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The Practical Journal for Emergency Physicians

Emergency Medicine Reports®

Volume 25, Number 4 February 9, 2004

Evaluation and Management of Patients with Blast Injuries in the ED

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Prior to 1995, United States civilian emergency physicians had little experience or interest in the effects of explosive devices. This abruptly changed with the destruction of the Alfred P. Murrah Federal building by a truck bomb in downtown Oklahoma City in 1995 that resulted in more than 750 casualties with 167 fatalities.[1] This was followed by the explosion of a satchel bomb in Atlanta at the 1996 Olympics.[2] Many smaller devices are found and/or exploded each year in the United States.[3] Although we have passed the second anniversary of the World Trade Center attack on Sept. 11, 2001, multiple new threats of violence within the United States have been discovered. U.S. physicians have been forced to consider that they, too, may face the specter of explosions and blast injuries due to terrorism[4] or accidents.

Recent terrorism incidents around the world have included an increasing use of suicidal/homicidal bombers who deliberately accompany the explosive device (often wearing it) to ensure that the maximum effect is derived from the explosive device. These bombers have walked or driven into buses, cafes, residential areas, guard-posts, and governmental buildings. The use of these devices in the United States has not yet occurred, but given the political climate, is very likely to occur. The emergency physician is likely to see the effects of these devices. Increasingly, information resources such as the Internet, terrorist training camps, and even library and television sources have made readily available the knowledge needed to construct these simple and very effective explosive devices.

The threat of delivered explosives is not confined to mail rooms or government facilities nor do they require a proponent such as the "Unabomber."[5] A substantial number of explosive devices have been placed in academic facilities and in public parks during events.[6,7] (See Figure 1.) *In addition, emergency physicians could encounter blast injury patients from industrial accidents or explosions at any time.*

This article will review the current literature about blast injuries. Explosions have the potential to cause multi-system injuries involving multiple patients simultaneously. The poten-

tial mechanisms of injury, early signs of these injuries, and the natural course of the problems caused by explosive blasts will be discussed.

Investigation into blast injury is not a new area of study for those interested in combat medicine. Present knowledge of the effects of blast injury date back to the Balkan wars in 1914 when Franchino Rusca, a Swiss researcher, observed that three soldiers had been killed by an explosion without evidence of any external injuries.[8] Using rabbits as an animal model, he demonstrated that the cause of death was pulmonary embolism. During World War I, blast injury was thought to be a nervous system disorder and labeled "shell-shock." (Psychological casualties were lumped together with those who had no visible injuries.)[9] In World War II, a noteworthy number of casualties were found among civilians in both German and British cities after bombing raids. The term blast lung was coined for massive pulmonary hemorrhage from disruption of the alveolar architecture and formation of alveolar-venous fistulas resulting in air embolism.[10] Following World War II, nuclear weapon blast injury was investigated intensively in the United States. Only since the onset of terrorist and suicide bombings in Ireland and the Near East have civilians been significantly concerned about the cause and treatment of blast injuries.[9]

Blast injury has an overall lethality of about 7.8% in open air.[9] This jumps to nearly 49% when the blast occurs in confined spaces.[11,12] About 70% of victims sustain minor soft-tissue injuries.[7,12] Traumatic amputations occur in about 11% of victims. The 2001 World Trade Center explosion and subsequent building collapse was unusual, as most victims had either few injuries or died as the buildings crashed down on them.

—The Editor

Mechanisms of Injury

Powerful explosions are the result of a change in chemical potential energy to kinetic injury in a very short period of time. As noted earlier, explosive agents cause both unique patterns of injury and very common injuries. Trauma caused by explosions traditionally has been divided into the injury caused by the direct effect of the blast wave (primary injuries); the effects caused by other objects that are accelerated by the explosive wave, (secondary injuries); the effects caused by movement of the victim (tertiary injuries); and miscellaneous effects caused by the explosion or explosives. (*See Figure 2.*)

The injury pattern following an explosion is partly random. This trauma results from a combination of the size of the explosive charge, the composition and nature of the explosive, the container, 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. Explosives are categorized as high-order explosives (HE) or low-order explosives (LE). HE and LE cause somewhat different injury patterns.

High-Order Explosives. High-order explosives are chemical materials that have an extremely high reaction rate. This reaction often is called a detonation. Examples of high-order explosives include nitroglycerin, dynamite, C-4, picric acid, Semtex, ammonium nitrate fuel oil mixture (ANFO), trinitrotoluene (TNT), and pentaeruthrotetranitrate (PETN).

When an HE detonates, it is converted almost instantaneously into a gas at very high pressure and temperature. For example, the major ingredient in Composition C-4 (Cyclotrimethylenetrinitramine or RDX [Royal Demolition eXplosive]) can gener-

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by Thomson American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
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GST Registration No.: R128870672

Periodicals postage paid at Atlanta, GA. **POSTMASTER:** Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $31. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, $359 each; 10 to 20 additional copies, $319 each.

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Figure 1. Historic Location of U.S. Bomb Incidents[6,7]

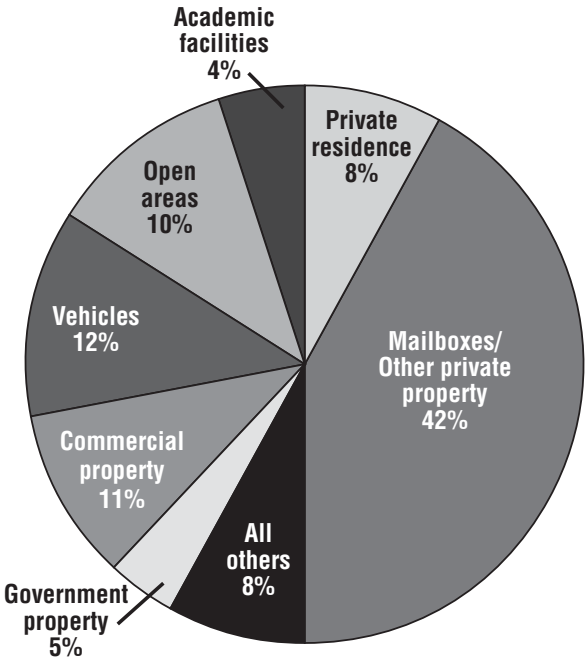


Figure 2. Diagram of Primary, Secondary, and Tertiary Blast Injuries

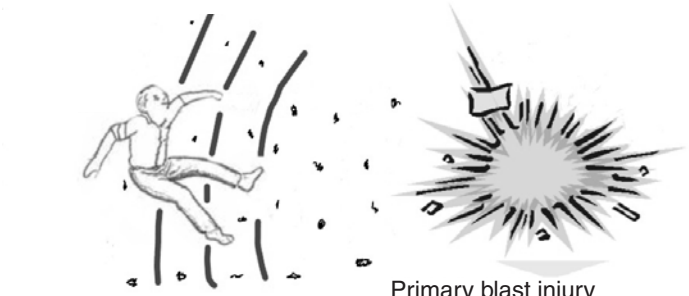


Illustration by Charles Stewart, MD.

ate an initial pressure of more than 4 million pounds per square inch (4x10E6 PSI).[13] 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 to the immediate surroundings.

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

The blast wave refers to an intense rise in pressure—often called over pressure—that is created by the detonation of a high explosive.[9] (*See Table 1.*) A typical pressure wave from a high explosive explosion in air is shown in the diagram in Figure 3. The pressure rises almost instantaneously in the ambient environment and then decays exponentially. The peak pressure and the duration of the initial positive phase of the blast wave depend on the size of the explosion and the distance from the center of the detonation. In air, the peak pressure is proportional to the cube root of the weight of explosives and inverse of the cube of the distance from the detonation. It also depends on the type of explosives used. 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; and
- focusing due to a confined area or walls.

This blast wave has three components:

1. a single spike of increased pressure;
2. an exponential decay with time; and
3. a much longer duration negative pressure wave with pressure below initial ambient pressure.

This increase in pressure can be so abrupt that it can shatter materials—also known as a shock wave. This effect is termed brisance and varies from one HE to another. 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.[9] 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, the damage to human structures is a function of the peak pressure and the duration of the initial positive phase.

The rapid production of immense amounts of gas creates a block flow of the surrounding medium away from the explosion. Blast wind refers to this rapid bulk movement of air and other gases from the explosion site. It occurs with both LEs and HEs.

Low-Order Explosives. LEs are designed to burn and subsequently release energy relatively slowly. These explosives often are called propellants because the most common use is to propel a projectile through a barrel. The principal military uses for LEs are as propellants and in fuses. Typical improvised LEs include pipe bombs, gunpowder, black powder, and petroleum-based bombs such as Molotov cocktails or gasoline tankers. Since LEs 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

Table 1. Blast Overpressure and Effects

OVERPRESSURE IN PSI	EFFECT
1-2	Frame house destroyed
3-5	Typical commercial construction destroyed
5	Tympanic membrane rupture
15	Tympanic membrane rupture in 50% of patients
30-40	Possible lung injury
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. *The Tactical Edge Journal* Summer 2000: 53-55.

Figure 3. Diagram of Pressure-Time History of an Explosion in Air

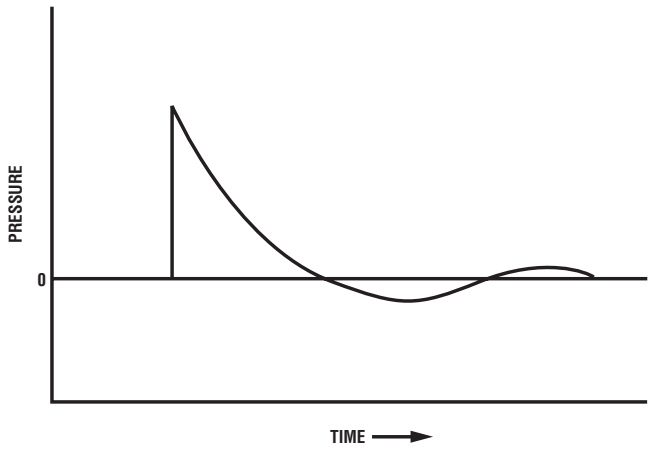


Diagram courtesy of Virtual Naval Hospital, Emergency War Surgery NATO Handbook.

slowly that when the LE is set off in the open, the gases push aside the air with only a flame and no appreciable disturbance. If the LE is confined, the speed of the reaction markedly is increased, but does not approach that of a high-order explosion. The explosion has more of a pushing effect than a shattering effect (blast wind without a blast wave).

