EMERGENCY MEDICINE PRACTICE
EBMEDICINE.NET
AN EVIDENCE-BASED APPROACH TO EMERGENCY MEDICINE

Blast Injuries: Preparing For The Inevitable

You have been working the evening shift in a Midwestern hospital and are preparing to leave at 7 am. It's been a quiet night, and you haven't used the "Q" word once. You are online reading about the improvised explosive devices (IEDs) being used in Iraq, when the phone rings. The nurse hands it to you, saying "it's for medic command." They have had an explosion at the grain storage facility on the other side of town, about 10 miles away. You take the phone and tell the charge nurse to contact the hospital administrator (he's the first number on the disaster call-up roster). The dispatcher says details are still sketchy, but the grain plant exploded just as the 18-person day crew assembled for the day's work. There are at least 4 known dead, with an unknown number of casualties. Fire has 4 trucks responding, and they have asked for activation of the local disaster plan. The first truck on the scene has requested 6 additional ambulances from county and mutual aid support. The medic on the scene is triaging and says that he will have at least 10 more casualties from this explosion. Quiet time is over. You take a deep breath and get ready to receive multiple simultaneous casualties.

OUR knowledge of the effects of blast injury dates from the Balkan wars in 1914, when Franchino Rusca, a Swiss researcher, observed 3 soldiers who had been killed by an explosion without evidence of any external injuries.[1] Rusca went on to use rabbits as an animal model and demonstrated that the cause of death was pulmonary embolism. During WWI, blast injury was thought to be a nervous system disorder and labeled "shell shock." (At that time, psychological casualties were lumped together with those who had no visible injuries.)[2]

In WWII, a noteworthy number of casualties were found among civilians in both German and British cities after bombing raids. "Blast lung" was the term coined for massive pulmonary hemorrhage from disruption of the alveolar architecture and formation of alveolar-venous fistulas resulting in air embolism.[3] Following WWII, blast injury was intensively investigated in

April 2006
Volume 8, Number 4

Author

Charles Stewart, MD, FAAEM, FACEP
Emergency Physician—Colorado Springs, CO.

Peer Reviewers

Andy Jagoda, MD, FACEP
Professor and Vice-Chair of Academic Affairs, Department of Emergency Medicine; Residency Program Director, Mount Sinai School of Medicine—New York, NY.

John M Howell, MD, FACEP
Clinical Professor of Emergency Medicine, George Washington University, Washington, DC; Director of Academic Affairs, Best Practices, Inc, Inova Fairfax Hospital—Falls Church, VA.

CME Objectives

Upon completing this article, you should be able to:

1. Present the physics of an explosion in order to understand the consequent injuries that result;
2. Differentiate high-order from low-order explosives and other types of explosive to help the clinician understand the potential medical injuries;
3. Provide a systematic approach to triage and management of victims of blast injuries;
4. Discuss both the immediate and delayed medical injuries that can result from a blast exposure; and
5. Discuss operational issues related to activating a disaster plan subsequent to a blast-related mass casualty.

Date of original release: April 10, 2006.
Date of most recent review: April 1, 2006.
See "Physician CME Information" on back page.

the United States, due to the perceived threat of nuclear warfare. But it is only since the advent of Islamic terrorist suicide bombings that civilian physicians have become significantly concerned about the cause and treatment of blast injuries.[2] Unfortunately, the threat of suicide bombing seems to have spread from the Near East to the Far East and back to Europe, as evidenced by both the Madrid and London attacks. The obvious concern is when, not if, the practice will spread to the United States.

Prior to 1995, most civilian emergency physicians in the US had neither experience of nor interest in the effects of explosive devices. This changed abruptly with the destruction of the Alfred P Murrah Federal Building by a truck bomb — the 1995 blast rocked downtown Oklahoma City and resulted in more than 750 casualties, with 167 fatalities.[4] Unfortunately, many smaller devices are exploded each year in the United States.[5-7] As a result of the casualties associated with September 11, 2001, more US physicians have had to face the specter of explosion and blast injuries filling their own EDs.[8,9] The London and Madrid bombings (on July 7, 2005 and March 11, 2004, respectively) have forced physicians in other countries to consider or reconsider their potential roles in explosions and blast injuries due to terrorism.

Bombings are clearly the most common cause of casualties from terrorist incidents.[10] Recent terror tactics include an increasing use of suicidal/homicidal bombers who deliberately accompany the explosive device (often wearing it) to ensure its maximum effect.[11] These bombers have walked or driven into buses, subways, cafes, residential areas, guard posts, and governmental buildings. The use of suicide devices in the US has yet to occur, but given the political climate, the scenario is very likely. Increasingly, information resources, such as the Internet, terrorist training camps, and even library and television sources, have made the knowledge needed to construct these simple and very effective explosive devices readily available.

Research on blast injury is not a new study for those interested in combat medicine. This issue of *Emergency Medicine Practice* will review the current literature, including the potential mechanisms of injury, early signs of these injuries, and the natural course of the problems caused by explosive blasts.

Abbreviations Used in This Article

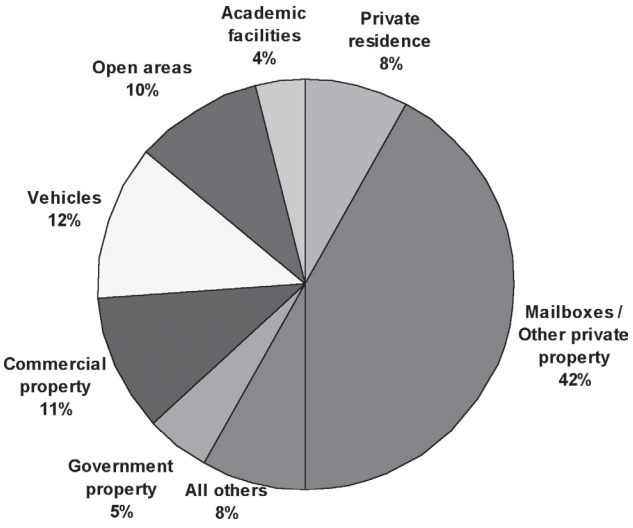
AGE — Arterial gas embolism
ANFO — Ammonium nitrate-fuel oil (explosive)
ATF — Bureau of Alcohol, Tobacco, Firearms and Explosives
BLEVE — Boiling liquid expanding vapor explosion
CT — Computed tomography
C-4 — Composition C-4 (explosive)
DPL — Diagnostic peritoneal lavage
FAST — Focused abdominal sonography for trauma
FBI — Federal Bureau of Investigation
HE — High-order explosive
IED — Improvised explosive device
LE — Low-order explosive
PETN — Pentaerythritol tetranitrate (explosive)
RDX — Royal demolition explosive
TATP — Triacetone triperoxide, also called TCAP or acetone peroxide (non-nitrate high explosive)
TM — Tympanic membrane
TNT — Trinitrotoluene (explosive)

Critical Appraisal Of The Literature

For this review, MEDLINE®, Ovid, BestBETs (Best Evidence Topics), Google Scholar, and Google were all searched using the terms blast injury, explosions, bombings, and explosives. The terms were used in Boolean combination and separately in each database, as appropriate.

As might be expected, there were no prospective, randomized, placebo-controlled studies of any treatment. There were, however, multiple retrospective studies, analyses of case reports, animal studies, and many individual case reports and short series. The literature of blast injuries is replete with case reports and data mining from trauma registries. There are few meta-analyses and even fewer prospective studies. This is due partly to the nature of the injury: sudden, random, and unpredictable. Another reason is the dispersion in both time and space of these kinds of injuries. Although recent bombings have had a widespread effect, they generally do not occur in the same location frequently enough to start a randomized study of any treatment methodology. (**Figure 1**) The exceptions would be England and Ireland in the 1980s and 1990s, and the present-day Middle East, specifically Israel and the US military in Iraq. (All of these exceptions are due to a markedly increased number of bombings in a short period of time in the given locales.) Many of the case reports cited in this article are from researchers in England, Israel, and the United States military. There are no evidence-based ATLS data about bombings and explosive injuries. The military

Figure 1. Historic location of United States bomb incidents.



has trauma registries, but this information is not open to public scrutiny. There is a published summary of the joint US Navy-Marine Corps Combat Trauma Registry available at http://www.stormingmedia.us.

Epidemiology And Etiology

An explosion is an event that occurs when a substance rapidly releases energy and produces a large volume of gaseous products. High-explosive, thermobaric, and nuclear detonations all provide this change in potential energy to kinetic injury in a very short period of time. The extreme compression of molecules by this change in energy creates the blast wave that moves outward from the epicenter of the blast. These blast waves travel faster than the speed of sound. Blast products — gas, particles, debris of the container, and items in proximity to the explosive (including human remains) — also spread outward, but travel much more slowly. Both the blast wave and the blast products can cause injuries as described below.

Trauma caused by explosions traditionally has been categorized according to the following scheme: injury caused by the direct effect of the blast wave (primary injuries); effects caused by other objects that are accelerated by the explosive wave (secondary injuries); effects caused by movement of the victim (tertiary injuries); and miscellaneous effects caused by the explosion or explosives (sometimes termed quaternary injuries). (**Figure 2**)

The injury pattern following an explosion is partly random. Explosions have the potential to cause multisystem injuries involving multiple patients simultaneously. The trauma that results from an explosion depends on the combination of the size of the explosive charge, the nature of the explosive, the container and surrounding or contained items, any shielding or protective barriers between the victim and the explosion, the surrounding environment, the method of delivery, and the distance between the explosion and the victim.

Nature of Explosives

A conventional explosion is the rapid chemical conversion of a solid or liquid into gas. Thermobaric explosives (commonly called fuel-air explosives) are either gases mixed with air or finely divided particles or droplets suspended in air. Explosives are categorized as either high- or low-order, and they cause somewhat different injury patterns. The explosive effects of nuclear weapons will not be discussed in this article.

Figure 2. Illustration of primary, secondary, and tertiary injury from blast.

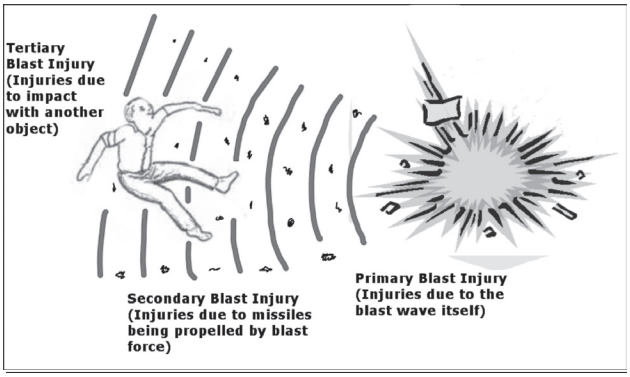


Illustration by Charles Stewart, MD. Used with permission of the author, © 2006, Charles Stewart. All rights reserved.

High-order Explosives

High-order explosives (HE) are chemical materials that have an extremely high reaction rate. This reaction is often called a detonation. (**Table 1**)

When a high explosive detonates, it is converted almost instantaneously into a gas at very high pressure and temperature. For example, the major ingredient in composition C-4 or RDX (cyclotrimethylenetrinitramine) can generate an initial pressure of over 4 million pounds per square inch ($4x10^6$ psi).[12] These high-pressure gases rapidly expand from the original volume and generate a marked pressure wave — the "blast wave" — that moves outward in all directions. The result is a sudden, shattering blow on the immediate surroundings.

Table 1. High-Order Explosives.

- Nitroglycerine
- Dynamite
- C-4 (Composition C-4 is a plastic explosive mixture of RDX explosive, plastic binders, and plasticizers)
- Picric acid
- Semtex is a general-purpose plastic explosive (first made by the Semtin Glassworks in the former Czechoslovakia, hence the name). It is similar to the US composition C-4 in characteristics and use.
- Dynamite (A mixture of diatomaceous earth and nitroglycerin patented by Alfred Nobel in 1867)
- Ammonium nitrate-fuel oil (ANFO) mixture
- TNT (trinitrotoluene)
- PETN
- TATP (triacetone triperoxide, a nonnitrate high explosive)

High explosives are further categorized as primary and secondary high explosives. The primary-high explosive is very sensitive, can be detonated very easily, and generally is used only in primary and electrical detonators. Secondary high explosives are less sensitive, require a high-energy shock wave to achieve detonation, and are generally safer to handle.

The blast wave refers to an intense rise in pressure — often called "overpressure" — that is created by the detonation of a high explosive.[2] A typical pressure wave from a high explosive explosion in air is shown in **Figure 3**.

The blast wave transfers energy to objects or bodies in its path. The extent of damage due to the pressure wave is dependent on:

- The peak of the initial positive pressure wave (an overpressure of 60-80 psi is considered potentially lethal)
- The duration of the overpressure
- The medium in which it explodes
- The distance from the incident blast wave
- Focusing due to a confined area or walls

As shown in **Figure 4**, the blast wave has 3 components:

1. A single spike of increased pressure. The leading edge of the blast wave is called the blast front and is represented by this spike. The actual blast wave is only a few millimeters thick. This spike is also the most important factor in the pathology of primary blast injury.
2. An exponential decay with time.
3. A much longer duration negative pressure wave, with pressure below initial ambient pressure.

In air, the peak pressure is proportional to the cube root of the weight of explosives and inversely proportional to the cube of the distance from the detonation.

This shock wave can be so abrupt that it shatters materials. This effect is termed brisance (the measure of the rapidity with which an explosive develops its maximum pressure) — a quality that varies from high explosive to high explosive. When craters are formed at the site of an explosion, this shock wave has disintegrated the material close to the explosion. Because the explosive gases continue to expand outward, the pressure wave rapidly deteriorates into an acoustic wave. Until the wave deteriorates enough to completely engulf the body simultaneously, tissue damage will depend on both the magnitude of the pressure spike and the duration of the force (represented by the area under the curve).

A blast wave that would cause only modest injury in the open can be lethal if the victim is in a confined area or near a reflecting surface, such as a solid wall or a building.[2] If the pressure wave is near a solid barrier, the pressure exerted at the reflecting surface may be many times that of the incident blast wave.

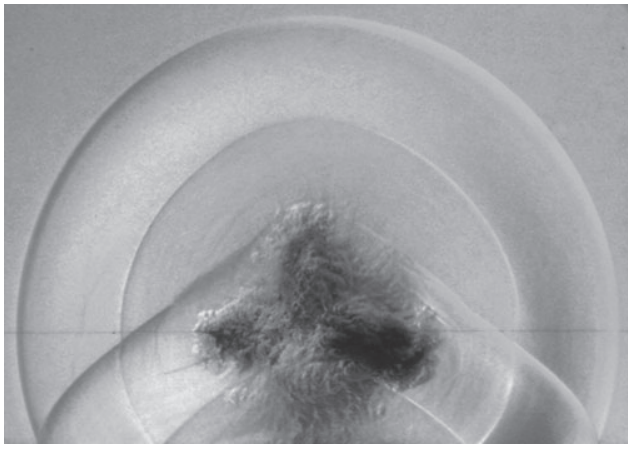
For a single, sharp rising blast wave caused by detonation of a high explosive, the damage to human structures is a function of the peak pressure and the duration of the initial positive phase. The greatest energy transfer occurs at points where tissue density changes. Energy transfer at a bone/soft tissue interface may partially amputate limbs.[13] **Figure 5** illustrates the estimated blast levels needed to cause damage in humans.

