substances, then decontamination procedures must be followed. For mass casualty incidents, triage systems such as the START system have been developed with the underlying goal of providing the maximum amount of benefit to the maximum number of patients. First receivers at hospitals need to be prepared to manage a vast array of injuries. Planning for mass casualty events using an all-hazards approach will help with coordination of the many resources required when such an event occurs.

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References

1. National Consortium for the Study of Terrorism and Responses to Terrorism. Annex of Statistical Information: Country Reports on Terrorism 2014. Available at www.state.gov/documents/organization/239628.pdf. Accessed Dec. 14, 2015.
2. Tu Y, Granados DV. 2014 Fireworks Annual Report: Fireworks-Related Deaths, Emergency Department-Treated Injuries, and Enforcement Activities During 2014. Washington, DC: US Consumer Product Safety Commission, June 2015. Available at www.cpsc.gov//Global/Research-and-Statistics/Injury-Statistics/Fuel-Lighters-and-Fireworks/Fireworks_Report_2014.pdf. Accessed Dec. 14, 2015.
3. Federal Bureau of Investigations; FBI Bomb Data Center General Information Bulletin 97-1. Washington, DC: U.S. Department of Justice, 1997.
4. Noji EK, Lee CY, Davis T, Peleg K. Investigation of Federal Bureau of Investigation bomb-related death and injury data in the United States between 1988 and 1997. *Mil Med* 2005;170:595-598.
5. Kapur GB, Hutson HR, Davis MA, et al. The United States twenty-year experience with bombing incidents: Implications for terrorism preparedness and medical response. *J Trauma* 2005;59:1436-1444.
6. Wolf SJ, Bebarta VS, Bonnett CJ, et al. Blast injuries. *Lancet* 2009;374:405-415.
7. Mitka M. IOM addresses ongoing effects of blast injury on soldiers. *JAMA* 2014;311:1098-1099.
8. Ritenour AE, Blackbourne LH, Kelly JF, et al. Incidence of primary blast injury in US military overseas contingency operations: A retrospective study. *Ann Surg* 2010;251:1140-1144.
9. Mathews ZR, Koyfman A. Blast injuries. *J Emerg Med* 2015;49:573-587.
10. Leibovici D, Gofrit ON, Shapira SC. Eardrum perforation in explosion survivors: Is it a marker of pulmonary blast injury? *Ann Emerg Med* 1999;34:168-172.
11. Rozenfeld M, Givon A, Shenhar G, et al. A new paradigm of injuries from terrorist explosions as a function of explosion setting type. *Ann Surg* 2015 Jul 1 [Epub ahead of print].
12. Wightman JM, Gladish SL. Explosions and blast injuries. *Ann Emerg Med* 2001;37:664-678.
13. Nguyen N, Hunt JP, Lindfors D, et al. Aerial fireworks can turn deadly underwater: Magnified blast causes severe pulmonary contusion. *Injury Extra* 2014;45:32-34.
14. Stewart C. Blast injuries: Preparing for the inevitable. *Emerg Med Pract* 2006;8:1-28.
15. Morrison JJ, Mahoney PF, Hodgetts T. Shaped charges and explosively formed penetrators: Background for clinicians. *J Royal Army Med Corps* 2007;153:184-187.
16. Baskin TW, Holcomb JB. Bombs, mines, blast, fragmentation, and thermobaric mechanisms of injury. In: Mahoney PF, Ryan JM, Brooks AJ, et al, eds. *Ballistic Trauma: A Practical Guide*, 2nd ed.

London: Springer-Verlag; 2005, Section 3, pp 45-66.

