EXPLOSIVES

What Happens in an Explosion?

During an explosion, gasses under extremely high pressures are produced. When these gasses expand, they form a blast wave, which spreads out in all directions. The magnitude and duration of the blast wave are major determinants of the severity of a blast injury.

See below for:

A. Blast Waves Defined
B. Nuclear vs. Conventional Weapons
C. Injury from Blast Waves

Specific Information:

A. Blast Waves Defined

The rapid chemical conversion of a solid or liquid into a gas following the detonation of an explosive device results in a blast, and is characterized by a release of large quantities of energy in the form of pressure and heat. As these high-pressure gasses rapidly expand, they displace and compress the surrounding medium causing it to move outwards in a radial fashion. This pressure pulse is known as the "blast wave." The blast wave begins as a single pulse of increased pressure that rises to peak levels within a few milliseconds, and then rapidly falls to a minimum pressure that is lower than the original atmospheric pressure creating a relative vacuum, as seen in Graph 1.

The duration and magnitude of the blast wave's peak may depend on a host of factors, including the type of explosive used, the conducting medium, and distance from the detonation site. The duration and magnitude of the peak of the blast wave's pressure also determine the overpressure that an object in its path is subjected to and constitute the main determinants of the severity of primary blast injury.

Blast waves are usually only a few millimeters thick. Due to the fact that they superheat molecules under high pressure at the wave front, blast waves travel at supersonic speeds. When the blast wave impacts an object, there is a virtually instantaneous increase in pressure to the peak overpressure.

B. Nuclear vs. Conventional Weapons

Nuclear blast waves differ from those of conventional munitions principally in their positive-phase duration, which may last as long as several seconds, compared to milliseconds with explosives such as TNT. Under certain conditions, nuclear explosions may also produce a precursor shock wave.

C. Injury from Blast Waves

As mentioned, a temporary relative vacuum is created as gasses continue to expand from their point of origin. A transient blast wind may travel immediately behind the blast front. In the vicinity adjacent to an explosion, this force can cause traumatic amputation, evisceration, or total disintegration of a body. Winds may also cause injury by accelerating debris and fragments that subsequently strike the victim, or by displacing the victim against a stationary object.

Types of Explosives

There are two basic types of explosives: low-order and high-order. Low-order explosives do not cause blast wave formation, and include black powder and petroleum (gasoline or jet fuel) based bombs. High-order explosives generate a reaction that results in the creation of a blast wave, the main culprit of primary blast injury. These include TNT, dynamite, and composition C4.

Specific Information:

An explosive is a substance that undergoes rapid decomposition or combustion when detonated, producing a tremendous amount of heat and a large volume of high-pressure gas. Chemical explosives can be classified as low-order or high-order explosives. Low-order explosives, such as black powder, burn by means of a process called deflagration. Mixtures of readily combustible substances, these explosives are used primarily for propelling, but may also be found in explosives such as pipe bombs.

Conversely, high-order explosives consist of substances that undergo rapid decomposition without any external sources of oxygen. With high-order explosives, products of the explosive reaction occupy a greater volume than that filled by the original reactants. The outcome is a rapidly expanding pressure wave, which compresses the surrounding medium. The pressure wave, unique to high-order explosives, is also known as an overpressure, or "blast wave."

Types of single-compound high-order explosives include ammonium nitrate, nitroglycerin, trinitrotoluene (TNT), pentaerythritol tetranitrate (PETN), clyclotrimethylene trinitramine (RDX), cyclotetramethylene tetranitramine (HMX) and nitrocellulose. These may be combined to form mixed-compound expolsives, such as dynamite, composition C4, ANFO and sheet explosives.

While explosives are used for many peaceful purposes, their major use has been in warfare. High-order explosives have been applied in bombs, missile warheads and explosive munitions. Low-order explosives have been employed as propellants for small arms fire and artillery shells. Low-order explosives can also cause severe damage as demonstrated by the explosion of the jet fuel filled planes striking the World Trade Center of September 11, 2001.

The Three Types of Blast Injury

Blast injuries are categorized into primary through tertiary types of injury. Each type has its own mechanism of injury, as displayed in the table below.