Blast wind refers to the rapid bulk movement of air and other gases from the explosion site. It occurs with both low-order and high-order explosives. Some explosives are manufactured to produce a relatively low-energy blast wave, but large amounts of gaseous products. These explosives produce a sustained blast wind and localized heaving with minimal blast. They are particularly useful in mining and demolition projects.

Low-order Explosives

Low-order explosives are designed to burn and subse-

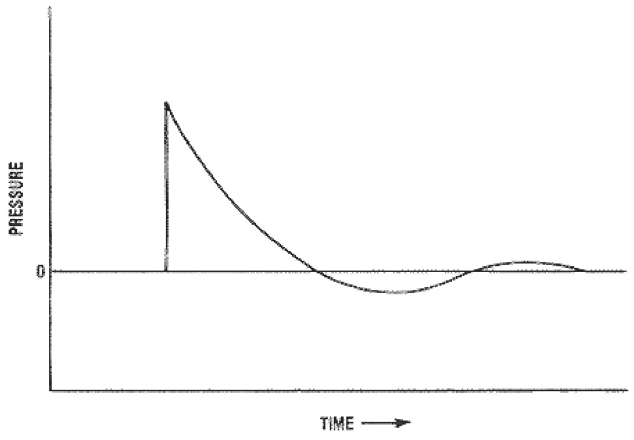
Figure 3. Blast waves from explosion.



Courtesy Harald Kleine. Used with permission, © 2006, Harald Kleine. All rights reserved.

Figure 4. Pressure-time graph of blast wave.

Idealized representation of pressure-time history of an explosion in air.



Courtesy Virtual Naval Hospital, Emergency War Surgery NATO Handbook. Available at: http://www.vnh.org.

quently release energy relatively slowly. These explosives are often called propellants, because their most common use is to propel a projectile through the barrel of a weapon. The principle military uses for low-order explosives are as propellants and in fuses. Typical improvised low-order explosives include pipe bombs, gunpowder, black powder, and petroleum-based bombs, such as Molotov cocktails or gasoline tankers. Since low-order explosives do not form shock waves, they do not have the quality of brisance.

The process of rapid, progressive burning of a low-order explosive is called deflagration. This burning takes place so slowly that when the low-order explosive is set off in the open, the gases push aside the air with only a flame and no appreciable disturbance. If the low-order explosive is confined, the speed of the reaction is markedly increased, but does not approach that of a high-order explosion. The explosion has more of a pushing effect than a shattering effect (ie, blast wind without a blast wave).

The explosion from low-order devices lacks the overpressure wave; thus, injuries are due to ballistics (fragmentation), blast wind from the expansion of the gases, and thermal injuries from the heat of the explosion. Obviously, it is clinically impossible to tell whether fragment wounds have occurred because the fragment was propelled by a high-order versus a low-order explosive. Likewise, if the victim is flung by a blast wind into a structure, it matters little to either the patient or the clinician that the explosion occurred from detonation of a high-order explosive or deflagration of a low-order explosive.

Special Explosives

Thermobaric or Fuel-Air Explosives

In this explosive device, a substantial quantity of fuel is vaporized and mixed with air. Fuel-air explosives represent the military application of the vapor cloud explosions and dust explosion accidents that have long plagued a variety of industries. Firefighters are all too familiar with the explosive effects of this device. (**Table 2**)

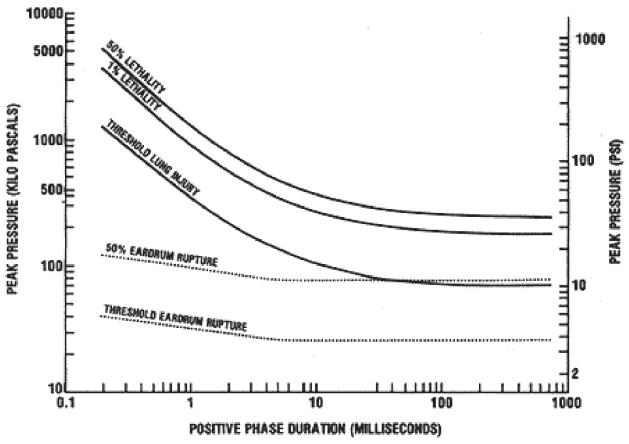
Since these explosive mechanisms are not uncommon in the civilian world, the emergency physician needs to know the special effects of this form of explosive. An astute terrorist could use these mechanisms to create a massive explosion.

In the military device, mixture of the fuel with air over the target may be accomplished by a dispersal charge. After the munition is dropped or fired, the first explosive charge bursts open the container at a predetermined height and disperses the fuel in a cloud that mixes with atmospheric oxygen (the size of the cloud varies with the size of the munition). The cloud of fuel flows around objects and into structures. After the fuel and air are mixed, a second detonation provides the spark needed for ignition.

There are dramatic differences between explosions involving fuel-air mixtures and high explosives at close distances. The shock wave from a trinitrotoluene (TNT) explosion is of relatively short duration, while the blast wave produced by an explosion of fuel-air mixture displays a relatively long duration. The duration of the positive phase of a shock wave is an important parameter in the response of structures to a blast. The temperature can be as high as 3000°C — more than twice that generated by a conventional explosive. The blast wave can travel at approximately 10,000 feet per second.

The blast effects from vapor cloud explosions are determined not only by the amount of fuel, but more importantly by the combustion mode of the cloud. Most vapor cloud explosions are deflagrations, not detona-

Figure 5. Estimated blast energy needed to cause damage in humans.



Courtesy Virtual Naval Hospital, Emergency War Surgery NATO Handbook. Available at: http://www.vnh.org.

Table 2. Thermobaric/Fuel-Air Explosives.

- Dust/air mixtures in grain silos and other storage or construction areas*
- Slowly escaping natural (or other flammable) gases†
- BLEVE (Boiling Liquid-Expanding Vapor Explosions)‡[14]

*Many materials form dust clouds that can ignite and explode, injuring personnel and damaging equipment. This is a well-publicized occurrence in the coal mining, grain storage, and the woodworking and paper industries. Many miners have been killed and injured and massive production losses have resulted from coal dust explosions in underground coal mining operations. Between 1987 and 1997, 129 grain dust explosions occurred nationwide. Of these dust explosions, about half involved corn and corn products, while 11 were caused by wheat dust and 10 by dust from soybeans.

†A Google search on this topic yielded over 265,000 entries.

‡A BLEVE is a type of pressure-release explosion that occurs when liquefied gases, which are stored in containers at temperatures above their boiling points, are exposed to the atmosphere, causing rapid vaporization. The result is the mixing of vapor and air that results in the characteristic fireball that occurs when the fire ignites the vapor. This happens when a container fails or is ruptured by an accident. A BLEVE can also occur when flame impinges on the tank shell at a point or points above the liquid level of the tank's contents. The heat from the fire causes the metal to weaken and fail as the internal pressure increases.

tions.[15] Flame speed of a deflagration is subsonic, with flame speed increasing in restricted areas and decreasing in open areas.

Flame propagation speed has a significant influence on the blast parameters, both inside and outside the source volume. High flame front speeds and resulting high blast overpressures are seen in accidental vapor cloud explosions, where there is a significant amount of confinement and congestion that limits flame front expansion and increases flame turbulence. These conditions are more difficult to achieve in the unconfined environment in which military fuel-air explosives are intended to operate.

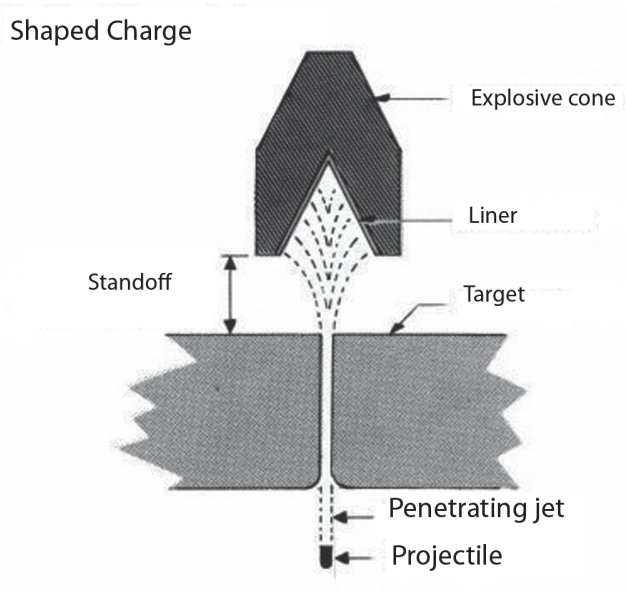
Since the fuel uses up the atmospheric oxygen, asphyxia for those who are not immediately killed by the explosive device can be a problem. Likewise, since the temperature of the burning fuel is greater than that of conventional explosives, extensive burns can occur in survivors.

Explosively Formed Projectiles and Shaped Charges

Charles Edward Munroe coined the term "The Munroe Effect" in 1885. He noted that a high explosive with a cavity facing a target left an indentation. The earliest known reference to the effect appears to be 1792, and there is some indication that mining engineers may have exploited the phenomenon over 150 years ago. A typical shaped charge consists of a solid cylinder of explosive with a conical hollow on one end, lined with a dense ductile metal, such as copper. (**Figure 6**) When detonated from the other end, the force of the explosive detonation wave is great enough to project the copper into a thin, effectively liquid stretching that has a tip speed of up to 12 km/sec. The enormous pressures generated cause the target material to yield and flow plastically.

Explosively formed projectiles (EFPs) are related to shaped charges, but form a fragment rather than a jet. A computer-designed, dish-shaped metal liner is placed in front of a shaped explosive charge. These explosive devices with wide-angle cones and other liner shapes, such as plates or dishes, do not jet, but instead give an explosively formed projectile. (**Figure 7**) When the explosive is detonated, the shock wave deforms the liner in a preset way to create a symmetric projectile traveling at very high speeds. Varying the liner shape and explosive confinement changes the shape and velocity. These sophisticated devices have been used in Iraq against Allied forces. They routinely defeat armor and can cause significant injuries. (**Figure 8**)

Figure 6. Shaped charge.

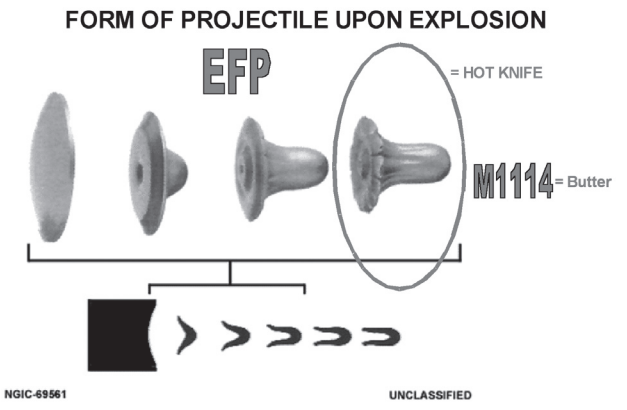


Source of Explosive

Explosive devices may also be characterized based on their source. The Bureau of Alcohol, Tobacco, Firearms and Explosives (ATF) categorizes explosives into "manufactured" and "improvised." A "manufactured" explosive implies a standard, mass-produced, and quality-tested weapon. "Improvised" describes the use of alternative materials, weapons produced in small quantities, or a device that is used outside of its intended purpose. Improvised explosive devices (IEDs) may be professional in appearance, and their operation may be quite lethal, if designed by someone with training in explosives. (Note that by this definition, any experimental explosive device is an "improvised" device, since it is not set to standards, mass-produced, and quality-tested. This rather unwieldy definition encompasses all experimental military devices produced by professional arms manufacturers.)

Improvised explosive devices (and many military munitions) can be triggered in a variety of ways, including electronic transmitters and switches, tilt switches, thermal switches, and various types of motion detectors. Improvised weapons vary in quality of the explosive used, from commercial explosives, TNT, Semtex, C-4, ammonia-based fertilizer, and fuel oil (used widely as an industrial explosive) to a simple, match-filled pipe bomb. High-quality IEDs may resemble military weapons in effect and appearance. The variety of initiation methods, explosive fillings, and fabrication techniques creates a threat that can be quite daunting to the professional explosive ordinance

Figure 7. Explosively formed projectile.



disposal crew.

Recent improvised devices have been manufactured with nonnitrogen explosives (TATP) in order to defeat explosive sniffing devices and dogs. These nonnitrogen explosives are often quite unstable and may spontaneously detonate — this means that, no matter how innocuous an improvised device may appear, the amateur should *never* touch the device.

Medium in Which the Explosion Occurs

Another important factor that defines blast injury patterns is the medium in which the blast occurs. An underwater blast wave causes far more damage, because water is essentially incompressible.[2,16] A wave resulting from an underwater blast travels farther and moves faster than a wave from a similar explosion in the air. Blast injuries in water occur at greater distances and may be much more severe.[17,18] Personnel treading water are at higher risk for abdominal than thoracic injury from an underwater explosion. Fully submerged victims are at equal risk of combined thoracic and abdominal blast injuries, but the blast injury occurs at 3 times the distance from the underwater explosion.[19]

Another characteristic of blast waves is that they are indeed true waves. (**Figure 3** on page 4) The injury patterns they produce are not only related to the medium through which they travel, but the position of the victim's body in relation to reflecting or deflecting objects that the wave strikes. For example, explosions near or within hard, solid surfaces become magnified 2-9 times as the shock wave is reflected.[20] In fact, victims located between the blast and a building generally suffer 2-3 times the degree of injury that an individual in an open environment would receive.[21]

Pathophysiology

Blast injury has an overall lethality of about 7.8% in open air; this jumps to 49% when the blast occurs in a confined space. One meta-analysis reported a 70% incidence of minor soft tissue injuries.[21] Traumatic amputations will occur in about 11% of cases. Traumatic amputations serve as a marker of severe multisystem trauma and subsequent high mortality.[13] The World Trade Center was an exception in that most victims had either few injuries or died as the building crashed down on them.

Primary Blast Injury

Primary blast injuries are caused only by high explosives and are due to the direct effects of the blast wave on the human body. (Since low-order explosives do not form a supersonic blast wave, they cannot cause primary blast injury. This is the sole clinical difference between wounds caused by a low-order explosive and a high-order explosive.)

The overall incidence of primary blast injury in soldiers in combat is about 5%.[22] Cernak retrospectively examined the records of 1303 patients and found evidence of primary blast injury in 51% of admitted patients.[23] (The victims of primary blast injury almost always have other types of injury, such as penetrating wounds from flying debris or blunt trauma from impact on immovable objects).[23]

Damage from an explosion (**Table 3**) depends upon:

- The peak of the initial positive wave, which is directly related to the magnitude of the explosion and to the proximity of the victim to the explosion
- The duration of the overpressure
- The medium in which it explodes
- The distance from the incident blast wave
- The nature and number of reflections in confined areas and with reflecting walls

Figure 8. Results of explosively formed projectile impact on a human being.

Photograph by Maj Paul Morton, USAF. Used with permission.

Table 3. Blast Injury Pressure Versus Injury.

Overpressure (psi)	Effect
1-2	Frame house destroyed
3-5	Typical commercial construction destroyed
5	Tympanic membrane rupture (threshold)
15	Tympanic membrane rupture in 50% of patients
30-40	Possible lung injury (threshold)
40	Reinforced concrete construction destroyed
75	Lung injury in 50% of patients
100	Possible fatal injuries
200	Death most likely

*Adapted from: Rice DC, Heck JJ. Terrorist Bombings: Ballistics, patterns of blast injury and tactical emergency care.[20]

Mechanism of Primary Blast Injury

In World War II, blast overpressures were thought to gain access to internal organs through natural orifices.[24] This has since been proven inaccurate. Other, more recent theories that have been proposed include implosion of gas-containing structures, inertial effects on tissues of different densities, and spalling at water-gas interfaces.

