REVIEW ARTICLE

CURRENT CONCEPTS

Blast Injuries

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N Engl J Med 2005;352:1335-42.

Although information about terrorism has emphasized chemical, biologic, and radiation events,[1-5] most terrorist attacks have involved explosive devices. To date, the number of lives lost, the number of people injured, and the consequences on the infrastructure are orders of magnitude higher after explosions[6] than after chemical or biologic incidents. Nonconventional weapons have been perceived as more dangerous than explosives,[7,8] probably because the effects of explosions are easier to comprehend. This review summarizes the mechanisms and describes the clinical consequences of blast injuries and outlines strategies for the immediate management of trauma and specific effects resulting from blast injuries.

To date, relatively few bombings in the United States (excluding the incendiary attacks of September 11, 2001) have caused mass casualties. In 1995, the large bomb composed of fuel oil and fertilizer that was set off against the Alfred P. Murrah Federal Building, in Oklahoma, caused 518 injuries and 168 deaths. Otherwise, there have consistently been fewer than 50 deaths per year from bombs in the United States.[9,10] Terrorists seek to injure or kill as many people as possible. Bombs large enough to cause the explosions at the Murrah Building and in the Madrid trains, on March 11, 2004, are easy to produce and can injure enough people to overwhelm the resources and expertise available in many communities. Improvised explosive devices currently used in insurgency warfare in Iraq and Israel are particularly challenging in that they are often loaded with metallic objects to inflict penetrating injuries in crowded civilian settings. Because of the increasing risk of terrorist attacks, physicians must become familiar with the characteristics of contemporary explosive devices and the spectrum of injuries inflicted by blasts and explosions.[11]

TYPES OF EXPLOSIONS

CONVENTIONAL BOMBS

The explosion of a conventional bomb generates a blast wave that spreads out from a point source. The blast wave consists of two parts — a shock wave of high pressure, followed closely by a blast wind, or air in motion. The physics of blast waves is nonlinear and complex. In general, damage produced by blast waves decreases exponentially with distance from the point source of the blast.[12] When explosions occur indoors, standing waves and enhanced differences in pressure occur because of the additive effects of reflections or reverberations from walls and rigid objects. As outward energy dissipates, a reversal of wind back toward the blast and underpressurization occur. The resulting pressure effect damages organs, particularly at air–fluid interfaces, and the wind propels fragments and people, causing penetrating or blunt injuries.

ENHANCED-BLAST EXPLOSIVE DEVICES

Enhanced-blast explosive devices, in contrast, can have more damaging effects. A primary blast from these devices disseminates the explosive and then triggers it to cause a secondary explosion. The high-pressure wave then radiates from a much larger area, prolonging the duration of the overpressurization phase, thus increasing the total energy transmitted by the explosion. Enhanced-blast devices cause a greater proportion of primary blast injuries than do conventional devices.

In confined spaces such as buildings and buses, irregular high-pressure waves from either conventional or enhanced-blast devices cause unpredictable patterns of injury. Clinicians should consider the type of explosive device and its location when evaluating victims of terrorist blasts.[13] Blast injuries should be suspected regardless of the distance the patient was from the blast center and the absence of injuries in other people who were near the patient.

TYPES OF BLAST INJURIES

The effects of blasts fall into the following four categories: primary (direct effects of pressure), secondary (effects of projectiles), tertiary (effects due to wind), and quaternary (burns, asphyxia, and exposure to toxic inhalants)[14-16] (Table 1). The types of injuries caused by blasts depend on whether the blasts occur outdoors in open air or within buildings and whether they cause the collapse of a building or other structure.

Table 1. Immediate Effects of Blasts and Explosions.

Primary — direct effects (e.g., overpressurization and underpressurization)
Rupture of tympanic membranes
Pulmonary damage
Rupture of hollow viscera
Secondary
Penetrating trauma
Fragmentation injuries
Tertiary — effects of structural collapse and of persons being thrown by the blast wind
Crush injuries and blunt trauma
Penetrating or blunt trauma
Fractures and traumatic amputations
Open or closed brain injuries
Quaternary — burns, asphyxia, and exposure to toxic inhalants

PRIMARY BLAST INJURIES

Primary blast injuries are caused by barotrauma — either overpressurization or underpressurization relative to atmospheric pressure. Primary blast injuries most commonly involve air-filled organs and air–fluid interfaces. Organs are damaged by dynamic pressure changes at tissue-density (e.g., air–fluid) interfaces due to the interaction of a high-frequency stress wave and a lower-frequency shear wave.[17] One or the other of these waves predominates, depending on the characteristics and location of the blast. Rupture of the tympanic membranes, pulmonary damage and air embolization, and rupture of hollow viscera are the most important primary forms of blast injury.

