Primary blast injury: Update on diagnosis and treatment

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Background: **Injuries from combat and terrorist explosions are increasing worldwide. As such, physicians can expect to treat more patients with complex and unique patterns of injury produced not only by fragments and blunt trauma, but also by high-pressure air expanding from the detonation center.**

Discussion: **Tissue damage from the blast wave or primary blast injury can be an important cause of occult trauma to the ocular, aural, pulmonary, cardiovascular, musculoskeletal, and neurologic systems. Awareness of the extensive corporal effects of the blast wave is an essential prerequisite to diagnosis.**

Summary: **This article focuses on the incidence, risk factors, diagnosis, management, and screening for primary blast injury. (Crit Care Med 2008; 36:[Suppl.]:S311–S317)**

KEY WORDS: primary blast injury; explosion; treatment; diagnosis

The use of explosive weaponry in military operations and in terrorist attacks on civilian targets has become all too common, placing the burden of care for blast injury casualties on military surgeons and civilian physicians alike. This article briefly discusses the mechanics and physiologies of primary blast injury and then focuses on diagnosis and management.

In the ongoing conflict in Iraq, the improvised explosive devise (IED) has been the most common cause of wounding in American soldiers and Iraqi civilians. As a result of the tactical deployment of the devices and the mechanics of blast waves, few primary blast injuries have been seen. Thus far, in Israel, where IEDs are more frequently exploded within closed spaces such as a bus, primary blast injuries are more frequent (1, 2). With the advent of new enhanced blast weapons, however, more primary blast injuries can be anticipated by both military and civilian physicians (3–5), and it is incumbent on us to be aware of the physiology and mechanics of this frequently devastating injury.

Pathophysiology

Explosions have the potential to inflict many different kinds of injuries on multiple victims. Injuries from explosions are traditionally classified into: a) primary blast injuries, that is, injuries due solely to the blast wave; b) secondary blast or explosive injury, which is primarily ballistic trauma resulting from fragmentation wounds from the explosive device or the environment; c) tertiary blast or explosive injury, which is the result of displacement of the victim or environmental structures, is largely blunt traumatic injuries; and d) quaternary explosive injuries or burns, toxins, and radiologic contamination (6).

An explosion creates a nearly instantaneous expansion of gas creating a shock wave, known as the *blast wave*, which travels away from the epicenter of the detonation at supersonic speed of 3,000 to 8,000 m/sec. The blast wave rapidly loses its pressure and velocity with distance and time (Fig. 1). The blast front has a shattering ability known as *brisance*, unless the weapon is detonated within a closed space in which case the blast overpressure is magnified by reflection off solid structures (7, 8). Conventional bombs or IEDs detonated in open spaces generally create few if any primary blast injuries as a result of the rapid decay of the blast wave. People who are close enough to detonation to experience primary blast injury generally die immediately from massive injuries. Enhanced blast weapons for air delivery, guided missiles, and handheld weaponry are readily available and have been used on the battlefields of Afghanistan, Chechnya, and possibly in Iraq. One needs only to peruse the pages of *Jane's Infantry Weapons* or to go online to see the RPO Shmel-M or Mikor MGL-140-MEI (http://jiw.janes.com/public/jiw/index.shtml). These weapons are designed to enhance the blast wave and also may have an added thermal effect. An enhanced blast weapon creates a lower peak pressure but a longer sustained time of blast overpressure that, with the additive effect of thermal injury, allows a greater time for damage to "soft" structures and to personnel.

The blast wave enters the body creating two types of energy, stress waves and shear waves. Stress waves are longitudinal pressure forces that move at supersonic speeds and create a "spalling" effect at air–tissue interfaces, much like boiling water, resulting in severe microvascular damage and tissue disruption (6–9). Shear waves are transverse waves that cause asynchronous movement of tissue and possible disruption of attachments (6, 9). Therefore, the organs most likely affected by primary blast injury are the ears, lungs, and colon or gas-filled organs with the damage originating at the tissue–gas interface. Ruptured tympanic membrane, ossicular disruption, alveolar hemorrhage, cerebral, coronary, retinal and lingual air emboli, ruptured viscus with pneumoperitoneum, and vagally mediated bradycardia, apnea, and hypotension are among the early signs of severe primary blast injury (10–16). Traumatic amputation is a marker of severe primary blast injury from which 50% of patients will die despite aggressive therapy (17, 18). Traumatic brain injury is also noted to occur and may be related to the development of posttraumatic stress disorder (PTSD) (10), whereas solid and fluid-filled organs seem to be relatively protected from injury (6).

