

Anesthesia Implications of Blast Injury

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BLAST INJURIES caused by a powerful explosion have occurred at a relatively steady rate in the North America and were often caused by home or workplace accidents.¹ The Federal Bureau of Investigation reported 17,579 intentional bombings in the United States (averaging 1,953 per year) between 1988 and 1997.² In contrast, there were only 46 reported deliberate bombings in Canada in 2003.³ Accidental detonations account for a higher total number of explosions in these 2 countries per year.

Recent intentional bombings around the world such as in Iraq, Jordan, London, Madrid, Bali, Istanbul, Israel, and the September 11th attacks have emphasized that no community is completely safe from deliberate explosions. Large urban centers seem to be at especially increased risk of being targets.⁴ Today, explosions are the most common cause of injury associated with terrorism affecting the general population, and blast victims have hence become a concern for civilian physicians. Accidental explosions at home or at the workplace continue to produce a significant number of casualties each year around the world. Fireworks are another source of injury as are antipersonnel landmines in some areas of the world.

This clear and grim reality makes it important for anesthesiologists in all communities to understand the physiology of primary blast injury (PBI) and injury pattern among explosion victims. Anesthesiologists' knowledge of physiology and pharmacology and their ability to resuscitate critically wounded patients puts them in the front line in a major explosion incident. This explains why anesthesiologists, along with other acute-care specialists, must be integrally involved in disaster management and planning. In Israel, anesthesiologists, trauma surgeons, and emergency physicians stand side by side and work closely as a team in order to treat blast casualties most effectively.^{5,6}

PHYSICS OF EXPLOSIONS

An explosion is defined as the release of mechanical, chemical, or nuclear energy in a sudden and often violent manner with generation of high temperatures and winds. The abrupt rise in atmospheric pressure is termed "blast overpressure" (BOP). Highly pressurized systems, materials, or gases that support detonation such as natural gas used for heating or cooking are the most common causes for accidental explosions at home. Explosive devices contain compounds, which are chemicals or chemical mixtures that can undergo rapid, abrupt combustion with generation of large amounts of gas and heat. A sudden increase of atmospheric pressure propagates radially from the site of detonation and is followed by a BOP wave (pressure or blast wave). The leading front of this pressure wave is termed "blast front" and is responsible for the peak high pressure.⁷ For example, 25 kg of trinitrotoluene will generate 150 pounds per square inch (psi) peak BOP for 2 milliseconds that moves at 3,000 to 8,000 m/s. The peak pressure, duration, and number of exposures determine the primary blast injury. Three pounds per square inch BOP typically will knock a human over, 35 psi will cause significant lung injury, and 65 psi will result in 99% mortality.⁷

The force of the blast is directly related to the size and the type of explosive compound and is inversely related to the square of the distance from the detonation. PBI are therefore limited to the relative proximity of the detonation itself.⁸ If the pressure wave is reflected by a solid surface, the reflected pressure can be several times that of the incident wave. Injury and mortality can therefore be much higher if the victim is in close proximity to solid structures.⁹ The same principle is valid for confined space explosions.⁷

Explosives can be grouped into 2 main classes: low-order explosives (LE) with combustion rates of centimeters per second; and high-order explosives (HE), with peak BOP traveling up to 9,140 m/s in air and at even much higher velocities in water.^{9,10}

The first explosive known was called black powder or gunpowder. Chinese alchemists discovered it as early as 850 AD. Nitrocellulose and nitroglycerine were discovered in 1846 and were the first modern high explosives. Alfred Nobel patented dynamite in 1867. Other examples of HE include trinitrotoluene, Semtex, C-4, and ammonium nitrate fuel oil; whereas the LE group consists of gunpowder, pipe bombs, and petroleum-based bombs such as Molotov cocktails. The main use of

