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## Introduction

Explosive devices have become a major weapon in current armed conflicts, antipersonnel landmines, and terrorist bombing. This has changed the trends of prevalence of the wounding mechanisms over the past several decades. Shrapnel injuries are now more common than bullet injuries in wars between armies and can cause up to 80% of casualties due to the preponderance of blasts and explosive devices in conflicts [1]. In addition, these explosive weapon systems have a greater distance range of injury compared to the close-range firearm systems [1]. The detonated explosives generate high winds and propel debris causing conventional blunt and penetrating trauma. However, explosive devices do not only cause injury through fragmentation which has similar wound ballistics as gunshot injuries discussed in the previous chapter. Explosive systems can cause a special set of lesions that have

particular pathology, their own diagnostic challenges, and specific management requirements known as primary blast injuries. This chapter discusses the biodynamics of blasts and their mechanisms of injury with an overview of the current understanding of primary blast injuries and their effects primarily on the respiratory, gastrointestinal, and auditory system.

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## Blast Physics

An explosive is a substance, solid or liquid, that once detonates will chemically convert instantaneously into gas through an intense exothermic reaction releasing large amounts of energy [2]. The gas expands radially outward from the location of explosion at supersonic speeds (usually greater than 5000 m/s) in a process termed detonation [3]. This expanding gas causes an instantaneous acute rise in pressure creating a supersonic wave called the blast wave or shock wave. The blast wave displaces the surrounding medium, be it air or water, generating winds of enormous velocity called blast winds that propel people and objects [4]. The displaced medium in front of the blast wave is compressed which heats and accelerates its molecules creating a pressure that exceeds atmospheric pressure called blast overpressure (BOP) [5]. The air molecules are compressed to such a density that the blast wave itself acts like a solid hitting the victim [6]. The

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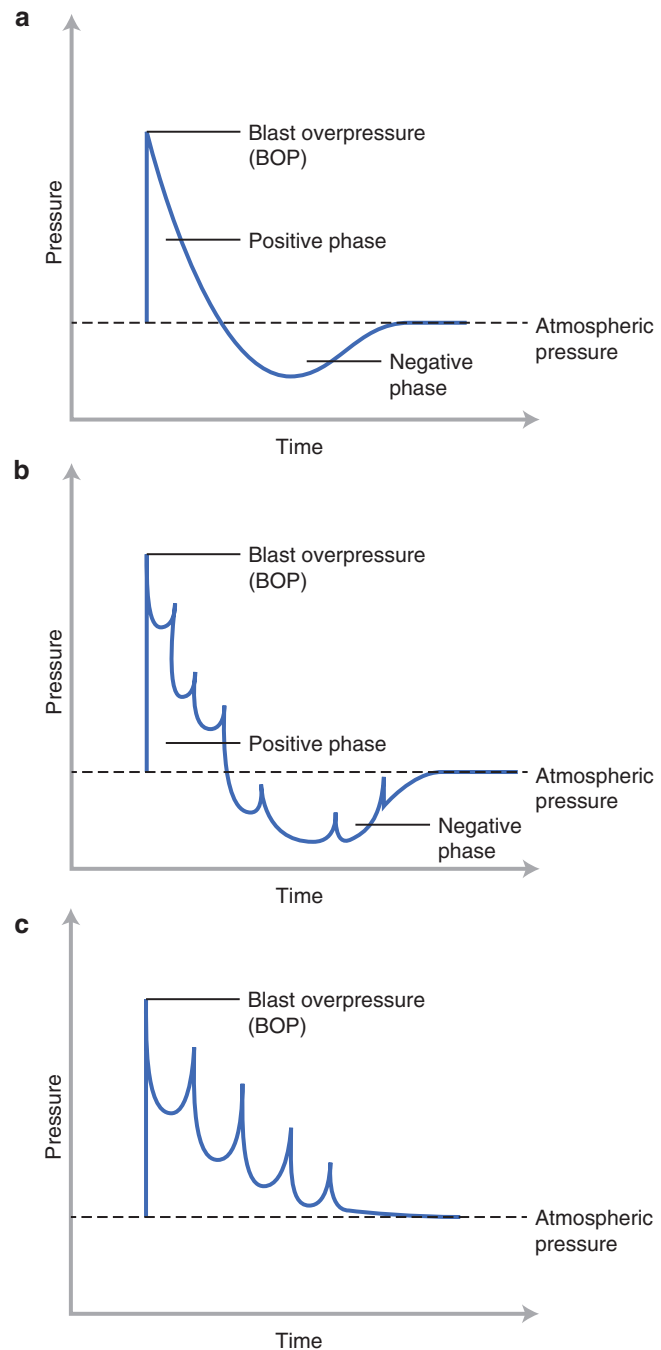
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blast pressure dissipates over time and space. These changes in pressure due to the blast wave vary depending on whether the detonation took place in open air or closed space. The classic Friedlander wave describes the characteristic pressure changes over time of a blast wave outdoors, the so-called free-field wave (Fig. 2.1a). It