Type of Blast Injury	**Mechanism of Injury**
Primary Blast Injury	Injury caused by the effect of the blast wave on the body. Primary blast injury occurs principally in the gas-filled organs, and results from extreme pressure differentials developed at body surfaces. Organs most susceptible include the middle ear, lung, and bowel.
Secondary Blast Injury	Injury caused by flying debris and fragments, propelled mostly by the blast winds generated by an explosion. Most commonly produces penetrating injury to the body. At very close distance to the explosion, may cause limb amputation or total body disruption.
Tertiary Blast Injury	Injury results from victim being diplaced through space by the blast wind and impacting a stationary object.

Primary Blast Injury

As a blast wave impacts the body, a pressure wave is transmitted through the body surface, affecting the underlying tissues. This transmitted wave may fragment tissues of different densities, compress air-filled organs, or tear organs from their points of fixation.

See below for:

A. How Blast Waves Damage the Body
B. Blast Wave Injury Terminology

Specific Information:

A. How Blast Waves Damage the Body

Most of the clinical syndromes resulting from primary blast injury are direct results of the "blast wave," or overpressure, created by an explosion. As the blast wave impacts the human body, extreme pressure differentials are generated at the body surface involved. This pressure differential subsequently accounts for a tremendous external force acting on the body surface, in turn creating a "stress wave" that is transmitted to the underlying tissues. A combination of stress and shear waves is produced by this process, and these affect mostly gas-containing organ systems, such as the lungs, the bowel and middle ear.

Blast wave impact >> Stress and shear waves >> Damage to gas-containing organs

B. Blast Wave Injury Terminology

There are several concepts describing how the blast wave injures the tissues it travels through. *Spalling*, or *spallation*, occurs as a blast wave passes from a tissue of higher density to one of lower density. At the interface of the two tissues, fragmentation of the higher density tissue may occur. *Implosion* occurs as high pressure in solid organs or fluid compresses gas-filled spaces within these organs or fluid. The pressure differential may force blood and fluid into the previously air-filled spaces, as seen in pulmonary contusion and hemorrhage.

The negative pressure wave that follows the initial pressure wave may cause additional injury, as the compressed gas forcefully re-expands. Finally, *shear* causes injury as tissues of different densities are accelerated and decelerated at different rates relative to one another. This acceleration and deceleration of organs relative to their points of fixation can produce shear forces that may tear or damage the organs.

Other Blast Injuries

Loose objects and bomb fragments striking a victim's body cause Secondary Blast Injury. The resultant penetrating trauma may cause significant internal injuries.

With Tertiary Blast Injury, blunt trauma is more common, as a victim is flung by the force of the explosion and impacts a stationary object.

See below for detailed discussions of:

A. Secondary Blast Injury
B. Tertiary Blast Injury

Specific Information:

A. Secondary Blast Injury is principally due to the blast wind generated in an explosion and encompasses all injury suffered as a result of flying debris and fragments impacting a victim's body.

During an explosion, the chemicals composing the explosive are consumed or incinerated, whereas bomb fragments from the casing or shrapnel within the explosive munitions essentially remain intact. The rapidly expanding gasses created during the explosion may catapult these secondary munitions at speeds in excess of 2000 miles per hour, striking victims thousands of feet away. In addition to projectiles intrinsic to an explosive device, the blast wind may accelerate loose objects in the vicinity of the explosion. Following the terrorist bombing of the US Embassy in Nairobi in 1998, people up to 1.5 miles away were wounded from flying debris.

Intentional shrapnel used in explosives include ball bearings, nuts, bolts, nails and various other objects. These may cause penetrating injuries similar to those seen with small arms fire. Although the outer appearance of such wounds may not be very impressive, significant internal injuries may occur. From recent experiences with terrorist homicidal bombings, clothing offers some degree of protection as secondary blast injuries to the head, neck, and extremities predominate in such cases. One should pay particular attention to foreign bodies to the eye.

Amputation may also be considered part of secondary blast injury, and is usually seen in victims who are in close proximity to the site of the explosion. As the blast wave impacts an extremity, tremendous pressure differentials may shatter the bone. The near simultaneous blast wind following the blast wave may subsequently avulse the extremity. On the whole, avulsions are observed mainly along the shaft of long bones and are most common among the dead and dying victims. These injuries have a high risk for exanguination and are rarely reattachable.

B. **Tertiary Blast Injury** also is caused by the blast wind generated in an explosion, but pertains to injuries suffered when a victim is hurled through space and impacts a stationary object. For the most part, manifestations of this form of injury include abrasions and contusions, as the victim tumbles along the ground. However, more significant blunt trauma from acceleration-deceleration type injuries may be seen if the victim is actually flung through the air from the force of the explosion.