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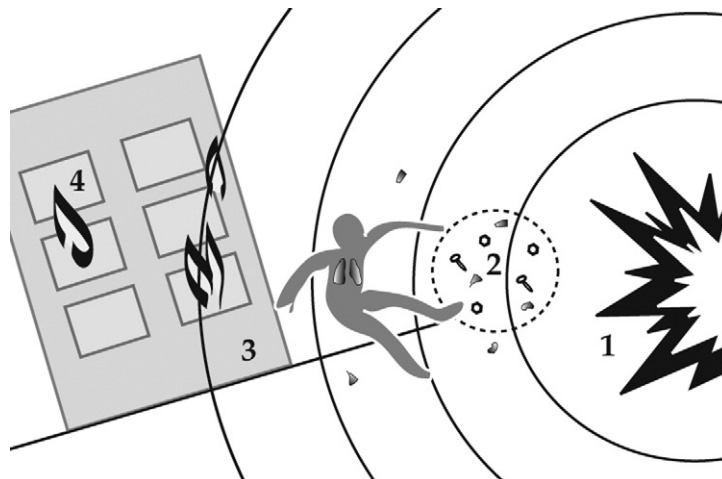


Fig 1. (1) Primary blast injury: blast over pressure (affects mostly air-filled organs such as ears, lungs, and gastrointestinal tract), (2) secondary blast injury: propelled objects/shrapnel, (3) tertiary blast injury: impact of displaced body against hard surface, and (4) quaternary blast injury: burns.

explosives during peacetime in the United States is coal mining.¹¹

Explosions can produce a unique pattern of injury to an organism that is commonly seen during armed conflict but seldom encountered outside of combat. Life-threatening multiorgan injuries may occur to many victims simultaneously. Organisms exposed to BOP mostly experience internal injury to the hollow organs, in particular the lungs, gastrointestinal tract, and the ears. Lung injury and the development of air emboli in the pulmonary and systemic circulations are the main mechanisms leading to death from the blast itself.

Because detonations are relatively infrequent, blast injuries can cause significant triage, diagnostic, and management problems for emergency physicians and anesthesiologists. The injury patterns depend on multiple characteristics of the explosion: blast type (bomb *v* accidental explosion), composition and amount of materials involved, HE versus LE, “manufactured” military-produced weapons versus “improvised” terrorist-produced devices, open versus closed space, distance to the blast, structural collapse, and presence of protective barriers. Shrapnel, poisons, chemicals (chemical weapons), infectious agents, or radiation (dirty or nuclear bomb) can add to the complexity of a bomb attack that can easily overwhelm the providers of emergency care.

TRIAGE AND INITIAL MANAGEMENT

Initial medical management and primary triage begin by emergency medical services at the site of the blast. In case of terrorist bombings, it needs to be remembered that a second explosion also known as “second-hit” principle may specifically target emergency personnel.^{12,13} Secondary triage is of utmost importance and occurs when the patients arrive at the hospital.^{14,15} It needs to protect the available resources from overtriage because large numbers of casualties can easily overwhelm existing medical resources and lead to confusion and increased mortality in patients otherwise regarded as salvageable.^{12,16-19} A report from Madrid describes an overtriage rate of

more than 50% at a university hospital after the 2004 Madrid train bombings.²⁰

After an attack, many victims arrive at the emergency department (ED) within minutes of the blast. An “upside-down” triage phenomenon must be expected, where the most severely injured arrive after the less injured, who often bypass the emergency medical services and go directly to the ED.²¹ About half of all initial casualties may arrive at the ED within the first hour after the detonation in bombing incidents, and this number can be used to estimate the total number of victims and recourses needed.²¹

Anesthesiologists may be involved in triage and assessing the survivability of the patient’s injuries. Patients are typically classified into 4 categories: expectant (death is inevitable), immediate (life-saving treatment required immediately), delayed (treatment can be delayed for a reasonable amount of time), and minor (not significantly injured, “walking wounded”).²²⁻²⁵ Some authors have recommended treating injuries of moderate severity first rather than the greatest injuries and “expectant” casualties.^{6,26} Several studies reported that 9% to 22% of the casualties in terrorist bombing incidents were critically injured requiring urgent care.^{6,12,26,27}