The absence of perforation of the tympanic membrane and lack of petechiae in

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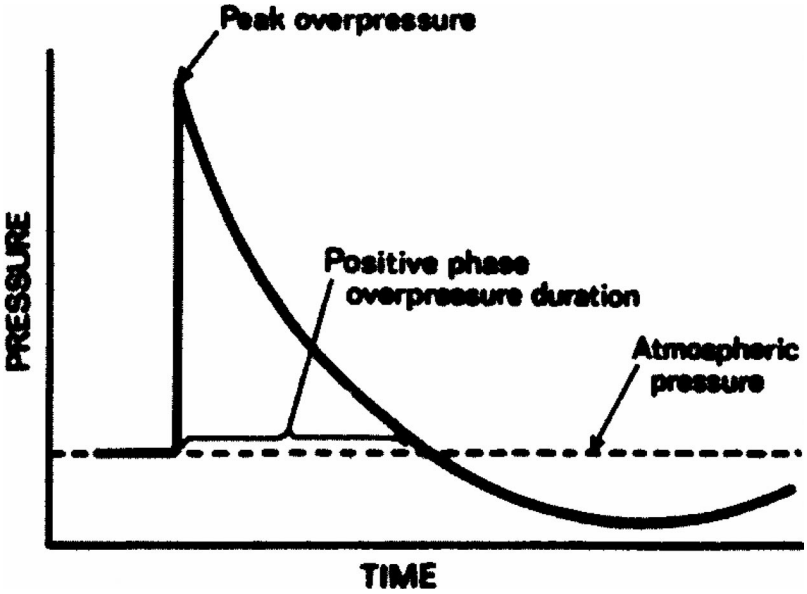

Figure 1. Idealized blast over pressure waveform.

the oropharynx have been said to mediate against primary blast injury of internal organs in the majority of cases (19), although this has not been born out by the Israeli experience. The Israeli literature clearly states that tympanic membrane rupture is found in only 60% of patients with clinically significant injuries with perforations in only 30% (9). However, when one considers the mechanism of the injury, and the presence of oral petechiae and perforated tympanic membrane together, this can be a valuable triage tool to alert the physician to keep a patient for further observation.

In the remainder of this article, we address the diagnosis and management of primary blast injury of each of the major organ systems primarily affected by the primary blast wave; the ocular, auditory, pulmonary, cardiovascular, gastrointestinal, musculoskeletal, and neurologic systems are discussed in detail. The article concludes with recommendations for patient assessment designed to detect the clues from history, physical examination, and laboratory and radiographic results that should increase the clinician's index of suspicion for the presence of primary blast injury.

Ocular Injury

Some studies estimate that as many as 10% of all blast survivors have significant eye injuries (20). Like in other organ systems, the eye is most commonly injured by penetrating fragments. Even small projectiles that would not harm the skin may cause significant ocular damage (21). Additionally, facial fractures caused by fragments or flying debris may cause muscular entrapment or compromise of the ocular nerve (19). Corneal or eyelid burns may result from the fireball in victims in close proximity to an explosion or from conflagration in those further away from the detonation center. Wearing simple eye protection such as military issue goggles or even commercially available sunglasses decreases the risk of direct eye injury resulting from fragments or burns from an explosion.