is an idealized blast overpressure waveform, with an acute instantaneous rise in pressure to a peak overpressure and then dissipation exponentially over time until back to atmospheric pressure in what is called the positive blast phase. This peak overpressure is the maximum pressure reached and is commonly referred to as BOP. It decreases

**Fig. 2.1** (a) Free-field wave—open-space wave. Classic Friedlander wave: An idealized blast overpressure waveform. (b) Simple free-field wave. A more realistic waveform. (c) Enclosed-space waveform. Blast overpressure is amplified, and positive pressure wave is prolonged



so rapidly (inversely proportional to the cube of the distance) as the distance from the detonation center increases, persons must be within tens of meters close to the epicenter to sustain a primary blast injury [2]. However, pressure keeps decreasing to subatmospheric pressures in what is called the negative-pressure suction wave before returning to ambient pressure. A more realistic wave-form of a simple free-field wave has both positive and negative phases roughly very similar to the Friedlander but with multiple peaks and troughs, very close in amplitudes, that represent vibration or reflection of the surrounding surfaces, at least the ground (Fig. 2.1b). In enclosed space explosion, however, the blast overpressure is significantly amplified and the positive pressure phase is prolonged. This is due to the confinement of the pressure waves that reflect back from the multiple surrounding solid surfaces which increases their force and causes multiple pressure peaks and troughs (Fig. 2.1c) [7]. This understanding of blast overpressure magnitude, positive pressure phase, and propagation speed of a blast wave is fundamental for the understanding of the biologic effects and management of blast injuries.

Many factors affect the likelihood and severity of blast injuries. One important factor is the medium in which the explosion takes place. For example, water molecules do not get as compressed by the blast wave as the air molecules do. Therefore, the blast wave propagates more rapidly and dissipates more slowly in a water medium causing more injury than an explosion does in an air medium [8]. Another important factor to consider is the distance at which a person or an object is from the detonation epicenter. This distance determines how exposed the victim is to the blast overpressure [9]. The blast energy dissipates and the pressure drops inversely proportional to the distance cubed. For example, if individual A is at a distance  $d$  from the detonation and individual B is at a distance  $2d$  double that of A's, then the BOP that individual B is exposed to is  $1/8$  that individual A is exposed to. A 1-kg explosive will generate blast overpressure of 500 Kpa at the site of detonation which is fatal and drops exponentially to 20 Kpa at 3 m from the center which causes minimal injury [4]. Another

substantial factor that determines blast overpressure exposure is the surrounding solid surfaces. These surfaces reflect the pressure waves and amplify their forces, hence exposing people next to them to a higher blast overpressure compared to those away from them and at the same radius from the detonation center. It is the reason behind which closed-space explosions have the potential to cause more severe injuries and higher mortality than open-field explosion [10, 11].

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## Mechanisms of Blast Injuries

Traditionally, blast injuries have been classified into four categories according to the mechanism by which the blast wave causes these injuries. A fifth type of blast injuries has been recently suggested.

*Primary blast injuries (PBI)* are the direct effects of the interaction of different organs in the body with the pressure changes of the blast wave. These injuries are unique to higher order explosives which make most civilian physicians unfamiliar with them. The organ damage in PBI is produced by the interaction of the blast wave at the interface between tissues of different densities or the interface between tissues and trapped air. Consequently, gas-containing structures, like the lung, GI tract, and ear, are most commonly affected by PBI [12]. These types of injuries are the main focus of this chapter and are discussed in great details in the following section.