The most likely mechanism of primary blast injury that fits current modeling techniques is the irreversible work effect related to the differences in tissue tensile strength and speed of the blast wave through the different tissues. This is currently thought to be the major cause of primary blast injuries.[25] The onset of damage occurs when the blast wave compresses the tissues. The resulting forces exceed the tensile strength of the material and cause shearing of vascular beds, pulmonary contusions, and gastrointestinal hemorrhages as the tissues are compressed and expanded.[26,27]

The illustration often used is of an aluminum can that is dented slightly and pushed back into shape. When the can is stressed beyond its tensile strength, it can no longer be restored to its original shape.[28]

Some combination of stress and shear waves is likely in all nonpenetrating blast trauma. Stress that exceeds tissue tensile strength probably predominates when blast surface loading exceeds velocities of 80-90 m/sec.[25]

Primary blast injury is common in the ear, the respiratory tract, and the gastrointestinal tract. Of the 3 organ systems, the ear is the most easily damaged.

Ear Damage

While the ear is the most easily damaged organ system in primary blast injury, it is also the easiest to protect. The structures of the ear are designed to collect and magnify sounds, so that the tympanic membrane will move with the sounds. Unfortunately, they also collect and magnify pressure waves. At a pressure of about 35 kilopascals (5 psi), the human eardrum may rupture. With an overpressure of 100 kilopascals (14 psi), almost all eardrums will be ruptured. The eardrum most frequently ruptures into the inferior pars tensa. At lesser pressures, the overpressure may cause hemorrhage into the drum without a rupture. With extremely high pressures, the drum may be destroyed and the ossicles dislocated or fractured.[29] Rupture of the eardrum will cause pain, hearing loss, and possibly tinnitus. Eardrum perforations, hearing loss, and dizziness may interfere with daily activities and may have a telling effect on the individual's quality of life.[29]

Physical examination may reveal blood in the external canal. Examination of the tympanic membrane with an otoscope may show evidence of the perforation or of a hematoma of the tympanic membrane.

It is often held as gospel that rupture of the tympanic membrane is a marker for serious gastrointestinal or pulmonary injury. If the patient has ear protection, this may not be the case. Likewise, if the patient is in the water, but with their head out of the water, the tympanic membranes may not be exposed to an underwater blast wave. Even in those exposed to simple blast injury, isolated eardrum rupture does not appear to be a good marker of concealed pulmonary blast injury or poor prognosis.[30]

Auditory barotrauma is quite common in blast injuries. In the Oklahoma City bombing, the incidence of auditory injury was 35%.[4,31] This does not include those patients with partial, temporary hearing losses or those who complained of tinnitus for an extended period of time.[29]

Figure 9. Illustration of pulmonary injury mechanisms.

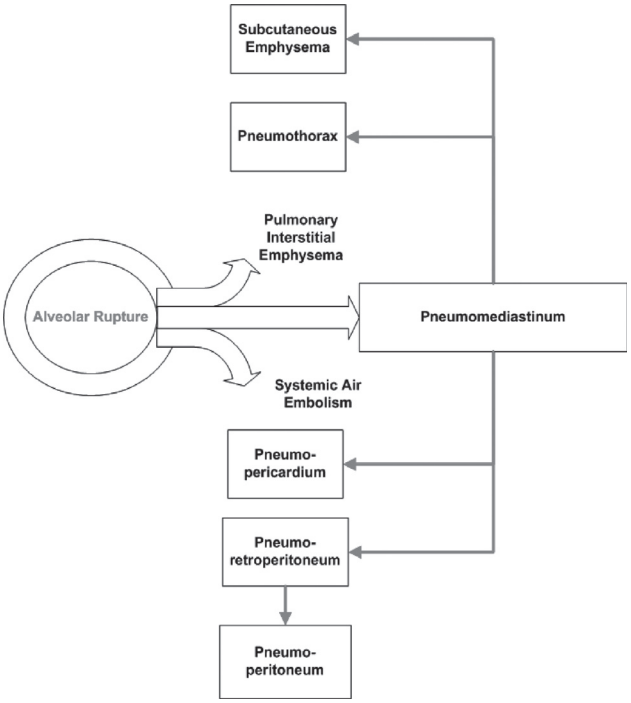


Illustration by Charles Stewart, MD. Used with permission of the author, © 2006, Charles Stewart. All rights reserved.

Pulmonary Damage

The lungs are particularly susceptible to damage due to the extensive air/lung tissue interfaces. Blast lung is a direct consequence of the supersonic pressure wave generated by a high explosive.[32] It is the most common fatal injury caused by the primary blast among the initial survivors of the explosion. These lung injuries may not be apparent externally or immediately, but may lead to death if not diagnosed and treated promptly. An overpressure of about 40 psi will cause lung injuries.

Pulmonary blast effects in survivors have been described as rare in the British literature, but are observed more often in the Israeli experience, with enclosed explosions that occur on a bus.[33,34]

Damage to the lungs can include pulmonary contusions and/or pulmonary barotrauma, such as pneumothorax, pulmonary interstitial emphysema, pneumomediastinum, or subcutaneous emphysema. The most common lung injury associated with a blast wave is a pulmonary

contusion.[35,36] This may take the form of microhemorrhages with perivascular/peribronchial disruption. It appears to be more common on the side closest to the explosion, but this may be influenced by the geometry of the surrounding area and reflected energy.[35-37] The alveolar wall may be torn, causing a blood-filled emphysematous change to the lung and subsequent hemoptysis. Pulmonary contusions may develop with or without a pulmonary laceration.

It should be assumed that if a patient is wheezing after a blast injury, then this wheezing is due to a pulmonary contusion. Other causes of wheezing may be pulmonary edema from myocardial contusion or infarction, or exacerbation of underlying disorders, such as asthma or COPD. If the patient has hemoptysis after a blast injury, the clinician must entertain a high suspicion for pulmonary blast injury.

Pulmonary contusions impair gas exchange at the alveolar level. The changes seen on microscopic examination closely resemble the pulmonary contusions seen in nonpenetrating blunt chest trauma. The histologic appearance of lung damage by blast overpressure is dominated by hemorrhage into the alveolar spaces. The degree of respiratory insufficiency depends on the magnitude of hemorrhage into the lung.

Parallel thoracic ecchymoses, once thought to be along the ribs, may be seen with larger blast loads.[28,36] These ecchymoses parallel the intercostal spaces. Rib fractures may occur due to blast injury, but are much more likely to be due to secondary or tertiary blast injury mechanisms, at least in survivors.[35,38]

The patient may have minimal or no symptoms initially. Blast lung is clinically characterized by the triad of dyspnea, bradycardia, and hypotension. The clinician should suspect blast lung in any victim who presents with dyspnea, cough, hemoptysis, or chest pain following blast exposure. Signs of blast lung are usually present at the time of the initial evaluation, but have been reported as late as 48 hours after the explosion occurs.

The occurrence of late pulmonary symptoms in primary lung blast injury has recently been questioned by Pizov et al, who described 15 patients with primary lung blast injury.[38] All of the patients required intubation and ventilation, either at the scene of the explosion or on admission to the ED. No patient in their series developed blast lung that did not require ventilatory support within the first 6 hours after the injury. It should be noted that all of the patients in this series were victims of blast injury within the enclosed confines of a "bus bombing." As noted earlier, blast pressures within enclosed areas are often much higher.

The overpressure may cause pulmonary barotrauma, including pneumothorax or pneumomediastinum. (See **Figure 9** for an illustration of pulmonary injury mechanisms.) The patient may develop pulmonary interstitial emphysema, subcutaneous emphysema, and systemic air embolism with larger blast loads.[26-28,39] Significant bronchopleural fistulae may lead to air embolism. Air emboli may present in a variety of ways, including shock, myocardial infarction, spinal infarction, or cerebrovascular accident. (See **Table 4** for correlation of severity and injury frequency.)

A simple frontal chest x-ray is diagnostic for most cases of pulmonary barotrauma from blast. (**Figure 10** on page 10) Blast lung produces a characteristic "butterfly" pattern on chest x-ray. The pulmonary injuries found may range from scattered isolated petechiae to confluent pulmonary hemorrhages. The radiographic evidence of pulmonary injury usually begins within hours of the explosion and begins to resolve within 1 week.[40]

Gastrointestinal Damage

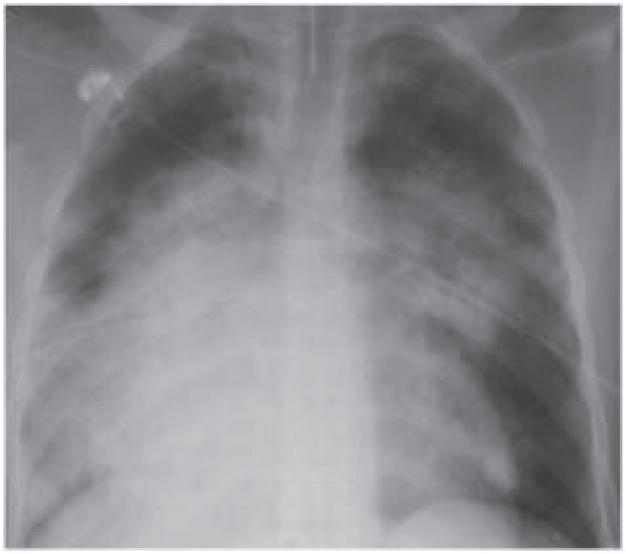
Gastrointestinal injuries were once thought to occur with the same frequency as lung injury. A recent, large Israeli case series found that abdominal injuries were seen only with massive trauma.[33] In this series, all of the patients

Table 4. Blast Injury Severity.

Status of Injury	Findings	Therapy
Insignificant	None	None
Mild	SpO_2 >75% on room air May have pneumothorax Rare bronchopleural fistulae	Low PEEP (<5 cm H_2O) and no positive pressure ventilation Unlikely to need PPV
Moderate	SpO_2 >90% on 100% supplemental oxygen Pneumothorax common Bronchopleural fistulae possible	PEEP (5-10 cm H_2O) and positive pressure ventilation
Severe	SpO_2 <90% on 100% supplemental oxygen Pneumothorax almost universal Bronchopleural fistulae common	PEEP (>10 cm H_2O) and pressure controlled ventilation

*Adapted from: Pizov R, Oppenheim-Eden A, Matot I, et al. Blast lung injury from an explosion on a civilian bus. *Chest* 1999;115:165-172 and Wightman J. Blast injuries: Recognition and management. Available at: http://www.brooksidepress.org/Products/OperationalMedicine/DATA/operationalmed/MilitaryMedicine/Blast%20Injuries/BlastInjuriesRecognitionandManagement.htm. Accessed April 4, 2006.

Figure 10. Radiograph of blast lung.



Reprinted from *Surg Clin North Am*, Vol 82(1), Wolf YG, Rivkind A, Vascular trauma in high-velocity gunshot wounds and shrapnel-blast injuries in Israel, pages 237-244, ©2002, with permission pending from Elsevier.

sustained their injuries in open air. The patient may have a greater risk for gastrointestinal injury when exposed to an underwater blast.[17]

The GI injury of primary blast injury is inconsistent in presentation. Gastrointestinal injuries may not be apparent externally. They have a great potential to cause death and may be much more difficult to protect against. The injury may consist of hemorrhage beneath the visceral peritoneum or may extend into the mesentery, colon, and cecum.[36,37] The colon is the most common site of both hemorrhage and perforation.[17,36] This is thought to be because the colon has the most bowel gas accumulation in the GI tract. Contused bowel may necrose and perforate several days after the initial trauma. The perforated bowel may be immediately apparent or may perforate only after a delay of up to 48 hours.[41,42]

Pneumoperitoneum is a known but relatively rare complication of GI barotrauma.[43] This complication has a wide differential diagnosis, ranging from perforated viscus to simple dissection of air through the retroperitoneum.

Solid organ laceration and testicular rupture are also seen due to primary blast injury, but they are less frequent and are often associated with large blast forces.[44] The most common solid organ lesions reported were subcapsular hematomas in the liver, spleen, and kidneys.[40] Mesenteric, scrotal, and retroperitoneal hemorrhages have been reported.[36]

These lesions can lead to the clinical signs of absent bowel sounds, bright red blood per rectum, guarding and rebound tenderness. The clinical symptoms can include abdominal pain, nausea, vomiting, diarrhea, and tenesmus. Blast injury to the gastrointestinal tract should be suspected in anyone who has abdominal pain, nausea, vomiting, hematemesis, rectal pain, testicular pain, unexplained hypovolemia, or any finding compatible with an acute abdomen after exposure to an explosion.

The clinician should be aware that the abundant high-velocity fragments associated with recent suicide bombs may also cause intraabdominal injuries. These injuries can certainly include penetrating bowel injuries.[45] Initial symptoms of penetration are the same as outlined above.

Brain Injury

Primary blast injury can cause concussion or traumatic brain injury, although this finding is difficult to distinguish from the concussion due to impact with another object. Likewise, high-velocity fragments can penetrate the skull. The clinician should be quick to consider CT or MRI in these patients.

Cardiac Injury

Although the heart is well protected and not subject to the air/fluid shear of primary blast injury, myocardial contusion can lead to either arrhythmia or hypotension.[25]

Secondary Blast Injury

Secondary blast injury is caused by the bomb fragments and other debris propelled by the intense energy release of an explosion. (These fragments are often erroneously referred to as "shrapnel." Shrapnel is actually the name for an artillery round containing multiple spherical projectiles and an explosive charge. This antipersonnel projectile was designed in 1784 by Major-General Henry Shrapnel, an English artillery officer. The round essentially functions as a very large shotgun, with several hundred half-inch lead balls.) As distance from the blast epicenter increases, the effect of the blast itself is reduced, and the effect of fragments and debris propelled by the explosive becomes

Figure 11. Multiple fragment wounds from blast injury.

Photo courtesy of Professor Zvi Gimmon, MD.

more important. Conventional military explosives may create multiple fragments, with initial velocities of up to 2500 m/sec (8202 feet per second).[46] (In comparison, the very fast-moving M-16 round has a muzzle velocity of 853 meters (2800 feet) per second.)[47]

These flying projectiles can produce both penetrating and blunt trauma, depending on the size of the projectiles and the speed at which they travel. With these velocities, the victim does not have to be in close proximity to the explosion. Individuals far from the scene of an explosion can be struck and injured by this debris. After the 1998 terrorist bombing of the US Embassy in Nairobi, flying glass wounded victims up to 2 kilometers away.[25] For US Air Force personnel wounded in the Khobar Towers in 1996, 88% of patients were injured by flying glass.[48] (The reason for the "stand-off distances," noted in **Table 5**, is to decrease to acceptable limits the number of injuries that occur from flying debris when the bomb explodes.) The farther away the explosion occurs, the less serious the injury.

Terrorist devices often have additional objects, such as nails, nuts, and bolts, added to the explosive mixture in order to increase the effects of secondary blast injury. (**Figure 11**) These fragments are of high mass and kinetic energy and the damage that they inflict at close range is considerable. Military devices, such as shells and grenades, may be designed in such a way as to increase the number of fragments flung by the explosion.