The explosion from low-order devices lacks the overpressure wave and, 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 an HE or an LE. Likewise, if the victim is flung by blast wind into a structure, it matters little to either the patient or the clinician that the explosion occurred from an HE or deflagration of an LE.

Source of Explosive

Explosive devices also may be characterized based on their source. The Bureau of Alcohol, Tobacco, and Firearms categorizes explosives as 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 may be professional in appearance and operation and 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 includes 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 the quality of the explosive used from use of commercial explosives, TNT, Semtex, C-4, ammonia-based fertilizer, and fuel oil (used widely as an industrial explosive), to a match-filled pipe bomb. High-quality improvised devices may resemble military weapons in effect. The variety of initiation methods, explosive filling, and fabrication techniques creates a threat that can be quite daunting to the professional explosive ordnance disposal crew. It also means that no matter how innocuous the appearance of an improvised device, the amateur should not touch the device.

Medium in Which the Explosion Occurs

Another important concept that defines blast injury patterns is the medium in which the blast occurs. An underwater blast wave causes far more damage because water is incompressible.[9,14] 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.

Another characteristic of blast waves is that they are indeed waves. The injury patterns they produce are related not only to the medium through which they travel, but also to 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.[15] In fact, victims located between the blast and a building generally suffer 2-3 times the degree of injury that individuals in an open environment receive.[12]

Body armor provides a false sense of security during an explosive detonation. The body armor does protect the victim from shrapnel and, to a lesser extent, from 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 the front and back of the armor. (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 risk of enhancing the blast wave.) The medical provider should not assume that body armor will protect the victim from an explosion-related injury.[16]

Pathophysiology

Primary Blast Injury. Primary blast injuries are caused only by HEs and are due to the direct effects of the blast wave on the human body. (Since LEs do not form a supersonic blast wave, they cannot cause primary blast injury. This difference is the sole apparent clinical difference between wounds caused by an LE and those caused by an HE.)

The overall incidence of primary blast injury is about 20%. This can be divided into about 1% for open-air explosions and 30-40% for closed-space explosions. (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).[17]

The extent of damage from the explosion depends upon:

- the peak of the initial positive wave (this is directly related to the magnitude of the explosion);
- the duration of the overpressure;
- the medium in which it explodes;
- the distance from the incident blast wave; and
- the nature and number of reflections in confined areas and with reflecting walls.

Three Possible Mechanisms of Injury of Primary Blast Injury. The first mechanism of injury usually described as the etiology of primary blast injury is the implosion of gas-filled spaces as the high pressure blast wave compresses them.[18,19] This theory states that the organs that are most vulnerable to blast injury are those containing air because the air readily is compressed. Hollow organs are compressed and disrupted by the rapid external pressure increase. The resulting force causes shearing of vascular beds, ear damage, pulmonary contusions, pneumothorax, and gastrointestinal (GI) hemorrhage. In some cases, the force of a pressure wave can be significant enough that it forces air into a blood vessel, creating air emboli. There isn't enough time during the passage of the overpressure phase of the blast wave for gas to transfer from the lungs to the outside world through the trachea.[20]

The second possible major mechanism of primary blast injury often is termed spalling. This is thought to occur when a blast wave moves from a dense medium such as water to a less dense medium such as air. This often is compared to the effect of striking the outside of a rusty bucket with a hammer and watching the flakes of rust fly off the inside of the bucket. In human tissues, the transfer of reflected blast injury through the dense substrates such as muscle and liver into the less dense material of the GI tract and lungs may cause spalling. Spalling also is believed to occur when the blast wave transits from the rib cage into the lung.

The third possible mechanism of primary blast injury is the inertial effect related to the differences in tissue density and speed of the blast wave through the tissues of different densities. This may be the most important effect of the blast injury and currently is thought to be the major cause of primary blast injuries. The differences in speed of movement result in shearing and tearing forces expressed as a stress wave propagated into the underlying tissues.[21,22] The resulting forces exceed the tensile strength of the material and cause shearing of vascular beds, pulmonary contusions, and GI hemorrhages.[23]

The true mechanism of primary blast injury likely is some combination of these theoretical mechanisms. Of these, the shearing and tearing forces appear to fit best. Primary blast injury is common in the ear, the respiratory tract, and the GI tract.

Ear Damage. Of the three organ systems, the ear is the most easily damaged, but it also is the easiest to protect. The structures of the ear are designed to collect and magnify sounds, so that the tympanic membrane moves with the sounds. Unfortunately, the ear's structures 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 kPa (14 PSI) almost all eardrums rupture. 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.

Rupture of the eardrum will cause pain, hearing loss, and may cause tinnitus. Eardrum perforations, hearing loss, and dizziness may interfere with daily activities and may affect the individual's quality of life.[24]

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

It often is 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 with his head out of the water, the tympanic membranes may not be exposed to an underwater blast wave. Isolated eardrum rupture does not appear to be a good marker of either concealed pulmonary blast injury or poor prognosis.[25]

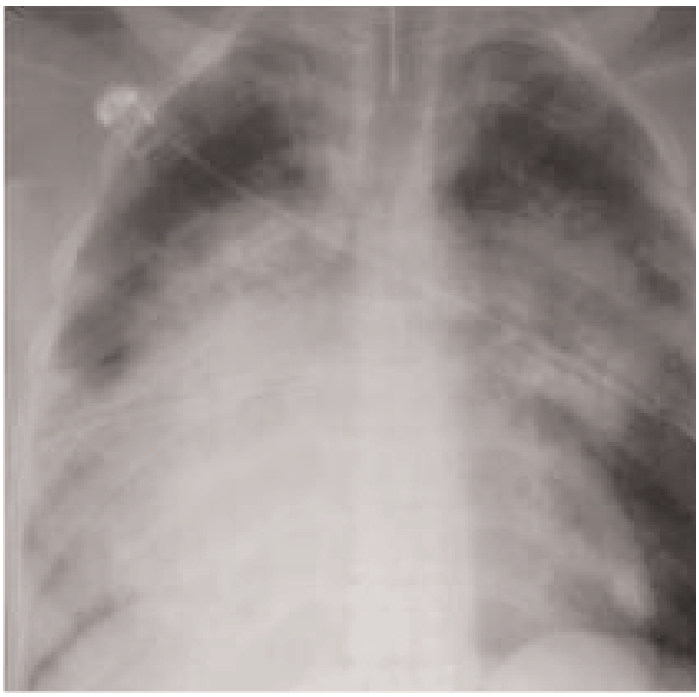
Auditory barotrauma is quite common in blast injuries. In the Oklahoma City bombing, the incidence of auditory injury was 35%.[1,13] This does not count those patients with partial, temporary hearing loss or those who complained of tinnitus for an extended period of time.[24]

Pulmonary Damage. The lungs have been considered to be the non-auditory organs most at risk for suffering primary blast injury. Blast lung is a direct consequence of the supersonic pressure wave generated by an HE.[26] (*See Figure 4.*) It is the most common fatal injury caused by the primary blast injury 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 causes lung injuries.

Damage to the lungs can include pulmonary contusions with or without a laceration, and/or pulmonary barotrauma such as pneumothorax, pulmonary interstitial emphysema, pneumomediastinum, or subcutaneous emphysema.

It is best to assume that if a patient is wheezing after a blast injury, that the wheezing is due to a pulmonary contusion. Other causes of wheezing may be pulmonary edema from myocardial

Figure 4. Blast Lung



Reprinted with permission from: Wolf YG. Vascular trauma in high-velocity gunshot wounds and shrapnel-blast injuries in Israel. *Surg Clin North Am* 2002;82:237-244.

contusion or infarction, or exacerbation of underlying disorders such as asthma or chronic obstructive pulmonary disease (COPD).

The most common lung injury associated with a blast wave is a pulmonary contusion. This may take the form of micro-hemorrhages 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.[27-29] The alveolar wall may be torn, causing a blood-filled emphysematous change to the lung. Pulmonary contusions may develop with or without a pulmonary laceration.

Pulmonary contusions impair gas exchange at the alveolar level. The changes seen on microscopic examination closely resemble the pulmonary contusions seen in non-penetrating blunt chest trauma.

Parallel thoracic ecchymoses, once thought to be along the ribs, may be seen with larger blast loads.[20,28] 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.[29,30]

The patient may have minimal or no symptoms initially. The patient also may complain of chest pain or respiratory distress. Signs of blast lung usually are present at the time of the initial evaluation, but have been reported as late as 48 hours after the explosion occurs.

The overpressure may cause pulmonary barotrauma, including pneumothorax or pneumomediastinum. The patient may develop pulmonary interstitial emphysema, subcutaneous emphysema, and systemic air embolism with larger blast loads.[20,22,23] 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.

Blast lung is characterized clinically by the triad of apnea, bradycardia, and hypotension. The clinician should suspect blast lung in any victim who presents with dyspnea, cough, hemoptysis, or chest pain following blast exposure.

A simple frontal chest x-ray is diagnostic for most cases of pulmonary barotrauma from blast. 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 one week.[31]

Gastrointestinal Damage. GI injuries may not be apparent externally. They have a great potential to cause death and may be much more difficult to protect against.

GI injuries once were 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.[32] In this series, all patients were injured from open air explosions. The patient may have a greater risk for GI injury when exposed to an underwater explosion.[33]

The GI injury of primary blast injury is inconsistent in presentation. It may consist of hemorrhage beneath the visceral peritoneum or may extend into the mesentery, colon, and cecum.[27,28] Contused bowel may necrose and perforate several days after the initial trauma. The perforated bowel may be apparent immediately, or may perforate only after a delay of up to 48 hours.[34,35]

Pneumoperitoneum is a relatively rare complication of GI barotrauma.[36] This complication has a wide differential diagnosis ranging from perforated viscus to simple dissection of air through the retroperitoneum.