*Secondary blast injuries* occur when objects energized by the explosion strike an individual, causing either blunt or penetrating trauma (e.g., bomb fragments, shrapnel). Fragments displaced by the blast winds travel a much longer distance than that traveled by the blast overpressure. This is why secondary blast injuries can occur up to thousands of meters away from the explosion site while PBI occurs within tens of meters only [13]. Penetrating secondary blast injuries from fragmentation of the detonated weapon or the secondary fragments resulting from the explosion are a leading cause of mortality in terrorist attacks not including building collapse [14].

*Tertiary blast injuries* occur when the victim's body or body parts are displaced by the blast winds

and then tumble impacting hard surfaces. They include injuries due to the structural collapse of buildings, crush injuries, traumatic amputations, closed head injuries, blunt abdominal trauma, tissue contusions, and fractures [15, 16].

*Quaternary blast injuries* involve the types of injuries that do not fit any of the three mechanisms above. They include flash burns including burns from hot gasses or fires, methemoglobinemia [17] due to inhalation of CO, inhalation of dust, smoke or cyanide, acute septicemic melioidosis [18], and psychological sequelae.

*Quinary blast injuries:* This recently suggested that classification is based on a case series, and it involves a “hyperinflammatory state” seen in patients postblast manifested clinically as hyperpyrexia, diaphoresis, low central venous pressure, and water retention [19].

The secondary, tertiary, and quaternary injuries are similar to injuries in civilian trauma and their management is no different than nonexplosive trauma treatment protocols whether penetrating or blunt.

## Primary Blast Injuries (PBI)

As the blast overpressure reaches the individuals in proximity to the detonation epicenter, forces will be transmitted into the body causing organ damage. These forces exert their maximum concentrated effect at air-tissue interfaces. Three explosive forces that cause PBI were first described in 1950 [20]: spallation, implosion, and inertia. These forces are the components of the blast-body interaction that eventually causes tissue damage.

Spallation happens when the blast wave passes from a dense medium to a less dense medium causing the fragmentation of the dense medium into the less dense. For example, in an underwater explosion, the pressure wave passes from the water into the air causing fragmentation of the denser medium, in this case the water, into the less dense medium, in this case the air. This is manifested as an upward splash of water into the air [2, 20]. From a physiologic standpoint, a blast wave passing through the interfaces between air,

alveolar tissue, and capillary wall will cause the alveolar wall to tear and the peri-alveolar capillary endothelium to be disrupted through spalling forces [21].

Implosion happens as a result of the air in air-containing organs getting compressed during the positive phase of a blast wave. Once the blast positive phase is over, the air will re-expand releasing large amounts of kinetic energy disrupting the structure containing it [22]. This is how a blast wave causes alveolar damage after air in alveoli gets compressed during the positive pressure phase and re-expands forcefully during the negative suction phase. Combined together, the spalling forces causing peri-alveolar capillary wall damage and the implosive forces causing re-expansion of air in the alveoli will force air emboli in the blood vessels leading to one of the most fatal primary blast feared complications, arterial air embolism [21]. Another example where implosive forces cause PBI is the implosion of compressed air in facial sinuses that leads to skeletal crush injuries of the naso-orbitoethmoid complex, maxillary sinuses, and nasal bones [23].

Inertial forces cause tissue damage based on the fact that different tissues of different densities will move at different speeds in response to blast overpressure. Similarly, different component structures of an organ of varying densities will move differently and get damaged by shear forces. The lighter structural components will move with higher acceleration than the heavier components causing major stress at the boundary [24].

It is imperative in the understanding of the blast front-body interaction to discuss the two types of waves that are generated by this interaction and that propagate through the body causing internal soft-tissue injuries: the stress waves and the shear waves.

Stress waves are longitudinal pressure waves (similar to acoustic waves) with high amplitude and velocity [25]. A shock wave can be considered a special form of stress wave that travels at supersonic speeds. These waves affect mostly organs with significant difference in the acoustic impedance of its structural components, thus affecting mainly gas-containing organs. When these stress waves reach an air-tissue interface, a component of

the compressive stress wave is reflected back at the interface as a tension wave [7]. It is when these stress waves equal and exceed the tensile strength of the tissue interface that their work done on the organs becomes an irreversible work of damage [2]. A stress wave also compresses air in air-filled organs that re-expands forcefully causing damage to the walls through implosive forces. All this interprets how for example small bowel wall injury or alveolar septum injury happens in thoracic and abdominal wall PBI.