Transfer of kinetic energy from the blast wave to the eye can result in rupture of the globe, serous retinitis, and hyphema (19). Additionally, exposure to high levels of overpressure may cause orbital fractures (22). Indirect ocular injury may also result from primary blast injury to the lungs, resulting in air emboli that travel to the ophthalmic artery as it branches off the internal carotid. Such emboli have been observed on funduscopic exam of blast-exposed animals (22). Symptoms of ocular injury include pain or irritation, altered vision, periorbital swelling, contusion, or foreign body sensation in the case of injury resulting from fragments (20). Some authors have claimed that the most common ophthalmic sign of blast overpressure injury is conjunctival hemorrhage (23). Other physical examination findings include diminished visual acuity, hyphema, globe rupture, presence of foreign body, or lid lacerations (20). A thorough physical examination should include examination of eyelids, globe, cornea, fundus, and visual acuity as well as oculomotor testing. Radiographic evaluation should be aimed at detecting intraocular foreign bodies as well as orbital fractures because 10% to 25% of orbital floor fractures are associated with globe injuries. Ophthalmology consultation should be obtained for suspected globe injuries, corneal foreign bodies or abrasions, orbital fractures, retinal detachments, hyphema, intraocular foreign bodies, corneal or eyelid burns, lid lacerations, subconjunctival hemorrhage, or head injuries that involve the orbit or may compromise vision (24).

Aural Injury

A recent retrospective study of more than 4000 U.S. casualties from Operations Iraqi Freedom and Enduring Freedom found that as many as 9% of explosion-injured patients had tympanic membrane rupture (Ritenour AE, Blackbourne LH, Kelly JF, et al., unpublished data). Other studies have reported an incidence of tympanic membrane rupture ranging between 9% and 47% in patients injured by explosion (25, 26). The design of the ear makes it the body's most sensitive pressure transducer. As such, tympanic membrane rupture is the most common primary blast injury. Although the tympanic membrane is more likely to be damaged at lower pressures (5–15 pounds per square inch) than the lungs or gastrointestinal tract (40–75 pounds per square inch) (6), the association between tympanic membrane rupture and blast lung or intestinal blast injury is inconsistent. Many factors may affect the likelihood of tympanic membrane rupture. Perpendicular orientation of the head to the blast wave is more likely to cause tympanic membrane rupture than parallel positioning to the wave (27). The presence of cerumen in the ear canal may protect the tympanic membrane while cerumen abutting the tympanic membrane will serve as a ramrod, making rupture more likely (28). Closed space explosion (1), prior injury, previous infections, advanced age (28), and insufficient pneumatization all increase an individual's chances of tympanic membrane rupture (29). Simple forms of hearing protection like earplugs or even headphones may decrease the peak pressure reaching the tympanic membrane to below the threshold needed to cause rupture (30).

The combined effect of these factors is to make tympanic membrane rupture an unreliable marker of lethal blast injury.

The most common symptoms of auditory injury are hearing loss, tinnitus, pain, and dizziness (31). All explosion victims, even those without symptoms, should be evaluated with an otoscopic examination not as a means of screening for other primary blast injuries, but simply to diagnose tympanic membrane rupture and ensure proper evaluation and treatment. Auditory injury may be easily overlooked in the most severely injured patients; therefore, clinicians should make otoscopic examination a routine part of the initial evaluation of explosion-injured patients. The otolaryngology service should be consulted to manage all tympanic membrane perforations to allow for proper evaluation and débridement of the middle ear to prevent cholesteatoma as a late complication from displacement of keratinizing squamous epithelium. Conductive hearing loss can also occur from dislocation of the ossicular chain as well as rupture of the oval or round windows. In time, perilymphatic fistulas can result.

Spontaneous healing rates after tympanic membrane perforation range from 78% to 88% (31). Studies have demonstrated an inverse relationship between size of perforation and healing with spontaneous healing in 92% of low-grade perforations and only 20% of high-grade ruptures. Location of the perforation also affects healing rates. Inferior perforations, reportedly, have the highest spontaneous healing rate with central kidney-shaped perforations having the lowest. Necessity for operative management will be determined by the otolaryngology service, but these findings have led some authors to suggest that early operative intervention be considered in patients with large perforations that are unlikely to heal (32).

Some studies have reported a high (30%) incidence of permanent high frequency hearing loss (>30 dB at 4,000 and 8,000 Hz) 1 yr after injury (31). For this reason, we recommend that all explosion-injured patients requiring inpatient admission undergo audiometric testing when clinically possible. Unidentified hearing loss may contribute to patient confusion and frustration and should be identified early to allow timely management and appropriate follow-up.