Undertriage, or the assignment of critically injured patients to a delayed care category, is a rare but a significant risk for patients who suffer mostly from primary blast injuries with minimal external injuries.^{6,12,28-34} Ongoing surveillance of all patients by the triage physicians is therefore important to recognize changing medical conditions.^{6,12,35} Anesthesiologists treating casualties must be prepared for a new quality of injuries and a new class of casualty different from conventional trauma.³⁶⁻³⁸ Blast lung injury (BLI) and multiple superficially appearing injuries often only become apparent later as being life-threatening.¹⁵ Triage protocols of bombing mass-casualty incidents have previously been described and differ from conventional trauma protocols.^{36,39-41} Familiarity with these triage systems is highly important for the anesthesiologist in order to treat and resuscitate the patients, es-

Table 1. The 4 Basic Mechanisms of Blast Injury

Primary
Injury is directly caused by blast overpressure
Blast lung injury
Rupture of hollow viscera
Tympanic membrane rupture
Cardiac arrhythmias, myocardial injury
Secondary
Injuries by objects set in motion by the blast
Shrapnel wounds
Penetrating and vascular trauma
Penetrating infectious biological material
Fragmentation injuries
Tertiary
Injuries produced by displacement of the victim against stationary objects or by structural collapse
Blunt trauma
Penetrating trauma
Crush injuries
Traumatic amputations and fractures
Compartment syndrome
Head injuries
Quaternary
Explosion-related injuries
Miscellaneous injuries
Thermal trauma and burns
Exposure to toxins or radiation
Asphyxiation (including carbon monoxide and cyanide)

pecially when surgery is part of this process and not something that follows the process.⁴²

PATTERN OF INJURY

Injury patterns after explosions are termed multidimensional because of complex injury mechanisms (Fig 1).^{7,36,43} These mechanisms have been well documented and can be divided into 4 groups: primary, secondary, tertiary, and quaternary blast injury. PBI are induced directly by the BOP, secondary blast injuries result from objects set in motion by the explosion, tertiary blast injuries are produced by the impact of the displaced victim against stationary objects, and quaternary blast injuries include burns from heat and fire production and miscellaneous injuries (Table 1). A fifth blast injury mechanism has recently been proposed as the patients' hyperinflammatory response, unrelated to injury severity and complexity.⁴⁴

Injuries associated with large blasts are markedly different from those seen in conventional trauma victims in North America. Injuries in peaceful times mostly consist of blunt trauma. Blast victims often show a combination of the 4 main groups of trauma (blast, blunt, penetrating, and thermal) as is more common in armed conflicts. The injuries found largely depend on the setting and type of explosion.

Three different settings have been targeted in recent terrorist bombing incidents: (1) open spaces (OS), such as open market areas or bus stops; (2) semiconfined spaces (SCS), such as restaurants or night clubs; and (3) confined spaces (CS), such as buses or trains. Each type produced different and unique patterns of injury, mortality, and hospitalization.⁴⁵

Explosions in OS usually injure victims in close proximity to

the blast because the energy of the detonation dissipates inversely with the distance to the second power.⁴⁶ Shrapnel and building collapse can be significant mechanisms of injury in this setting. Attacks in SCS often result in a high casualty count and mortality among the victims. Injuries are typically severe and are caused by penetrating, blast, and thermal injuries. CS detonations usually show the most severe forms for primary blast injuries, with a large number of fatalities because of the additive effects of the reflections and reverberations of the BOP from the walls.^{6,47-49} Leibovici et al⁴⁷ reported a 7.8% mortality in OS and a 49% mortality in CS bombings in Israel. A high dead-to-critically wounded ratio of >2:1 has been reported for CS explosions.^{20,24}

PBI occurs as a result of the interaction of the BOP wave and the body.⁵⁰ These interactions differ from organ to organ, with air-filled organs and air-fluid interfaces more commonly involved. Pathology of the BOP has been described previously.⁵⁰⁻⁵⁴ The Injury Severity Score has been used to evaluate patients after terrorist bombings.^{20,55} Blast casualties experience more severe injuries compared with conventional trauma, as is indicated by a higher Injury Severity Score.⁵⁶

In a report following the Madrid train bombings, rupture of the tympanic membrane (41% of 243) was the most frequent injury followed by chest injuries (40%) and shrapnel wounds (36%). Fractures (18%), burns (18%), eye lesions (18%), head trauma (12%), and abdominal injuries (5%) were also commonly described.²⁰ Katz et al⁴⁸ reported perforated eardrums in 76%, BLI in 38%, and abdominal PBI in 14% of patients of a terrorist bomb explosion in a bus in Jerusalem.⁴⁸ The following organ damages are commonly found after explosions.