Shear waves are transverse waves with long duration and low velocity, traveling perpendicularly to the longitudinal stress waves and tangentially to body surfaces. They are generated from body wall displacement. Different solid organs with different densities move asynchronously with different inertias causing shearing of solid organs [25].

## Biological Effects of Primary Blast Injuries

The true incidence of primary blast injuries is unknown despite the various reports of incidence published. This is because PBI tend to be commonly overlooked especially in situations of mass casualty where the health care teams are faced with amputations, crush injuries, burns, toxic inhalations, and penetrating trauma. Delay in the diagnosis of PBI can complicate patient care especially in patients with isolated PBI who do not manifest external body trauma [26].

Primary blast injuries involve mainly gas-containing structures, namely the pulmonary, gastrointestinal, and auditory systems. The ear is the most commonly affected organ because for primary blast injury of the ear to occur, the blast overpressure threshold required is lower than that required for lungs and the bowels to be injured [27]. However, blast injuries are not exclusive to gas-containing structures. Other systems are affected as well though less common: the heart, vascular system, eye and orbit, and central nervous system among others.

Other than specific organ injuries, PBI have a systemic effect, a global physiologic response in

the form of a cardiogenic shock in the absence of hemorrhage uncompensated by vasoconstriction. It is mediated by pulmonary C-fiber receptors that are thought to initiate this vagal reflex. It usually occurs following thoracic PBI within seconds and lasts between minutes to hours but often resolves by 2 h. It is characterized by transient bradycardia, bradypnea, and hypotension [28, 29].

## Pulmonary System

As with all primary blast injuries, the lungs are more likely to get injured after a blast whereby the blast overpressure is high and the positive blast phase is prolonged. Uncomplicated blast lung injury has a favorable prognosis at 1-year follow-up. Hirshberg et al. reported that people who are discharged after surviving a lung blast injury had no pulmonary complaints, normal pulmonary function tests, and resolution of the chest radiography findings at 1-year follow-up [30]. Pulmonary PBI is essentially manifested as pulmonary contusions [5]. The spallation and implosion of the stress wave at the different air-alveolar-capillary wall interfaces cause alveolar wall, capillary wall, and interalveolar space disruption [2, 22]. This causes the pooling of blood perivascularly and alveolar hemorrhage. It can range all the way from petechiae to confluent hemorrhage [31]. Also, the extravascular fluid is compressed and driven into the alveolar space which causes pulmonary edema manifested as bilateral pulmonary infiltrates on chest radiography [32]. Implosive forces can also drive air into the interstitial spaces causing interstitial emphysema [26]. Shearing forces can disrupt the bronchovascular tree and create bronchopulmonary fistulas. These tears in the air-tissue interface can lead to arterial air emboli (AAE) development either immediately after the blast causing rapid death or delayed with the initiation of positive pressure ventilation [33]. AAE when big enough can cause MI, stroke, spinal cord infarction, intestinal ischemia, and death [34]. Even when microscopic, AAE can still cause symptoms like confusion, mental status changes, vision disturbances, pain, and weakness. Clinical signs like air in the retinal arteries, tongue blanching, or

livedo reticularis can be indicators of emboli [35]. Pleural tears and lacerations can also be caused by pulmonary barotrauma as a blast effect or due to positive pressure ventilation and can give rise to pneumothoraces, hemothoraces, or pneumomediastinum [7].

Pulmonary contusions are usually bilateral in closed-space explosions but tend to be worse on the side of the impact of the blast wave in open-field explosions [5]. The ribs protect the lung parenchyma from the full force of the blast overpressure. This results in stripes of hemorrhagic congestion corresponding to the intercostal spaces where there is no rib protection. These parallel bands of ecchymoses used to be called mistakenly “rib markings” as they were thought to be occurring under the ribs but were proven to occur along the intercostal spaces with the ribs providing protection to the underlying parenchyma. Perimediastinal lung parenchyma especially the azygos lobe and lung regions in the costophrenic angles are more severely involved by the blast injury. This unequal distribution is justified by the reflection and augmentation of the stress wave within the chest [7].