Blast Lung

When a blast wave impacts the lung, the lung tissue is compressed faster than the air within it. As a result, the lung tissue may bruise or bleed. Bruising within the lung may be widespread, or it can assume a characteristic "butterfly pattern." Other injuries, seen with high magnitude blasts, include collapsed lungs and bleeding within the chest cavity. Another potential complication is acute air embolism, which is most common during initiation of mechanical ventilation.

Since few care providers in the United States have extensive experience with blast injuries, we provide a detailed clinical discussion of the management of those injuries, beginning with blast lung (below).

Specific Information:

An external force acting on the chest wall may compress the lungs slowly enough to allow air contained in the alveoli to be expelled through the trachea. However, when a significant blast wave impacts the chest wall, there is little time for equilibration, and lung intaparenchymal pressures can match or exceed the pressure of the blast wave. This is because lung tissue is less compressible than the air within it. As mentioned earlier, pressure differentials develop at the interface between media of different densities. These pressure differentials tear the alveolar walls and disrupt the alveolar-capillary interface, resulting in emphysematous spaces filled with blood.

Primary blast injury of the lung, or blast lung injury, primarily presents as pulmonary contusions (similar to pulmonary contusions from blunt trauma, but without the rib fractures), impairing gas exchange at the alveolar level and resulting in decreased oxygen diffusion. The manifestations of contusion may develop over the course of hours and may have the appearance of a local or diffuse infiltrate. Pleural petechiae and ecchymoses may be seen on pathologic examination of victims of mild blast injury, while multifocal hemorrhages are more prevalent with significant and higher energy blast waves. Near the mediastinum and diaphragm, conducted stress waves may summate and reflect, resulting in the characteristic "butterfly" pattern of lung injury.

Blast lung injury may result in activation of pulmonary C-fiber receptors, in turn resulting in a vagus nerve-mediated type of cardiogenic shock, lacking compensatory vasoconstriction. The clinical syndrome appears as bradycardia and hypotension, and may partially resolve over the course of the first few hours.

Higher magnitude blast waves can cause significant lung injury, which may result in pneumothorax, hemothorax, alveolovenous fistulas and traumatic emphysema. Complications of such injuries include bronchopleural fistulas and arterial air embolism. In cases of low intravascular pressures due to hemorrhage or increased inspiratory pressures during mechanical ventilation with positive pressure ventilation, arterial air embolism is more likely to occur. Acute air embolism to the brain or heart may be the most common cause of rapid death solely caused by primary blast injury and often occurs at the moment of initiation of mechanical ventilation. In the absence of blood loss from external, intrathoracic, intraabdominal, pelvic, or long bone fractures, coronary acute air embolism with myocardial infarction and cardiogenic shock should be considered as the cause of shock.

Management of Blast Lung

All patients with suspected blast lung injury should be given the highest available fraction of inspired oxygen (FiO_2) possible, preferably by non-rebreather facemask (NRFM).The recommendations for treatment of blast lung injury are prevention of intubation and positive-pressure ventilation.

See below for:

- A. Blast Lung Injury Management Overview
- B. Symptoms of Blast Lung Injury
- C. Diagnosis and Radiographs
- D. Upper Respiratory Complications
- E. Mechanical Ventilation
- F. Other Methods of Blast Lung Injury Management

Specific Information:

A. Blast Lung Injury Management Overview

Diagnosis and management of patients with severe primary blast injury to the lung (blast lung injury) is challenging not only because of the injury to the lung itself, but also because severe primary blast lung is frequently accompanied by shock and unconsciousness. Resuscitation of a blast injury patient is complex and relies on general principles of trauma care not discussed here. Instead, only issues unique to blast lung injury will be considered in this section.

Blast lung Injury may cause:

- Parenchymal lung injury resulting in pulmonary contusion
- Pulmonary barotrauma giving rise to pneumothorax or pneumomediastinum
- Pulmonary lacerations producing hemopneumothorax or bronchopleural fistula.

B. Symptoms of Blast Lung Injury

Symptoms that should make the clinician suspicious of injury to the lung include:

- Dyspnea
- Chest pain
- Hemoptysis
- Cough
- Inability to carry out a conversation.