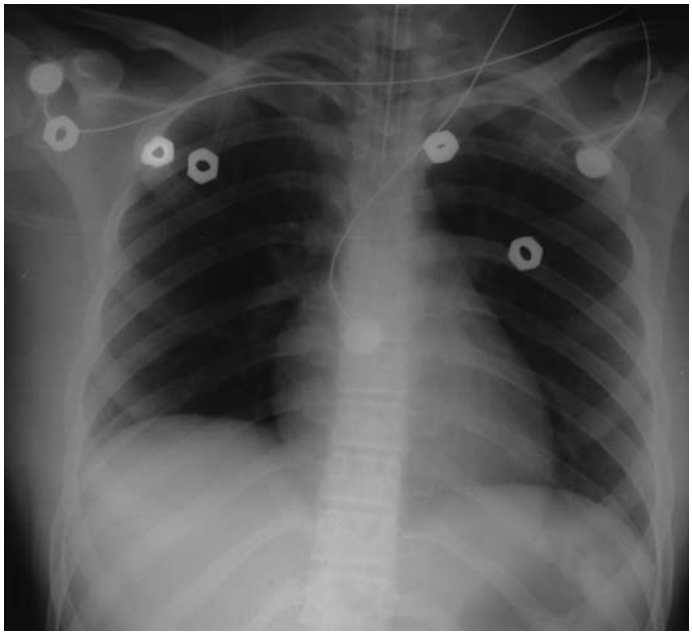
The colon is the most common site of both hemorrhage and perforation.[33] This is thought to be because the colon has the most bowel gas accumulation in the GI tract.

Solid organ laceration and testicular rupture also are seen due to primary blast injury, but are less frequent and often are associated with large blast loads.[37] The most common solid organ lesions reported were subcapsular hematomas in the liver, spleen, and kidneys.[31] Mesenteric, scrotal, and retroperitoneal hemorrhages have been reported.[28]

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 GI tract should be suspected in anyone exposed to an explosion who has abdominal pain, nausea, vomiting, hematemesis, rectal pain, testicular pain, unexplained hypovolemia, or any finding compatible with an acute abdomen.

The clinician should be aware that the abundant high-velocity fragments associated with recent suicide bombs also may cause intra-abdominal injuries. These injuries certainly can include penetrating bowel injuries.[38] Initial symptoms of penetration are the same as outlined above.

Figure 5. Secondary Blast Injury: Projectiles Thrown by the Force of the Blast



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

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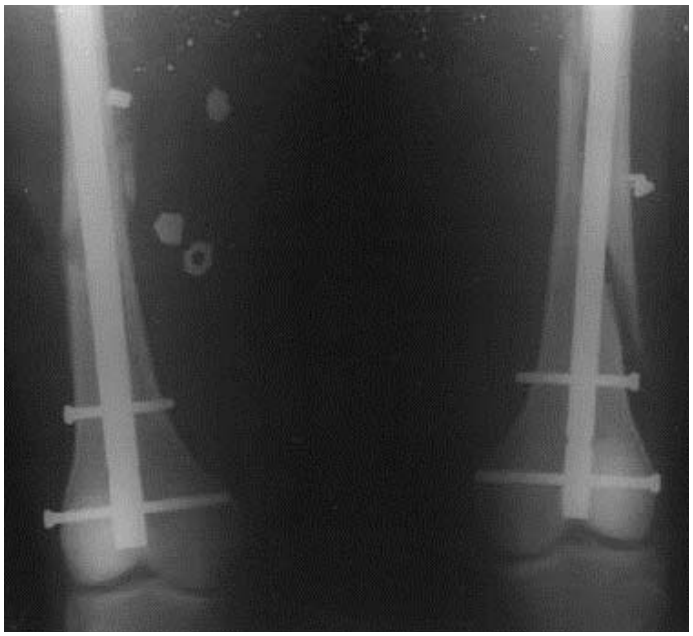
Figure 6. Secondary Blast Injury: Projectiles Thrown by the Force of the Blast



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

Reprinted with permission from Zvi Gimmon, MD.

Brain Injury. Primary blast injury can cause concussion or traumatic brain injury, although this finding is difficult to differentiate from the concussion due to impact with another object. The clinician should be quick to consider computed tomography (CT) or magnetic resonance imaging (MRI) in these patients.

Cardiac Injury. Myocardial contusion may occur with arrhythmia or hypotension.[39]

Secondary Blast Injury. Secondary blast injury is caused by the bomb fragments and other debris that are propelled by the intense energy release of the explosion. (These fragments often erroneously are referred to as "shrapnel." Shrapnel is the name for an artillery round containing multiple round lead balls that was designed during World War I by then-Lt. Shrapnel. This round essentially functions as a very large shotgun with several hundred half-inch lead balls.) (*See Figures 5-7.*) Conventional military explosives may create multiple fragments with initial velocities of up to 2500 m/second (8202 feet/second).[40] (In contrast, the very fast moving M-16 round has a muzzle velocity of 2800 feet [853 meters] per second.)[41]

These flying projectiles can produce both penetrating and blunt trauma, depending on the size of the projectile and the speed at which it travels. With these speeds, 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 U.S. Embassy in Nairobi, flying glass wounded victims up to 2 kilometers away.[39] (The reason for the "stand-off distances" noted in the accompanying tables is to decrease to acceptable limits the number of injuries that occur from flying debris when the bomb explodes.) The farther away a person is from the explosion, the less serious the injury.

Terrorist devices often have additional objects such as nails, nuts, and bolts added to the explosive mixture to increase the effects of secondary blast injury. 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 to increase the number of fragments (shrapnel) flung by the explosion.

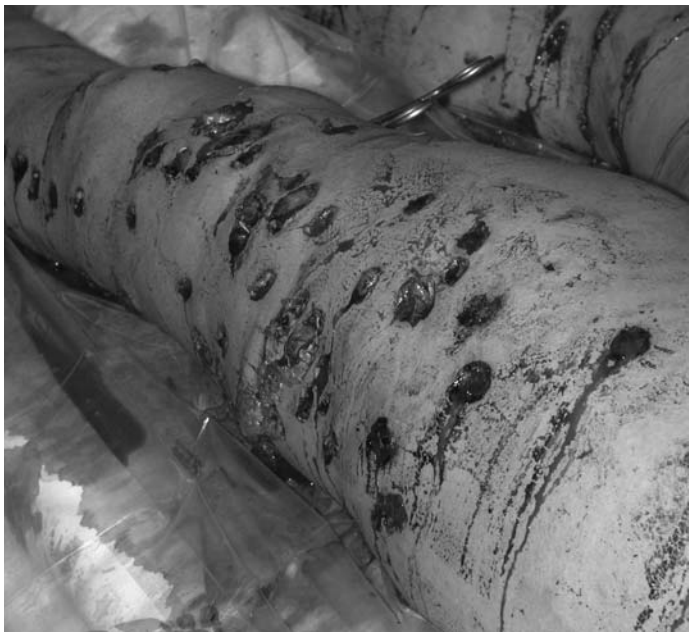
Secondary blast injuries are much more common than primary blast injuries. Indeed, secondary blast injury is the most common cause of death in blast victims. The penetrating injuries occur most often in the exposed areas such as the head, neck, and extremities.

Glass causes many of the secondary blast injuries (up to 50% of all blast injuries). Victims who are peppered with glass often are difficult to distinguish from victims who are peppered with glass and have penetrating injuries.[42]

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

Up to 10% of blast survivors have significant eye injuries.[43] (*See Figure 8.*) These injuries may be perforations from high-velocity projectiles. Glass is notorious for causing these ocular injuries. Window fragments often don't kill, but they can cause blindness and ruptured globes. At the speed that explosively propelled fragments of glass travel, there is no time for the blink reflex to operate. These injuries may occur with minimal initial

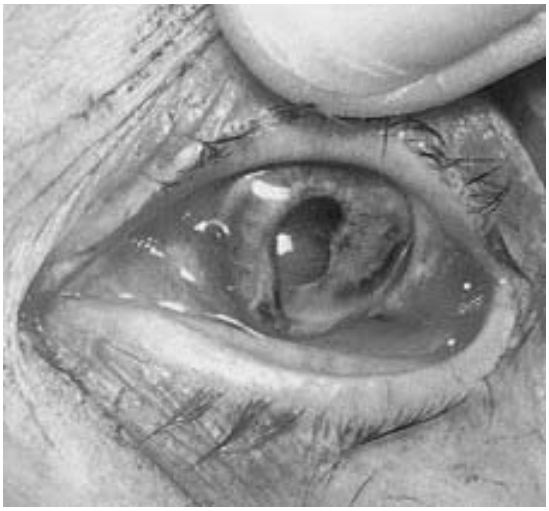
Figure 7. Fragment Injuries from a Bomb



Fragment injuries from a suicide/homicide bomber in Israel. The device has large metal nuts contained in an explosive vest.

Photo courtesy of Professor Zvi Gimmon, MD.

Figure 8. Eye Rupture



This image shows rupture of the eye with prolapsing iris due to an explosion and direct trauma.

Photo courtesy of Richmond Eye Associates at www.richmond-eye.com/fireworks.htm.

discomfort and may 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.[20,44] Tertiary effects result from the bulk flow of gas away from the explosion. Blast winds can generate a body acceleration of more than 15 gs. 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 positioned in the path 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.

The deceleration caused by impact into a rigid structure causes the majority of injuries. A person who is flung into a fortified immovable object with a velocity greater than 26 feet/second (7.92 meters/second) has a mortality rate of about 50%.[45] 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 tumble along the ground, sustaining abrasions, contusions, and "road rash."

Miscellaneous Blast Effects (Quaternary Blast Injuries). This category of injury 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.

The unprotected human body can survive a blast with a peak overpressure of 30 PSI (206 kPa), but buildings and other structures collapse with the stress of only a few pounds per square inch. This means that people may survive the effects of the blast only to be injured by collapsing buildings.

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

Patients who have been exposed to a blast in an enclosed area should have carboxyhemoglobin levels obtained. Inhalation of irritant gases or dusts also may trigger wheezing in these patients.