The brain is less susceptible to primary blast injury than are hollow organs because the brain is relatively homogeneously encapsulated within the dura and protected by the skull. Penetrating shrapnel, flying debris, displacement of the victim, and structural collapse more commonly cause brain injury. In one study of 5,600 terrorist bombing incidents, 6% of the casualties (327 cases) sustained brain or skull injury, and 91% of these died within 24 hours.⁵⁰ The brain may be directly traumatized, resulting in cerebral contusion, skull fracture, coup-counter-coup injuries, epidural and subdural hematomas, cerebral hemorrhage, and edema.^{57,58} Cerebral infarction might be a result of cerebral air and fat embolism.⁵⁰

The most common injuries to the eye are conjunctival hemorrhage and abrasion. Air embolism might ophthalmoscopically be visualized in the retinas of casualties.⁹ More severe eye injury, intraocular hemorrhage, and enucleation might make early surgical therapy necessary.

The ear as an air-filled organ is the most susceptible to BOP. Tympanic membrane, middle ear, and inner ear are often involved. Tympanic membrane rupture is the most common injury after BOP exposure.^{49,59}

The upper respiratory tract consists of the nasopharynx, larynx, and trachea and is frequently involved after BOP exposure. Hemorrhage in the form of single petechia and confluent ecchymosis are the classical findings after severe BOP. Laryngeal injury, thermal injury, symmetrical hemorrhages on the vocal cords, dislocation of cartilages, or laryngeal fracture are described and might make intubation difficult. Inspiratory

Table 2. Severity Categories of Blast Lung Injury⁷²

Blast Lung Injury	Mild	Moderate	Severe
Chest radiograph	Localized lung infiltrates	Bilateral asymmetric lung infiltrates	Severe diffuse bilateral infiltrates
Bronchopleural fistula	Not present	Sometimes present	Present
PaO ₂ /F _i O ₂	>200	60-200	<60
Respiratory need for mechanical ventilation	Unlikely	Highly likely	Highly likely, difficult to ventilate, requiring unconventional ventilation strategies
PEEP requirement	Unlikely	5-10 cmH ₂ O usually needed	>10 commonly needed
Air embolism	Low risk	Increased risk	High risk

stridor is a warning sign.⁶⁰ Surgical experience showed that acute laryngeal trauma should be treated within the first 24 hours so that complications and uncontrolled airways are avoided.⁶¹ Tracheal and bronchial injury can lead to pneumothorax, hemothorax, and subcutaneous emphysema and the inability to ventilate the patient adequately. A foreign body blocking the trachea after an explosion has been described.⁶²

The lung is the organ that is second most commonly injured in association with an explosion. Blast lung is characterized by the clinical triad of apnea, bradycardia, and hypotension.²¹ BLI should be suspected in the presence of dyspnea, cough, hemoptysis, or chest pain.

Primary blast, shrapnel, and crush injuries involving the lungs have been described and might lead to pneumothorax, hemothorax, pneumomediastinum, subcutaneous emphysema, and pulmonary contusion. BLI produces a characteristic “butterfly” pattern on chest x-ray.²¹ DePalma et al⁴⁹ suggest chest radiography for all patients with tympanic membrane rupture to rule out BLI.

Pressure differences caused by the BOP can cause disruption of the alveolar-capillary interface. These interruptions can lead to life-threatening acute air embolism because the air can occlude blood vessels in the heart, lungs, abdomen, brain, and spinal cord.^{21,49,63,64} Arterial air emboli are believed to be responsible for most sudden deaths within the first hour after the blast.⁶⁵ Pizov et al⁶⁶ have classified BLI into mild (PaO₂/F_iO₂ >200, localized lung infiltrates on chest radiograph, no evidence of bronchopleural fistula), moderate (PaO₂/F_iO₂ 60-200, bilateral and asymmetric chest infiltrates), and severe (PaO₂/F_iO₂ <60, severe bilateral infiltrates, and bronchopleural fistula present) (Table 2). Ventilatory support is likely to be necessary in moderate-to-severe BLI because of lung or coexisting injury.⁶⁶ Desaturation might be the first sign of BLI even before other symptoms appear.⁴⁹ It has been proposed that subtle biochemical changes such as free-radical-mediated oxidative stress and antioxidant depletion contribute to BLI.^{67,68} These mechanisms can also cause methemoglobinemia and oxoferryl hemoglobin.^{69,70} The exposure to inhaled toxins and poisonings (eg, carbon monoxide and cyanide) is a considerable risk.²¹