17. Yen NH, Wang LY. Reactive metals in explosives. *Propellants, Explosives, Pyrotechnics* 2012;37:143-155.
18. Abbasi T, Abbasi SA. The boiling liquid expanding vapour explosion (BLEVE): Mechanism, consequence assessment, management. *J Hazard Mater* 2007;141:489-519.
19. Lemonick DM. Bombings and blast injuries: A primer for physicians. *Am J Clin Med* 2011;8:134-140.
20. Zuckerman S. Discussion on the problem of blast injuries. *Proc Roy Soc Med* 1941;34:171-192.
21. Schardin H. The physical principles of the effects of a detonation. In: German Aviation Medicine, World War II, Vol 2. Washington, DC: US Government Printing Office;1950:1207-1224.
22. Ho AMH. A simple conceptual model of primary pulmonary blast injury. *Med Hypotheses* 2002;59:611-613.
23. Phillips YY. Primary blast injuries. *Ann Emerg Med* 1986;15:1446-1450.
24. Kang DG, Lehman RA, Carragee EJ. Wartime spine injuries: Understanding the improvised explosive device and biophysics of blast trauma. *Spine J* 2012;12:849-857.
25. DePalma RG, Burris DG, Champion HR, et al. Blast injuries. *N Engl J Med* 2005;352:1335-1342.
26. 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;4:528-529.
27. Champion HR, Holcomb JB, Young LA. Injuries from explosions: Physics, biophysics, pathology, and required research focus. *J Trauma* 2009;66:1468-1477.
28. Edwards MJ, Lustik M, Eichelberger MR, et al. Blast injury in children. *J Trauma Acute Care Surg* 2012;73: 1278-1283.
29. Kluger Y, Nimrod A, Biderman P, et al. The quinary pattern of blast injury. *Am J Disaster Med* 2007;2:21-25.
30. Baker MS. Creating order from chaos: Part I: Triage, initial care, and tactical considerations in mass casualty and disaster response. *Mil Med* 2007;172:232-236.
31. Balazs GC, Blais MB, Bluman EM, et al. Blurred front lines: Triage and initial management of blast injuries. *Curr Rev Musculoskeletal Med* 2015;8:304-311.
32. Cone DC, Koenig KL. Mass casualty triage in the chemical, biological, radiological, or nuclear environment. *Eur J Emerg Med* 2005;12:287-302.
33. Kahn CA, Schultz CH, Miller KT, et al. Does START triage work? An outcomes assessment after a disaster. *Ann Emerg Med* 2009;54:424-430.e1.
34. Eastridge BJ, Mabry RL, Sequin P, et al. Death on the battlefield (2001-2011): Implications for the future of combat casualty care. *J Trauma Acute Care Surg* 2012;73(6 [suppl 5]):S431-S437.
35. King DR, Larentzakis A, Ramly EP, Boston Trauma Collaborative. Tourniquet use at the Boston Marathon bombing: Lost in translation. *J Trauma Acute Care Surg* 2015;78:594-599.
36. King DR, van der Wilden G, Kragh JF, et al. Forward assessment of 79 prehospital battlefield tourniquets used in the current war. *J Spec Oper Med* 2012;12:33-38.
37. Kragh JF, Burrows S, Wasner C, et al. Analysis of recovered tourniquets from casualties of Operation Enduring Freedom and Operation New Dawn. *Mil Med* 2013;178:806-810.
38. Kragh JF Jr, Littrel ML, Jones JA, et al. Battle casualty survival with emergency tourniquet use to stop limb bleeding. *J Emerg Med* 2011;41:590-597.
39. Bulger EM, Snyder D, Schoelles K, et al. An evidence-based prehospital guideline for external hemorrhage control: American College of Surgeons Committee on Trauma. *Prehosp Emerg Care* 2014;18:163-173.