Immediate Death. Fatal injuries may occur due to blast effects involving the head, chest, and abdomen and often are seen in victims who are close to the detonation.[46] Indeed, in some of these victims close to the site of the blast, parts of the victim (or perpetrator) may become missiles that kill or wound other victims.[47] Immediate death may occur from massive pulmonary bleeding with rapid suffocation despite good care. The patient may develop a massive air embolism or may sustain a significant brain injury. The patient 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 without signs of life;
- there are no effective respirations;
- there is no palpable pulse; and
- there are dilated pupils.

Persons with immediate, severe respiratory insufficiency that is caused by a blast effect have far less chance of survival.

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

Evaluation and Management

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. For a rough prediction of the number of "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, which may include EMS, fire, police, and media.

Most of the injuries seen after a conventional explosive detonates are blunt, penetrating, and thermal trauma that is well known to prehospital providers, emergency physicians, and trauma surgeons.[48] Much of this trauma includes soft-tissue, orthopedic, or head injuries.[11,49,50] The approach to the casualty with blast-related injury, therefore, is the same as for any other trauma victim.

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. A thorough physical examination then should be performed. The clinician should look for sentinel signs of potentially significant blast exposure. (*See Tables 2 and 3.*) 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 does not consider the possibility of primary blast injury, the patient's care may be complicated further.

For each patient, attempt to determine the distance from the explosion and whether the victim was in the open air or in an enclosure. If the patient was wearing body armor, this should be noted in the record. Body armor increases the chance and severity of primary blast injury, but provides significant protection against fragment injuries.

Distance obviously decreases the risk of primary blast injury (at least in the open). (*See Table 4.*) If the patient was in water, this should be noted, and the suspicion for intra-abdominal blast injury heightened.

Laboratory and Radiographic Studies

There are few screening studies that are of any benefit in the casualty with primary blast injury. A chest x-ray, however, should be obtained in all patients who have been near a significant explosion. The clinician should look for evidence of pulmonary contusion (as noted earlier) and barotrauma. A chest x-ray also may show free air under the diaphragm, signifying hollow viscus rupture in the abdomen.

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 unconscious, these CT studies are not optional.

The only laboratory study that is useful is serial hemoglobin determinations. These appear to be useful in casualties who have severe bleeding. 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. (*See Figure 9 for an algorithm on assessment of patients with blast injury.*)

Table 2. 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* • Tinnitus* • Earache*
Cardiovascular	• Tachycardia • Fall of mean arterial blood pressure
Gastrointestinal	• Nausea* • Abdominal tenderness* • Abdominal rigidity*
Neurologic system	• Vertigo • Retrograde amnesia
Ocular injury	• Eye irritation** • Hyphema** • Distorted pupil** • Decreased vision** • Blindness** • Funduscopic findings of retinal artery air embolism**
Respiratory system	• Cyanosis* • Ecchymosis or petechiae in hypopharynx* • Cough (often dry)* • Tachypnea (often preceded by a short period of apnea)* • Dyspnea* • Hemoptysis* • Rales or moist crepitation in lung fields* • Chest pain*

* Most common findings
** Common findings

Table 3. 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 central nervous system 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.

Adapted from CDC Mass Trauma Preparedness and Response web site available at http://www.cdc.gov/masstrauma/preparedness/primer.htm. Accessed 1/26/04.

Treatment

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

Debris gently should be removed from the external canal. The canal may be irrigated with an antiseptic solution if necessary. Neither antibiotics nor ear drops are recommended, particularly if the patient has a ruptured tympanic membrane. Tympanoplasty is reserved only for failures of conservative therapy.

Pulmonary. Blast lung is treated by correcting the effects of barotrauma if any is found. Gas exchange is supported. The provider should be aware that positive pressure ventilation may exacerbate pneumothorax and cause air embolism in the presence of bronchopleural fistula. The patient's body should be positioned to ensure that the effects of air embolism are minimized.

In victims with mild respiratory distress, supplemental oxygen by nasal cannula is appropriate. Those patients with significant respiratory distress or hemoptysis should have an endotracheal tube placed. This is not without hazard, however.

Positive pressure ventilation markedly increases the possibility of both air embolism and pulmonary barotrauma. The provider should take the least invasive measure that still provides appropriate airway support in these patients.[51] Avoid peak end-expiratory pressure (PEEP) and high ventilation pressures.

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.[52] Patients with more than 28% contusion always required ventilation.

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. If the patient needs air evacuation, this becomes more desirable. If a patient with a blast lung injury abruptly decompensates, the clinician should presume that the patient has a tension pneumothorax and treat accordingly.

If the patient survives the blast lung and other trauma, there is a good chance that he will regain lung function within a year.[53]

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 intra-abdominal solid organ rupture;
- hypotension from compression of vessels and heart by pneumothorax;
- hypotension due to the cardiovascular effects of an air embolism; and
- hypotension due to vagal reflexes.

The patient's fluid volume should be supported without excessive fluid replacement. Often, blood products or colloid solutions should be used rather than crystalloid. Too much fluid replacement of course can cause increased respiratory distress as either congestive heart failure or acute respiratory distress syndrome.

Table 4. 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	
Pipe 4" x 12"	20 pounds			
Pipe 8" x 24"	120 pounds (uncommon)			
Bottle, 2 liter	10 pounds			
Bottle, 2 gallon	30 pounds			
Bottle, 5 gallon	70 pounds (uncommon)			
Box Shoebox	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
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 or cargo van	4000 pounds 1818 kilos	200 feet 61 meters	2750 feet 838 meter	2750 feet 838 meters
Small box van	10,000 pounds 4545 kilos	300 feet 91 meters	3750 feet 1143 meters	3750 feet 1143 meters
Box van or water/fuel truck	30,000 pounds 13,636 kilos	450 feet 137 feet	6500 feet 1982 meters	6500 feet 1982 meters
Semi trailer	60,000 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, bomb makers are not limited to these dimensions and may use different materials with smaller or larger capacities. Remember that for pipe bombs, in particular, bomb makers often may use multiple containers. Stand-off distances for improvised devices that are not given may be estimated from given distances for smaller and larger devices. When estimating, a larger stand-off distance is safer. The stand-off distance is the closest distance that an unprotected human should be to an explosive device. This distance is calculated/approximated to minimize the probability of damage from bomb fragments and debris hurled by the bomb. In all cases, it still is safer to seek cover behind something solid such as a retaining wall or another building.

** The table is built from several ATF and TSWG publications. It is unclear 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.

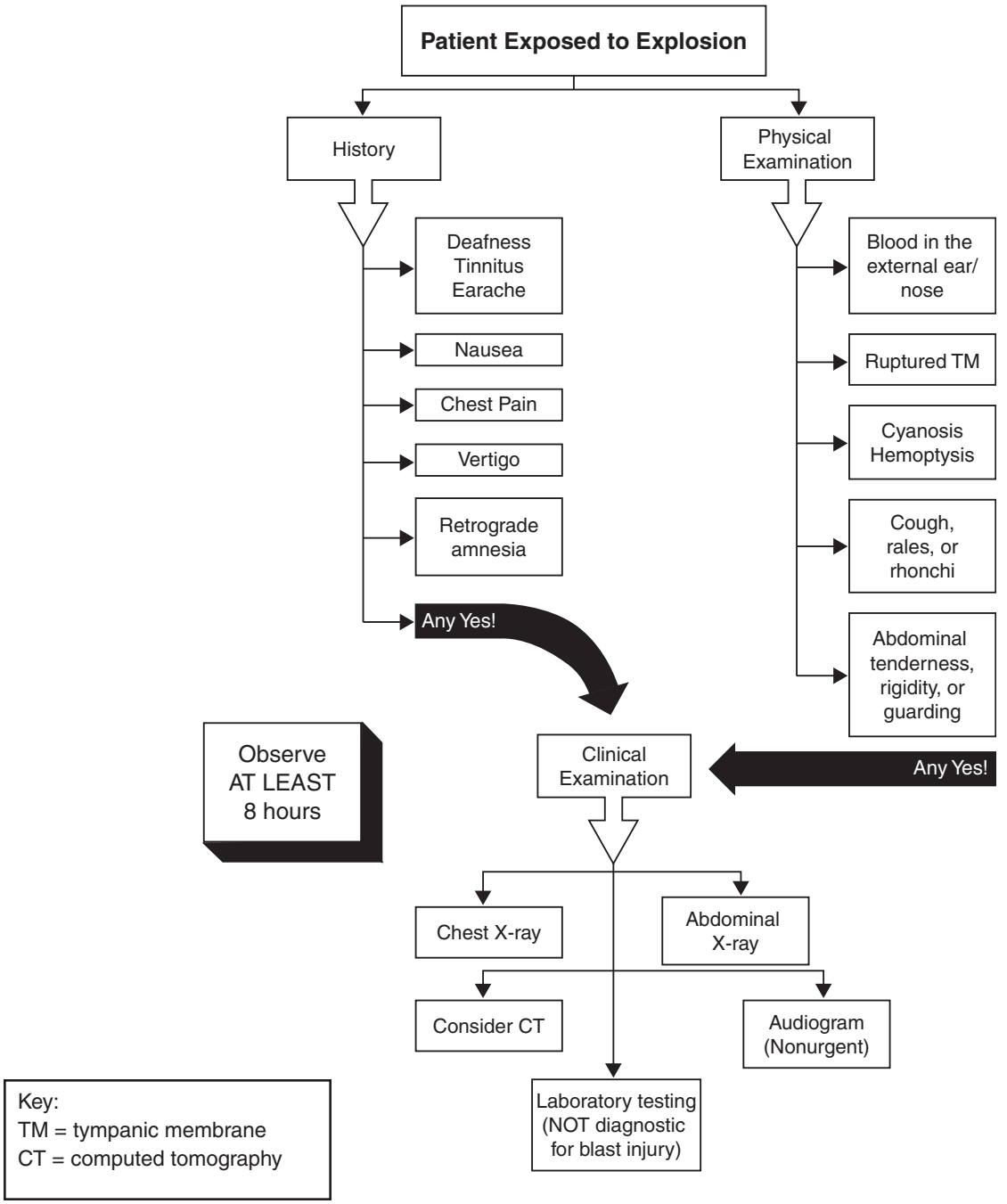
Information from Department of the Treasury, Bureau of Alcohol, Tobacco, and Firearms (ATF) and the Technical Support Working Group[59,60]

Gastrointestinal. Blast injury of the GI 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 unconscious but hemodynamically stable or is conscious with abdominal complaints and is hemodynamically unstable, then fluid resuscitation should be undertaken. If the patient's blood pressure stabilizes and remains stable, then a CT scan of the abdomen is appropriate. If the blood pressure does not improve, then urgent surgical management is indicated.