Blast Lung Injury

Perhaps the most discussed and feared primary blast injury is blast lung because it is the most common fatal injury among initial survivors of explosions. The blast wave causes thoracic acceleration and propagates through lung parenchyma; complex interactions of kinetic energy transfer occur and cause tissue disruption at the capillary–alveolar interface. This may result in minor or massive parenchymal hemorrhage, pulmonary edema, pneumothorax, or air embolism from alveolovenous fistulas. The reflection of the blast wave off mediastinal structures causes the characteristic perihilar or "batwing" infiltrates seen on chest x-ray or computed tomography scan in patients with blast lung injury (33). Like other primary blast injuries, it is more common in victims in close proximity to large explosions in enclosed spaces (1). Burned patients also have a higher incidence of pulmonary blast injury than explosion-injured patients without burns (34). Although at least one study has suggested that body armor is associated with an increased incidence of blast lung injury, it seems more likely that body armor prevents penetrating injuries, thus making victims who would have been killed by penetrating thoracic injury more apt to survive to seek medical care for blast lung injury. The incidence of pulmonary blast injury as reported in the literature is highly variable, ranging from 3% to 14% depending on the study (1, 26).

Clinical diagnosis of blast lung injury is based on the presence of respiratory distress, hypoxia, and "butterfly" or batwing infiltrates. Symptoms and signs include tachypnea, dyspnea, cyanosis, and hemoptysis. On physical examination, the patient may have diminished breath sounds and crepitance resulting from subcutaneous air. Hypoxia (oxygen saturation <90% on room air) is present and reaches its nadir within the first 24 hrs. Avidan et al. reported that chest x-ray findings of the batwing (bilateral central) lung infiltrates were the most common radiographic finding in their series and were usually present on admission. The central location of infiltrates may help distinguish blast lung injury from blunt etiologies of pulmonary contusion, which usually causes peripheral lesions. Additionally, radiographs may reveal pneumothorax or pneumomediastinum (33).

The classic teachings in management of blast lung injury are to avoid positive pressure ventilation, if possible, minimize positive end-expiratory pressure ventilation, and use judicious fluid resuscitation strategies. In one series with a low mortality of 3%, mechanical ventilation was required in 76% of patients, and several patients needed positive end-expiratory pressure >10 cm H_2O to allow sufficient oxygenation (33). Pressure-limited, volume-controlled ventilation with permissive hypercapnia has been advocated in patients sustaining blast lung to minimize mean airway pressure and the chance of air embolism as well as to reduce the risk of further pulmonary trauma (35). High-frequency ventilation and nitric oxide inhalation have also been successfully used (33). Other sophisticated modes such as airway pressure release ventilation, jet ventilation, oscillatory ventilation, and independent lung ventilation may be useful. When all else fails, the physician may resort to salvage methods like extracorporeal membrane oxygenation (Michael Stein MD, Chairman, Israel Trauma Society, Director of Trauma, The Rabin Medical Center, Beilinson Campus, Tel-Aviv University, Tel-Aviv, Israel, personal communication).

In their series, Avidan et al. reported no cases of clinical deterioration with need for mechanical ventilation in patients who were more than 2 hrs postinjury. These authors reject the concept that blast lung injury may cause respiratory failure after a latent period and concluded that prolonged observation of asymptomatic patients was not necessary (33). Tympanic membrane rupture is an inconsistent finding in patients with blast lung injury (36) with a sensitivity of 29% (37). In accordance with the recommendations of Avidan, Ashkenazi, and Leibovici, we do not suggest that otoscopic examination be used as a screening tool for blast lung injury (9, 33, 36). Instead, we advocate patient risk stratification based on history, active questioning about symptomatology, and careful physical examination followed by chest radiograph if symptoms or physical examination findings are consistent with blast lung injury. Blast lung injury should be diagnosed when a patient has dyspnea or hemoptysis, hypoxia and pulmonary infiltrates, or pneumothorax or pneumomediastinum without penetrating or blunt thoracic injury.

Intestinal Blast Injury

Primary blast injury to the gastrointestinal tract is rare with an incidence of 0.3% to 0.6% (1, 36) among survivors and seldom without other evidence of primary blast injury. It is more likely to occur in victims injured in underwater explosions than in air blasts. However, when intestinal blast injury occurs, the rapid compression and subsequent expansion of air in gas-filled organs may cause contusions, intramural hemorrhages, and perforation. Also, shear stress may tear mesenteric vessels (38). Solid organ injury, including laceration or rupture of the liver, spleen, or kidneys, occurs less frequently (39). All of these injuries are more likely to occur as a result of tertiary blast injury when a victim is propelled into an object as a result of the blast wind than by the blast wave alone. However, both primary and tertiary blast injury mechanisms are possible and the resulting injuries are treated the same way regardless of mechanism (38).