40. Defense and Veterans Brain Injury Center. DoD Worldwide Numbers for TBI, August 2015. Available at http://dvbic.dcoe.mil/dod-worldwide-numbers-tbi. Accessed December 15, 2015.
41. Wojcik BE, Stein CR, Bagg K, et al. Traumatic brain injury hospitalizations of U.S. army soldiers deployed to Afghanistan and Iraq. *Am J Prev Med* 2010;38(1 suppl):S108-S116.
42. North CS, Nixon SJ, Shariat S, et al. Psychiatric disorders among survivors of the Oklahoma City bombing. *JAMA* 1999;282:755-762.
43. Jones E, Fear NT, Wessely S. Shell shock and mild traumatic brain injury: A historical review. *Am J Psychiatr* 2007;164: 1641-1645.
44. Säljö A, Arrhén F, Bolouri H, et al. Neuropathology and pressure in the pig brain resulting from low-impulse noise exposure. *J Neurotrauma* 2008;25: 1397-1406.
45. Chen YC, Smith DH, Meaney DF. In-vitro approaches for studying blast-induced traumatic brain injury. *J Neurotrauma* 2009;26:861-876.
46. Finkel MF. The neurological consequences of explosives. *J Neurol Sci* 2006;249:63-67.
47. Fitzpatrick-Swallow VL, Kneubuehl BP, Schroter RC, et al. Lung injury similar to blast lung in a case of shotgun wound of the head. *Am J Forensic Med Pathol* 2012;33:286-288.
48. Tsokos M, Paulsen F, Petri S, et al. Histologic, immunohistochemical, and ultrastructural findings in human blast lung injury. *Am J Resp Crit Care Med* 2003;168:549-555.
49. Argyros GJ. Management of primary blast injury. *Toxicology* 1997;121:105-115.
50. Avidan V, Hersch M, Armon Y, et al. Blast lung injury: Clinical manifestations, treatment, and outcome. *Am J Surg* 2005;190:945-950.
51. Hare SS, Goddard I, Ward P, et al. The radiological management of bomb blast injury. *Clin Radiol* 2007;62:1-9.
52. Briel M, Meade M, Mercat A, et al. Higher vs lower positive end-expiratory pressure in patients with acute lung injury and acute respiratory distress syndrome: Systematic review and meta-analysis. *JAMA* 2010;303:865-873.
53. Sorkine P, Szold O, Kluger Y, et al. Permissive hypercapnia ventilation in patients with severe pulmonary blast injury. *J Trauma* 1998;45:35-38.
54. Bridges EJ. Blast injuries: From triage to critical care. *Crit Care Nursing Clin North Am* 2006;18:333-348.
55. Pizov R, Oppenheim-Eden A, Matot I, et al. Blast lung injury from an explosion on a civilian bus. *Chest* 1999;115:165-172.
56. Mackenzie IMJ, Tunnicliffe B. Blast injuries to the lung: Epidemiology and management. *Philos Trans R Soc Lond B Biol Sci* 2011;366:295-299.
57. Lavery GG, Lowry KG. Management of blast injuries and shock lung. *Curr Opin Anaesthesiol* 2004;17:151-157.
58. Schultz JM, Trunkey DD. Blunt cardiac injury. *Crit Care Clin* 2004;20:57-70.
59. Elie M-C. Blunt cardiac injury. *Mt Sinai J Med* 2006;73:542–552.
60. Guy RJ, Kirkman E, Watkins PE, et al. Physiologic responses to primary blast. *J Trauma* 1998;45:983-987.
61. Ohnishi M, Kirkman E, Guy RJ, et al. Reflex nature of the cardiorespiratory response to primary thoracic blast injury in the anaesthetised rat. *Experimental Physiology* 2001;86:357-364.
62. Irwin RJ, Lerner MR, Bealer JF, et al. Shock after blast wave injury is caused by a vagally mediated reflex. *J Trauma* 1999;47:105-110.
63. Weekes AJ, Hwang J, Ghali S. Focused cardiac ultrasonography in the emergent patient. *Ultrasound Clinics* 2014;9: 143-171.