A recent study by Almogly et al⁷¹ suggests a correlation between external injury and BLI. Victims of 15 bombing incidents in Israel were more likely to develop BLI if skull fractures, penetrating wounds to head and torso and burns covering more than 10% of body surface area, were found. It is impor-

tant to consider BLI in patients with these injury patterns because symptoms of BLI might be subtle at the beginning but can quickly deteriorate requiring interventions and more sophisticated forms of ventilation.^{66,71}

BOP can cause injury to the heart by multiple mechanisms including cardiac contusion; pathologic neurocardiac reflexes; cell-mediated pathways of injury; and coronary artery obstruction from embolism of air, fat, or fibrin.⁵⁰ Myocardial ischemia and infarction caused by air or other emboli are thought to be major causes of death.^{50,72} Of 249 animals exposed to a complex-blast wave environment, 5 of 249 (2%) showed lethal air embolism. All 5 had coronary artery air embolism, and 2 had cerebral air embolism.⁷³

Cardiac arrhythmias are common including asystole, bradycardia, tachycardia, and ventricular fibrillation.⁵⁰ Blast-induced shock may result from immediate myocardial depression without a compensatory vasoconstriction.⁷⁴ Penetrating cardiac trauma is one of the most serious injuries but can be survived if treated promptly.⁷⁵ Transesophageal echocardiography may be helpful to diagnose cardiac tamponade, wall motion abnormalities, air embolism, and hypovolemia,⁷⁶ but care must be taken when inserting the probe because rupture of the esophagus has been reported after an explosion.⁷⁷

The organs of the abdomen most commonly affected by PBI are the hollow viscous organs. Hemorrhage, perforation, and lacerations may occur, especially in the colon where gas tends to accumulate.^{43,50,78} Slow disruption of the bowel wall might lead to clinical symptoms several days after the blast.⁴³ Shrapnel injuries might cause abdominal injury as well. Splenic and hepatic ruptures can cause life-threatening bleeding.

Shrapnel injuries are reported in 20% to 42% of suicide-bombing victims in Israel, with a higher incidence in closed spaces.⁷⁰ Flying shrapnel can penetrate any part in the body and severity of the injury is related to the entry site. Shrapnel fragments are frequently large with an irregular shape, and their path within the body is often unpredictable. Injury to the brain, heart, and large vessels carries a high mortality. Vascular damage often demands early surgical intervention. The most common severe vascular injuries reported by Wolf and Rivkind⁷⁹ were vascular extremity injuries with skeletal fracture and severe soft-tissue damage. A multidisciplinary vascular-orthopedic-plastic surgical approach was frequently necessary to save the damaged limbs. The most common surgical procedures were vascular repair with arterial and venous shunts, skeletal fixation, open fasciotomy, and soft-tissue coverage.

Tourniquets applied to bleeding limbs can cause ischemia and distal thrombosis. Washout of ischemic products, embolism of thrombotic material, and fat embolism after fractures can complicate anesthesia management after the tourniquet is released.

Traumatic limb amputations are a grim prognostic marker, and victims are often dead at the bombing site.^{6,80} Typically, the victims were in close proximity to the actual bomb, and limb amputations were a direct result of shearing forces of the BOP.⁸¹ Severe coexisting injuries are common. Other causes of limb amputation are large shrapnel and building collapse.⁴⁷ One young man survived the London train bombings from July 7, 2005, with an amputated leg after the train driver tied his belt around the remains of the leg while waiting for rescue personnel to arrive.⁸²