Patients with primary blast injury to abdominal viscera may present with abdominal pain, nausea, vomiting, hematemesis, melena, and peritoneal signs of injury. Hemodynamic instability may also be seen in the case of mesenteric hemorrhage or solid organ injury. Peritoneal signs resulting from visceral perforation may be present immediately after injury or in a delayed fashion after devascularized bowel wall becomes ischemic and necrotic and perforates days after injury (38). Radiographic evidence of abdominal blast injury on computed tomography includes pneumoperitoneum, free intraperitoneal fluid not consistent with blood, and a "sentinel clot" seen adjacent to bowel wall or mesentery. Pneumoperitoneum alone may be a nonspecific sign only associated with bowel perforation in 44% of patients (40).

Mesenteric or mural hematoma in hemodynamically stable patients without peritoneal signs may be managed with bowel rest, nasogastric tube decompression, and resuscitation. However, massive hemorrhage or obvious hollow viscus perforation should be treated with laparotomy for hemostasis and control of spillage of enteric contents (38). The ileocecal region seems to be particularly susceptible to primary blast injury and should be carefully inspected during exploratory laparotomy (13).

Cardiovascular Effects of Blast

A less frequently discussed aspect of primary blast injury is the hemodynamic effect of the blast wave. The heart may be damaged by contusion from thoracic acceleration or from air embolization to the coronary arteries (38). Additionally, the blast wave can induce hemodynamic effects apart from those resulting in direct thoracic injury. Animal studies have demonstrated a triad of immediate bradycardia, hypotension, and apnea (41) that is a partially vagally mediated response to thoracic blast (42). Rat studies have shown that the consistent finding of bradycardia is immediate and severe but short-lived with recovery to baseline in 15 mins. Higher-intensity blasts induce a more profound bradycardia (15). Of particular clinical relevance, another study found that normal hemorrhage-induced tachycardia was absent and that bradycardia was augmented in rats exposed to thoracic blast injury. Additionally, investigators found that blood pressure began to decline as soon as hemorrhage began (43). The most common blast-induced arrhythmias, in addition to bradycardia, are premature ventricular contractions and asystole (15). Hypotension has been associated with low cardiac index and stroke volume but normal systemic vascular resistance. In rats, the cardiac index falls within seconds of the explosion but recovers to control values after 45 mins (15). Interestingly, blast-injured patients have higher mean concentrations of inflammatory mediators in the 5 days after injury when compared with severely injured patients without blast injury (44).

The clinical application of these findings is unclear, except that a physician should be aware that hemorrhaging explosion-injured patients may not have the expected compensatory tachycardia and may become hypotensive without rapid resuscitation. Atropine may be a useful adjunct in patients with blast-induced bradycardia who do not respond as predicted to resuscitation efforts. More research is certainly needed in this area to determine the effects of hypotensive resuscitation on explosion-injured patients with penetrating and primary blast injuries as well as to investigate pharmacologic means of attentuating the bradycardia and hypotension caused by the blast wave.

Traumatic Amputations

Traumatic amputations from primary blast injury are uncommon and controversial as to whether the blast wave alone is the cause. The transmission of energy from the blast wave to the tissues of the extremity may cause fracture resulting from axial stress, usually through the diaphysis rather than the joint (39). Soft tissue injury is minimal with damage confined to the level of amputation (10). Principles of management of the combat-injured extremity such as early tourniquet use should be applied in the care of these patients regardless of precise mechanism of injury. Clinicians should have a high clinical suspicion for occult explosive injuries to the central nervous system, thorax, and abdomen in these patients and should search for them in the patient who does not respond appropriately to resuscitation once control of extremity hemorrhage is achieved.