64. Bernardin B, Troquet J-M. Initial management and resuscitation of severe chest trauma. *Emerg Med Clin North Am* 2012;30:377-400.
65. Pasquale M, Fabian TC. Practice management guidelines for trauma from the Eastern Association for the Surgery of Trauma. *J Trauma* 1998;44:941-957.
66. Clancy K, Velopulos C, Bilaniuk JW, et al. Screening for blunt cardiac injury. *J Trauma Acute Care Surg* 2012;73:S301-S306.
67. Mathew WE. Notes on the effects produced by a submarine mine explosion. *J R Nav Med Serv* 1917;4:108-109.
68. Golan R, Soffer D, Givon A, et al. The ins and outs of terrorist bus explosions: Injury profiles of on-board explosions versus explosions occurring adjacent to a bus. *Injury* 2014;45:39-43.
69. Owers C, Morgan JL, Garner JP. Abdominal trauma in primary blast injury. *Br J Surg* 2011;98:168-179.
70. Lance RM, Capehart B, Kadro O, et al. Human injury criteria for underwater blasts. *PloS One* 2015;10:e0143485.
71. Singleton JAG, Gibb IE, Bull AMJ, et al. Primary blast lung injury prevalence and fatal injuries from explosions. *J Trauma Acute Care Surg* 2013;75:S269-S274.
72. Wani I, Parray FQ, Sheikh T, et al. Spectrum of abdominal organ injury in a primary blast type. *World J Emerg Surg* 2009;4:46.
73. Shamir D, Ardekian L, Peled M. Blowout fracture of the orbit as a result of blast injury: Case report of a unique entity. *J Oral Maxillofac Surg* 2008;66: 1496-1498.
74. Remenschneider AK, Lookabaugh S, Aliphas A, et al. Otologic outcomes after blast injury: The Boston Marathon experience. *Otol Neurotol* 2014;35:1825-1834.
75. Dougherty AL, MacGregor AJ, Han PP, et al. Blast-related ear injuries among U.S. military personnel. *J Rehab Res Dev* 2013;50:893-904.
76. Garth RJ. Blast injury of the ear: An overview and guide to management. *Injury* 1995;26:363-366.
77. Darley DS, Kellman RM. Otologic considerations of blast injury. *Disaster Med Public Health Prep* 2010;4:145-152.
78. Sridhara SK, Rivera A, Littlefield P. Tympanoplasty for blast-induced perforations: The Walter Reed experience. *Otolaryngol Head Neck Surg* 2013;148: 103-107.
79. Lou Z-C, Lou Z-H, Zhang Q-P. Traumatic tympanic membrane perforations: A study of etiology and factors affecting outcome. *Am J Otolaryngol* 2012;33:549-555.
80. Seaman RW, Newell RC. Another etiology of middle ear cholesteatoma. *Arch Otolaryngol* 1971;94:440-442.
81. Kronenberg J, Ben-Shoshan J, Modan M, et al. Blast injury and cholesteatoma. *Am J Otol* 1988;9:127-130.
82. Hull JB, Cooper GJ. Pattern and mechanism of traumatic amputation by explosive blast. *J Trauma* 1996;40 (3 suppl):S198-S205.
83. Patel HDL, Dryden S, Gupta A, et al. Pattern and mechanism of traumatic limb amputations after explosive blast: Experience from the 07/07/05 London terrorist bombings. *J Trauma Acute Care Surg* 2012;73:276-281.
84. Shuker ST. The effect of a blast on the mandible and teeth: Transverse fractures and their management. *Br J Oral Maxillofacial Surg* 2008;46:547-551.
85. Brown KV, Murray CK, Clasper JC. Infectious complications of combat-related mangled extremity injuries in the British military. *J Trauma* 2010;69(suppl):S109-S115.
86. Gordon W, Kuhn K, Staeheli G, et al. Challenges in definitive fracture management of blast injuries. *Curr Rev Musculoskelet Med* 2015;8:290-297.
87. Bombings: Injury Patterns and Care. Blast Injuries Seminar Curriculum Guide, December 2006. Available at www.bt.cdc.gov/masscasualties/word/blast_curriculum_3h.doc. Accessed Dec. 15, 2015.
88. Mulligan D, Levy L, Rokusek C. Preparation for terrorist threats: Explosive devices. *Clin Pediatr Emerg Med* 2009;10:140-143.
89. Yazbeck-Karam VG, Aouad MT, Kaddoum RN, et al. Methemoglobinemia after a blast injury. *Anesthesiology* 2004;100:448-449.
90. Koenig KL. Preparedness for terrorism: Managing nuclear, biological and chemical threats. *Ann Acad Med Singapore* 2009;38:1026-1030.
91. Salter CA. Psychological effects of nuclear and radiological warfare. *Mil Med* 2001;166(12 Suppl):17-18.
92. Bedwell P, Mortimer K, Wellings J, et al. An assessment of the doses received by members of the public in Japan following the nuclear accident at Fukushima Daiichi nuclear power plant. *J Radiol Prot* 2015;35:869-890.
93. 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;67:210-211.
94. 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;357:830-831.