Secondary blast injury is much more common than primary blast injuries. Indeed, secondary blast injury is the most common cause of death in blast victims. The penetrating injuries occur most often in the exposed areas, such as the head, neck, and extremities. (**Figure 12** on next page) Thoracic and intraabdominal injuries may occur when fragments penetrate.[45] (**Figure 13** on next page)

Glass causes many of the secondary blast injuries (up to 50% of all blast injuries). Victims who are peppered with glass are often difficult to distinguish from victims

Table 5. Bomb "Stand-Off" Distances.*

	Container or Vehicle Description	Maximum Explosives Capacity	Lethal Air Blast Range	Minimum Evacuation Distance	Falling Glass Hazard
Pipe	2" x 12"	5-6 pounds		850 feet 259 meters	
	4" x 12"	20 pounds			
	8" x 24"	120 pounds (uncommon)			
Bottle	2 liter	10 pounds			
	2 gallon	30 pounds			
	5 gallon	70 pounds (uncommon)			
Boxes	Shoe box	30 pounds			
	Briefcase Satchel bomb	50 pounds		1850 feet† 564 meters	1250 feet 381 meters
	1 cubic foot box	100 pounds (uncommon)			
	Suitcase	225 pounds (uncommon)		1850 feet† 564 meters	1250 feet 381 meters
Vehicles	Compact sedan	500 pounds (227 kilos) in trunk	100 feet 30 meters	1500 feet 457 meters	1250 feet 381 meters
	Full size sedan	1000 pounds (455 kilos) in trunk	125 feet 38 meters	1750 feet 534 meters	1750 feet 534 meters
	Passenger van or cargo van	4000 pounds 1818 kilos	200 feet 61 meters	2750 feet 838 meters	2750 feet 838 meters
	Small box van	10000 pounds 4545 kilos	300 feet 91 meters	3750 feet 1143 meters	3750 feet 1143 meters
	Box van or water/fuel truck	30000 pounds 13636 kilos	450 feet 137 meters	6500 feet 1982 meters	6500 feet 1982 meters
	Semi-trailer	60000 pounds 27,273 kilos	600 feet 183 meters	7000 feet 2134 meters	7000 feet 2134 meters

*Although bomb "capacities" have been listed for improvised devices, the reader should realize that the bomb maker is not limited to these dimensions and may use different materials with smaller or larger capacities. Remember that, for pipe bombs, in particular, bomb makers may often use multiple containers. Stand-off distances for improvised devices that are not given may be estimated from given distances for smaller and larger devices. Whenever estimating, a larger stand-off distance is safer!

†The table is built from several ATF and TSWG publications. The author is unsure why they have a larger stand-off distance for a briefcase than for a small car. Certainly by explosive weight, this distance should be about midway between the car and the pipe bomb...

who are peppered with glass *and* have penetrating injuries.[49] The clinician must be suspicious of any penetrating torso or abdominal injury.

Secondary blast injuries may not be initially obvious. A seemingly small abrasion or wound may mask the entrance wound for a substantial fragment.

Up to 10% of blast survivors will have significant eye injuries.[50] (**Figure 14**) These injuries may be perforations from high-velocity projectiles. Glass is notorious for causing these ocular injuries. While window fragments are not often lethal, they can cause blindness and ruptured globes. At the speeds at which explosively propelled fragments of glass travel, there is no time for the blink reflex to operate. These injuries can occur with minimal initial discomfort and can present days after the event. Symptoms include eye pain and irritation, foreign body sensation, alterations of vision, periorbital swelling, or periocular contusions. Signs can include loss of vision, decreased visual acuity, globe perforation or rupture, lid lacerations, and subconjunctival hemorrhage around the point of entry.

Tertiary Blast Injuries

Tertiary blast injuries are caused when the victim's body is propelled into another object by the blast winds.[28,51] Tertiary effects result from the bulk flow of gas away from the explosion. Blast winds can generate a body acceleration of over 15 g's. They most often occur when the victim is quite close to the explosion.

This displacement of the victim can take place relatively far from the point of detonation if the victim is unfortunately positioned in the path that gases must take to vent from a structure, such as a doorway, window, or hatch. Likewise, if the patient is in an alley, magnification of the blast wind may occur due to the configuration of the buildings.

It is the deceleration caused by impact into a rigid structure that causes the majority of injuries. A person who is flung into a fortified immovable object with a velocity greater than 26 ft/sec will have a mortality rate of about 50%.[52] The most common injuries are fractures and closed head injuries. Isolated body parts may be broken, dislocated, or even amputated. Injuries from this mechanism also depend on what the victim hits in the environment and can range from simple contusions to impalement. Victims may also tumble along the ground, sustaining abrasions, contusions, and "road rash."

Miscellaneous Blast Effects (Quaternary Blast Injuries)

This category of blast trauma includes burns from fire or radiation, crush injury associated with structural collapse, poisoning from carbon monoxide or other toxic products of the explosion, and inhalation of dust or chemicals from the explosion. This category would include the burns sustained from thermobaric weapons.

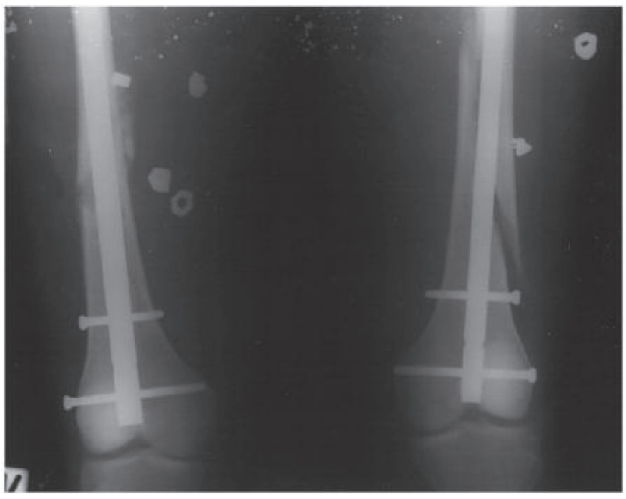
The unprotected human body can survive a blast with a peak overpressure of 30 psi, but buildings and other structures collapse with stress of only a few psi. (**Table 3** on page 7) This means that people can survive the effects of a blast, only to be injured by collapsing buildings.

The blast may be a vector for both chemical warfare agents and biological warfare agents. The effects of these agents on the body may well overshadow any effects from the explosive energy.

Patients who have been exposed to a blast in an enclosed area should have carboxyhemoglobin levels

Figure 12. Blast injury due to bolts as missiles.

Multiple nuts in both thighs. The force imparted was sufficient to fracture both femurs, which have been repaired.



Reprinted from *Surg Clin North Am*, Vol 82(1), Wolf YG, Rivkind A, Vascular trauma in high-velocity gunshot wounds and shrapnel-blast injuries in Israel, pages 237-244, ©2002, with permission pending from Elsevier.

Figure 13. Radiograph of chest injury from bolts as missiles.

Chest x-ray showing bolts used as missiles in a suicide/homicide bomb.

Photo courtesy of Professor Zvi Gimmon, MD.

obtained. Inhalation of irritant gases or dusts may also trigger wheezing or even delayed pulmonary edema in these patients.

Prehospital Care

The job of the prehospital provider confronted with a major explosion becomes extremely difficult. The emergency services provider needs to ensure appropriate on-scene management, including triage, transportation to medical care, and appropriate distribution to hospitals with both facilities and skills to care for the victims. There may be additional problems due to unsafe or collapsed buildings, the dangers of further explosions, and civilian panic.[10]

An explosion that occurs in a confined space (including vehicles, mines, buildings, and subways) is associated with greater morbidity and mortality. If the structure collapses, this markedly increases the mortality associated with the event (assuming that there are people in the structure).

The early presentation of victims can be deceiving, because the initial manifestations of significant blast injury can be subtle. Blast lung injury is the most common fatal injury among initial survivors of the blast.[53]

The prehospital provider can aid the preparation of the hospital for reception of the victims by giving a preliminary needs assessment for the hospital(s). Identification of the site of the explosion is particularly helpful. Terrorist targets tend to be highly visible and may play an important operational or symbolic role in the community. The site also suggests the number of potential victims that may be involved. The location establishes the proximity to the hospital and the potential for those injured to arrive at the hospital within a few minutes by alternative transportation. Mutual aid agreements and location of the explosion may dictate transportation to other hospitals. Further information about the delivery system in a terrorist attack and whether the explosion occurred in open air or a confined space may help estimate needs for other resources.

Field Medical Care

- Initial care is similar to regular trauma care.
- Identify and correct ABCs of trauma care as rapidly as possible given constraints of multiple casualties.
- Identify and correct life-threatening external hemorrhage at once. This is the most common cause of preventable death on the battlefield.
- Liberal use of field tourniquets is encouraged in recent battlefield medicine guidelines.
- Ear and GI injury do not need special care in the field.
- Rapid evacuation increases the chance of survival.
- Do not attempt definitive care in triage.
- Do not attempt extensive resuscitation in the field. (As noted below, CPR at the scene of a mass casualty is not indicated.)
- Early (ie, from the field) normal vital signs may be an inaccurate guide to the severity of injury in patients with blast lung, barotrauma, and/or hemorrhage and rupture of gas-filled hollow organs.

Figure 14. Blast injury of the eye.

Rupture of eye with prolapsing iris due to explosion and direct trauma.

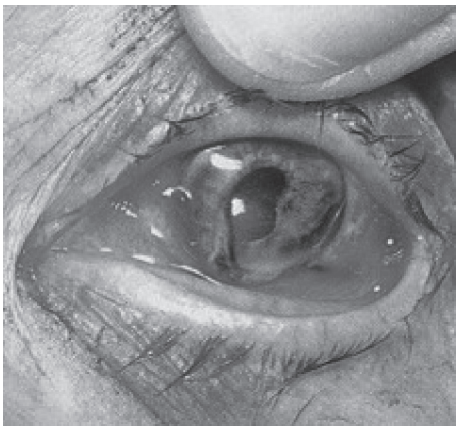


Photo from Richmond Eye Associates, PC. Available at: http://www.richmondeye.com/fireworks.htm. Permission pending.

If the blast casualty is ambulatory, it is critical to minimize physical activity. Exertion after blast injury can markedly increase the severity of the primary blast injury. This was seen in WWII, when some blast casualties appeared well, but died after vigorous activity.[54]

The EMS provider should be wary of secondary (and, on rare occasion, tertiary) devices and explosions. Foreign experience has shown that terrorists often will set a second device timed to explode some 30 to 100 minutes after the first device has detonated.[55,56] This second device is designed to injure EMS, fire, and police personnel who may be at the scene. This second device may often be larger than the first. In some cases, the perpetrator of the explosion may be watching over the area of the explosion, and will either remotely detonate the second explosive or employ high-powered rifle fire to injure or kill responders.

Remember to check all victims for weapons, booby traps, and explosives. It is quite common for a bomber to become a victim of his own device.

Fatal injuries can occur due to blast effects involving the head, chest, and abdomen and are often seen in victims who are close to the detonation.[57] Indeed, close to the site of the blast, parts of the victim (or perpetrator) can become missiles that kill or wound other victims.[58] Immediate death may occur from massive pulmonary bleeding with rapid suffocation, despite good care. The patient may develop a massive air embolism, may sustain a significant brain injury, or may suffer a traumatic amputation and exsanguinate before help arrives. Finally, the patient may have a crush injury or impalement injury that causes rapid death before extrication can occur.

The field physician or paramedic should consider a patient dead in the field when there is:

- An amputated body part in a patient without signs of life
- No effective respirations

- No palpable pulse
- Dilated pupils
- Immediate, severe respiratory insufficiency that is caused by a blast effect has far less chance of survival. (These cases may be triaged according to usual protocols when there are resources to care for all victims.)

CPR at the scene is never indicated. There will be too many injured, not enough medical providers, and no significant chance of successful resuscitation in this blunt trauma patient.

Finally, evacuation from the blast site to medical care can be problematic. The blast that caused the injuries may also degrade routes to and from the site of the explosion. Air transport can pose special problems. The barotrauma that results from primary blast injury can be exacerbated by air evacuation. Pneumothorax and arterial gas emboli will enlarge with ascent. Regardless of the altitude and distance of the flight, casualties with field evidence of pneumothorax must have a chest tube placed. Evacuation aircraft should fly at the lowest possible altitude. Evacuation by long-distance, high-altitude flights should be avoided. Evacuation aircraft should be pressurized to at least 8000 feet (preferably 5000 feet).

If the victim has marginal oxygenation (PO_2 <60 mm Hg), the clinician should recognize that oxygenation will worsen with ascent in an aircraft (with the increase in altitude and subsequent decreased barometric pressure) and consider intubation prior to transport.

ED Evaluation

The first priority of the emergency physician faced with the aftermath of an explosion is to activate the hospital's external disaster plan. During the period before the arrival of the first patients, the physician should clear the ED of all possible patients by either discharge or admission. The administration should simultaneously cancel all elective surgery cases, clear the recovery room, and clear as many intensive care beds as possible.

If your hospital is close to the explosion, expect that the most severely injured patients will arrive after the less injured. The less injured often skip EMS and proceed directly to the closest hospitals.[4] For a rough prediction of the "first wave" of casualties, double the first hour's casualty count.

Remember that a secondary device may be employed that can cause substantial additional casualties to include EMS, fire, police, and media.

Most casualties within the injury radius of a conventional explosive detonation or deflagration will have common penetrating, blunt, and burn injuries that are managed no differently than similar nonblast trauma.[59] Much of this trauma will be soft tissue, orthopedic, or head injuries.[31,60,61] The first and most important step of management is assessment of life support needs and ensuring that the patient has an adequate airway, appropriate ventilation, and adequate circulation. Identify and correct life-threatening external hemorrhage at once. Arrhythmias (particularly bradycardia), hypotension, and apnea are frequently observed after blast injury to the thorax and have been associated with primary blast wave effects on the myocardium and vagal stimulation.[3,62]

A thorough physical examination should then be performed. The emergency physician should look for sentinel signs of potentially significant blast exposure. (**Table 6**)

Unfortunately, when the health care provider is faced with dramatic injuries, such as amputations, fragment injuries, and multiple critically ill patients, it is altogether too easy to miss the subtle signs of blast injury. If the clinician overlooks the possibility of primary blast injury, this may further complicate the patient's care.

Important Historical Questions

In addition to the usual questions about medications, allergies, tetanus immunization, prior surgeries, and past/current illnesses, there are specific questions that may guide your management of the patient who has been near an explosion.

Can you hear me? Do you have ear pain? Tympanic membrane rupture and temporary hearing loss is common in blast injury, but should not be life-threatening (that is, unless the casualty cannot hear life-saving commands or communications!).

Are you short of breath? Do you get short of breath with walking? A pulmonary contusion will inhibit oxygen diffusion and will cause dyspnea. Pneumothorax and hemothorax can decrease the volume of inspired air, with resultant subjective dyspnea. Shock from other causes can give the sensation of dyspnea caused by lactic acidosis from poor tissue perfusion. The more exertion required to elicit dyspnea, the less likely that there is a lung injury.

Do you have pain in your chest? Chest pain may occur from penetrating or blunt trauma, pneumothorax, pneumomediastinum, or myocardial ischemia or infarction due to coronary AGE.

Do you have nausea, abdominal pain, urge to defecate, or blood in your stools? Penetrating or blunt abdominal trauma can cause pain, or the patient may have primary blast injury to gas-filled abdominal organs, ruptured colon, or small bowel.

Do you have eye pain or problems with your vision? Evaluate the patient for blunt or penetrating eye trauma.