Specific ultrastructural manifestations have been reported in lung primary blast injury. On light microscopy, pulmonary capillaries are seen dilated [36]. On electron microscopy, increased pinocytosis, blebbing, and ballooning in pulmonary capillary endothelial and type I epithelial cells are seen in experiments done on rats exposed to a blast. Also, loss of structure or enlargement was noted in the lamellated bodies of type II epithelial cells [37]. These changes occurred not only in areas of the lung with apparent damage but also in apparently normal regions of lung parenchyma. So patients with no clinical or radiologic evidence of injury could still have sustained a lung blast.

At the macroscopic level, respiratory mucosa is very sensitive to blast effect. Damage occurs at overpressures below those that would cause parenchymal lung injury. Mucosal injury includes loss of cilia and flattening of epithelial cells. More severe injury can also occur with stripping of the mucosal epithelium off the basal lamina the so-called stripped epithelium lesion, with the

resultant intraluminal hemorrhage. This stripping of the epithelium is postulated to be due to the spalling forces at the epithelial tissue-air surface. These injuries generally resolve spontaneously and should be sought while examining a patient subjected to a blast. Their presence is an indicator of possible primary parenchymal lung blast injury and other organ blast injuries [38].

Clinically, the triad of dyspnea, cough, and hypoxia is referred to as “blast lung syndrome” and is due to ventilation mismatch, vascular shunting, and impaired gas exchange [39]. Focal pulmonary edema and hemorrhage in the alveoli cause ventilation perfusion mismatch with increased intrapulmonary shunt, hypoxia, reduced lung compliance, and increased work of breathing [40]. Clinical symptoms include dyspnea, cough, hemoptysis, chest pain, or discomfort. Clinical signs include tachypnea, cyanosis, reduced breath sounds and dullness to percussion, coarse crepitations, rhonchi, subcutaneous emphysema, features of hemopneumothorax or pneumothorax, retrosternal crunch, or retinal artery emboli.

Any blast-exposed patient is worth a chest radiograph. Bilateral pulmonary infiltrate is typically seen on chest radiography in primary blast injuries [32]. Usually, these infiltrates develop within few hours, become maximal at 24–48 h, and tend to resolve within a week. Infiltrates that continue to worsen beyond 48 h may be indicative of pneumonia or adult respiratory distress syndrome [7]. Pneumothorax, hemopneumothorax, interstitial emphysema, subcutaneous emphysema, pneumomediastinum, or pneumoperitoneum might be evident on chest radiography. Most blast injuries develop immediately, but sometimes, progressive vascular leak and inflammatory changes develop over 12–24 even up to 48 h contributing to delayed presentation [31]. Hence, patients with pulmonary symptoms and negative chest radiographs should be observed for 8 h before discharge [14]. However, the majority of patients with blast lung injury will manifest radiologic or clinical findings on admission [41]. If the symptoms are persistent or severe with a negative chest radiograph then a chest CT should be done [42]. A study showed that the ratio of  $\text{PaO}_2$  to  $\text{FiO}_2$ , the presence or absence of

chest radiograph infiltrates, and bronchopleural fistulas can help identify the severity of the lung injury in terms of mortality or progression to adult respiratory distress syndrome (ARDS) and help determine the respiratory management [43].

Management of primary blast injuries of the lung can be quite challenging. On one side, these patients are most often hemodynamically unstable requiring volume resuscitation yet excessive fluid resuscitation can lead to or exacerbate pulmonary edema in patients suffering from contusions [32]. To optimize the patient's respiratory status adequate pain management and noninvasive ventilation techniques are used. Avoiding positive pressure ventilation (PPV) as much as possible could not be emphasized enough. Positive pressure ventilation especially with high positive end-expiratory pressure (PEEP) is thought to increase the risk of pulmonary barotrauma, namely pneumothorax and arterial air emboli [9, 44]. Drainage of air, fluid, and blood through chest tube thoracostomy is very important in optimizing the pulmonary status of the patient and helps minimize the need for PPV. Prophylactic chest tube thoracostomy is recommended for patients who suffer from severe lung blast injury that will need positive pressure ventilation or need air transportation [45]. In blast lung injuries, lung compliance is poor and if positive pressure ventilation is to be needed then protective measures must be used like low PEEP, low  $O_2$  saturation, low tidal volumes, and permissive hypercapnia [43, 46]. Reversion to spontaneous breathing by intermittent mechanical ventilation and continuous positive airway pressure should be done as soon as the patient's pulmonary status allows it.