Clinical signs observed with significant lung injury include cyanosis, tachypnea, crackles, diminished breath sounds, dullness to percussion, increased resonance, retrosternal crunch, subcutaneous crepitus, and tracheal deviation. Rapid shallow respirations are characteristic of blast victims.

C. Diagnosis and Radiographs

A chest radiograph should be obtained on all patients with suspected blast lung injury as the characteristic pulmonary infiltrates can help confirm the diagnosis, as well as further define the severity and extent of injury. Table 2 describes a classification system used by Israeli investigators studying blast lung injury, which may help predict the necessity for use of positive pressure ventilation (PPV) and positive end expiratory pressure (PEEP) based on radiographic findings. Radiographs may also help in diagnosis of pneumothorax, hemothorax and other chest pathology, as well as confirm placement of chest tubes, endotracheal tubes, and central venous catheters.

D. Upper Respiratory Complications

With regard to a patient's airway, upper respiratory tract injury and compromise due solely to primary blast injury is not common. Mucosal edema has not been described as a significant cause of airway obstruction following blast injury. However, upper airway obstruction can be seen in patients presenting with altered mental status and massive hemoptysis from an underlying lung injury. It is also possible to see facial and laryngeal trauma due to secondary blast injury.

Patients who present with decreased or asymmetric breath sounds and evidence of circulatory compromise mandate an attempt at needle thoracostomy to decompress a potential tension pneumothorax. Similarly, if a simple pneumothorax or hemothorax is diagnosed, a tube thoracostomy should be placed. Because of large air leaks associated with bronchopleural fistulas, short large-bore thoracostomy tubes are preferred in management of blast lung injury. Conversely, failure of a properly placed large-bore chest tube to fully re-inflate a lung should raise the suspicion of bronchopleural fistula. Although there are no clearly established guidelines, some sources recommend a prophylactic thoracostomy tube be placed in all patients with primary blast injury to the lung.

E. Mechanical Ventilation

One of the most difficult issues to contend with in blast lung injury is the initiation of mechanical ventilation. The recommendations for treatment of blast lung injury are *prevention* of intubation and positive-pressure ventilation. Acute air embolism (AAE), discussed below, is one of the major complications seen with mechanical ventilation of blast-injured lungs, and its incidence can be decreased if spontaneous respirations are maintained. All patients with suspected blast lung injury should receive the highest available fraction of inspired oxygen (FiO_2) possible, preferably by non-rebreather facemask (NRFM).

Blast victims who have extreme respiratory embarrassment should be intubated endotracheally to handle massive hemoptysis in anticipation of mechanical ventilation. As previously mentioned, mechanical ventilation is not without risk for the blast victim. If too much positive pressure ventilatory support is used, there is a high risk of developing a tension pneumothorax or acute air embolus. Once again, some sources recommend placement of bilateral prophylactic chest tubes prior to initiation of mechanical ventilation.

In spite of these risks, mechanical ventilation should not be withheld in cases of respiratory insufficiency. When positive pressure ventilation is used, parameters for tidal volume, respiratory rate and inspiratory flow rate should be chosen so as to minimize the peak airway pressure during the machine delivered breaths. Pressure controlled ventilation and permissive hypercapnia, to facilitate oxygen exchange but keep transalveolar pressures less than 35 cm H_20, have been recommended.

F. Other Methods of Blast Lung Injury Management

Although beyond the scope of this discussion, successful management of patients with severe blast lung injury has also included such methods as independent lung ventilation, inverse inspiratory/expiratory ratios, extracorporeal membrane oxygenation, high flow jet ventilation, and nitric oxide inhalation. Bronchoscopy may be necessary for management of hemoptysis.

Air emboli may be liberated from the lacerated lung into the arterial circulation, where they may cause occlusions with disastrous results. Though they can affect any organ, air emboli may dislodge in the cerebral vessels causing a cerebrovascular accident, or in the coronary vessels resulting in a myocardial infarction. Symptoms and signs of air embolism develop very early in the clinical picture of blast lung injury, and the emergency clinician must be aware of this potentially fatal complication. Symptoms of AAE include blindness, chest pain, focal neurological deficits, loss of consciousness or vestibular disturbances, whereas signs include air in the retinal vessels, dysrhythmias or cardiac ischemia on EKG, focal neurological deficits, and livedo reticularis.