The collapse of buildings and other structures by large explosions causes a high rate of death because of crush injuries and entrapment.^{31,49,83} Although mortality is high after structural collapse, the patients who survive may show less severe injuries compared with other blast victims.⁴⁹ For example, only 2% of survivors of the 1995 Oklahoma City bombing, which involved structural collapse, needed endotracheal intubation compared with 42% after CS bombings in Israel.^{47,49,84} Patients with crush injuries are in danger of acute renal failure because of release of myoglobin from damaged muscle. Treatment includes hydration and alkalization.^{49,85}

Thermal injury is common after explosions. Burns were an important outcome after the attacks on the Pentagon in 2001 and the 2002 Bali bombing. Patients with large burns need to be covered to prevent heat and fluid loss because of disruption of dermal integrity. Aggressive fluid therapy is normally indicated to prevent cardiovascular collapse. High blood loss is a significant problem associated with burn surgery.⁸⁶

ANESTHESIA DEPARTMENT AND ORGANIZATION

A mass-casualty terrorist event can easily overwhelm hospitals and medical providers. Careful planning and mass-casualty drills can help to decrease the chaos that occurs after such an event. Anesthesia departments need to participate in these planning efforts and organize themselves to be able to respond efficiently to a bombing incident. Shamir et al⁵ have provided helpful examples of how an anesthesia department can improve the care of bombing victims and have emphasized 2 basic personnel elements: perioperative anesthesia management and a chain of command. In the Israeli experience, perioperative anesthesia management for blast victims includes a forward deployment of anesthesiologists to the ED. Anesthesiologists together with ED physicians and surgeons are involved in treating the severely injured. The anesthesiologist's ability to manage the airway, initiate mechanical ventilation, administer fluids, insert intravenous and arterial catheters, and treat shock is of importance in this setting. The anesthesiologist can continuously assess, treat, and prepare the patients for the operating room (OR), radiology suite, or the intensive care unit (ICU). Shamir et al⁵ reported a mean time from arrival in the ED to skin incision in the OR of about 2 hours because only few patients with severe life-threatening injuries had to be operated on immediately. The anesthesiologist can stay close to the patient and move with the patient from the ED to imaging

stations, the preoperative area, recovery room, the OR, and the ICU.

Casualties need to continually move from the ED to areas in the hospital to free up the ED for new arrivals. Minimal acceptable care was suggested during the initial phase, and more optimal care is to be sought in the definite phase, which takes place when casualties are no longer arriving in the ED.¹⁴ Patients who are already in the hospital should, if possible, be discharged or moved if clinically feasible out of the ED or ICU.^{13,20} All elective surgery and imaging studies should be cancelled immediately and the operating rooms prepared for the injured. Lesser injured casualties can be moved from the ED to the wards or transferred to other hospitals. The holding area of the OR and recovery room have been described as good locations for unstable or ventilated patients awaiting surgical treatment or a bed in the ICU.^{5,20}

Organizing this type of anesthesia response is crucial, and a clear chain of command can help to decrease confusion and chaos. Shamir et al⁵ have recommended a senior anesthesiologist to be stationed in the ED as an ED anesthesiology coordinator to help with triage and to keep a log of the most severely injured patients. This ED anesthesiology coordinator can assign other anesthesiologists in the ED to critically injured patients and needs to keep close contact with the clinical coordinating anesthesiologist in the OR. This OR clinical coordinator needs to organize the OR and recovery-room response. He/she needs to call for backup, have other anesthesiologists prepare the operating rooms, or look after patients in the OR holding area or recovery room. He/she needs to keep close contact with an OR supervising nurse and keep an updated OR schedule. Frequent contact with the ED coordinating anesthesiologist is very important in order not to be surprised by newly arrived patients and to avoid chaos in the OR. Because bombing victims can be very challenging to treat, it must be anticipated that some patients will need more than 1 anesthesiologist caring for them. This is especially important for remote areas outside the OR that are unfamiliar to the anesthesiologist (computed tomography or angiography suite). Therefore, free anesthesiologists need to be available to meet patients and help their colleagues. This type of response is more challenging during evening and nights because fewer anesthesiologists are in house and the clinical coordinator needs to alert an adequate number of anesthesiologists to come to the hospital. A prepared calling list during disasters can help to organize and activate an anesthesia department. Cellular phone service might not be available for various reasons. Anesthesia technicians are helpful in order to move equipment to locations where they are needed (eg, anesthesia equipment or rapid-infusing devices to the angiography suite).