Traumatic Brain Injury

Without a doubt, one of the most controversial questions about primary blast is its effect on the central nervous system. Nonspecific symptoms of organ dysfunction previously attributed to "shell shock" or PTSD are now being reexamined to determine whether these may be, at least in part, a result of cerebral primary blast injury. Investigators have postulated that kinetic energy of the blast wave transferred to the central nervous systems causes shearing, resulting in diffuse or focal axonal injury and initiating secondary injury mechanisms that may result in both acute and delayed symptoms (45). Primary blast injury can also result in cranial fractures around air-filled sinuses (38) and focal neurologic deficits as a result of air embolism (46, 47). At least one source has suggested that because of the increased use of body armor among military personnel, central nervous system injury should be increasingly attributed to primary blast injury (46, 47). The implication is that secondary or tertiary mechanisms are unlikely in a victim wearing a helmet at the time of injury. We have not found any data to support this assumption and believe that blunt trauma mechanisms still play a large role in traumatic brain injury even in blast-injured patients who were wearing protective equipment.

Data from both animal and human studies support the concept of blast-

induced brain injury. Rats subjected to whole body or chest blast injury demonstrated cognitive impairment and biochemical changes, including oxidative stress in the hippocampus that correlated with blast injury severity (45). Animal studies suggest that the overpressure wave is transferred to the central nervous system, causing diffuse axonal injury (48). In humans, electroencephalographic irregularities consistent with cortical dysfunction have been observed days after blast injury (39). Veterans with a history of blast concussion have been found to have electroencephalographic abnormalities and attentional dysfunction (46). Autopsies of blast victims have demonstrated diffuse punctate hemorrhages and disintegration of Nissl substance (chromatolysis) in both the brain and spinal cord. Focal neuronal atrophy and axonal degeneration have also been observed (10). Yehuda has suggested linking memory dysfunction and the neuroendocrine alteration of PTSD with the neuroanatomic findings of reduced hippocampal volume in male combat veterans (49). A single institutional study of 1303 patients injured by explosive munitions found that after rigorous screening 51% had evidence of primary blast injury and that significantly more primary blast-injured than nonblast-injured patients (30% versus 4%) had persistent central nervous systems disorders. The increased incidence of PTSD in the blast compared with the nonblast group suggested blast may be the etiology of symptoms usually attributed to PTSD and that the signs and symptoms of postconcussion syndrome overlap with those of PTSD (44).

Symptoms of central nervous system injury may be psychologic, such as excitability, irrationality, retrograde amnesia, apathy, lethargy, poor concentration, insomnia, psychomotor agitation, depression, anxiety (44), or physical such as fatigue, headache, back and diffuse pains, vertigo, transient paralysis, and "heavy" feeling extremities (10). The history should include specific questioning about these symptoms. Physical examination should include a thorough neurologic examination to include checking for positive Romberg's sign (44) as well as funduscopy to look for evidence of air emboli (38). Computed tomography scan should be used to search for evidence of blunt head injury and intracranial hemorrhage. Psychologic evaluation and cognitive testing should be performed on stabilized patients. At our institution, even asymptomatic blast-injured patients are provided with education about the effects of traumatic brain injury as well as the resources available to them. Counseling and treatment is provided to symptomatic patients during their hospitalization and asymptomatic patients are given contact information so that they may seek treatment for delayed symptoms that may manifest themselves after discharge. Medical treatment and counseling should focus on managing an individual's symptoms to optimize the functional outcome and quality of life.

Patient Risk Stratification

When the situation allows, a focused history is of great assistance to the physician in patient risk stratification for primary blast injury (Fig. 2). The ideal (although rarely obtainable) history should include:

1. Explosive device details: type and weight of explosive, improvised versus commercially available, suicide bomber, time of detonation
2. Geography: device location, open vs. closed space detonation, surrounding structures (urban vs. rural setting)
3. Victim: distance of the victim from the detonation center, specific location of the victim with orientation of body in relation to explosive and surrounding structures, personal protective equipment
4. Status of other casualties: cause of any on-scene deaths, primary blast injury in other surviving victims

These type of data, in order of importance, in a patient's history can determine the likelihood of primary blast injury. Details about the explosive device such as type and weight can be helpful. A larger, more powerful explosive would be more likely to produce sustained overpressure capable of causing primary blast injury at a distance from the detonation center. Commercially available explosives can be expected to cause more predictable injury patterns than IEDs that are highly variable in casings and types and amounts of explosive. A suicide bomb is usually detonated in close proximity to a large group of people. The bomber's fragmented body can form projectiles that produce injury and infection in the victims. Time of detonation will allow determination of time of injury and helps place a patient's current hemodynamic status in context.