CME/CE Questions

1. Which characteristic of an explosion environment is most likely to raise the peak overpressure of a blast, and, thus, give a greater potential for injury?
   A. Greater ambient air temperature
   B. Greater enclosure around blast space
   C. Greater barometric pressure
   D. Lower humidity index
2. The Hopkinson's scaling law states that as the distance from the explosion doubles, the peak overpressure experienced by the patient decreases by what factor of the original?

TRAUMA REPORTS

CME/CE Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

A. 1/2
B. 1/4
C. 1/6
D. 1/8

3. An underwater blast increases the risk of injury due to what mechanism?
   A. Incompressible nature of water
   B. Chemical reaction between the explosive and water
   C. Lower boiling point
   D. Risk of hypothermia
4. All of the following are typically characteristic of a high-order explosive *except*:
   A. deflagration.
   B. supersonic blast overpressure.
   C. primary blast injury.
   D. virtually instant chemical conversion.
5. Acute gas embolism occurs when air enters the pulmonary capillary vasculature of the lung. In terms of blast injury, this is an example of:
   A. spalling.
   B. implosion.
   C. "shimmy effect."
   D. brisance.
6. The blast injury classification that results in the victim being physically displaced is:
   A. primary.
   B. secondary.
   C. tertiary.
   D. quaternary.
7. Zuckerman's classification of blast injury from primary to quaternary has been used since the 1940s. Some authors describe a "quinary" blast injury. The best example of this is:
   A. shrapnel lodged in the soft tissue.
   B. delayed hypersensitivity reaction.
   C. tympanic membrane perforation.
   D. asphyxiation when all available oxygen is consumed in combustion.
8. "Walking wounded" are best described as victims who:
   A. were walking within the blast radius at the time of detonation.
   B. have extreme injuries, despite being able to ambulate.
   C. often self-present to healthcare facilities, bypassing on-scene triage.
   D. ambulate only with EMS assistance.
9. The "signature" injury of the military operations of the Middle East in the 21st century is:
   A. blast lung injury.
   B. traumatic amputation.
   C. tympanic membrane perforation.
   D. traumatic brain injury.
10. A proposed means of classifying blast lung injury involves which measure?
    A. PF ratio
    B. Maximum PEEP requirement
    C. Diameter of "butterfly" infiltration on chest radiograph
    D. Time to requiring intubation

We need your help!

Please take a few moments to provide your input in our brief two-question survey:

https://www.surveymonkey.com/r/W2S22XB

Your responses will help in planning future issues. Thank you for your feedback!

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This CME/CE activity is intended for emergency, family, osteopathic, trauma, surgical, and general practice physicians and nurses who have contact with trauma patients. It is in effect for 36 months from the date of publication.

Trauma Reports

2016 Reader Survey

In an effort to learn more about the professionals who read *Trauma Reports,* we are conducting this reader survey. The results will be used to enhance the content and format of *Trauma Reports.*

Instructions: Fill in the appropriate answers. Please write in answers to the open-ended questions in the space provided. Please insert this survey in the provided envelope along with your continuing education evaluation, fax it to 678-974-5419, or fill out online at: https://www.surveymonkey.com/r/TR_2016_survey. The deadline is **July 1, 2016.**

1. Are the articles in *Trauma Reports* written about issues of importance and concern to you?

- O A. Always
- O B. Most of the time
- O C. Some of the time
- O D. Rarely
- O E. Never

2. How would you rate your overall satisfaction with your job?

- O A. Very satisfied
- O B. Somewhat satisfied
- O C. Somewhat dissatisfied
- O D. Very dissatisfied

3. What are you most dissatisfied with in your job?

- O A. staffing
- O B. heavy workload
- O C. low morale in your department or facility
- O D. impact of cost-cutting on quality of care
- O E. other _____

Questions 4-9 ask about coverage of various topics in *Trauma Reports.* Please mark your answers in the following manner:

A. very useful B. fairly useful C. not very useful D. not at all useful

4. Damage Control Resuscitation (July/Aug 2015)	O A	O B	O C	O D
5. Pediatric Head Injury (Sept/Oct 2015)	O A	O B	O C	O D
6. Man-made Disaster: In-hospital Management (Nov/Dec 2015)	O A	O B	O C	O D
7. Ultrasound for Trauma (Jan/Feb 2016)	O A	O B	O C	O D
8. Electrical and Lightning Injuries (Mar/Apr 2016)	O A	O B	O C	O D
9. Blast Injuries (May/June 2016)	O A	O B	O C	O D

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- O A. I am a paid subscriber (proceed to question 11)
- O B. I receive it as a supplement to another publication (skip to question 12)

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If not, why? _____

12. How would you describe your satisfaction with your subscription to *TR*?

- O A. Very satisfied
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13. What is your title?

- O A. Practicing emergency medicine physician
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- O D. Physician assistant
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14. On average, how much time do you spend reading each issue of *TR*?

- O A. fewer than 30 minutes
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17. How large is your hospital?

- O A. fewer than 100 beds
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Please rate your level of satisfaction with the following items.
A. excellent B. good C. fair D. poor

18. Quality of newsletter	○ A	○ B	○ C	○ D
19. Article selections	○ A	○ B	○ C	○ D
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27. To what other publications or information sources do you subscribe?

28. Has reading *Trauma Reports* changed your clinical practice? If yes, how?

29. What do you like most about *Trauma Reports*?

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31. What specific topics would you like to see addressed in *Trauma Reports*?

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