The tympanic membrane (Fig. 1) is the structure injured most frequently, and at the lowest pressure, by blasts. The eardrum thus represents a site for detecting primary effects of blasts. An increase in pressure of as little as 5 psi above atmospheric pressure (1 atm is equivalent to 14.7 psi, or 760 mm Hg) can rupture the human eardrum.[18] Temporary neurapraxia in the receptor organs of the ear, manifested by deafness, tinnitus, and vertigo, characterizes rupture of the eardrum, which should be suspected even when the tympanic membrane cannot be seen after a blast incident. If dynamic overpressures are high enough, the ossicles of the middle ear can be dislocated. Traumatic disruption of the oval or round window can cause permanent hearing loss. In contrast, pressure gradients of 56 to 76 psi (3.8 to 5.2 atm) are needed to cause damage to other organs.[19] If there is no rupture of the tympanic membrane, then primary effects of blasts on other air-containing organs is unlikely.

Although rupture of the tympanic membranes serves as a convenient and sensitive marker for blast injuries, data have shown that delayed pulmonary complications of blast injuries may not develop in persons with ruptured tympanic membranes, and that some patients with lung injuries did not have ruptured membranes.[19] The researchers of this study of 647 survivors of explosions on buses used immediate radiography of the chest to screen for pulmonary injuries from the blasts. Primary injuries, in some form, were found in 193 persons: 142 had isolated perforation of the eardrum, and 51 had other forms of primary blast injuries, including 18 with isolated pulmonary injuries, 31 with combined otic and pulmonary injuries, and 2 with intestinal injuries.

In a recent report of the train bombing in

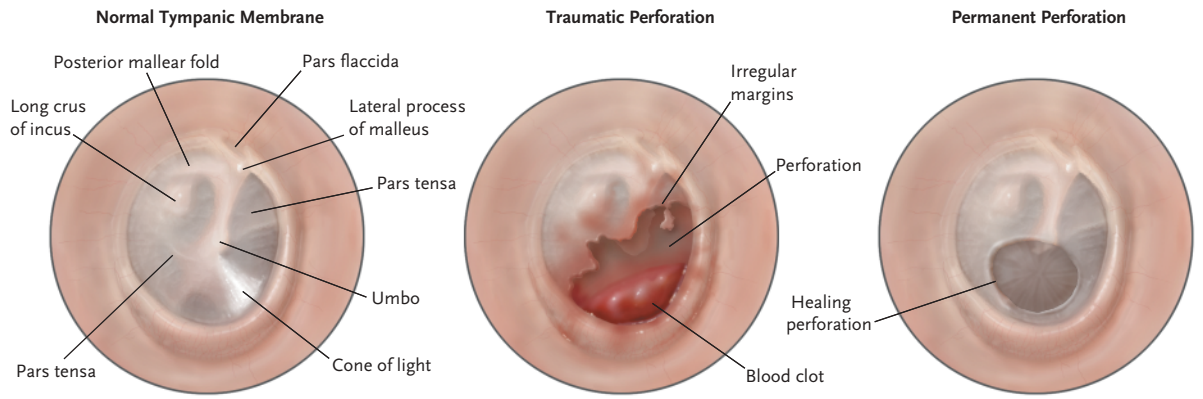

Figure 1. Normal and Perforated Right Tympanic Membranes.
The drawing of a traumatic perforation shows an irregular margin or rim with blood or a blood clot, and the drawing of a permanent central perforation shows a tympanocele.

Madrid,[20] rupture of the tympanic membranes occurred in 99 of 243 victims, chest injuries in 97, shrapnel wounds in 89, fractures in 44, burns in 45, eye injuries in 41, abdominal injuries in 12, and traumatic amputations in 5. Among 17 critically ill victims with pulmonary injuries from the blast, 13 had ruptured tympanic membranes and 4 did not. Rupture of tympanic membranes occurred in 18 of 27 critically injured victims; 17 of these were bilateral.

The lung is the organ that is second most susceptible to primary blast injury. Pressure differentials across the alveolar–capillary interface cause disruption, hemorrhage, pulmonary contusion (appearing as a bihilar "butterfly" pattern on chest radiographs), pneumothorax, hemothorax, pneumomediastinum, and subcutaneous emphysema.[21-23] Pulmonary injuries are life-threatening; for example, the immediate onset of pulmonary edema with frothing at the mouth (associated with bilateral radiographic "whiteout") carries a grave prognosis.