With arterial air embolism, if the patient is not intubated administration of oxygen should be initiated promptly and if available hyperbaric oxygen is the definitive treatment [7]. If the patient is intubated, then the ventilator settings should be adjusted to low PEEP and 100%  $FiO_2$ . Some recommend putting the patient in left lateral decubitus position to decrease the risk of systemic embolization [35, 47]. Organ transplant teams should be aware of the fact that normally looking organs could be unusable due to AAE [48].

Other less conventional techniques including extracorporeal membrane oxygenation, independent lung ventilation, nitric oxide ventilation, and high-frequency jet ventilation have been used in a small number of patients with varying degrees of success [43].

### Gastrointestinal System

The gastrointestinal system like the pulmonary system is at an increased risk of primary blast injury due to its gas-containing structures and similar blast overpressure to the lung [5]. The most common site of the GI tract of both hemorrhage and perforation is the colon and ileocecal region where gas accumulates most in the tract and ruptures the wall due to implosive forces of the blast [49]. Solid abdominal organs injuries can be PBI but arise more commonly as secondary or tertiary blunt and penetrating blast injuries [50, 51]. Solid intra-abdominal organs like the spleen, liver, or kidneys are made of relatively similar liquid densities [52]. Therefore, solid organ injuries during a blast are less likely due to compression by the stress wave but rather due to body-wall displacements causing acceleration effects at organ attachments. Shear forces can therefore cause subcapsular petechiae, contusions, or even frank ruptures [25]. Bowel PBI are caused by the implosive and shearing forces rupturing the bowel walls. These forces cause the wall's structural layer to separate resulting in intramural edema and hemorrhage with microthromboses [49]. Hemorrhages can range in size from small petechiae to large confluent submucosal hematomas and can progress into more severe transmural hemorrhages [2]. This can compromise the perfusion putting the bowels at risk for delayed perforation. At laparotomy it was found that small bowel contusions more than 15 mm in diameter and large bowel contusions of more than 20 mm were at significantly higher risk of perforation. Some considered that finding such contusions intraoperatively warrants resection while a more conservative approach is reserved to smaller contusions [53]. Mesenteric, retroperitoneal, and scrotal hemorrhages can also occur [2]. Shearing forces can disrupt the blood supply leading to intestinal ischemia. Arterial air embolism can be a cause of intestinal ischemia as well [35].

Clinically, abdominal PBI can present with abdominal pain, hematemesis, nausea and vomiting, rectal pain, tenesmus, and testicular pain. Clinical signs include peritoneal signs, absent bowel sounds, and evidence of hypovolemia [7]. In some cases the diagnosis of intra-abdominal injury can be obvious, yet in most cases as with all PBI it is a diagnostic challenge. Focused abdominal sonography can be used to assess for free fluid in the abdomen when assessing abdominal complaints for abdominal life-threatening injuries [54]. But a negative FAST does not exclude an abdominal primary blast injury. If hemodynamically stable, an abdominal CT can be of help [55]. However, CT is specific for solid organ damage and perforation but it lacks sensitivity to rule out intestinal contusions and mesenteric injury [56]. Doppler can also be used for investigation of abdominal perforating injury. Patients subjected to blast with abdominal complaints should be observed for 8 h and reexamined even if the mentioned imaging modalities are negative for findings [26]. Victims found to have an abdominal primary blast injury requiring operative intervention should be assessed for primary lung blast injury as these patients will require intubation and positive pressure ventilation intraoperatively [4].

Patients with suspected abdominal injury that could not be stabilized and have unexplained signs of hemorrhage would need urgent laparotomy. Pneumoperitoneum, diaphragmatic rupture, signs of peritoneal irritation on physical exam, and significant persistent GI bleed are all indications for urgent laparotomy. Tension pneumoperitoneum has also been reported. It is a complication of pulmonary barotrauma due to blast. It causes severe cardiovascular and respiratory collapse with severe hypoxemia, hypercarbia, and shock [57].