If the patient is conscious with abdominal findings and is hemodynamically stable, then an abdominal CT scan should be obtained. If the patient is stable, then an abdominal CT scan with oral and intravenous contrast is a reasonable screening procedure.

While abdominal CT scan is appropriately specific, it may not be sufficiently sensitive to identify hollow viscus injury.[31] If

Figure 9. Algorithm for Evaluation of Patients Exposed to Explosions*



* Note: Obvious trauma requires management as per usual trauma protocols.

Adapted from: Cernak I, Savic, J, Ignajatovic D, et al. Blast injury from explosive munitions. *J Trauma* 1999;47:96-103.

patients who have been scanned continue to have signs of abdominal pathology, then a diagnostic peritoneal lavage is appropriate. If the effluent contains significant red blood cells, bacteria, bile, or fecal matter, then urgent laparotomy is indicated. CT *must* precede peritoneal lavage 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 available 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.[54]

Wound Management. For lacerations and fragment wounds, avoid primary closure and consider the use of delayed primary closure in these wounds. There is about an 80% rate of infection when fragment wounds are sutured. All debris that is flung by the explosion is not radiopaque, and the wise provider carefully should explore injuries and consider CT, ultrasound, or MRI of wounds to evaluate for radiolucent foreign bodies. Update the tetanus status as appropriate.

Air Embolism. Air embolism should be treated as soon as the diagnosis is considered. The first step should be to place the patient on high flow oxygen. Next, the patient should be positioned properly. The usual recommended positioning is the left lateral decubitus position with the head down. If only one lung is injured, 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. It should be noted that a recent review article about gas embolism opined that a flat position would be more appropriate.[55] The review article also discusses use of increased fluids, heparin, and corticosteroids as treatments for gas embolism. This review article does not cite any work about blast injury in its bibliography and does not mention blast lung injury as an etiology of gas embolism. The author feels that there isn't enough evidence specific to blast lung as an etiology of gas embolism to make a more specific recommendation.

The definitive treatment for air embolism is hyperbaric oxygenation, which often is not available in a timely fashion. Hyperbaric oxygenation will reduce the bubble size (by Boyle's gas law), increase tissue oxygenation, and increase the solubility of the gas. The United States Navy protocols for gas embolism and decompression sickness would be an appropriate reference.

Disposition

The disposition of these patients depends on the injury sustained by each victim. Those who were close to the center of the explosion should be considered for observation for at least 24 hours.

Precautions

Ensure that physical activity of the victims is minimized after the blast explosion. Exertion after the blast explosion can increase the severity of primary blast injury. This was seen in World War II where some blast casualties appeared well, but died after vigorous exercise.[56]

The barotrauma that results from primary blast injury can be exacerbated by evacuation. Regardless of the altitude and distance of the flight, casualties with 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 mmHg), the clinician should recognize that oxygenation will worsen with ascent in an aircraft (with the increase in altitude and subsequent decreased barometric pressure).

Acute air embolism (AAE) may be the most common cause of rapid death in initial survivors of a blast. It often occurs when positive pressure ventilation is started.[39,57,58]

If the patient requires general anesthesia for any reason, a chest x-ray should be obtained, looking for evidence of barotrauma. It has been reported that blast victims have a higher morbidity rate when they receive general anesthesia during the first 24 hours after surgery. This may well be due to unrecognized primary blast injury and subsequent barotrauma from positive pressure ventilation during anesthesia.[39] If barotrauma is noted and the patient requires general anesthesia, bilateral chest tubes are appropriate.[58] If possible, local or spinal anesthesia may be better.

The evaluation of a patient with blast injuries depends on the nature of the explosion and fragments produced, the distance of the victim from the explosion, and the protection that the victim has from the blast wave and fragments. Most injuries result from fragments and debris hurled by the blast. When a victim is close and/or the explosion is large, the evaluation must include the possibility of blast-induced damage to air-filled or hollow structures. Explosive devices also may cause traumatic amputations from blast effects or from debris. If collapsed structures are involved, many injuries may be due to crushing from falling building debris and support structures. Management of most victims will follow usual trauma protocols for penetrating or crushing injuries. For victims close to the blast, the patient must be observed and evaluated for the possibility of primary blast injuries to air-filled structures.

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Physician CME Questions

31. Positive pressure ventilation markedly increases the possibility of both air embolism and pulmonary barotrauma.
    A. True
    B. False

32. The pipe bomb usually is made from which type of explosive?
    A. High-order explosive
    B. Low-order explosive
    C. Fuel-air explosive
    D. Nitroglyerin

33. Blast injury is much more lethal in the open air.
    A. True
    B. False
    C. There is little difference for either exposure.

34. Which of the following agents is a low-order explosive?
    A. Black powder
    B. Semtex
    C. Composition C-4 (RDX)
    D. Dynamite

35. The extent of damage due to the pressure wave is dependent upon which of the following?
    A. The trough of the initial negative pressure wave
    B. The fourth power of the distance of the incident blast wave
    C. The medium in which it explodes
    D. An exponential decay with time

36. Which of the following often is considered a marker for injury to other organs?
    A. Lung
    B. GI tract
    C. CNS
    D. Ear

37. Which of the following often is considered to be the non-auditory organ at highest risk for primary blast injury?
    A. Lung
    B. GI tract
    C. CNS
    D. Musculoskeletal system

38. Which of the following statements is true?
    A. Lung injury rarely is found with primary blast injury.
    B. Pneumoperitoneum is a common complication of GI barotrauma.
    C. GI injury usually is confined to solid organ laceration.
    D. GI injury associated with primary blast injury is inconsistent in presentation.

39. A common presentation of a terrorist blast injury is:
    A. primary blast injury to the lung.
    B. secondary blast injury from objects added to the explosive mixture, such as nails, nuts, and bolts.
    C. tertiary blast injury.
    D. quaternary blast injury.

40. Which of the following statements is true?
    A. Tertiary blast injuries are caused when the victim's body is propelled into another object by the blast winds.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

B. Primary blast injuries are more common than secondary blast injuries.
C. Primary blast injuries are confined to the pulmonary system.
D. Ear injuries are a very good marker for occult tertiary blast injury.

In Future Issues:

Stroke

CME Answer Key

31.	A	36.	D
32.	B	37.	A
33.	B	38.	D
34.	A	39.	B
35.	C	40.	A

Emergency Medicine Reports
CME Objectives

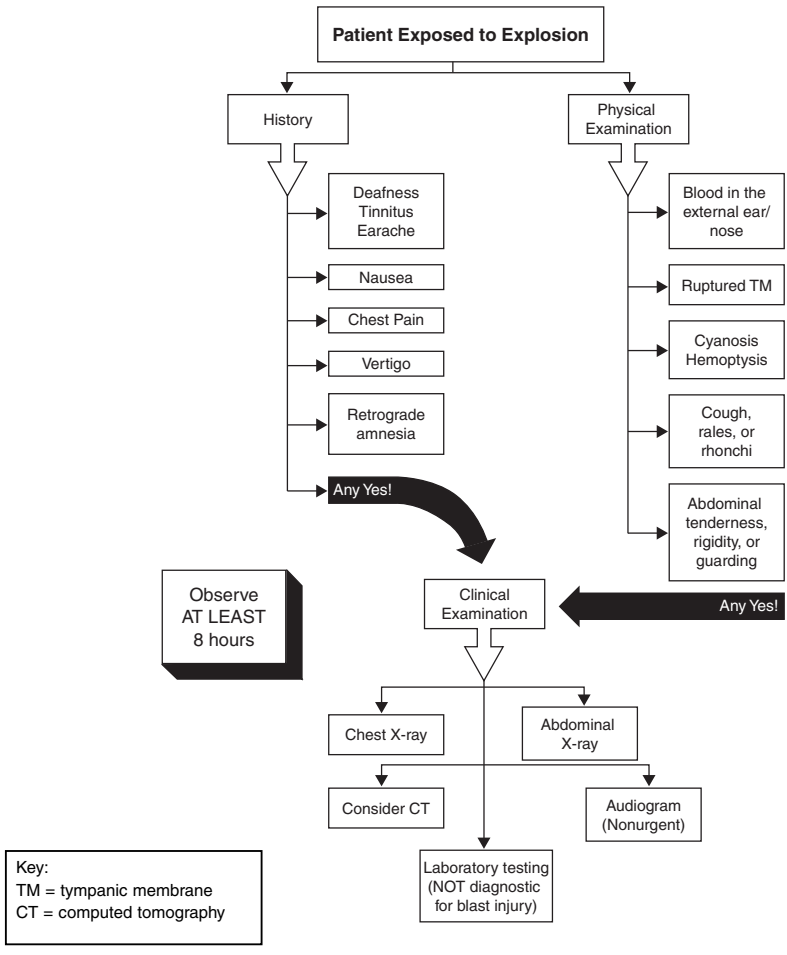
To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Blast Injuries

Algorithm for Evaluation of Patients Exposed to Explosions



* Note: Obvious trauma requires management as per usual trauma protocols.

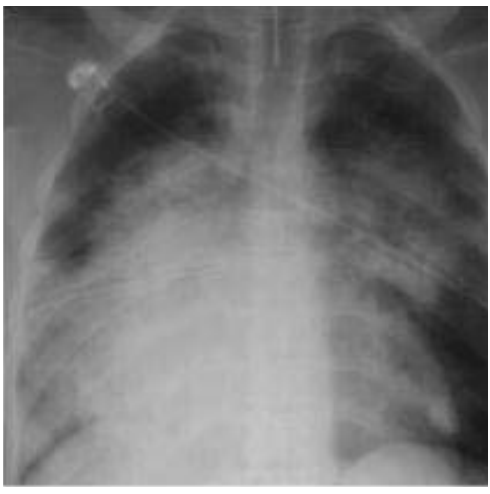
Adapted from: Cernak I, Savic, J, Ignajatovic D, et al. Blast injury from explosive munitions. *J Trauma* 1999;47:96-103.