Body armor has been shown to protect military personnel from most ballistic projectiles to the torso, thus increasing survival. However, body armor does not protect against the barotrauma of primary blast injury.[24] Pulmonary barotrauma is the most common critical injury to people close to a blast center, whether civilian or military. Systemic acute gas embolism from pulmonary disruption is believed to occlude the blood vessels of the brain or spinal cord. Injury to the central nervous system due to acute gas embolism must be differentiated from the direct effects of head trauma and concussion.

The colon is the visceral structure most frequently affected by a primary blast injury.[25-27] Rupture of the colon and, less frequently, the small intestine may occur as an immediate result of a blast. Mesenteric ischemia or infarct can cause delayed rupture of the large or the small intestine; these injuries are difficult to detect initially. Rupture, infarction, ischemia, and hemorrhage of solid organs such as the liver, spleen, and kidney are generally associated with very high blast forces or proximity of the patient to the blast center.

Primary blast injuries to the eye include rupture of the globe, serous retinitis, and hyphema. Facial fractures are commonly due to flying objects or direct trauma. Primary blast injuries to the brain include concussion as well as barotrauma caused by acute gas embolism. Loss of consciousness and coup and contrecoup injuries formerly were considered secondary or tertiary injuries, but with the increased use of body armor in the military, damage to the central nervous system after an explosion has been increasingly attributed to the direct effects of the blast.[28,29] Serious late effects of traumatic brain injuries, such as central nervous system residua, have brought attention to the need for rehabilitation of the central nervous system after blast exposure.

SECONDARY BLAST INJURIES

Many explosive devices contain metallic and other fragments. These and the disintegrated casing are designed to cause penetrating wounds. Such fragments are sometimes incorrectly called shrapnel,

after Henry Shrapnel, the developer of specific military antipersonnel munitions based on the same principle. Penetrating injuries from primary fragments (fragments that are part of the weapon) and secondary fragments (those that result from the explosion) are the leading cause of death and injury in both military and civilian terrorist attacks, except in cases of a major building collapse.

TERTIARY BLAST INJURIES

Blasts cause structural collapse and fragmentation of buildings and vehicles. The types and severity of injuries depend on whether they were caused by collapse or fragmentation. The collapse of buildings and other structures causes a higher rate of death (due to crush injuries and entrapment) than does fragmentation. Fewer people survive structural collapse.[13] Structural collapse and large airborne fragments lead to crush injuries and extensive blunt trauma, whereas flying fragments cause penetrating trauma.

The crush syndrome in victims of structural collapse is a metabolic derangement resulting from damage to muscle tissues and the subsequent release of myoglobin, urates, potassium, and phosphates. Oliguric renal failure, the most severe end point, causes the retention of potassium above and beyond that released from the damaged muscle. Appropriate treatment includes hydration and alkalization.[30]

The compartment syndrome results from the compression that a damaged, edematous muscle exerts within its inelastic sheath. Such confined swelling promotes local ischemia, which then continues a vicious cycle of swelling, increased compartment pressures, decreased tissue perfusion, and further ischemia. Left untreated, compartment syndrome causes local tissue death and also presages development of the crush syndrome. The compartment syndrome usually involves the extremities, and fractures of the long bones commonly give rise to the syndrome. Occasionally, the compartment syndrome may involve the buttocks[31,32] and the abdominal musculature such as the rectus muscle.[33] Pelvic fractures also may induce intraabdominal hypertension, which requires laparotomy and decompression as lifesaving measures.[34] In these cases, urgent application of external fixators should be used to stabilize the pelvis to reduce blood loss.

The characteristic sign of the compartment syndrome is pain out of proportion to the injury. Passive movement of an affected extremity exacerbates pain when the affected compartment of the swollen muscle is placed under tension. Fasciotomy or compartment decompression should be performed as soon as possible. In the unconscious or obtunded patient, monitoring of compartment pressure is useful to determine the need for fasciotomy.[35] A perfusion pressure of less than 35 mm Hg is a reasonable threshold when used in conjunction with clinical assessment.[35]

After the Hanshin–Awaji earthquake,[36] in 1995, which produced 5500 fatalities among 41,000 injured people, the rate of death increased sharply from less than 20 percent among people who had been trapped for less than 24 hours to almost 40 percent among people who had been trapped for longer periods, even if they reached hospitals alive. This increase in mortality was due largely to the sequelae of crush and compartment injuries.