Special Considerations

Attempt to determine the distance from the explosion for each patient and whether the victim was in the open air or in an enclosure during the blast. Distance obviously decreases the risk of primary blast injury (at least in the open). Sharing a confined space with an explosion, including the inside of a vehicle, increases the magnitude of the blast wave to the victim. If the patient was in water, this should be noted, and the suspicion for intraabdominal blast injury heightened. If the patient was wearing body armor, this should be noted in the record. While body armor provides significant protection against fragment injuries, it also increases the chance and severity of primary

blast injury.[63]

Important Physical Findings

All injured survivors need to be evaluated with suspicion of primary blast injury. A full set of vital signs is essential and must include the pulse oximetry in all patients. These vital signs should be repeated frequently, as deterioration of the patient may occur over relatively short time periods. This deterioration may be from occult injury to either pulmonary or abdominal pathology. The subtle findings of tachycardia and a narrowed pulse pressure may be the first signs of this pathology. Signs of specific pathology are found in **Table 7** on page 17.

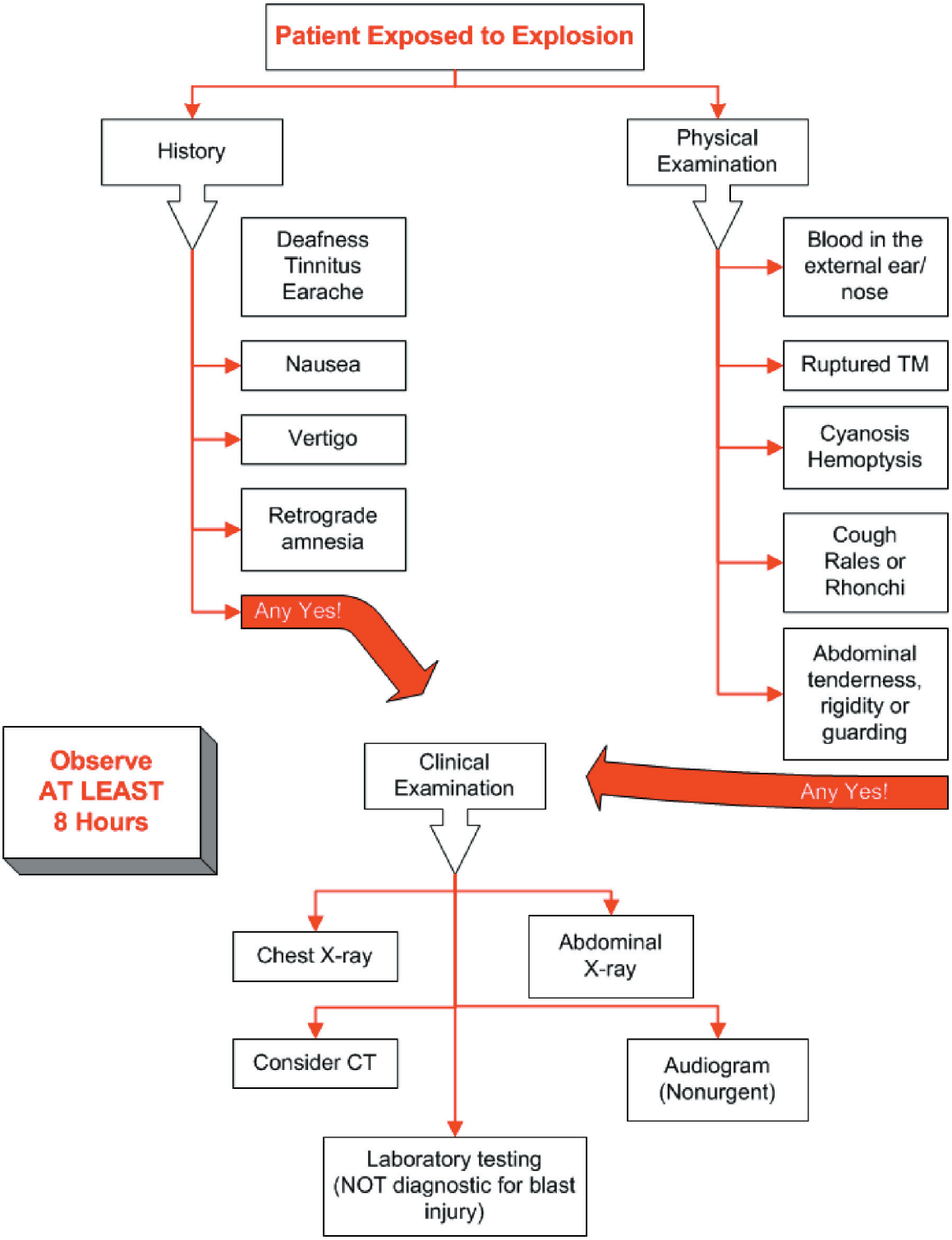
Continued on page 17

Table 6. Clinical Signs/Symptoms Of Significant Explosion-Related Injuries.*

System	Injury or Condition
Auditory System	Blood oozing from the mouth, nose, or ears Eardrum hyperemia, hemorrhage, or rupture Deafness (may persist) Tinnitus Earache
Cardiovascular	Tachycardia (stress, hemorrhage, hypoxia, exertion, or dehydration) Bradycardia (may be transient due to blast-induced vasovagal reaction) Delayed capillary refill Fall of mean arterial blood pressure (hemorrhage, AGE, vasovagal reaction) Arrhythmia (cardiac irritability due to shock or coronary AGE)
Gastrointestinal	Nausea Vomiting Abdominal tenderness (particularly progressive tenderness) Abdominal rigidity Hematochezia Hematemesis
Neurologic System	Vertigo (vertigo is *not* usually due to auditory trauma) Coma Altered mental status (may be due to head trauma, shock, or cerebral AGE) Focal numbness Paresthesias Seizures Retrograde amnesia Apathy
Ocular Injury	Eye irritation Difficulty focusing Blindness Funduscopic findings of retinal artery air embolism Loss of red reflex on funduscopic examination
Respiratory System	Cyanosis Ecchymosis or petechiae in hypopharynx Asymmetric breath sounds Cough (often dry) Tachypnea (often preceded by a short period of apnea) (Rapid shallow respirations are common after blast exposure) Dyspnea (respiratory difficulty) Hemoptysis Rales or moist crepitation in lung fields Wheezes Chest pain Asymmetric chest movement Subcutaneous emphysema (open wound or rupture of air-containing internal structure)
Miscellaneous	Tongue blanching (may indicate AGE) Mottling of nondependent skin (may indicate AGE or hypotension) Subcutaneous emphysema (open wound) Pharyngeal petechiae (this has a better predictive value for blast lung than tympanic rupture) Abrasions

*Dark gray shading indicates most common findings; light gray shading indicates common findings.

Clinical Pathway: Treatment Of Blast Injuries



This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Diagnostic Studies

There are only a few screening studies that are of any benefit in the casualty with primary blast injury.

Pulse oximetry may indicate some degree of lung injury. A falling pulse oximetry should prompt additional monitoring and raise suspicion of pulmonary injury or shock from another injury. With multiple casualties, continuous pulse oximetry may not be possible in all patients.

Serial hemoglobin determinations are useful in select cases where internal hemorrhage is suspected. The data may be used as a guide for blood transfusion requirements. Victims of major trauma should have baseline blood counts, hematocrit, hemoglobin, and crossmatching for potential transfusion.

Although most casualties with primary bowel injury have bleeding, it is usually gross hematochezia. A guaiac positive stool can indicate occult penetrating, blunt, or blast trauma to the bowel.

X-ray

An immediate chest x-ray should be obtained in all patients who have been near a significant explosion. The clinician should look for evidence of pulmonary contusion (as noted above) and barotrauma. A chest x-ray may also show free air under the diaphragm, signifying hollow viscus rupture in the abdomen from primary blast injury.[40]

Puncture wounds should be presumed to be due to high-speed missiles and examined accordingly. Any puncture wound of the thorax, abdomen, or extremities should prompt a radiograph, at least. In many cases, CT may be more appropriate.

CT Scans

A CT of the head, chest, or abdomen should be obtained if the history or physical examination suggests pathology in these areas. If the patient is or was unconscious, these CT studies are not optional.

Unfortunately, the CT scanner may also be the biggest bottleneck in the treatment of multiple patients with blast injuries.[64] After the Oklahoma City bombing, 19% of 338 patients treated in the ED had CT scans.[4] The CT scan is often slow and requires contrast use and equipment availability. Often, CT scanning requires transport away from the emergency care area.

The emergency physician must prioritize CT scans based on the urgency of finding a *remedial* problem in a survivable patient. A dedicated radiologist and a resuscitation team in the CT scanner suite can both obviate the problems associated with moving the patient from an emergency care area and expedite the movement of patients through the CT scanner suite.

Sonography

Sonography has become an extension of the physical examination of the abdomen and should be performed whenever available and when abdominal injury is suspected. Focused abdominal sonography for trauma (FAST) aids in the prioritization of penetrating injury patients for the operating room, indicates which cavity to open first in patients with thoracoabdominal injuries, identifies pericardial fluid, and may assist in the diagnosis of hemopneumothorax and hemopericardium.

The 3.5- to 5-MHz curved probe is optimal for the performance of the FAST examination. The abdomen is examined through 4 standard sonographic windows. A FAST examination assists the surgeon in determining the need for laparotomy in blunt-injured patients, but it does *not* identify specific injuries. A FAST examination does not

Table 7. Overview Of Explosion-Related Injuries.*

System	Injury or Condition
Auditory System	Ruptured tympanic membrane, disruption of the ossicles, damage of the cochlea
Cardiovascular	Myocardial contusion, myocardial infarction from air embolism, cardiogenic shock, peripheral vascular injury, peripheral ischemia from air embolism, shock
Extremity Injuries	Fractures, amputations, crush injury, compartment syndrome, burns, cuts, lacerations, acute occlusion of an artery, air embolism-induced injury
Gastrointestinal	Viscus perforation, hemorrhage, fracture/rupture of liver or spleen, mesenteric ischemia from air embolism, sepsis
Neurologic System	Concussion, closed brain injury, open CNS injury, stroke from air embolism, spinal cord injury. **Primary blast injury can cause concussion without a direct blow to the head.**
Ocular Injury	Perforated globe, foreign bodies, air embolism, and orbital fractures. **Up to 10% of blast injury survivors have significant eye injuries.**
Renal Injury	Renal contusion, kidney laceration, acute renal failure due to shock or rhabdomyolysis, testicular rupture
Respiratory System	Blast lung, hemothorax, pneumothorax, pulmonary contusion, pulmonary hemorrhage, arteriovenous fistula (air embolism), airway epithelial damage, aspiration pneumonitis, sepsis. **Blast lung is a direct consequence of the HE overpressure wave. It is the most common fatal primary blast injury among initial survivors of an explosion.**

*Dark gray shading indicates most common findings; light gray shading indicates common findings. Modified from Centers for Disease Control and Prevention Mass Trauma Preparedness and Response Web page. Available at: http://www.cdc.gov/masstrauma/preparedness/primer.htm. Accessed April 4, 2006.

identify or stage solid organ or hollow viscus injury, but reliably identifies free intraperitoneal fluid.

Treatment

Hypotension

Hypotension in blast injury victims can be due to several mechanisms:

- Blood loss due to wounds (otherwise not related to the cardiovascular system)
- Blood loss due to gastrointestinal hemorrhage
- Blood loss due to intraabdominal solid organ rupture
- Hypotension from compression of vessels and heart by pneumothorax
- Hypotension due to the cardiovascular effects of an air embolism
- Hypotension due to vagal reflexes

The patient's fluid volume should be supported without excessive fluid replacement. Too much fluid replacement can, of course, cause increased respiratory distress. Often blood products or colloid solutions are more appropriate in the acute trauma patient than crystalloid infusions.

Auditory

There is no specific treatment for blast-related ear injuries. The physician should caution the victim to avoid any further auditory injury, if possible. The patient should be transferred to a quieter environment, where available, and the ears should be evaluated within 24 hours.

Debris should be gently removed from the external canal. Neither antibiotics nor ear drops are recommended, particularly if the patient has a ruptured tympanic membrane.[65] Tympanoplasty is reserved only for failures of conservative therapy.[65] In at least 1 retrospective study of 147 patients, nearly 70% of perforated tympanic membranes healed within 10 months.[65]

Pulmonary

Blast lung is treated by correcting the effects of barotrauma, if any are found. If available, supplemental oxygen should be started on any patient who does not have or cannot maintain a normal oxygen saturation, or those who have any external injuries. Those patients with significant respiratory distress or hemoptysis should have an endotracheal tube placed. This is *not* without its hazards, however. The provider should opt for the least invasive measure that will still provide appropriate airway support in these patients.[66]

In one study using thoracic CT scans of patients with pulmonary contusion (not blast injury), patients with less than 18% contusion did not require intubation or ventilation.[67] Patients with more than 28% contusion always required ventilation. Although this was a small study, the findings are persuasive.

Positive pressure ventilation markedly increases the possibility of both air embolism and pulmonary barotrauma.[21,38,68] Avoid positive end-expiratory pressure (PEEP) and high ventilation pressures.[66] Preventative strategies include using limited peak inspiratory pressures, pressure-controlled ventilation, high-frequency jet ventilation, and permissive hypercapnia.[10,38,52,68]

Because the combination of positive pressure ventilation and blast lung injury poses such a high risk for tension pneumothorax, some authors suggest bilateral prophylactic chest tubes after intubation.[69] If the patient needs air evacuation, this becomes an important consideration. If a patient with a blast lung injury abruptly decompensates, the clinician should presume that the patient has a tension pneumothorax and treat accordingly.

Experimental techniques, such as high-frequency jet ventilation or nitric oxide, do not seem to confer any particular benefit to victims of blast lung.[38] The use of extracorporeal circulation is associated with catastrophic pulmonary hemorrhage.[70]

Data on the short- and long-term outcomes of patients with pulmonary blast injury are currently limited. In a retrospective review of 11 patients, Hirshberg et al found that, if the patient survives the blast lung and other trauma, there is a good chance that they will regain full lung function within a year after the injury.[71]

Air Embolism

Arterial gas embolism (AGE) may be the most common cause of rapid death in initial survivors. It often occurs when positive pressure ventilation is started.[24,25,39] Symptoms of an air embolism depend on where the bubbles lodge. Air embolism can present as stroke, MI, acute abdomen, blindness, deafness, spinal cord injury, or claudication.

Air embolism should be treated as soon as the diagnosis is considered. The first step is to place the patient on high-flow oxygen. Next, the patient should be properly positioned — case studies involving divers support the use of the Trendelenburg position in the immediate management of gas embolism, and it is appropriate to use this in blast injury-induced gas embolism.[72] The injured lung should be placed in the dependent position (which may override the left side down position, described above.) By placing the injured lung down, the alveolar oxygen pressure is lower, with a subsequent decreased risk of air entering the lungs.

The definitive treatment for air embolism is thought to be hyperbaric oxygenation (HBO), which is often not available in a timely fashion. Randomized, controlled trials demonstrating efficacy of hyperbaric oxygenation have yet to be performed, but the physiological mode of action seems entirely sufficient to warrant the application of HBO, despite this lack of research.[72] Hyperbaric oxygenation will reduce the bubble size (according to Boyle's law), increase tissue oxygenation, and increase the solubility of the gas. The US Navy Dive Table 6 and 6a protocols for gas embolism and decompression sickness would be a good clinical start. (**Table 8**) Consultation with an experienced hyperbaric physician or dive physician is appropriate for all HBO patients.