## The Ear

As discussed before, the auditory system is the most sensitive to blast overpressure injury [58]. The part of the tympanic membrane most frequently injured is the pars tensa [59]. Small tympanic membrane ruptures can be managed conservatively as they usually heal spontane-

ously. Ruptures involving beyond 5% of the membrane's surface will more likely require surgical intervention [60]. Isolated eardrum perforation has been shown to be a poor marker of latent pulmonary or gastrointestinal PBI and explosion survivors with eardrum perforations and no signs of PBIs can be discharged after monitoring and normal chest radiography [61]. Because the tympanic membrane ruptures at BOP lower than that required to cause lung or intestinal injury, it has been postulated that patients with intact tympanic membrane are probably exposed to little BOP and will not need further assessment. However, studies have shown that victims with and without tympanic membrane rupture were found to have lung blast injury [62, 63].

A temporary shift in the threshold for audible noises can cause a transient tinnitus or sensorineural deafness that resolves over several hours to days [64]. This is due to stunning of the receptor organs of the inner ear. The severity of these symptoms decreases as the distance from the detonation epicenter increases [65]. However, injury to the inner ear can sometimes cause permanent hearing loss in case of severe structural damage to the organ of Corti causing permanent threshold shifts [66]. This sensorineural hearing loss is different which is usually of high frequency and is a different entity than the usual 4-kHz noise-induced and reported trauma-induced hearing loss [67].

Ossicular injuries including incudomalleolar and incudostapedial joint disruption, fractures of the stapes superstructure, dislocation of the stapes footplate, and dislocation of the incus are also features of middle-ear primary blast injuries [68, 69]. Cholesteatoma of the middle ear and mastoid air-cell system is a late complication in blast-induced tympanic membrane perforations. The incidence of cholesteatoma is related to the grade of perforation. For example, a grade 1 perforation (<2 mm in diameter) has a 2% incidence of cholesteatoma whereas a grade 4 perforation (subtotal) has a 20% incidence [70]. Vertigo postblast can be due to benign paroxysmal positional vertigo, perilymph fistulas, or more commonly associated head injuries [4, 7].

All patients exposed to a blast should have an audiometric assessment just as they should have a chest radiograph. Clinically, the survivors can present with hearing loss, tinnitus, otalgia, vertigo, bleeding, or mucopurulent otorrhea. Temporal bone fracture can be an associated injury and cannot be excluded by plain skull radiography but rather will need CT, MRI, or angiography for assessment [7]. Prophylactic antibiotics are not indicated [71]. Early surgical intervention is preferably reserved for clearing debris and removal of foreign material. Most otologists would prefer to wait up to 1 year before doing an elective tympanoplasty on nonhealing perforations [72]. Long-term follow-up is needed to monitor any cholesteatoma formation occurring as a late complication [70]. A study showed that antioxidant treatment can improve recovery and decrease damage to the mechanical and neural components of the auditory system when given shortly after the blast [73]. There is little evidence on whether systemic steroids, vitamins, antiplatelets, low-molecular-weight dextrans, or vasodilators do help in improving the outcome for blast-induced hearing loss [7]. Yet, the administration of one or more of these is still a common practice.

## Conclusion

Explosive devices are becoming the prevalent weapon of military combats and explosions are no longer confined to battlefields but rather are becoming very common in civilian areas especially in countries of armed conflicts. Terrorist attacks and industrial accidents are other causes of civilian victims' injuries that get referred to nonmilitary hospitals. Accordingly, the knowledge of blast injuries is becoming a necessity to civilian physicians. An understanding of the biodynamics of blast injuries by surgeons, internists, anesthesiologists, nurses, and all health care providers plays a great role in improving patient care and management outcomes of blast victims. Blast injuries include every organ system in the body. However, injuries caused by primary mechanism constitute a diagnostic and therapeutic challenge. Primary blast injuries can affect any organ but

primarily include the lung, bowels, and ear. A solid knowledge of the pathophysiology, clinical manifestations, and management as well as maintenance of a high index of suspicion in any victim subjected to blast will help reach an early diagnosis and therefore a more robust health care, which can save many lives.

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Reconstructing the War Injured Patient

Abu-Sittah, G.S.; Hoballah, J.J.; Bakhach, J. (Eds.)

2017, VIII, 212 p. 207 illus., 153 illus. in color.,

Hardcover

ISBN: 978-3-319-56885-0