Tertiary blast injuries also result from people being thrown into fixed objects by the wind of explosions. Any body part may be affected, and fractures, traumatic amputations, and open and closed brain injuries occur.

QUATERNARY BLAST INJURIES

Quaternary blast injuries refer to explosion-related injuries, illnesses, and diseases not due to primary, secondary, or tertiary injuries. Quaternary blast injuries encompass exacerbations or complications of persisting conditions, such as might occur in women who are pregnant or in patients receiving anticoagulants. Quaternary injuries include burns (chemical or thermal), toxic inhalation, exposure to radiation, asphyxiation (including carbon monoxide and cyanide after incomplete combustion of materials), and inhalation of dust containing coal or asbestos.

Burns and other quaternary injuries were an important outcome of the attack on the Pentagon on September 11, 2001, in which terrorists used a fuel-laden aircraft. Incendiary bombs, like those used as major weapons in attacks on cities during World War II, caused widespread destruction. To hinder firefighters, delayed-action high-explosive bombs had been dropped with them.

Napalm, used in incendiary bombs, consists of powdered aluminum soap or similar compounds, which gelatinize, or thicken, oil or gasoline; these compounds increase stickiness and burning time. Incendiary bombs have been used by terrorists, particularly in South America, and have been found as booby traps in clandestine drug laboratories in the

United States. Nuclear detonations produce considerable primary thermal effects. Conventional explosives generally do not cause primary fires, because most of the available oxygen is consumed during the explosion itself. After incendiary bomb attacks, the number of burn injuries should be ascertained as early as possible and alternative national burn management resources should be alerted, because large numbers of burn victims can quickly overwhelm local medical resources.

TREATMENT STRATEGIES

GENERAL CONSIDERATIONS AND IMMEDIATE TREATMENT

Once the emergency department of a hospital or a medical facility receives notice of a blast or explosion, a triage area should be established at the entrance to the main emergency department.[37] This area might also be needed for decontamination procedures. Midlevel clinicians should staff this triage area, since specialty surgeons will be needed for operative interventions. An area for treatment of minor injuries should be organized apart from the emergency department. Information about the type of explosion and the target helps to predict the types of injuries. After an explosion in a confined space, such as a bus, one would anticipate, in addition to penetrating injuries, more victims with primary blast injuries and lung damage than would be expected after an explosion in an open space. For example, in two bus bombings in Israel described in 1996,[38] 22 of 52 injured survivors (42 percent) needed endotracheal intubation and 10 (19 percent) required chest-tube thoracostomies. After two open-air bombings in Israel that year, only 13 of 190 victims (7 percent) were intubated, and 5 (3 percent) required chest tubes. In the 1995 Oklahoma City bombing, which involved structural collapse,[39] only 7 of 388 survivors (2 percent) required intubation, and 3 (1 percent) needed chest tubes. Most of the victims in the Oklahoma City bombing had soft-tissue injuries and injuries requiring radiography; 19 percent underwent computed tomographic scanning to determine whether they needed surgical interventions.

Terrorist attacks in civilian settings tend to have a biphasic distribution of mortality — high immediate rates of death followed by low early and late mortality rates. Rarely have civilian providers faced a battlefield approach to triage or sorting. The battlefield approach requires "the greatest good for the greatest number." A situation involving mass casualties of battlefield magnitude in a civilian setting has not happened yet but is certainly possible. This situation requires triage according to the model of urgent, immediate, delayed, minimal, or expectant care. Expectant care is reserved for victims whose survival is unlikely even in the presence of adequate resources. Patients in this category will probably include those in full cardiac arrest as well as those with burns over 100 percent of their body surface. The challenge in civilian blasts and terrorist attacks so far has been identifying critically injured persons with correctable conditions among the many people with minor injuries.

Initial stabilization of victims of blast injury, like that of other trauma victims, includes assessment and management of the airway, breathing, and circulation. Recommended circulatory support includes the infusion of fluid to maintain a systolic blood pressure of 100 mm Hg, a palpable radial pulse of less than 120 beats per minute, and normal mentation. Fluids should not be "pushed" before surgical intervention — clinical and experimental evidence suggests that rapid infusion may increase bleeding.[40,41] When treating patients with the crush syndrome, therapy with fluids must maintain renal perfusion while avoiding fluid overload. Subsequent dialysis may be needed if renal failure progresses with an increasing serum creatinine level of 1.5 mg per deciliter (132.6 μmol per liter) per day.