ANESTHESIA MANAGEMENT

Anesthesia and surgery should be truncated according to principles of damage control, allowing for rapid turnover of rooms.^{12,20} Anesthesia management highly depends on the patient's injuries and type of surgery. Trauma and life support protocols, such as Advanced Trauma Life Support and Advanced Cardiac Life Support, should be followed first when caring for a severely injured victim because they are designed to identify and treat the most life-threatening problems first. As

in conventional trauma, the anesthesiologist's main concern is to maintain adequate respiratory gas exchange, to maintain adequate circulation, and to preserve central nervous system function.^{87,88} Once these objectives have been established, a more complete history and physical examination should be obtained. A chest radiograph, laboratory workup, and if necessary a computed tomography scan or other diagnostic studies (such as angiogram) should be obtained if feasible before induction of anesthesia.

Patients should be monitored with American Society of Anesthesiologists standard monitors. Large-bore intravenous catheters are indicated; arterial catheters and central venous and pulmonary artery catheters are helpful in the severely injured. The CXR can give valuable information about pulmonary contusion, pneumothorax, and PBI and should be assessed before induction of anesthesia when available. Hemoglobin levels can be used to guide transfusion requirements.⁶⁵ Maintenance of temperature homeostasis is important, and the operating room should be warm upon arrival of the patient. Hypothermia can be a leading cause of coagulopathy.⁶ Intravenous fluids should be warmed and warming blankets applied. Humidification of inspired gases reduces evaporative heat loss and can help to warm the patient.

Perioperative broad-spectrum antibiotic therapy should be started liberally.⁷⁹ Unusual infections with *Candida species*, which needed antifungal therapy, have been reported after explosions in Israel.^{43,89}

Endotracheal intubation should be performed in patients with significant deoxygenation and respiratory distress, for comatose patients to protect the airway, and when inducing general anesthesia for surgery.^{43,65,87} Initiating general anesthesia should be accomplished by choosing medications that minimize cardiovascular depression and intracranial hypertension.⁸⁷ Small doses of fentanyl, etomidate, or propofol can be titrated to desired anesthesia effect. Adequate cardiovascular homeostasis and cerebral perfusion have to be balanced with the need for hypnosis and amnesia.⁸⁷

All patients need to be considered at risk for aspiration on induction and full stomach precautions taken. Succinylcholine for rapid-sequence induction is initially safe, but potentially catastrophic hyperkalemia might occur 24 hours after major thermal and denervation injuries.^{87,90,91} A large dose of rocuronium can be used for rapid-sequence induction. Anesthesia can be maintained with halogenated hydrocarbon vapors or with total intravenous anesthesia. Nitrous oxide should be avoided in blast victims because of the potential of worsening air embolism and diffusion into closed air spaces such as a pneumothorax. Prophylactic chest tube (thoracostomy) placement is recommended by the Centers for Disease Control and Prevention before induction of general anesthesia if BLI is suspected.²¹

Respiratory management and ventilation in patients with BLI are challenging in that positive-pressure ventilation (PPV) increases the risk of arterial and venous air embolism, barotrauma, and pneumothorax.⁶⁵ The current recommendation for treatment of BLI is to prevent intubation and PPV when feasible.^{9,66} For example, victims who are hypoxemic but not hypercarbic can be treated with high inspired oxygen concentrations.⁶⁵ If intubation and PPV are deemed necessary, me-

chanical ventilation should use respiratory parameters of tidal volume, respiratory rate, inspiratory flow rate, and the ratio of inspiratory and expiratory time to keep airway pressures as low as possible and to allow permissive hypercapnia.^{49,65,92} Sorkine et al⁹² limited peak inspiratory pressures (<40 cmH₂O) in 17 blast victims, and no victim developed barotrauma; all were hemodynamically and metabolically stable despite permissive hypercapnia. When treating victims with suspected or proven head injuries, permissive hypercapnia might be less acceptable because of fear for increased intracranial pressure.^{43,66} More unconventional respiratory strategies such as inverse inspiratory/expiratory ratios, high-frequency jet ventilation, use of nitric oxide, and extracorporeal membrane oxygenation have been reported in patients with severe BLI.^{1,66}