Gastrointestinal

Blast injury of the gastrointestinal tract can be managed in much the same way as blunt trauma of the abdomen. If the patient has an obvious penetrating wound of the abdomen, then urgent surgical management is indicated. If the patient is not conscious and hemodynamically unstable, or conscious with abdominal complaints and hemodynamically unstable, then fluid resuscitation should be undertaken. If the patient's blood pressure stabilizes and remains stable, then a noncontrast CT scan of the abdomen is appropriate. If the blood pressure does not improve, then urgent surgical management is indicated. Fatal splenic rupture has been reported in at least 1 victim who had no sign of external injury.[73] This suggests a role for FAST ultrasonography in symptomatic patients, or for those patients who cannot be adequately evaluated.

While abdominal CT scan is appropriately specific, older scanners may not be sufficiently sensitive to identify hollow viscus injury.[40] If patients who have been scanned continue to have signs of abdominal pathology, then a repeat FAST examination followed by diagnostic peritoneal lavage (DPL) is appropriate. If the DPL effluent contains significant red blood cells, bacteria, bile, or fecal matter, then urgent laparotomy is indicated. CT must precede DPL, or false-positive air and fluid will be introduced.

In the context of a mass casualty incident, there should be a low threshold for laparotomy when a hollow viscus injury is suspected. Close observation may not be possible, because of the number of casualties. Clinical signs and symptoms of early bowel injury, particularly in children, may be so subtle as to be easily missed in the patient with multiple injuries.[74]

Wound Management

There is about an 80% rate of infection when fragment wounds are sutured. For lacerations and fragment wounds, avoid primary closure and consider the use of delayed primary closure in these wounds.[61] Delayed primary closure is the technique of cleaning the wound, leaving the wound open under a moist dressing for approximately 4 to 5 days, and then suturing the wound if there is no evidence of infection. The first step in delayed primary

Table 8. US Navy Treatment Table 6A: Initial Air and Oxygen Treatment of Arterial Gas Embolism.*¶

Depth (feet)	Time (minutes)	Breathing Media¥	Elapsed Time (h:min)
165‡	30#	air	0:30
165 to 60	4	air	0:34
60	20	O_2§	0:54
60	5	air	0:59
60	20	O_2	1:19
60	5	air	1:29
60	20	O_2	1:44
60	5	air	1:49
30	15	air	3:49
30	60	O_2	4:49
60 to 30	30	O_2	2:19
30	15	air	2:34
30	60	O_2	3:34
30 to 0	30	O_2	5:19

*Treatment of arterial gas embolism where complete relief obtained within 30 min at 165 feet. Use also when unable to determine whether symptoms are caused by gas embolism or severe decompression sickness.
†Descent rate — as fast as possible. Ascent rate — 1 ft/min. Do not compensate for slower ascent rates. Compensate for faster ascent rates by halting the ascent.
‡Time at 165 feet — includes time from the surface.
§If oxygen breathing must be interrupted as a result of adverse reaction, allow 15 minutes after the reaction has subsided and resume schedule at the point of interruption.
¥Caregiver breathes oxygen during ascent from 30 feet to the surface, unless he has had hyperbaric exposure within the past 12 hours, in which case he breathes oxygen at 30 feet.
¶Extensions: Table 6A can be lengthened up to 2 additional 25-minute periods at 60 feet (20 minutes on oxygen and 5 minutes on air) or up to 2 additional 75-minute oxygen breathing periods at 30 feet (15 minutes on air and 60 minutes on oxygen), or both. If Table 6A is extended either at 60 or 30 feet, the tender breathes oxygen during the last half at 30 feet and during ascent to the surface.
#If complete relief is not obtained within 30 min at 165 feet, switch to Table 4, consulting with a Diving Medical Officer, if possible.

Adapted from the US Navy Diving Manual.

closure is thorough cleansing and removal of debris and devitalized tissue. Heavily contaminated wounds resulting from high-energy missile injuries are ideal for delayed primary closure.

All debris that is flung by the explosion is not radiopaque, and the wise provider should carefully explore injuries and consider CT, ultrasound, or MRI of wounds to evaluate for radiolucent foreign bodies. Update the tetanus status, as appropriate. (For an evidence-based approach to wound care, see *Emergency Medicine Practice*, Volume 7, Number 3, Wound Care: Modern Evidence In The Treatment Of Man's Age-Old Injuries, March 2005.)

Special Circumstances

Minimize the physical activity of blast victims after an explosion. Exertion after the blast explosion can increase the severity of primary blast injury. This was seen in WWII,

Ten Pitfalls To Avoid

1. "The firefighter wanted to go back and help after he was evaluated in the aid station. He said his buddies were still on the scene and he knew where they were. He had a normal oxygen saturation and had no evidence of external injury — just a ruptured tympanic membrane. How was I to know he would decompensate?"

The potential victim of blast injury should rest while being observed by a qualified medical provider for at least 6 hours after the injury. Increased exertion is associated with a poor outcome in blast lung injury.

2. "The police officer was wearing body armor. She had some injuries on her extremities, but no torso injuries from the fragments. How could I know she was going to develop a pulmonary blast injury?"

Body armor provides good protection from fragment injuries, but may actually increase the incidence of pulmonary blast injury due to reflection of the blast wave off the armor.

3. "The medics on the scene were ignoring the patient who was hypotensive, until she became pulseless and apneic. I thought we could get another save if we could work her up."

Triage is a hard science to master. One of the most common failings of the newcomer to triage is an inability to shift from "we can try to save every cardiac arrest patient" to "we will do the most we can for the greatest number of people with the resources at hand." In this case, the medics were correct in writing off the hypotensive arresting patient as nonsalvageable, while providing care to more people.

4. "How was I to know that she would develop a tension pneumothorax in the helicopter? It seemed like the quickest way to get her to the trauma center."

Helicopter transport in the blast injury victim is fraught with hazards. Any pulmonary barotrauma will be exacerbated by even modest altitude changes. In many cases, chest tubes before transport are recommended.

5. "The bomb went off across the street from the hospital. Many of us went out to bring patients back to the ED. I never even thought about a secondary device — I just reacted."

The use of secondary bombs to wound, kill, or disable medical providers, emergency workers, and police is quite common. Unless you are wearing protective gear, have been trained to look for secondary devices, and are armed, you should consider waiting for the scene to be rendered safe or cleared before approaching — even if it is across the street.

6. "I'm finding nails, nuts, and bolts in wounds in these patients. Was the blast so powerful that it stripped these off other objects?"

Actually, the addition of ball bearings, nuts, bolts, screws, and nails is quite common. These objects are designed to increase the mayhem caused by the blast.

7. "The officer said that the blast was due to gunpowder. Gunpowder isn't going to cause this kind of damage, is it?"

Gunpowder is a low-order explosive. It is often referred to as a propellant, since it is used to propel bullets out of the barrel of a weapon. The explosions caused by gunpowder are somewhat less intense than the explosions caused by an equivalent weight of high explosive, but not by much. Gunpowder can cause extensive damage!

8. "The fireman said something about a boiling liquid. Propane isn't going to cause an explosion injuring hundreds of people like this one did, is it? This was a huge explosion. There must have been something else here."

BLEVE (boiling liquid expanding vapor explosion) is one of the thermobaric weapons that can cause extensive damage.

9. "We've gotten nearly 50 walking wounded in the last 20 minutes from the explosion 3 blocks away. I'm just glad nobody got hurt badly in this explosion."

Oops! You may want to rethink that sense of relief here, if the explosion is only 3 blocks away and the victims know where your ED is. In an uncontrolled incident, the walking wounded will self-evacuate and come to you. This reverse triage effect can swamp your emergency services before the critically ill arrive. The critically ill will need stretchers, gurneys, and ambulances, and it takes time to get them to the ED. Your optimism may well be misplaced. You need to prepare for the critically ill, not let your guard down.

10. "The patient had an oxygen saturation of 92%, ecchymosis about the oral pharynx, ruptured tympanic membranes, and was short of breath. I started him on oxygen and his saturation climbed to 100%. I didn't see any external wounds. He never complained about his abdomen."

You've described a patient with primary pulmonary blast injury. You need to ensure that there are no other injuries. This patient was close enough to the blast that other injuries are entirely possible. ▲

where some blast casualties appeared well, but died after vigorous exercise.[28,54]

If the patient requires immediate anesthesia for any reason, the patient needs a chest x-ray to look for any evidence of barotrauma. It has been reported that blast victims have a higher morbidity rate when they receive general anesthesia.[25] This may well be due to unrecognized primary blast injury and subsequent barotrauma from positive pressure ventilation during anesthesia.[25] If barotrauma is noted and the patient requires general anesthesia, bilateral chest tubes are appropriate.[24] If possible, local or spinal anesthesia may be better.

Controversies/Cutting Edge

Ear Injury as a Marker for Blast Injury

New findings based on recent meta-analyses and large-scale retrospective studies have disabused us of the notion that a marker for primary blast injury is ear damage (tympanic membrane rupture).[30] Both use of hearing protection and partial immersion in water may affect the incidence of ear damage associated with primary blast injury. A recent study showed that primary blast injury is more reliably associated with skull fractures, burns covering more than 10% of the body surface area, and penetrating injuries to the head or torso.[75]

Protective Effects of Body Armor

Body armor provides a false sense of security during an explosive detonation. The body armor does protect the victim from bomb fragments and, to a lesser extent, objects picked up and flung by the blast wave, but it also provides a reflecting surface that can concentrate the power of the explosion as the blast wave reflects off of the armor front and back.[25,27,76,77] (Since the bulk of injuries from an explosive device are from secondary objects flung by the blast wave, the advantages of body armor outweigh the risks of enhancing the blast wave.) The medical provider should not assume that body armor will protect the victim from an explosion-related injury.[77]

Improvised Explosive Devices

As previously mentioned, a "manufactured" explosive refers to a standard, mass-produced, and quality-tested weapon, while "improvised" describes the use of alternative materials, weapons produced in small quantities, or a device that is used outside of its intended purpose. Improvised explosive devices now found in Iraq are often professional in appearance and operation. These devices have been unexpectedly lethal in action. The emergency physician must not yield to the idea that the "improvised" explosive device is crude and of low efficacy. The rather unwieldy government definition includes professional military devices produced by explosives experts.

Disposition

There are no definitive guidelines for observation, admission, or discharge following ED evaluation for patients with possible blast injury sustained in an explosion. The disposition of these patients depends on the injuries specific to each victim. Those victims with penetrating secondary blast injury or tertiary blast injury need treatment guided by the nature of their injury.

In general, patients with normal chest radiographs and arterial blood gases who have no complaints that would suggest pulmonary blast injury can be considered for discharge after 4-6 hours of observation. Persons exposed to significant closed-space explosions, in-water explosions, and those who were close to the center of the explosion should be considered for observation for at least 8 hours, as they are at higher risk of delayed complications. For those who are sent home, return instructions should include shortness of breath, abdominal pain, vomiting, or development of other symptoms.

Patients with any complaints or findings suspicious for pulmonary or abdominal blast injury should be observed in the hospital. Admit to the hospital all patients with significant burns, abnormal vital signs, abnormal lung examination findings, clinical or radiographic evidence of pulmonary contusion or pneumothorax, abdominal pain, vomiting, evidence of renal contusion, or penetrating injuries to the thorax, abdomen, neck, or cranial cavity. Patients with only penetrating injuries to the extremities should be admitted or discharged, as appropriate to the clinical situation. Patients diagnosed with pulmonary or abdominal blast injury may require complex management and should be admitted to an intensive care unit. Patients with penetrating wounds to head, chest, and abdomen must be treated in accordance with their wounds and good surgical practice.

Transfer of the patient to another medical facility can exacerbate the original injuries. The pulmonary barotrauma from primary blast injury can be exacerbated by air evacuation. As noted earlier, both pneumothorax and arterial gas emboli will enlarge with ascent. All casualties with any evidence of pneumothorax must have a chest tube placed, regardless of the altitude and distance of the flight. Evacuation helicopters should fly at the lowest possible altitude. Long-distance evacuation aircraft should be pressurized to at least 8000 feet (preferably 5000 feet). If the victim has marginal oxygenation (PO_2 <60 mm Hg), oxygenation will worsen with ascent in an aircraft. Intubation prior to transport is recommended for all seriously ill patients.

Summary

When confronted with the scenario that opens this review, you will be facing a busy and very long day. Fortunately, you, your ED, and your hospital have practiced your mass casualty protocols. Rather than being overwhelmed by circumstances, you assume command of the incident within the hospital, and assign your most experienced emergency physician to triage as the casualties start to appear. You

Continued on page 24

Supplemental Material: Terrorism

Profile of Suicide Bomber and Possible Physical Appearance

The official United States position is that there "is no specific profile for those who have engaged in suicidal/homicidal bombings."[78] The Israelis have had significantly more experience with this phenomenon and have established several guidelines and profiles.[79] The Israeli profile for suicide/homicide bombers is as follows:

- 47% have an academic education, and an additional 29% have at least a high school education.
- The suicide bomber undergoes a process of indoctrination that lasts for months. By the time that they are ready to don their explosives, they have reached a hypnotic state and believe that by blowing themselves up, they have opened a direct gateway to heaven.
- 83% of the suicide/homicide bombers are single.
- Most are males aged 16-30, although older males and females have been known to carry these devices.
    - 64% of the suicide bombers are between the ages 18-23 — most of the rest are under 30.

Demeanor and appearance of a suicide/homicide bomber prior to attack:

- The effectiveness of a suicide/homicide bombing attack is often more related to the training and purpose of the bomber than the equipment used by the bomber.
    - May appear nervous, preoccupied, or have a blank stare.
    - Appears to be focused and vigilant.
    - May be fervently praying to him/herself, giving the appearance of whispering to someone.
    - No response to authoritative voice or direct salutation.
    - Behavior may be consistent with no future — unconcerned about receiving purchases or change.
    - The subject may walk with deliberation — but not running — towards a visible objective.
    - Demonstrates forceful actions (to reach a desired target by pushing their way through a crowd or into a restricted area).
    - Stiff movements, lack of mobility of lower torso or decreased flexibility (from wearing bomb device — although backpack devices are increasingly common).
    - May shave his or her head or have a short haircut. A short haircut or recently shaved beard or moustache may be evidenced by differences in skin complexion on the head or face. (This may be done to disguise appearance or to be better groomed when going to paradise.)
    - May smell of unusual herbal/flower water (in order to smell better when going to paradise).
- Clothing may be out of sync with the weather. The perpetrator may wear excess clothing to hide the device.
- Clothing is often loose — clothing may give the impression that the body is disproportionately larger than the head or feet.
    - Devices are generally concealed within an article of clothing worn close to the body, such as a vest, belt, or jacket. (See **Figures 15-19**. **Figures 15-19** all courtesy United States Coast Guard.)
    - Backpack, bag, luggage, or briefcase may be carried.
    - The bomber is often holding a push-button or toggle switch to detonate the explosives. Alternative manual devices include a pull-type wire leading to the main device that triggers the detonation.
    - Many devices have a backup trigger system, such as an electronic timer, pager, cell phone, or booby-trap type switch. If the attacker is killed, apprehended, or attempts to abort the attack, an accomplice/supervisor may remotely trigger the device.
    - The device will likely be filled with ball bearings, nails, screws, nuts, or other small metal pieces. Dispersal of these fragments is the primary "kill" mechanism of the suicide/homicide bomber.