Blast wave enhancement occurs when it reflects off fixed structures and reverberates within a closed structure. These complex interactions of the blast wave and its environment increase the likelihood of primary blast injury in closed spaces. For this reason, determination of

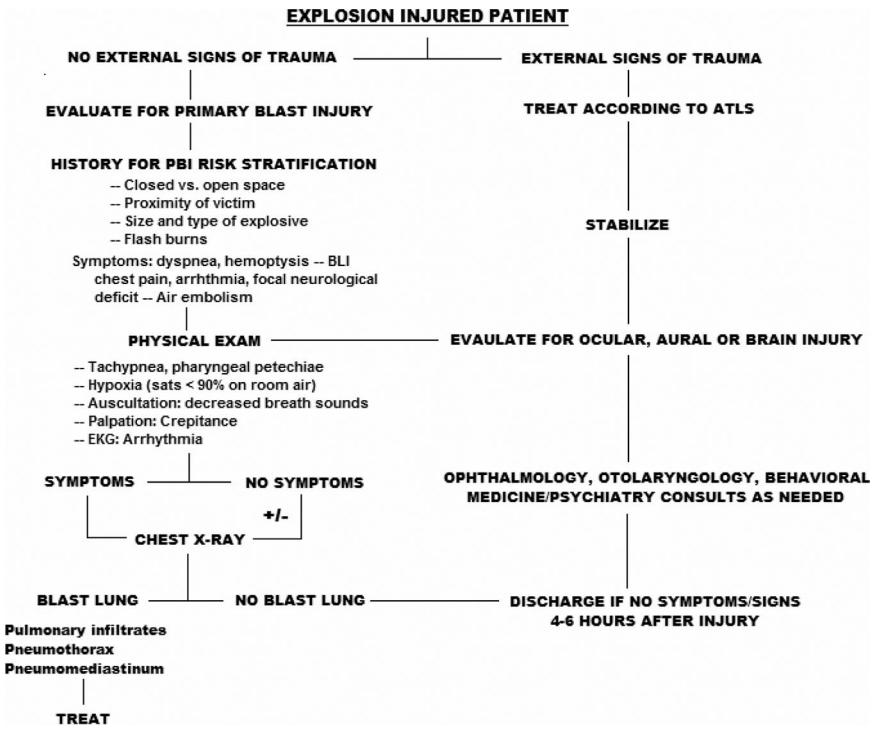

Figure 2. Primary blast injury algorithm. *PBI*, primary blast injury; *BLI*, blast lung injury.

explosion location (closed vs. open space) may be helpful. Victims in closer proximity to explosions are more likely to sustain primary blast injury because they will be exposed to larger overpressure than those further away. Victim orientation may help predict injury pattern. For example, the ear or lung closest to the explosion would be expected to be injured before the contralateral one. However, this may not be true of a patient standing near a wall or in a closed space.

Finally, when no detailed history is obtainable, burns have been associated with increased rates of primary blast injury. Thermal injury from an explosion may result from exposure to the explosion fireball or from conflagration. Patients with flash burns from exposure to the fireball were likely to have been close enough to have been injured by overpressure exposure. Therefore, clinicians should have a higher index of suspicion for primary blast injury in patients sustaining burns.

CONCLUSION

Unfortunately, injuries from explosions are increasing in frequency worldwide. It is incumbent on civilian and military physicians alike to have a working knowledge of the variety of injuries that may be caused by explosives. Physicians should treat patients according to current trauma principles, because penetrating and blunt trauma mechanisms remain the most common causes of injury after explosion. Using a focused history and physical examination and in conjunction with basic radiographic studies, physicians may be confident in their ability to diagnose and treat primary blast injuries. Targeted physical examination and radiographic findings will allow accurate and timely diagnosis of blast injury to the lung and gastrointestinal tract. We recommend routine ocular examination, otoscopic examination, audiogram, and traumatic brain injury screening in all explosion-injured inpatients.

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