Air embolism can be produced in mechanically ventilated patients for hours to days after blast exposure.⁶⁵ Therapy of air embolism is treatment in a hyperbaric chamber. Reduction of airway pressures can reduce the amount of air entering the blood stream. Positioning the patient recumbent in left lateral decubitus position with his/her head down may help to prevent air embolism to the brain and coronary arteries. The Trendelenburg position might predispose to coronary artery air embolism, whereas the upright position may predispose air embolism to the head.⁶⁵

Management of severe hemoptysis includes blocking the involved side with a bronchial blocker or double-lumen tube.¹ Pneumothorax should be treated with chest tubes, and needle thoracocentesis can be used initially to decompress a suspected tension pneumothorax.¹

Fluid management after blast injury can be a major challenge. Infusion of intravenous fluids is recommended to maintain a systolic blood pressure of 100 mmHg and a palpable radial pulse of <120 beats/min.⁴⁹ Fluid overload must be avoided because of the potential of worsening respiratory status and increasing bleeding,^{49,76,93} whereas suboptimal resuscitation may exacerbate tissue damage. Fluid therapy must maintain renal perfusion, especially in patients with crush injury, in order to avoid myoglobin-induced renal failure.^{49,85} Patients with burns need to be hydrated more aggressively, and fluid replacement within the first 24 hours usually follows well-established burn guidelines and formulas.⁸⁶

Massive blood loss might need to be replaced by the anesthesiologist. Patients frequently develop dilutional thrombocytopenia, prolonged coagulation times, and decreased serum calcium levels. Coagulopathy needs to be treated with factor, platelet, and calcium replacement. Normothermia should be aimed for. Hypotensive victims of suicide-bombing attacks with abdominal and/or thoracic penetrating injuries are often taken to the OR immediately.⁶ Often, it is unclear which of the many injuries and shrapnel wounds causes the hypotension. Many of the shrapnel entry sites might be on the back of the patient because suicide bombers often attack from behind.⁶ The lateral decubitus position might be necessary to pack some of the wounds to achieve adequate hemostasis. Packing shrapnel wounds quickly might lessen the degree of soft-tissue damage and hypothermia and achieve better hemostasis and survival.⁶

Recombinant activated factor VIIa has been recommended to treat exsanguinating victims as an adjunct to surgical hemostasis.⁶ Recombinant activated factor VIIa has been used success-

fully in patients with severe injury and profound bleeding after surgical hemostasis and massive transfusions of red blood cells and coagulation factors.⁹⁴

CONCLUSIONS

Anesthesiologists routinely manage critically ill patients and trauma victims. Their detailed knowledge of pathophysiology and pharmacology and their ability to resuscitate seriously wounded patients are factors that make anesthesiologists well prepared in coping with the aftermath of a major explosion incident. Therefore, anesthesiologists play an important role in the initial treatment of blast casualties and in their continued management in the critical care unit.

Unfortunately, terrorist mass casualty bombing incidents are increasing in frequency around the world, and no community is completely safe. Although large urban centers have been targeted recently, it must be emphasized that all hospitals need to plan and prepare in advance for mass casualty events. Anesthesiology departments must be involved in disaster planning and training to help to decrease the amount of chaos that even

in the best of circumstances follows a mass casualty bombing incident.

Coordination and communication within the anesthesia department and with other physicians and departments are vital. Hospital triage and distribution are important, and attention must be given to the moderately injured patients because these seemingly stable patients might harbor life-threatening injuries. Only casualties with immediately life-threatening injuries should be brought to the operating room right away.

Anesthesiologists must be prepared for especially complex operations and procedures with patients who have sustained multiple injuries. Blunt, penetrating, and thermal injuries can be complicated by primary blast injuries, making the treatment of these casualties more complex and difficult. Primary blast injury to the lung must always be suspected because the implications can be lethal. Initiating positive-pressure mechanical ventilation can help and harm these patients at the same time. Specific consideration must be given to prevent and treat air embolism.

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