The first responsibility of responding officials should be to disperse any crowds. A search for a second device or perpetrator should be immediately started. If a suspicious person or item is identified, do not try to apprehend or move the object/person. Back off, try and get behind solid shielding, and call for security personnel who are trained to deal with the threat.

Figure 15. Suicide vest.

Suicide vest used by the LTTE — dual electric firing system, military-grade explosives, ball bearings.

Figure 16. Cutaway view of suicide vest.

Cutaway showing packages that contain main explosive charge and ball bearings for fragmentation.

Possible Bomb Indicators

- Unusual device attached to a container or cylinder of flammable material.
- Unusual or misplaced mailing containers leaking oil or with wiring attached.
- Abandoned vehicles or vehicles that do not appear to belong in the area.
- Strong chemical odors.
- An unusual or out of place container.
- Obvious bomb-making materials: blasting caps, nitroglycerin, dynamite, other explosives, bags of high nitrogen fertilizer, wire or clock-timers, pipe with fuse or electrical wires.
- Unattended packages or packs hanging on hooks in restrooms.

If a suspected bomb is discovered, do not touch or disturb it. Note the location, floor, and room. Report this at once to your fire or police and *EVACUATE THE AREA*!

Figure 17. Close up of Iraqi suicide vest.

Exterior view of Iraqi suicide vest found in Baghdad.

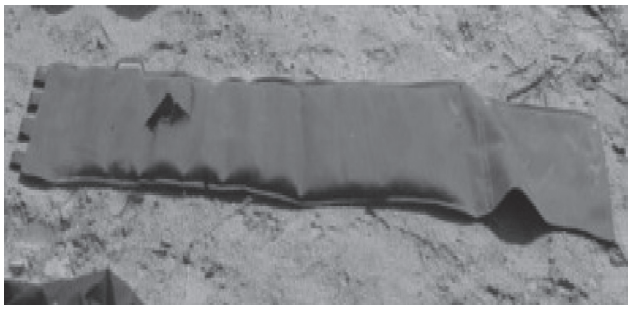
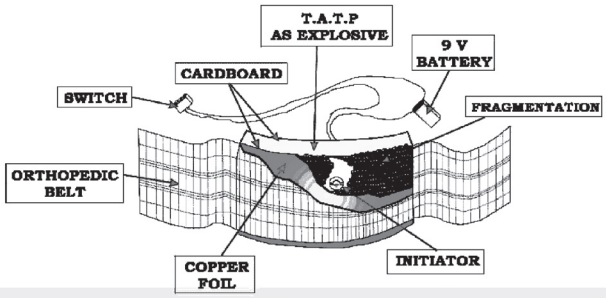


Figure 18. Suicide belt diagram.



Types of Terrorist Bombs (See **Figure 20**.)

Pipe Bomb

A pipe bomb is a fragmenting bomb that is easily made. This type of bomb can be identified by a section of pipe capped at both ends. A fuse may extend from one end.

Bottle Bomb

A bottle bomb is often called a Molotov cocktail, after the Russian government official. It was a popular WWII antitank weapon. There are many different ways to make this device. The simplest is to fill a glass bottle with gasoline, use a sock or other rag stuffed in the end, and light the sock. When the bottle breaks, the gasoline is ignited.

Vehicle Bomb

A vehicle is filled with explosives. When the explosives are detonated, a powerful explosion results. A small boat was used against the USS Cole, while in Oklahoma City, a van was used as a bomb.

Satchel (Bag) Bomb

Several sticks of dynamite or other explosive are placed in a suitcase, briefcase, backpack, or shoulder strap bag. If 1 or 2 liquid propane cylinders are added to the bag, it increases the explosion and creates a fire. The bag can also be packed with antipersonnel material, such as nails, screws, bolts, or nuts, to inflict additional casualties. This kind of bomb can be left on a hook inside a restroom stall or in a locker. The 1996 Olympic Centennial Park bomb was a satchel bomb.

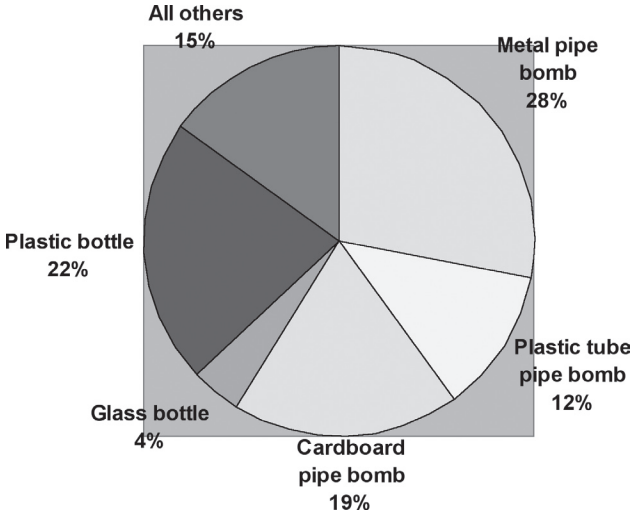
Suicide Bomb

While any of the devices listed above (and many others) can be used as a suicide bomb, the classic suicide bomb is a vest or belt filled with explosives and other material designed to increase fragment wounds.

Figure 19. Firing mechanism for suicide vest.

Firing device with lanyard port, two toggle switch safety system, and 9V battery.

Figure 20. Types and frequencies of small improvised explosive devices.



point out to the staff that they can expect a high percentage of burns as well as blast injuries, because this is a fuel-air explosion (grain dust). You empty all of your ED rooms so that you can start processing the casualties as they arrive. Within 15 minutes of the dispatcher's notification, the first of your reinforcements arrives, and the trauma teams assemble. At the same time, the first of your casualties arrive... in a pickup truck. You thank your lucky stars that this is only a grain elevator, not a terrorist attack. ▲

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references.

1. Rusca F. *Deutsche Ztschr f Chir* 1915;132:315. **(Historical article first describing primary blast injury)**
2. Elsayed NM. Toxicology of blast overpressure. *Toxicology* 1997;121(1):1-15. **(Review article)**
3. Irwin RJ, Lerner MR, Bealer JF, et al. Cardiopulmonary physiology of primary blast injury. *J Trauma* 1997;43(4):650-655. **(Animal study)**
4. Hogan DE, Waeckerle JF, Dire DJ, et al. Emergency department impact of the Oklahoma City terrorist bombing. *Ann Emerg Med* 1999;34(2):160-167. **(Retrospective review of 338 patients)**
5. US Department of Justice General Information Bulletin 96-1. *1996 Bombing Incidents*. Washington, DC: US Department of Justice; 1996. **(Data mining article; bombing reports to FBI during 1991)**
6. Noji EK, Lee CY, Davis T, et al. Investigation of Federal Bureau of Investigation bomb-related death and injury data in the United States between 1988 and 1997. *Mil Med* 2005;170(7):595-598. **(Data mining statistical compilation)**
7. Kapur GB, Hutson HR, Davis MA, et al. The United States twenty-year experience with bombing incidents: Implications for terrorism preparedness and medical response. *J Trauma* 2004;59:1436-1444. **(Data mining review)**
8. Eiseman B. Combat casualty management for tomorrow's battlefield: Urban terrorism. *J Trauma* 2001;51:821-823. **(Review)**
9. Feliciano DV, Anderson GV Jr, Rozycki GS, et al. Management of casualties from the bombing at the centennial Olympics. *Am J Surg* 1998;176(6):538-543. **(Retrospective review of 111 victims)**
10. Stein M, Hirshberg A. Medical consequences of terrorism. The conventional weapon threat. *Surg Clin North Am* 1999;79(6):1537-1552. **(Review article based on Israeli experiences)**
11. Gutierrez de Ceballos KP, Fuentes FT, Diaz DP, et al. Casualties treated at the closest hospital in the Madrid, March 11 terrorist bombings. *Crit Care Med* 2005;33(1):S107-S112. **(Retrospective review)**
12. Bailey A, Murray SG. The chemistry and physics of explosions. In: Bailey A, Murray SG. *Explosives, Propellants, and Pyrotechnics (Land Warfare, Vol 2)*. 2nd ed. London, UK: Brassey's UK Ltd; 1989:1-19. **(Textbook chapter)**
13. Hull JB, Cooper GJ. Pattern and mechanism of traumatic amputation by explosive blast. *J Trauma* 1996;40(3 Suppl): S198-205. **(Retrospective review coupled with animal studies)**
14. Wolf A. BLEVE kills two. *NFPA J* 1998(Nov/Dec):42-47. **(Case report, description of BLEVE)**
15. Wildegger-Gaissmaier AE. Aspects of thermobaric weaponry: *ADF Health* 2003;4(1):3-6. **(Review article)**
16. Landsberg PG. Underwater blast injuries. *Trauma Emerg Med* 2000;17(2). Available at: http://www.scuba-doc.com/uwblast.html. Accessed April 4, 2006. **(Review)**
17. Huller T, Bazini Y. Blast injury of the chest and abdomen. *Arch Surg* 1970(100):24-30. **(Case report/historical)**
18. Cameron GR, Short RHD, Wakely CPG. Abdominal injuries due to underwater explosion. *Br J Surg* 1943;31:52-56. **(Historic case report)**
19. Petri NM, Dujella J, Definis-Gojanovic M, et al. Diving-related fatalities caused by underwater explosions: a report of two cases. *Am J Forensic Med Pathol* 2001;22(4):383-386. **(Case report)**
20. Rice D, Heck J. Terrorist Bombings: Ballistics, patterns of blast injury and tactical emergency care. *Tactical Edge J* 2000(Summer):53-55. **(Review article)**
21. Boffard K, MacFarlane C. Urban bomb blast injuries: patterns of injury and treatment. *Surg Annu* 1993;25 Pt 1:29-47. **(Meta-analysis)**
22. Galarneau MR, Hancock WC, Konoske P, et al. U.S. Navy-Marine Corps Combat Trauma Registry Operation Iraqi Freedom-1 Preliminary Findings. Interim report Oct 2003-Jun 2004; Jun 2004. Available at: http://www.stormingmedia.us/33/3383/A338334.html. Accessed April 4, 2006. **(Retrospective database search)**
23. Cernak I, Savic J, Ignjatovic D, et al. Blast injury from explosive munitions. *J Trauma* 1999;47(1):96-103; discussion 103-104. **(Retrospective review, 1303 patients, 665 with primary blast injury)**
24. Phillips Y, Richmond D. Primary blast injury and basic research: A brief history. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistic, Blast, and Burn Injuries*. Washington DC: Surgeon General Department of the Army; 1991:221-240. **(Textbook chapter)**
25. Wightman JM, Gladish SL. Explosions and blast injuries. *Ann Emerg Med* 2001;37(6):664-678. **(Review article)**
26. Cooper G, Taylor D. Biophysics of impact injury to the chest and abdomen. *J R Army Med Corps* 1989;135(2):58-67. **(Review/mathematical description of injuries)**
27. Cooper C, Townsend D, Cater S et al. The role of stress waves in thoracic visceral injury from blast loading; Modification of stress transmission by foams and high-density materials. *J Biomech* 1991;24:273-285. **(Review/athematical description)**

28. Stuhmiller J, Phillips Y, Richmond D. The physics and mechanisms of primary blast injury. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistic, Blast, and Burn Injuries*. Washington DC: Surgeon General Department of the Army; 1991:241-270. **(Textbook chapter)**

29. Cohen JT, Ziv G, Bloom J, et al. Blast injury of the ear in a confined space explosion: auditory and vestibular evaluation. *Isr Med Assoc J* 2002;4(7):559-562. **(Retrospective review, 17 patients)**

30. Leibovici D, Gofrit ON, Shapira SC. Eardrum perforation in explosion survivors: is it a marker of pulmonary blast injury? *Ann Emerg Med* 1999;34(2):168-172. **(Retrospective review, 647 patients, 193 primary blast injuries)**

31. Mallonee S, Shariat S, Stennies G, et al. Physical injuries and fatalities resulting from the Oklahoma City bombing. *JAMA* 1996;276(5):382-387. **(Retrospective review, analysis of all injuries)**

32. Yelverton J. Blast biology. In: Cooper C, Dudley H, Gann D, eds. *Scientific Foundations of Trauma*. 1st ed. Oxford, UK: Butterworth-Heinemann; 1997:189-199. **(Textbook chapter)**

33. Leibovici D, Gofrit ON, Stein M, et al. Blast injuries: bus versus open-air bombings--a comparative study of injuries in survivors of open-air versus confined-space explosions. *J Trauma* 1996;41(6):1030-1035. **(Retrospective review, 297 patients)**

34. Mellor SG. The relationship of blast loading to death and injury from explosion. *World J Surg* 1992;16:893-898. **(Retrospective study, 828 patients from Northern Ireland)**

35. Maynard R, Coppel D, Lowry K. Blast injury of the lung. In: Cooper G, Dudley H, Gann D, eds. *Scientific Foundations of Trauma*. Oxford UK,: Butterworth-Heinemann, 1997: 214-224. **(Review)**

36. Sharpnack D, Johnson A, Phillips Y. The pathology of primary blast injury. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistic, Blast, and Burn Injuries*. Washington DC: Surgeon General Department of the Army; 1991:271-294. **(Textbook chapter)**

37. Mayorga MA. The pathology of primary blast overpressure injury. *Toxicology* 1997;121(1):17-28. **(Review)**

38. Pizov R, Oppenheim-Eden A, Matot I, et al. Blast lung injury from an explosion on a civilian bus. *Chest* 1999;115(1):165-172. **(Case report/retrospective analysis of 15 total patients from 2 bus bombings)**

39. Ho A-H, Ling E. Systemic air embolism after lung trauma. *Anesthesiology* 1999;90:564-575. **(Review)**

40. Argyros GJ. Management of primary blast injury. *Toxicology* 1997;121(1):105-115. **(Review)**

41. Paran H, Neufeld D, Shwartz I, et al. Perforation of the terminal ileum induced by blast injury: delayed diagnosis or delayed perforation? *J Trauma* 1996;40(3):472-475. **(Case report, 3 patients)**

42. Cripps NP, Cooper GJ. Risk of late perforation in intestinal contusions caused by explosive blast. *Br J Surg* 1997;84(9):1298-1303. **(Animal study)**

43. Oppenheim A, Pizov R, Pikarsky A, et al. Tension pneumoperitoneum after blast injury: dramatic improvement in ventilatory and hemodynamic parameters after surgical decompression. *J Trauma* 1998;44(5):915-917. **(Case report, 2 patients)**

44. Clemedson C, Pettersson H. Propagation of a high explosive air shock wave through different parts of an animal body. *Am J Physiol* 1956;84:119-126. **(Historic animal study)**

45. Almogy G, Makori A, Zamir O, et al. Rectal penetrating injuries from blast trauma. *Isr Med Assoc J* 2002;4(7):557-558. **(Case report)**

46. Royal School of Artillery. *Warheads*. Available at: http://www.army.mod.uk/royalartillery/units/royal_school_of_artillery/basic_science/warheads.htm. Accessed April 4, 2006. **(Review of warhead technology)**

47. GlobalSecurity.org. M16 (history and weapon characteristics). Available at: http://www.globalsecurity.org/military/systems/ground/m16.htm. Accessed April 4, 2006. **(Review article)**

48. Thompson D, Brown S, Mallonee S, et al. Fatal and Nonfatal injuries among U.S. Air Force personnel resulting from the terrorist bombing of the Khobar Towers. *J Trauma* 2004;57:208-215. **(Retrospective review)**

49. Wong TY, Seet MB, Ang CL. Eye injuries in twentieth century warfare: a historical perspective. *Surv Ophthalmol* 1997;41(6):433-459. **(Review article/historical)**

50. Centers for Disease Control and Prevention. *Explosions and Blast Injuries: A Primer for Clinicians*. Available at: http://www.bt.cdc.gov/masstrauma/explosions.asp. Accessed April 4, 2006. **(CDC review article)**

51. de Candole CA. Blast injury. *Can Med Assoc J* 1967;96:207-214. **(Review article/historical)**

52. Stapczynski J. Blast injuries. *Ann Emerg Med* 1982;11:687-694. **(Review article/historical)**

53. Guy RJ, Glover MA, Cripps NP. Primary blast injury: pathophysiology and implications for treatment. Part III: Injury to the central nervous system and the limbs. *J R Nav Med Serv* 2000;86(1):27-31. **(Review)**

54. Hutton JJ. Blast lung: history, concepts, and treatment. *Curr Concepts Trauma Care* 1986;9:8-14. **(Review)**

55. Karmy-Jones R, Kissinger D, Golocovsky M et al. Bomb-related injuries. *Mil Med* 1994;159:536-539. **(Case reports/review)**

56. Anonymous. *Emergency Response to Terrorism Manual*. US Department of Justice and the Federal Emergency Management Agency; 1997.