MANAGEMENT OF SPECIFIC INJURIES

Figure 2 shows a way to check for blast injuries once immediate life support measures have been taken. Treatment of patients after blasts focuses on two specific examinations. First, portable otoscopes are used to identify rupture of the tympanic membranes. If the tympanic membranes are intact, serious primary blast injuries can be conditionally excluded, in the absence of other symptoms such as dyspnea, respiratory distress, and acute abdominal pain. Patients with rupture of the tympanic membranes should undergo radiography of the chest and should be observed for at least eight hours, as clinically indicated. Primary blast injuries are notorious for their delayed onset.

Second, these victims should be monitored by sequentially measuring their oxygen saturation by pulse oximetry. Decreased oxygen saturation probably signals early "blast lung" (pulmonary barotrauma) even before symptoms begin. Treatment of

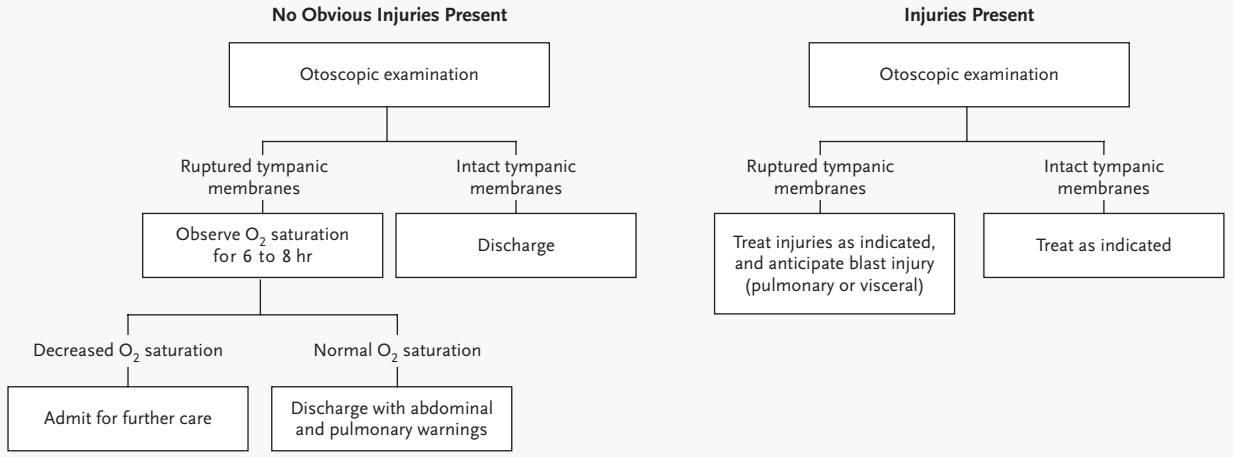

Figure 2. Initial Evaluation for Life-Threatening Injuries.

After evaluation for basic life support and after immediate treatment of life-threatening penetrating or blunt injuries, examination of the tympanic membrane can be used to identify persons who may be at risk for late primary blast injuries, particularly in a setting of mass casualties. Rupture of the tympanic membranes suggests possible pulmonary injury. A period of observation with sequential measurement of oxygen saturation is useful for victims who show signs of no other injuries and might help predict delayed pulmonary or visceral injuries for those patients who have begun treatment.

blast lung is challenging in that ventilation with high peak inspiratory pressures increases the risk of air embolism or pneumothorax.[37] Ventilation should use limited peak inspiratory pressures and permissive hypercapnia; when available, high-frequency ventilation may be of value.[42] Fluid levels must be managed to avoid overload, because damaged lung tissue is particularly susceptible to the development of edema; definitive roles for corticosteroids and antibiotics have yet to be delineated. The patient should be continuously monitored for developing pneumothorax and treated promptly with tube thoracostomy should this ensue.

Treatment of rupture of the tympanic membranes generally is expectant: patients should avoid probing or irrigating the auditory canal, avoid swimming or immersing the head under water, take ototopical antibiotics as indicated, and be referred for potential repair if healing does not occur. Small perforations typically heal within a few weeks. When the ear canal is full of contaminated debris, as has often been the case with soldiers exposed to bomb blasts in Iraq, antibiotics in the form of eardrops should be started as soon as possible to help clear the canal. Otic injuries can be quite severe, with extensive perforations that require tympanoplasty.[43] Evidence of dysfunction of the seventh cranial nerve or vestibular damage suggests severe trauma, and patients with these injuries should be referred for further evaluation. Sensorineural hearing loss after a blast, in the absence of contraindications, may respond to a brief course of steroids.