Blast Overpressure and Effects

OVERPRESSURE IN PSI	EFFECT
1-2	Frame house destroyed
3-5	Typical commercial construction destroyed
5	Tympanic membrane rupture
15	Tympanic membrane rupture in 50% of patients
30-40	Possible lung injury
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. *The Tactical Edge Journal* Summer 2000: 53-55.

Blast Lung



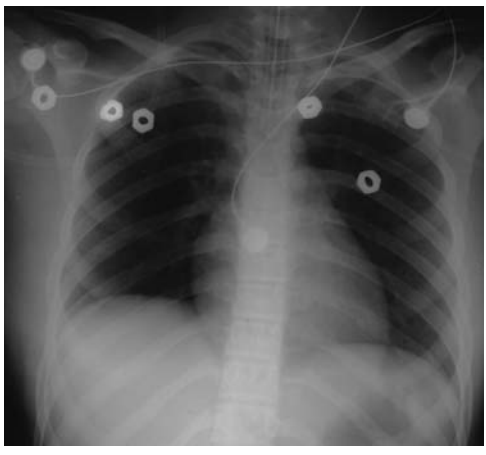
Reprinted with permission from: Wolf YG. Vascular trauma in high-velocity gunshot wounds and shrapnel-blast injuries in Israel. *Surg Clin North Am* 2002;82:237-244.

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* • Tinnitus* • Earache*
Cardiovascular	• Tachycardia • Fall of mean arterial blood pressure
Gastrointestinal	• Nausea* • Abdominal tenderness* • Abdominal rigidity*
Neurologic system	• Vertigo • Retrograde amnesia
Ocular injury	• Eye irritation** • Hyphema** • Distorted pupil** • Decreased vision** • Blindness** • Funduscopic findings of retinal artery air embolism**
Respiratory system	• Cyanosis* • Ecchymosis or petechiae in hypopharynx* • Cough (often dry)* • Tachypnea (often preceded by a short period of apnea)* • Dyspnea* • Hemoptysis* • Rales or moist crepitation in lung fields* • Chest pain*

* Most common findings
** Common findings

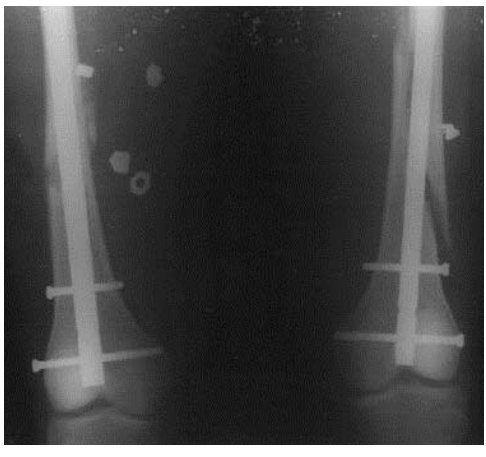
Secondary Blast Injury: Projectiles Thrown by the Force of the Blast



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

Reprinted with permission from Zvi Gimmon, MD.

Secondary Blast Injury: Projectiles Thrown by the Force of the Blast



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

Reprinted with permission from Zvi Gimmon, MD.

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 central nervous system 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.

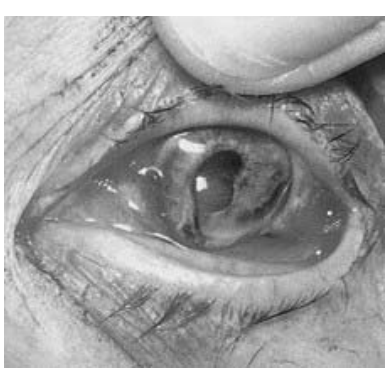
Adapted from CDC Mass Trauma Preparedness and Response web site available at http://www.cdc.gov/masstrauma/preparedness/primer.htm. Accessed 1/26/04.

Fragment Injuries from a Bomb

Fragment injuries from a suicide/homicide bomber in Israel. The device has large metal nuts contained in an explosive vest.

Photo courtesy of Professor Zvi Gimmon, MD.

Eye Rupture



This image shows rupture of the eye with prolapsing iris due to an explosion and direct trauma.

Photo courtesy of Richmond Eye Associates at www.richmond-eye.com/fireworks.htm.

Diagram of Primary, Secondary, and Tertiary Blast Injuries

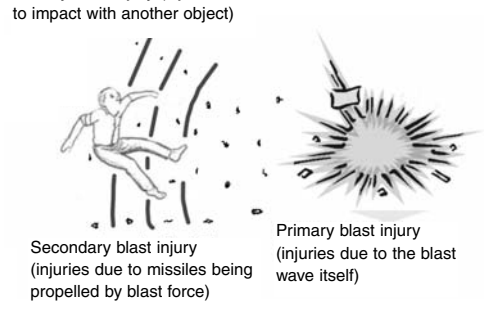


Illustration by Charles Stewart, MD.

Supplement to *Emergency Medicine Reports*, February 9, 2004: "Evaluation and Management of Patients with Blast Injuries in the ED."
Author: **Charles Stewart, MD, FACEP,** Emergency Physician, Colorado Springs, CO.

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PEDIATRIC Influenza Update

S04110

A supplement to *Pediatric Emergency Medicine Reports, Emergency Medicine Reports, Primary Care Reports, Internal Medicine Alert, and Infectious Disease Alert*

February 2004

Influenza Update: Focus on Children

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From October 2003 to Jan. 9, 2004, the Centers for Disease Control and Prevention (CDC) received reports of 93 influenza-associated deaths among children younger than 18 years. The demands the annual flu season places on emergency department (ED) and urgent care facilities and the voracity of the current year's epidemic have overwhelmed many physicians. High-risk adults long have been the focus of public health programs and early diagnostic and therapeutic interventions. Influenza is responsible for significant morbidity and mortality in infants and young children, with influenza-associated hospitalization rates similar to those in adults with chronic medical conditions.[1,2]

During influenza epidemics, a diagnostic test that provides an accurate diagnosis in infants younger than 6 months of age with fever enables the clinician to provide the family with specific expectations for the disease process and minimizes unnecessary use of antibiotics.

This article reviews the current status of diagnostic testing, vaccine indications, and antiviral therapies for pediatric patients with influenza infections.

— The Editor

Epidemiology

Children account for nearly two-thirds of diagnosed cases of influenza during a typical season, with more than 30% of children living in affected communities.[3] Adults contract the illness from children, and infection rates dramatically increase in households with school-age children.

Two population-based studies highlight the direct effect of influenza on children. Evaluation of hospitalization rates in Group Health Seattle and Kaiser Northern California indicated dramatically increased rates of hospitalization in healthy children younger than 2 years of age.[1] Analysis of Tennessee Medicaid patients indicated hospitalization rates of children younger than age 2 that were similar to high-risk adults with substantive excess use of antibiotics.[2]

Influenza strikes hard in Japan. The population is long-lived, and many elderly live in homes with schoolchildren present. From 1962 to 1987, most Japanese schoolchildren were vaccinated against influenza. The vulnerable elderly were considered secondary targets for immunization. Excess influenza and pneumonia deaths dropped 40%, with 37,000-49,000 excess deaths per year averted. The laws mandating this effort were relaxed in 1987 and repealed in 1994. Subsequent vaccination rates dropped to low levels, leading to a sharply rising number of deaths.[4]

Similar findings are emerging from a study of immunizing schoolchildren with attenuated live intranasal influenza vaccine in a Texas community. Immunization rates of 50% have demonstrated the ability to prevent community epidemics and dramatically reduce excess mortality in the elderly. Immuniza-

Pediatric Influenza Ulpdate™ (ISSN 1082-3344) is published by Thomson American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
Editorial Group Head: Valerie Loner
Managing Editor: Allison Mechem
Marketing Manager: Schandale Kornegay

Periodicals Postage Paid at Atlanta, GA 30304.

POSTMASTER: Send address changes to ***Pediatric Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Accreditation

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Intended Audience

This program is intended for pediatricians, emergency medicine practitioners, internists, and family practitioners.

Effective Dates

This activity is approved for release Feb. 1, 2004 until Jan. 31, 2005.

Questions & Comments

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Objectives

After completing this activity physicians should be able to:

1) recognize the historical and clinical symptoms associated with influenza infection;
2) integrate appropriate laboratory diagnostic testing for suspected influenza into clinical practice; and
3) understand and implement into practice utilization of antiviral therapies.

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tion of schoolchildren was shown to be cost-effective when considering indirect costs of illness.[5] In another, less recent study by Monto and colleagues in Tecumseh, MI, school-age immunization reached 85%, and the incidence of influenza-like illness was one-third that of neighboring communities.[6]

Prior to the 2003-2004 influenza season, laboratory-confirmed influenza illnesses and deaths were not nationally reportable conditions. Due to an increased concern about the morbidity (especially encephalitis cases) and mortality associated with influenza in pediatrics, the CDC requested that all influenza-associated deaths be reported to state and local health departments in the 2003-2004 season. Since October 2003, a total of 93 influenza-associated deaths in children have been reported. The median age of the 93 children was 4 years, with 26% between 6 months and 23 months of age and 59% of the children younger than 5 years of age.[7] Although 38% of the children were reported to have a chronic underlying medical condition, 44% had no report of any pre-existing conditions. Another series reported that an underlying medical condition (asthma, neurologic deficits, or malignancy) was documented in 25% of children hospitalized with influenza A or B.[8]

Clinical Picture and Diagnosis

The clinical picture of influenza in young children often is subtle, with signs and symptoms mimicking other common childhood diseases. A series evaluating the prevalence of influenza in children 0-11 months of age, during the peak of an influenza epidemic, found that every third patient seen in a pediatric ED had virologically confirmed influenza infection.[9] The younger the child, the more difficult it is to distinguish influenza from other febrile illnesses.[8] In one series, the most common symptoms of influenza included fever, cough, and rhinorrhea, all nonspecific symptoms.