57. Shuker ST. Maxillofacial blast injuries. *J Craniomaxillofac Surg* 1995;23(2):91-98. **(Review)**

58. Braverman I, Wexler D, Oren M. A novel mode of infection with hepatitis B: penetrating bone fragments due to the explosion of a suicide bomber. *Isr Med Assoc J* 2002;4(7):528-529. **(Case report)**

59. Weiner S, Barrett J. Explosions and explosive device-related injuries. In: Wiener S, ed. *Trauma Management for Civilian and Military Physicians*. Philadelphia, PA: Saunders; 1986:13-26. **(Textbook chapter)**

60. Hadden W, Rutherford W, Merrett J. The injuries of terrorist bombing: A study of 1532 consecutive patients. *Br J Surg* 1978;65:525-531. **(Retrospective review)**

61. Frykberg E, Tepas J, Alexander R. The 1983 Beirut Airport

terrorist bombing: Injury patterns and implications for disaster management. *Am Surg* 1989;55:134-141. **(Retrospective review)**

62. Guy RJ, Watkins PE, Edmondstone WM. Electrocardiographic changes following primary blast injury to the thorax. *J R Nav Med Serv* 2000;86(3):125-133. **(Animal study)**

63. Roberts JC, O'Connor JV, Ward EE. Modelling the effect of non-penetrating ballistic impact as a means of detecting behind-armor blunt trauma. *J Trauma* 2005;58:1241-1251. **(Animal study)**

64. Hirshberg A, Stein M, Walden R. Surgical resource utilization in urban terrorist bombing: a computer simulation. *J Trauma* 1999;47(3):545-550. **(Computer simulation of disaster practice)**

65. Kronenberg J, Ben-Shoshan J, Wolf M. Perforated tympanic membrane after blast injury. *Am J Otolaryngol* 1993;14(1):92-94. **(Retrospective study, 147 patients)**

66. Sorkine P, Szold O, Kluger Y, et al. Permissive hypercapnia ventilation in patients with severe pulmonary blast injury. *J Trauma* 1998;45(1):35-38. **(Case report, 17 patients)**

67. Wagner R, Jamieson P. Pulmonary contusion evaluation and classification by computed tomography. *Surg Clin North Am* 1989;69:31-40. **(Review/case reports)**

68. Uretzky G, Cotev S. The use of continuous positive airway pressure in blast injury of the chest. *Crit Care Med* 1980;8(9):486-489. **(Case report)**

69. Lavery GG, Lowry K. Management of blast injuries and shock lung. *Curr Opin Anaesthesiol* 2004;17:151-157. **(Review article)**

70. Dopfmer UR, Braun JP, Grosse J, et al. Treatment of Severe Pulmonary Hemorrhage After Cardiopulmonary Bypass by Selective, Temporary Balloon Occlusion. *Anesth Analg* 2004;99(5):1280-1282. **(Case report)**

71. Hirshberg B, Oppenheim-Eden A, Pizov R, et al. Recovery from blast lung injury: one-year follow-up. *Chest* 1999;116(6):1683-1688. **(Retrospective review, 11 patients)**

72. van Hulst RA, Klein J, Lachmann B. Gas embolism: pathophysiology and treatment. *Clin Physiol Funct Imaging* 2003;23(5):237-246. **(Very extensive review)**

73. Biancolini CA, Del Bosco CG, Jorge MA. Argentine Jewish community institution bomb explosion. *J Trauma* 1999;47(4):728-732. **(Case report, 86 patients)**

74. Maxson R. Management of pediatric trauma: Blast victims in a mass casualty incident. *Clin Pediatr Emerg Med* 2002;3:256-261. **(Review article discussing lessons learned from Oklahoma City/WTC attacks)**

75. Almogy G, Luria T, Richter E, et al. Can external signs of trauma guide management? Lessons learned from suicide bombing attacks in Israel. *Arch Surg* 2005;140:390-393. **(Retrospective analysis of 15 suicide bombing attacks during 1994-1997)**

76. Cooper GJ. Protection of the lung from blast overpressure by thoracic stress wave decouplers. *J Trauma* 1996;40(3 Suppl):S105-110. **(Animal study)**

77. Nerenberg J, Makris A, Klein H. The effectiveness of different personal protective ensembles in preventing injury to the thorax from blast-type anti-personnel mines. *J Mine Action* 2000. Available at: http://maic.jmu.edu/Journal/4.2/Focus/Effectiveness/effect.htm. Accessed April 4, 2006. **(In vitro evaluation of protective equipment)**

78. Department of Homeland Security. Information Bulletin: Department of Homeland Security. 21 October 2003. **(Responder guideline)**

79. Shurman E. What makes suicide bombers tick? Available at: http://www.israelinsider.com/channels/security/articles/sec_0049.htm. Accessed April 4, 2006. **(Review article describing the strategy and tactics of suicide bombing)**

80. National Institute of Justice. *NIJ Final Report on Law Enforcement Robot Technology Assessment: 2.0 Background*. National Institute of Justice, 2000. Available at: http://www.nlectc.org/jpsg/robotassessment/background.html. Accessed April 4, 2006. **(Review)**

81. TSWG Technical Support Working Group. *Bomb Threat Standoff Card*. Available from: http://www.tswg.gov/tswg/prods_pubs/newBTSCPress.htm. Accessed April 4, 2006. **(Responder guideline)**

Physician CME Questions

49. The blast wave that occurs as a result of detonation of a high explosive moves:
a. Slower than the speed of sound
b. In all directions
c. Straight up
d. Toward the nearest solid structure
e. At a 90-degree angle to the trajectory of the missile containing the high explosive

50. Secondary blast injuries result from:
a. Movement of the victim's body by the blast wave
b. High explosives only
c. Inhaled gases or toxins secondarily produced by the chemical reaction of the explosion
d. Submucosal dissection of the alveolar tissue
e. Flying debris and bomb fragments

51. Which of the following injuries/findings are *NOT* associated with early mortality from a blast injury?
a. Multiple head and torso trauma
b. Pharyngeal ecchymosis and petechiae
c. Abrasions
d. Frankly bloody hemoptysis
e. Traumatic blast amputations of a limb

52. The body part that is most sensitive to the effects of the blast wave and is most often affected by an explosion is the:
a. Eye
b. Ear
c. Bladder
d. Liver
e. Large intestine

53. Which of the following statements about blast injury and the lung are true?

a. It is not necessary to monitor the oxygen saturation in blast victims without external evidence of trauma or respiratory symptoms, since the symptoms will precede the development of oxygen abnormalities.
b. Visceral structures, such as the liver, are more susceptible to blast injury than is the lung.
c. Rapid aeromedical evacuation is safe in patients who have signs of pulmonary blast injury.
d. The chest x-ray of patients with blast injury to the lung may often have a pulmonary "butterfly" pattern.
e. Ventilation of the patient with "blast lung" (pulmonary barotrauma) is best accomplished with high peak inspiratory pressures.

54. Which of the following appears to be the best marker for pulmonary blast injury?

a. Hematochezia
b. Tympanic membrane trauma
c. Oropharyngeal ecchymosis or hemorrhage
d. Tender and guarded abdomen
e. Epistaxis

55. Which of the following statements is true regarding the care of patients with blast injury?

a. Objects that are impaling a person should be removed or manipulated only in an operating room.
b. Chemical burns of the eye should be treated by at least 5 minutes of continuous irrigation with saline or tap water.
c. Direct injury to the fetus is common when a pregnant woman (with pregnancy under 16 weeks) is injured in an explosion.
d. Submucosal dissection of the trachea during intubation is common in patients who have been injured in an explosion.
e. In suicide bombings, objects such as nuts and bolts are often added to the explosive mixture or device in order to increase the number of casualties.

56. Which of the following statements is true regarding blast injuries?

a. The kidneys are the visceral structures that are most frequently injured by a primary blast.
b. In order to avoid infection, nothing should be placed on burns sustained during an explosion.
c. Pain out of proportion to the injury is a characteristic sign of a gas embolus due to blast injury.
d. An explosion in a confined space is generally associated with more fatalities and blast injuries than an explosion in an open space.
e. Hematemesis is a good marker of intraabdominal blast injury.

57. The most common presentation of a terrorist blast injury is:

a. Primary blast injury to the lung
b. Secondary blast injury from fragments of the bomb
c. Tertiary blast injury
d. Quaternary blast injury
e. None of the above

58. Which of the following agents is a low-order explosive?

a. Gunpowder (black powder)
b. Semtex
c. Composition C-4 (RDX)
d. Ammonium nitrate-fuel oil (ANFO) mixture
e. All of the above

59. Which of the following are symptoms/signs of barotrauma to the ears?

a. Headache
b. Epistaxis
c. Ringing in the ears
d. Shortness of breath
e. None of the above

60. Compression injuries to air-filled organs, such as the lungs, would be expected as a result of:

a. Primary blast injury
b. Secondary blast injury
c. Tertiary blast injury
d. Quaternary blast injury
e. None of the above

61. Which of the following is *NOT* true about an explosion of a high-order explosive?

a. The physical shock wave pushes ahead of the expanding gases of the explosion.
b. Expanding gases, heat, and flying debris are potential mechanisms of injury.
c. As in the movies, people can easily outrun the blast wave.
d. Fifty tons of TNT can cause up to 1 million pounds of pressure per square inch in less than 2 millionths of a second.
e. All of the above answers are true.

62. At a blast overpressure of 15 pounds per inch, 50% of victims will:

a. Die
b. Have ruptured tympanic membranes
c. Have implosion injuries to the small bowel and colon
d. Will probably have pulmonary injury (blast lung)
e. All of the above

Physician CME questions conclude on back page

63. The extent of damage due to the pressure wave is dependent upon which of the following?

a. The fourth power of the distance of the incident blast wave from the explosion
b. The trough of the initial negative pressure wave
c. The medium in which the blast wave travels
d. The reflection from the ground wave
e. All of the above

64. Which of the following statements is true?

a. Lung injury is rarely associated with primary blast injury.
b. Pneumoperitoneum is a common complication of GI barotrauma.
c. Injuries to the abdominal contents due to primary blast injuries are more common in solid organs.
d. Gastrointestinal injury associated with primary blast injury is inconsistent in presentation.

Coming in Future Issues:

Seizures • Pain Control In The ED

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case-
- control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA* 1992;268(16):2289-2295.

Physician CME Information

This CME enduring material is provided by Mount Sinai School of Medicine and has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education. Credit may be obtained by reading each issue and completing the printed post-tests administered in December and June or online single-issue post-tests administered at EBMedicine.net.

Target Audience: This enduring material is designed for emergency medicine physicians.

Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Date of Original Release: This issue of *Emergency Medicine Practice* was published April 10, 2006. **This activity is eligible for CME credit through April 1, 2009.** The latest review of this material was April 3, 2006.

Discussion of Investigational Information: As part of the newsletter, faculty may be presenting investigational information about pharmaceutical products that is outside Food and Drug Administration approved labeling. Information presented as part of this activity is intended solely as continuing medical education and is not intended to promote off-label use of any pharmaceutical product. *Disclosure of Off-Label Usage*: This issue of *Emergency Medicine Practice* discusses no off-label use of any pharmaceutical product.

Faculty Disclosure: It is the policy of MSSM to ensure objectivity, balance, independence, transparency, and scientific rigor in all CME-sponsored educational activities. All faculty participating in the planning or implementation of a sponsored activity are expected to disclose to the audience any relevant financial relationships and to assist in resolving any conflict of interest that may arise from the relationship. Presenters must also make a meaningful disclosure to the audience of their discussions of unlabeled or unapproved drugs or devices. The information received is as follows: Dr. Stewart, Dr. Jagoda, and Dr. Howell report no significant financial interest or other relationship with the manufacturer(s) of any commercial product(s) discussed in this educational presentation.

Accreditation: Mount Sinai School of Medicine is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

Credit Designation: Mount Sinai School of Medicine designates this educational activity for up to 4 hours of Category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit actually spent in the educational activity. *Emergency Medicine Practice* is approved by the American College of Emergency Physicians for 48 hours of ACEP Category 1 credit (per annual subscription). *Emergency Medicine Practice* has been approved by the American Academy of Family Physicians as having educational content acceptable for Prescribed credit. Term of approval covers issues published within one year from the distribution date of July 1, 2005. This issue has been reviewed and is acceptable for up to 4 Prescribed credits. Credit may be claimed for three years from the date of this issue. *Emergency Medicine Practice* has been approved for 48 Category 2-B credit hours by the American Osteopathic Association.

Earning Credit: Two Convenient Methods
- **Print Subscription Semester Program:** Paid subscribers with current and valid licenses in the United States who read all CME articles during each *Emergency Medicine Practice* six-month testing period, complete the post-test and the CME Evaluation Form distributed with the December and June issues, and return it according to the published instructions are eligible for up to 4 hours of Category 1 credit toward the AMA Physician's Recognition Award (PRA) for each issue. You must complete both the post-test and CME Evaluation Form to receive credit. Results will be kept confidential. CME certificates will be delivered to each participant scoring higher than 70%.
- **Online Single-Issue Program:** Paid subscribers with current and valid licenses in the United States who read this *Emergency Medicine Practice* CME article and complete the online post-test and CME Evaluation Form at EBMedicine.net are eligible for up to 4 hours of Category 1 credit toward the AMA Physician's Recognition Award (PRA). You must complete both the post-test and CME Evaluation Form to receive credit. Results will be kept confidential. CME certificates may be printed directly from the Web site to each participant scoring higher than 70%.

***Emergency Medicine Practice* is not affiliated with any pharmaceutical firm or medical device manufacturer.**

CEO: Robert Williford. **President & General Manager**: Stephanie Williford. **Publisher**: Cheryl Strauss. **Director of Member Services**: Charlotte Pratt.

Direct all editorial or subscription-related questions to EB Practice, LLC: **1-800-249-5770** • Fax: 1-770-500-1316 • Non-U.S. subscribers, call: 1-678-366-7933
EB Practice, LLC • 305 Windlake Court • Alpharetta, GA 30022
E-mail: **emp@empractice.net** • Web Site: **EBMedicine.net**

Emergency Medicine Practice (ISSN Print: 1524-1971, ISSN Online: 1559-3908) is published monthly (12 times per year) by EB Practice, LLC, 305 Windlake Court, Alpharetta, GA 30022.