Multiple severe trauma, amputation, and head, thoracic, and abdominal penetrating injuries are the traumatic lesions associated with death after most terrorist attacks.[37] Each injury requires accurate diagnosis and prompt intervention. Specialty and senior surgeons must be available. Impalement injuries, penetrating wounds, burns, and long-bone fractures can also be produced by blasts. Objects that are impaling a person should be removed or manipulated only in an operating room. To facilitate the transport of impaled patients, the objects can be cut or shortened.

Before a patient is transported to a burn unit, burns should be covered to prevent heat loss and to minimize fluid loss due to disruption in dermal integrity. Also, fluid resuscitation with an appropriate burn formula should be started. The larger the burn, the more heat and fluid are lost. Covering the burn area, ideally with a sterile material, prevents further contamination.

Transporting patients with long-bone fractures requires temporary splinting to manage pain and also to avert further soft-tissue damage. Effective splinting minimizes further neurovascular compro-

mise and bleeding.[44-46] Obvious gross deformities may be gently realigned before splinting. Open fractures must be immobilized and covered with bulky sterile dressings, and therapy with systemic broad-spectrum antibiotics should be begun. Tetanus prophylaxis or booster injections should be given during initial treatment.

Up to 28 percent of blast survivors may have serious eye injuries, particularly if the blast caused shattering glass.[47] These injuries include corneoscleral lacerations, orbital fractures, hyphema, lid lacerations, traumatic cataracts, injury of the optic nerve, serous retinitis, and rupture of the globe itself. Emergency enucleation is not advised. In the case of an exposed globe, provisional repair of eyelid laceration can be considered. Objects penetrating the eye should not be removed in an emergency setting; the eye can be covered with a paper cup or other clean object that will not exert pressure on the globe, and the patient can be referred for definitive surgical treatment. Chemical burns of the eye should be treated by at least 60 minutes of continuous irrigation with sterile saline.

Terrorist attacks can injure children and pregnant women. In the Oklahoma City bombing, which involved the child care center in the Murrah Building, there was a high incidence of traumatic amputation, fractures, and cranial injury among children.[48] In contrast, experience in Israel has shown that penetrating injuries of the trunk are more common in children who are victims of vehicular bombings than they are in children who have other types of trauma.[49] Children who are victims of terrorism require more resources of intensive care units, have higher Injury Severity Scores (scores for severity of injury in patients with multiple injuries), and have longer hospital stays than children who survived traumatic events unrelated to terrorism. Intraabdominal and thoracic injuries in children require prompt acute surgical intervention. The overall care of children is more resource intensive than is the care of adults.

During pregnancy, direct injury of the fetus by a blast is said to be uncommon. The fetus is protected by amniotic fluid, but its attachment to the placenta is at risk if the blast wave affects the high-density uterine wall and the lower-density placental medium, causing placental abruption. For women exposed to blasts in the second and third trimester of pregnancy, admission to the labor and delivery area for fetal monitoring is recommended. If the Kleihauer–Betke assay for maternal hemorrhage is positive, pelvic ultrasonography, fetal monitoring, and obstetrical consultation are recommended.[50]

CONCLUSIONS

In this review, we have outlined mechanisms of blast injuries and provided guidelines for initial treatment. On the basis of experience, many — but by no means all — eventualities have been considered. Updated Web-based sources with appropriate links are also available.[51,52] Although it seems likely that certain injury patterns will repeat themselves, other types of terrorist attacks could provoke different challenges.

The opinions expressed herein are those of the authors and not necessarily those of the Department of Veterans Affairs or the U.S. government.

We are indebted to Abid Rahman, Ph.D., and Stephen Sloan, M.H.A., who coordinated the efforts of the Veterans Health Administration, the Department of Defense, and the Uniformed Services University of the Health Sciences toward the development of educational materials under Public Law 107-287; and to Susan M. Mather, M.D., M.P.H., Jerry L. Mothershead, M.D., M.P.H., Creighton B. Wright, M.D., Nichole B. Amundsen, R.N., M.S., Virginia W. Hayes, R.N., M.S., Artie L. Shelton, M.D., Col. (ret.), M.C., U.S. Army, and Brian J. McKinnon, M.D., Commander, M.C., U.S. Navy, for their substantive contributions to this article.