A retrospective 20-year review of pediatric patients with nasopharyngeal aspirates that were positive for influenza A or B, conducted in Finland, characterized the diversity of presentations. The majority of children had high fevers, and febrile seizures occurred in 12% of the children with influenza A and 9% with influenza B. Rhinorrhea and cough were present in 60% of the children and gastrointestinal symptoms (vomiting and diarrhea) were documented in 24% of the children. The classic adult symptoms of influenza (myalgias, headache, and malaise) are challenging to elicit from children younger than 3 years, secondary to normal developmental limitations. In assessments of children older than 3 years of age, 25% conveyed the presence of a headache and only 6% reported myalgias.[8]

Further confounding the ability of the clinician to make an accurate diagnosis is the diversity of clinical syndromes that may be caused by influenza. Croup, bronchiolitis, a febrile disease mimicking bacterial sepsis, and encephalitis all have been reported in association with influenza. Differentiation from parainfluenza or respiratory syncytial virus (RSV) infec-

Table 1. Commercially Available Rapid Influenza Diagnostic Tests

TEST NAME	INFLUENZA DETECTED	TIME FOR RESULTS	SENSITIVITY	SPECIFICITY	CLIA CATEGORY LAB SETTING
Now Flu A Now Flu B	A, B (distinguishes A and B)	15 min	N/A	N/A	Mod complex Hospital or referral lab
FLU OIA	A, B (does not distinguish A and B)	15 min	71.8% (range 36.7-93%)	82% (range 65.2-95.7%)	Mod complex Hospital or referral lab
QuickVue Influenza	A, B (does not distinguish A and B)	10 min	79.2% (range 74-95%)	91.9% (range 76-98%)	CLIA waived
ZstatFlu	A, B (does not distinguish A and B)	30 min	68.8% (range 48.1-96%)	83% (range 62.7-92.4%)	CLIA waived
Directigen Flu A	A	15 min	87.2% (range 39-100%)	98.1% (range 84-100%)	Mod complex Hospital or referral lab
Directigen Flu A + B	A, B (distinguishes A and B)	15 min	89.8% (A) 87.5% (B)	98.7% (A) 96.8% (B)	Mod complex Hospital or referral lab

Adapted from Uyeki TM. Influenza diagnosis and treatment in children: A review of studies on clinically useful tests and antiviral treatment for influenza. *Pediatr Infect. Dis* 2003;22:164-177.

tions requires culture or immunoassay. In addition, the young infant may require significant laboratory testing and procedures to assure a nonbacterial focus of infection.[8,10-12]

In the past, the lack of effective treatment coupled with delayed laboratory confirmation limited the ability of practitioners to develop a timely, specific diagnosis. The viral diagnosis paradigm was simply an exclusion of treatable bacterial illness in the differential diagnosis. The emergence of effective diagnostic techniques and antiviral therapy mandates a reconsideration of the evaluation of children with potential influenza-associated illnesses.

Complications

Influenza may be severe and may even lead to death, especially in children with underlying medical conditions. Secondary infections, including pneumonia and otitis media, may complicate influenza. One series reported 24% of the children developed otitis media, and pneumonia occurred in approximately 9% of the patients.[8] Pneumonia was reported in 25 of the 93 fatal cases reported for the 2003-2004 influenza season, and 15 of the children had an invasive bacterial co-infection.[7] Severe presentations and complications also have been associated with influenza and include encephalitis, Guillain-Barré type polyradiculopathy, and myositis.[8]

Diagnostic Testing

Exclusively pediatric studies comparing the accuracy of a clinical diagnosis to laboratory-confirmed influenza virus infections have not been conducted. Studies consisting primarily of adult patients have shown the highest predictive value of any case definition for influenza was 40% when compared with viral culture.[13] Immunofluorescence staining may be performed on respiratory specimens (nasopharyngeal swabs, aspirate, or nasal swab with adequate epithelial cells) for the detection of influenza viruses.

Direct immunofluorescence antibody (DFA) tests and indirect immunofluorescence antibody (IFA) tests may be performed on respiratory samples to detect influenza viruses. These tests usually can be performed at a hospital or reference lab within 2-4 hours, but the specimen quality (adequate number of epithelial cells in the specimen) and technician experience are important. Three studies of the use of DFA for influenza A (compared to viral culture), revealed a median sensitivity of 62% (range 45-65%) and the median specificity was 98% (range 92-99.7%).[14-17] Three studies that compared IFA for influenza A and B to viral culture revealed a median sensitivity of 73.9% (range 59.8-90%) and a median specificity of 97% (range 93-97%).[17-19] Both IFA and DFA only are available to hospital-based physicians, have a high rate of false negative results, and require a minimum of 2-4 hours for test results.

The availability of CLIA (Clinical Laboratories Improvement Act) made rapid diagnostic kits for influenza available for diagnosis and timely intervention. Rapid influenza testing is reasonably accurate for detecting influenza infections in pediatric patients. *(See Table 1.)* False negative results may occur, but false positives are infrequent. Therefore, if the diagnosis of influenza is suspected in a moderately to severely ill child, more definitive testing may be indicated.

The development of a selective strategy for the use of the rapid influenza test is pragmatic. Each patient is not necessarily tested; rather, the test is used to calibrate the clinician's clinical acumen and to provide confirmation of local disease presence.

Table 2. Antiviral Drugs for Treatment of Influenza

DRUG MECHANISM	INDICATION	ACCEPTABLE AGE GROUPS	DOSAGE FOR ACUTE TREATMENT	POTENTIAL ADVERSE EFFECTS
Amantadine M2 inhibitor	Influenza A	Treatment > 1 year of age Prophylaxis > 1 year of age	1-9 years of age: 5 mg/kg/day divided BID (maximum dose 150 mg)	CNS side effects Rapid resistance
Rimantadine M2 inhibitor	Influenza A	Treatment > 14 years of age Prophylaxis > 1 year of age	> 14 years of age: 100 mg BID	Rapid resistance
Zanamivir neuraminidase inhibitor	Influenza A and B	Treatment > 7 years of age (orally inhaled powder)	> 7 years 10 mg BID for 5 days	Caution with history of bronchospasm. Not approved for prophylaxis.
Oseltamivir neuraminidase inhibitor	Influenza A and B	Treatment > 1 year of age Prophylaxis > 13 years of age	< 15 kg: 30 mg BID for 5 days > 15 kg and < 23 kg: 45 mg BID for 5 days > 23 kg and < 40 kg: 60 mg BID for 5 days > 40 kg: 75 mg BID for 5 days	Mild GI side effects
Ribavirin Unknown	In vitro activity against RSV, influenza, and other viruses	Not approved	No approved dosage	Not approved for treatment of influenza infections.

Key: RSV—respiratory syncytial virus; CNS—central nervous system; GI—gastrointestinal

In certain situations, use of the rapid test may confirm a specific viral diagnosis; avert unnecessary diagnostic testing; provide for early, effective antiviral treatment; and reassure concerned parents about the lack of indication for antibiotics.

The use of diagnostic tests has led to a significant decrease in the unnecessary use of antibiotics, and logically should lead to a reduction in the emergence of antibiotic resistance.[20,21] The use of laboratory and radiographic testing also has been shown to be significantly reduced through the use of the rapid influenza tests, leading to decreased patient charges.[10,21] Especially in infants and younger children, a positive influenza test has been shown to decrease the number of complete blood counts, blood cultures, urine cultures, cerebral spinal fluid studies, and chest radiographs.[10] In addition, the use of rapid diagnostic testing has been shown to decrease the length of stay in the ED for patients with a positive rapid test for influenza.[10]

Treatment

Currently, three antiviral drugs—amantadine, oseltamivir, and zanamivir—are approved for the treatment of influenza in children, and two other drugs—rimantadine and ribavirin—have been used. *(See Table 2.)*

Amantadine. About 30 years ago, specific antiviral therapy of influenza began with the introduction of amantadine, a drug that targets the M2 membrane protein of influenza A. The drug has not been studied in an exclusively pediatric population, but two studies have shown a reduction in the mean duration of fever.[22,23] It also has been shown to decrease the frequency of headaches and gastrointestinal symptoms (nausea and vomiting)[22,23] and to reduce the duration of uncomplicated influenza A and B illness by one day when compared to placebo. Amantadine has not been demonstrated to be effective for the prevention of serious influenza-related complications, and the majority of the studies have been conducted in patients with uncomplicated illnesses. Although the development of amantadine resistance has not been adequately assessed, a trend toward a rapid emergence of resistance and lack of activity against influenza type B reduces its usefulness.

It also is effective as a prophylactic agent during epidemics in both adults and children older than 1 year.[24] Neurologic and gastrointestinal side effects may be significant in very young and elderly patients. Resistance has been documented within single households, with treated index cases transmitting resistant virus to other family members.[25]

Rimantadine. Although rimantadine currently only is approved for chemoprophylaxis of influenza type A infections among children, some experts consider it acceptable treatment for the illness. Rimantadine, similar to amantadine, has been

shown to decrease the duration of uncomplicated influenza A illness when administered within 48 hours of the onset of illness.[26] Resistance has been shown to develop rapidly with the use of rimantadine, and one study demonstrated a prolonged viral excretion in children treated with rimantadine.[26] Rimantadine has been shown to have a decreased incidence of central nervous system (CNS) side effects when compared to amantadine.