To minimize the likelihood of acute air embolus, a partial left lateral decubitus position, tilted slightly more towards prone, has been recommended as this position will place the coronary ostia in their lowest positions. Prone positioning places the left atrium in its highest position, in theory making it more difficult for air bubbles to enter the left ventricle and the systemic circulation. However, positioning of the patient can be complicated by concomitant injuries to other parts of the patient and the security of the airway.

Treatment in a hyperbaric chamber is the definitive therapy for arterial air embolism, as an increase in ambient pressure will decrease the size of the emboli and promote their rapid absorption. However, as most such interventions took place hours after blast exposure, data on the effectiveness of such therapy for human AAE is lacking. Nitrates and thrombolytics are not indicated for ECG findings of ischemia and infarction in patients with acute air embolism.

Table 2. Severity Categories for Primary Blast Injury of the Lung

	Mild	**Moderate**	**Severe**
Radiographic Infiltrates	Unilateral	Asymmetrical Bilateral	Diffuse
PFR (mm Hg)	>200	60-200	<60
Broncholpleural Fistula	No	Yes	Yes
Positive Pressure Ventiliation (PPV) Requirement	Unlikely for respiratory problem	Highly likely but usually conventional methods	Universal and unconventional methods common
Positive End Expiratory Pressure (PEEP) Requirement (cm H_2O)	<5 if PPV needed	5-10 usually needed	>10 commonly needed

Severity categories as reported by Pizov et al **39**, which may help predict the necessity for use of PPV and PEEP.

Eye and Facial Injuries

Likely because of the uniform density of the eye, ocular injury from a blast wave is rare. In rare cases, blast victims may experience transient blindness after exposure, as well as hyphema and conjunctival hemorrhage. Perforation and other ocular trauma is much more likely to be due to secondary mechanism of injury. If visual deficits do exist, the diagnosis of acute air embolism within the retinal arteries should be considered.

Fractures to the sinuses of the facial bones can occur in victims who are in close proximity to a blast. Experimental evidence is lacking, but these injuries are assumed possible with high magnitude blast waves at a super lethal blast dose. Such fractures cannot be easily detected, and may be missed on routine clinical examination.

Ear, Nose, and Throat Injuries

Perforation of the eardrum is the most common ear injury seen after an explosion. Most perforations tend to heal spontaneously. More significant blasts can damage the inner parts of the ear, including the bones behind the eardrum and the nerves within the ear. Injury to the eye purely from a blast wave is rare, but often results from debris propelled into the eye by the blast itself.

It is common that persons injured in a blast will have hearing damage or deficits. This must be kept in mind as you are triaging, assessing, and giving patient direction.

See below for:

A. Tympanic Membrane Perforation
B. Middle Ear Injury

Specific Information:

The ear is most frequently affected organ during an explosion, with the ear nearest the explosion usually suffering more severe damage. The powerful blast wave can overwhelm the extremely delicate structures within the ear, causing tympanic membrane rupture, fracture or dislocation of the ossicles, or permanent inner ear damage. Awareness of these injuries is important so that their deleterious consequences can be prevented.

A. Tympanic Membrane Perforation

Perforation of the tympanic membrane is a familiar finding on physical examination. The most susceptible portion of the tympanic membrane is the pars tensa; the least susceptible the pars flaccida. By itself, the rupture of a tympanic membrane by a blast wave is not likely to be the cause of significant disability, and the majority of perforations will heal spontaneously without intervention. Of note, it is incorrect to assume that absence of tympanic membrane damage precludes a significant blast exposure, particularly if the ear was not as exposed as other parts of the body.

B. Middle Ear Injury

Less common middle ear injuries include fractures of the ossicles and dislocations of the incudomalleal or incudostapedial joints. Though usually in conjunction with tympanic membrane perforation, these can occur independently. Injury to the inner ear may also occur, and includes perilymphatic fistulae in the oval window and ruptures of the saccule, utricle, and basilar membrane. Sensoryneural hearing loss may be seen with loss of hair cell integrity. Similarly, damage to the vestibular apparatus may occur and manifest as vertigo.

Treatment of Ear, Nose, and Throat Injuries

It is important to recognize injury to the auditory system to prevent the complications that develop from undiagnosed injury.

Patients with primary blast injury to the auditory system may present with complaints of tinnitus, hearing loss, ear pain, aural fullness, or vertigo. The patient's ears should be examined by direct otoscopy, looking for perforation of the tympanic membrane, disruption of the ossicular chain, and foreign material in the auditory canal. The presence of CSF or blood may be associated with a basilar skull fracture.