REFERENCES

1. Bush LM, Abrams BH, Beall A, Johnson CC. Index case of fatal inhalational anthrax due to bioterrorism in the United States. N Engl J Med 2001;345:1607-10.
2. Jernigan DB, Raghunathan PL, Bell BP, et al. Investigation of bioterrorism-related anthrax, United States, 2001: epidemiologic findings. Emerg Infect Dis 2002;8:1019-28.
3. Torok TJ, Tauxe RV, Wise RP, et al. A large community outbreak of salmonellosis caused by intentional contamination of restaurant salad bars. JAMA 1997;278:389-95.
4. Crompton R, Gall D. Georgi Markov — death in a pellet. Med Leg J 1980;48:51-62.
5. Okumura T, Takasu N, Ishimatsu S, et al. Report on 640 victims of the Tokyo subway sarin attack. Ann Emerg Med 1996;28:129-35.
6. Clark MA. The pathology of terrorism: acts of violence directed against citizens of the United States while abroad. Clin Lab Med 1998;18:99-114.
7. Boholm A. Comparative studies of risk perception: a review of twenty years of research. J Risk Res 1998;1:135-64.
8. Covello VT, Peters RG, Wojtecki JG, Hyde RC. Risk communication, the West Nile virus epidemic, and bioterrorism: responding to the communication challenges posed by the intentional or unintentional release of a pathogen in an urban setting. J Urban Health 2001;78:382-91.
9. Karmy-Jones R, Kissinger D, Golocovsky M, Jordan MH, Champion HR. Bomb-related injuries. Mil Med 1994;159:536-9.
10. Federal Bureau of Investigation. 1999 Bombing incidents. Washington, D.C.: Department of Justice, 2003. (FBI Bomb Data Center publication no. 0367.)
11. Champion HR, Oschner MG, Bellamy R. Surgery of victims of conflict. In: Moore EE, Mattox KL, Feliciano DV, eds. Trauma. 5th ed. New York: McGraw-Hill, 2004:1161-93.