Zanamivir. Zanamivir is a neuraminidase inhibitor and has activity against both influenza A and B. Clinical studies in experimental and natural infection demonstrated decreased length of viral shedding, symptoms, and severity in both type A and B influenza illnesses.[27,28] Ongoing studies of the neuraminidase inhibitors have shown efficacy in childhood. A double-blind, placebo-controlled study of zanamivir in the 1998-1999 northern hemisphere flu season recruited 471 children with flu-like symptoms. Three hundred forty-six had culture-proven influenza, and inhaled diskhaler therapy significantly shortened time to alleviation of symptoms and time to resumption of normal activity. The treatment group also used less relief medications and there was a reduction in associated complications (16%) and antibiotic use (12%).[29]

Questions were raised regarding respiratory function deterioration in patients with existing chronic obstructive pulmonary disease (COPD) and asthma. Bronchospasm has occurred in patients with asthma.[28,30] The package insert contains important precautionary information regarding the use of zanamivir with underlying airway disease. The drug is taken as a five-day course using a proven diskhaler design. It is indicated for patients ages 7 and older who have signs and symptoms of influenza A and B of fewer than 48 hours duration.

Oseltamivir. The desire for an orally active drug led to the development of oseltamivir (Tamiflu). Oseltamivir is a neuraminidase inhibitor that has activity against both influenza A and B. Oseltamivir has been approved for treating uncomplicated influenza infections in children older than 1 year and also has been approved for use as a chemoprophylaxis agent in children older than 13 years.

A study of 695 patients ages 1-12 years showed a 36-hour, or 26%, reduction in duration of influenza. The incidence of otitis media was reduced by 44%.[31] Specific efficacy was demonstrated with influenza B infection in other studies, with a decrease of symptom duration by 25%.[30,32] Oseltamivir was well-tolerated in clinical trials, with no safety issues raised. In adult, adolescent, and child studies, nausea was reported, with a greater incidence of emesis over placebo of 5.8%. The recipients described the gastrointestinal symptoms as transient and mild.[33,34] Discontinuation of medication due to adverse events was 1.8% in the oseltamivir group vs. 1.1% with placebo.[31] Prior studies with adolescents and adults indicate significant reduction of gastrointestinal symptoms with concomitant consumption of food.[33] Resistant strains were uncommon and represented viruses with limited infectivity in humans.

Ribaviron. Minimal information is available regarding the use of ribaviron for the treatment of influenza in children. Two randomized controlled studies compared ribaviron to the use of placebo in children with influenza. Aerosolized ribaviron, given to hospitalized children who had fewer than 48 hours of symptoms reduced the mean duration of fever.[35] Oral ribaviron was evaluated in a single double-blinded controlled study in girls 8-16 years of age and found significantly reduced symptoms 24 hours after starting the medication and decreased viral shedding. Neither study reported any significant adverse effects.[36]

Prophylaxis. Chemoprophylactic drugs are an adjunct to vaccination in the prevention and control of influenza. The neuroaminidase inhibitors have been shown effective for prevention of influenza infection. Zanamivir once a day was 79% effective for the prevention of influenza transmission within families with a confirmed index case.[37] Orally administered oseltamivir 75 mg once a day protected close family contacts against influenza by 92% and interrupted transmission within households by 89%.[38,39] Post-exposure, the placebo group had a 12% incidence of influenza, compared with a 1% incidence in the prophylaxis group. The U.S. Food and Drug Administration (FDA) has indicated oseltamivir for prophylaxis in adolescents and adults ages 13 and older.

It has been suggested that use of family prophylaxis after treatment of the index case may be the most effective use of the medication. It not only protects familial contacts but also can serve to reduce community exposure. Family physicians are in a unique position to treat the whole family when an index case is identified. Pediatricians will need to form effective alliances with internists and family physicians to effectively reach the parents of children with influenza.

Reducing Complications and Antibiotic Use

The serious complications of influenza include bacterial pneumonias, Reye syndrome, and prolonged recovery of high-risk patients. The increased frequency of otitis media and other respiratory infections in children with influenza is under-appreciated. With proper antiviral treatment of influenza, a substantive reduction of antibiotic usage has been demonstrated. This reflects a real decrease in otitis media occurrence, as well as the desired reduction in the overuse of antibiotics for primary viral infection.

In the oseltamivir trials in children ages 1-12 years, 21% of placebo recipients and only 12% of treated subjects had documented otitis media.[30] The 44% reduction in clinical diagnosis was paralleled by a 40% reduction in antibiotic usage.[31] The zanamivir trials of children ages 5-12 years showed a 30% reduction in bacterial complications, with a 20% reduction in antibiotic use.[29] Effective treatment of primary viral infections can reduce otitis morbidity and antibiotic usage.

In pivotal clinical trials, the neuraminidase inhibitors showed efficacy with one- to two-day decreases in time to alleviation of all significant symptoms of influenza. Early FDA examination

and subsequent professional commentary questioned this apparent marginal benefit from therapy. Health maintenance organizations and other third-party payers also questioned utility, and frequently excluded the medications from their panels. This marginality of efficacy contrasted strongly with clinical observations of patients, physicians, and investigators using the medications. In an effort to reconcile clinical impressions in practice with clinical trial data, investigators followed 1408 patients using prescribed zanamivir in Australia during the 1999 flu season.

Symptom relief was reported by more than 50% of patients within 24 hours and by 77% within 48 hours.[40] Of the 400 elderly patients, 78% were satisfied with their treatment, with 59% experiencing symptom relief within 24 hours.[41] The survey concluded that zanamivir was associated with an early return to normal activities. The investigators also noted that patients with influenza had a protracted cough. Even in treated influenza patients, the cough persisted after systemic symptoms of fever, headache, myalgia, and malaise had resolved. They speculated that residual cough prolonged the end point in the clinical studies and, thus, caused an underestimation of the clinical effect of treatment.

The identification and treatment of primary viral infections remains a significant challenge to pediatric medicine. It also represents a significant opportunity to reduce an ongoing burden of illness. The technology for effectively preventing, diagnosing, and treating influenza has been demonstrated. Outpatient clinics, emergency rooms, and urgent care centers, as well as private physician offices, need to organize specifically to meet the challenge of early intervention in influenza epidemics. Telephone triage systems need to efficiently screen those with classic symptoms of influenza and promptly direct them to where they can be evaluated and treated with minimal delay. Specific time slots dedicated to prompt evaluation and treatment of infectious disease must be set aside during anticipated flu seasons.

The widespread implementation of influenza prevention and treatment in pediatric populations would provide benefit not only to the index cases but also to household contacts and vulnerable fragile elderly in the community. The antiviral treatment and chemoprophylaxis of contacts of influenza victims will serve as a model for treatment of other specific viral illness as newer antiviral agents that are readily visible in the drug pipeline become available to the practitioner.

Immunization. The U.S. Advisory Committee on Immunization Practices (ACIP) has issued its recommendations for the 2004-2005 influenza season. ACIP has recommended that all children 6-23 months of age be vaccinated annually against influenza beginning in the fall of 2004. Children younger than 9 years of age who previously have been unvaccinated should receive two doses one month apart. ACIP continues to strongly recommend vaccination of children with chronic medical problems. The recommendation to vaccinate healthy children in the 6-23 month age group is based on studies that showed that previously healthy young children account for the majority of hospitalizations for pediatric influenza.[42] Cost calculations have suggested that, for healthy children, vaccination against influenza would be cost-effective[5] and would decrease influenza-associated morbidity and mortality in the adult population because of the important role of children in the dissemination of influenza.[4] Vaccination programs should start in October and target adults older than 50 years, children 6-23 months of age, high-risk patients of any age and their household contacts, and health care workers. The attenuated live intranasal vaccine (FluMist) is an expensive option ($55.20/dose) for healthy individuals 5-49 years of age who do not have contact with immunocompromised patients. Children 6-35 months may receive 0.25mL/dose of Fluzone, and patients older than 35 months may receive 0.50 mL/dose of Fluvirin or Fluzone.

Future Directions and Considerations

The use of real-time influenza reporting systems enables clinicians to either include or exclude the possibility of influenza through community epidemiology. The use of quick diagnostic kits makes a positive diagnosis of the viral infection possible, and increased usage may prevent many needless exposures to unnecessary antibiotics.

With proper local epidemiologic surveillance and available laboratory tests, more precise diagnosis will be facilitated, and enhanced use of antiviral agents promoted. More than ever, precision in defining the etiologic agents is not only desirable, but also necessary.

(The authors wish to acknowledge the contributions of Katalin Koranyi, MD, Professor of Clinical Pediatrics, Section of Infectious Disease, Department of Pediatrics, College of Medicine, The Ohio State University, Columbus.)

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CME Objectives

The CME objectives for *Pediatric Influenza Update* are to help physicians:

1) recognize the historical and clinical symptoms associated with influenza infection;
2) integrate appropriate laboratory diagnostic testing for suspected influenza into clinical practice; and
3) understand and implement into practice utilization of antiviral therapies.

evaluation of 1-beta-D-ribofuranosyl-1,2,4-triazole-3-carboxamide (ribavirin) in a double-blind study during an outbreak of influenza. *Ann NY Acad Sci* 1977;284:272-277.
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Physician CME Questions

1. Gastrointestinal symptoms of vomiting and diarrhea occur in how many children with influenza?
   A. Approximately 24%
   B. More than 50%
   C. More than 75%
   D. Almost all

2. The findings of a Texas study with a 50% immunization rate for schoolchildren demonstrated:
   A. immunization of schoolchildren was shown to be cost effective when considering indirect costs of illness.
   B. immunization made no difference.
   C. immunization prevented community epidemics.
   D. immunization dramatically reduced excess mortality in the elderly.
   E. A, C, and D only are correct.

3. Which of the following statements about the presentation of influenza in children is correct?
   A. The infection only presents as classic bronchiolitis.
   B. Differentiation from parainfluenza or respiratory syncytial virus (RSV) infections may be accomplished without culture or immunoassay.
   C. Febrile seizures may occur in association with an influenza illness.

4. Severe presentations and complications associated with influenza include encephalitis, Guillain-Barré type polyradiculopathy, and myositis.
   A. True
   B. False

5. Which of the following may be a complication of influenza?
   A. Bacterial co-infection
   B. Otitis media
   C. Pneumonia
   D. All of the above

6. Effective treatment of primary viral infections can reduce otitis media occurrence and antibiotic usage.
   A. True
   B. False

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the enclosed evaluation form and return it in the reply envelope provided to receive a certificate of completion. When your evaluation is received, a certificate will be mailed to you.

Answer Key

1. A
2. E
3. C
4. A
5. D
6. A