Tympanic membrane rupture is by far the most common aural injury, and barring infection, the vast majority of ruptures will heal without intervention. It should be mentioned that the presence of tympanic membrane perforation is not associated with delayed-onset blast lung injury, as once thought. Sensorineural hearing loss usually resolves within the first few hours, but some degree of permanent hearing loss may occur in up to a third of blast victims. Recognition of possible injury to the ear may help in planning the optimal treatment for its manifestations and prevent the consequences of unrecognized injury.

Abdominal Injuries

Injury to the abdomen is more commonly noted in the gas containing organs, such as small and large intestine, than solid organs, such as liver, spleen, and kidney. Small injuries to the bowel can progress with time resulting in perforations in the bowel.

Specific Information:

The characteristics of blast injury in the abdominal cavity resemble those of blast injury in the thoracic cavity. The blast wave strikes and displaces the body wall, causing distortion of the tissues within that results in their stress and failure. Although no external injury is seen on the body wall, tissues containing air are especially vulnerable.

Significant injury may be found in gas-containing structures in the abdomen due to blast wave effects at the tissue-gas interface. The small intestine and large intestine may sustain petechiae and ecchymoses. Hemorrhage into the wall and lumen of the intestine, often with perforation, tends to be a multifocal process, but is most often found in the lower small intestine and the cecum. Acute rupture is most common in the colon, where an increased quantity of gas exists, and at the ileocecal junction. More importantly, initial mild injury to the bowel may progress to perforation within hours to days, as injured tissue becomes ischemic and necrotic.

Solid organs, principally the liver, spleen, and kidney, have a relatively uniform liquid density. When a blast wave impacts these organs, little compression occurs, and significant injury to the tissue is not common. Solid intra-abdominal organs are more likely to be injured through secondary or tertiary mechanisms. However, blast waves can cause shear forces to develop at points of attachments of organs or at the surfaces of the organs. In the former case, an organ may tear off of its point of attachment, while in the latter case, subcapsular petechiae, contusions, lacerations, or rupture may occur.

Management of Abdominal Injuries

Patients with abdominal injuries may have an unimpressive clinical picture initially, but may develop signs of severe abdominal injury hours to days after a blast.

See below for:

A. Signs and Symptoms of Gastrointestinal Tract Injury
B. Evaluation and Management of GI Tract Injury

Specific Information:

A. Presentation of Gastrointestinal Tract Injury

Injury to the gastrointestinal tract (GI) is often overshadowed by the more immediately life-threatening pulmonary contusions and lacerations and their resultant complications. However, gastrointestinal tract injury may be more dramatic at the time of presentation. Patients with primary blast injury to the GI tract may complain of abdominal pain, nausea, testicular pain, tenesmus, or a temporary loss of motor control in the legs. Physical examination may reveal signs that are similar to those found in blunt abdominal trauma from any cause, the main difference being that solid organ injury is less common. Signs of GI injury on examination include absent bowel sounds, bright red blood from the rectum, hypotension, guarding, and rebound tenderness. Patients with an unimpressive physical examination initially may develop peritonitis hours or days later, when intestinal perforation has occurred.

B. Evaluation and Management of GI Tract Injury

The work up for primary blast injury to the abdomen includes serial hemoglobin determinations, which are essential if the patient is likely to have suffered intra-abdominal hemorrhage. However, serum chemistry and enzyme analyses fail to correlate with the presence or extent of GI tract involvement in primary blast injury. Focused abdominal sonography for trauma (FAST) should be used when available, and is especially useful in mass casualty incidents. Stable patients may be amenable to further diagnostic imaging with computed tomography of the abdomen and pelvis, though this technique may not identify all intra-abdominal free air or bowel injury. In unstable patients, a diagnostic peritoneal lavage (DPL) may play a role in the diagnosis of intra-abdominal hemorrhage. A major disadvantage of DPL is its insensitivity to retroperitoneal hemorrhage and mesenteric hematomas. In addition, DPL may fail to detect subcapsular injuries to the liver or spleen.

Unlike respiratory barotrauma and air embolism, gastrointestinal primary blast injury often requires surgical intervention for repair. A chest radiograph should be obtained on all patients likely to undergo laparotomy, anticipating evidence of unidentified and concomitant lung injury.