12. Cullis IG. Blast waves and how they interact with structures. J R Army Med Corps 2001;147:16-26.
13. Arnold JL, Halperin P, Tsai MC, Smithline H. Mass casualty terrorist bombings: a comparison of outcomes by bombing type. Ann Emerg Med 2004;43:263-73.
14. Gans L, Kennedy T. Management of unique clinical entities in disaster medicine. Emerg Med Clin North Am 1996;14:301-26.
15. Miller K, Chang A. Acute inhalation injury. Emerg Med Clin North Am 2003;21: 533-57.
16. Lioy PJ, Weisel CP, Millette JR, et al. Characterization of the dust/smoke aerosol that settled east of the World Trade Center (WTC) in lower Manhattan after the collapse of the WTC 11 September 2001. Environ Health Perspect 2002;110:703-14.
17. Guy RJ, Glover MA, Cripps NP. The pathophysiology of primary blast injury and its implications for treatment. Part I: the thorax. J R Nav Med Serv 1998;84:79-86.
18. Jensen JH, Bonding P. Experimental pressure induced rupture of the tympanic membrane in man. Acta Otolaryngol 1993;113: 62-7.
19. Katz E, Ofek B, Adler J, Abramowitz HB, Krausz MM. Primary blast injury after a bomb explosion in a civilian bus. Ann Surg 1989; 209:484-8.
20. Gutierrez de Ceballos JP, Fuentes FT, Diaz DP, Sanchez MS, Llorente CM, Guerrero Sanz JE. Casualties treated at the closest hospital in the Madrid, March 11, terrorist bombings. Crit Care Med 2005;33:S107-S112.
21. Leibovici D, Gofrit ON, Shapira SC. Eardrum perforation in explosion survivors: is it a marker of pulmonary blast injury? Ann Emerg Med 1999;34:168-72.
22. de Candole CA. Blast injury. Can Med Assoc J 1967;96:207-14.
23. Coppel DL. Blast injuries of the lungs. Br J Surg 1976;63:735-7.
24. Mellor SG, Cooper GJ. Analysis of 828 servicemen killed or injured by explosion in Northern Ireland 1970-84: The Hostile Action Casualty System. Br J Surg 1989;76: 1006-10.
25. Irwin RJ, Lerner MR, Bealer JF, Mantor PC, Brackett DJ, Tuggle DW. Shock after blast wave injury is caused by a vagally mediated reflex. J Trauma 1999;47:105-10.
26. Guzzi LM, Argyros G. The management of blast injury. Eur J Emerg Med 1996;3: 252-5.
27. Yang Z, Wang Z, Tang C, Ying Y. Biological effects of weak blast waves and safety limits for internal organ injury in the human body. J Trauma 1996;40:Suppl 3:S81-S84.
28. Trudeau DL, Anderson J, Hansen LM, et al. Findings of mild traumatic brain injury in combat veterans with PTSD and a history of blast concussion. J Neuropsychiatry Clin Neurosci 1998;10:308-13.
29. Haberstroh J. Focus on head injuries. Newsday. March 29, 2004.
30. Abassi ZA, Hoffman A, Better OS. Acute renal failure complicating muscle crush injury. Semin Nephrol 1998;18:558-65.
31. Brumback RJ. Traumatic rupture of the superior gluteal artery, without fracture of the pelvis, causing compartment syndrome of the buttock: a case report. J Bone Joint Surg Am 1990;72:134-7.
32. Su WT, Stone DH, Lamparello PJ, Rockman CB. Gluteal compartment syndrome following elective unilateral iliac artery embolization before endovascular abdominal aortic aneurysm repair. J Vasc Surg 2004;39: 672-5.
33. O'Mara MS, Semins H, Hathaway D, Caushaj PF. Abdominal compartment syndrome as a consequence of rectus sheath hematoma. Am Surg 2003;69:975-7.
34. Hessman M, Rommens P. Does the intrapelvic compartment syndrome exist? Acta Chir Belg 1998;98:18-22.
35. Vassalos A, Rana B, Patterson PRN, Grigoris P. Compartment syndrome — current trends in Scottish practice. Scott Med J 2003;48:82-4.
36. Kuwagata Y, Oda J, Tanaka H, et al. Analysis of 2,702 traumatized patients in the 1995 Hanshin-Awaji earthquake. J Trauma 1997;43:427-32.
37. Halpern P, Tsai MC, Arnold JL, Stok E, Ersoy G. Mass-casualty, terrorist bombings: implications for emergency department and hospital emergency response. Prehospital Disaster Med 2003;18:235-41.
38. Leibovici D, Gofrit ON, Stein M, et al. Blast injuries: bus versus open-air bombings — a comparative study of injuries in survivors of open-air versus confined-space explosions. J Trauma 1996;41:1030-5.
39. Hogan DE, Waeckerle JF, Dire DJ, Lillebridge SR. Emergency department impact of the Oklahoma City terrorist bombing. Ann Emerg Med 1999;34:160-7.
40. Leppaniemi A, Soltero R, Burris D, et al. Fluid resuscitation in a model of uncontrolled hemorrhage: too much too early, or too little too late? J Surg Res 1996;63:413-8.
41. Burris DG, Rhee P, Kaufmann CR, et al. Controlled resuscitation for uncontrolled hemorrhagic shock. J Trauma 1999;46:216-23.
42. Stein M, Hirshberg A. Medical consequences of terrorism: the conventional weapon threat. Surg Clin North Am 1999;79: 1537-52.
43. Miller IS, McGahey D, Law R. The otologic consequences of the Omagh bomb disaster. Otolaryngol Head Neck Surg 2002; 126:127-8.
44. Bone LB, Johnson KD, Weigelt J, Scheinberg R. Early versus delayed stabilization of femoral fractures: a prospective randomized study. J Bone Joint Surg Am 1989; 71:336-40.
45. Abarbanell NR. Prehospital midthigh trauma and traction splint use: recommendations for treatment protocols. Am J Emerg Med 2001;19:137-40.
46. Wood SP, Vrahas M, Wedel SK. Femur fracture immobilization with traction splints in multisystem trauma patients. Prehosp Emerg Care 2003;7:241-3.
47. Odhiambo WA, Guthua SW, Macigo FG, Akama MK. Maxillofacial injuries caused by terrorist bomb attack in Nairobi, Kenya. Int J Oral Maxillofac Surg 2002;31:374-7.
48. Quintana DA, Parker JR, Jordan FB, Tuggle DW, Mantor PC, Tunell WP. The spectrum of pediatric injuries after a bomb blast. J Pediatr Surg 1997;32:307-11. [Erratum, J Pediatr Surg 1997;32:932.]
49. Aharonson-Daniel L, Waisman Y, Dannon YL, Peleg K. Epidemiology of terror-related versus non-terror related traumatic injury in children. Pediatrics 2003;112:280-91.
50. Lavanoas E. Blast injuries. (Accessed March 7, 2005, at http://www.emedicine.com/emerg/topic63.htm.)
51. Treatment of primary blast injury. In: Emergency war surgery NATO handbook. Washington, D.C.: Department of Defense. (Accessed March 10, 2005, at http://www.vnh.org/EWSurg/EWSTOC.html.)
52. Wightman JM, Gladish SL. Explosions and blast injuries: a primer for clinicians. Atlanta: Centers for Disease Control and Prevention. (Accessed March 7, 2005, at http://www.cdc.gov/masstrauma/preparedness/primer.pdf.)