Blast Injury Patient Disposition

Observation, admission, and discharge of patients with blast injury depend to a large extent on patient complaints and injuries.

- Patients with complaints of shortness of breath or chest pain should be observed in the emergency department for a prolonged period of time prior to discharge.
- Patients with abdominal pain and tenderness should be admitted to the hospital for inpatient observations.
- Patients with isolated tympanic membrane rupture can be discharged home with ENT follow up.

Specific Information:

There are no official, established guidelines detailing the duration of the observation period or admission criteria in blast injury victims.

Patients who have no complaints of chest pain or dyspnea and normal chest radiographs are candidates for discharge from the emergency department. With adequate return indications, these patients may come back to the emergency department at a later time if missed pulmonary contusions develop. However, as previously mentioned, significant blast lung injury is likely to present within the first few hours following a blast. Patients with complaints of dyspnea or chest pain and those with any abnormality on chest radiographs should not be discharged from the emergency department without an adequate observation period.

Patients with complaints of abdominal pain and tenderness should be admitted to the hospital, regardless of the imaging study results or other diagnostic work up. Such patients should be observed for a period of several days as inpatients while receiving serial abdominal examinations.

Patients with isolated rupture of the tympanic membrane may be discharged home with specific instructions on how to protect their ear canals from further debris and water. Antibiotics are not indicated initially, and follow up should be arranged with an otolaryngologist.

Blast Injury Summary

Explosive Summary

- Low-order and high-order explosives can cause serious damage to the human body.
- The expansion of gasses produced in an explosion form a blast wave, which is the source of many injuries caused by high-order explosives.

Blast Injury Summary

The injuries produced by explosives fall into four categories:

- Primary Blast Injury is caused by the blast wave when it impacts the body and creates a pressure wave that injures tissues and organs. The magnitude and duration of the blast wave are the primary determinants of the severity of primary blast injury.
- Secondary Blast Injury is principally due to the blast wind generated in an explosion, and encompasses all injury suffered as a result of flying debris and fragments impacting a victim's body.
- Tertiary Blast Injury is also caused by the blast wind generated in an explosion, but pertains to injuries suffered when a victim is hurled through space and impacts a stationary object.

Blast Lung

- When blast waves impact the lung, the lung tissue is compressed faster than the air around it. Bruising and bleeding result from this compression.
- Bruising within the lung may be widespread, or it can assume a characteristic "butterfly pattern."
- Other potential complications include collapsed lungs, bleeding within the chest cavity, and acute air embolism, which is most common during initiation of mechanical ventilation.

Abdominal Injuries

- Injury to the abdomen is more commonly noted in the gas containing organs, such as the small and large intestines, than solid organs, such as liver, spleen, and kidney. Small injuries to the bowel can progress with time resulting in perforations in the bowel.

- The characteristics of blast injury in the abdominal cavity resemble those of blast injury in the thoracic cavity.

- Solid intra-abdominal organs are more likely to be injured through secondary or tertiary mechanisms.

ENT and Eye Injuries

- The ear is the most frequently affected organ during an explosion, with the ear nearest the explosion usually suffering more severe damage.

- The most susceptible portion of the tympanic membrane is the pars tensa; the least susceptible is the pars flaccida.

- Tympanic membrane rupture is by far the most common aural injury, and barring infection, the vast majority of ruptures will heal without intervention.

- In rare cases, blast victims may experience transient blindness after exposure, as well as hyphema and conjunctival hemorrhage. Perforation and other ocular trauma is much more likely to be due to secondary mechanism of injury.

GI Track Injury

- Patients with abdominal injuries may have an unimpressive clinical picture initially, but may develop signs of severe abdominal injury hours to days after a blast.

- Signs of GI injury on examination include absent bowel sounds, bright red blood from the rectum, hypotension, guarding, and rebound tenderness. Patients with an initially unimpressive physical examination may develop peritonitis hours or days later, when intestinal perforation has occurred.

- Unlike respiratory barotrauma and air embolism, gastrointestinal primary blast injury often requires surgical intervention for repair.

- A chest radiograph should be obtained on all patients likely to undergo laparotomy, anticipating evidence of unidentified and concomitant lung injury.

Disposition of Patients with Primary Blast Injury

Observation, admission, and discharge of patients with blast injury depend to a large extent on patient complaints and injuries.

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- Patients with isolated tympanic membrane rupture can be discharged home with ENT follow up.