Chapter 9

THE MANAGEMENT OF PRIMARY BLAST INJURY

YANCY Y PHILLIPS 111, M.D., FCCP[*] AND JOAN T. ZAJTCHUK, M.D., FACS[**]

INTRODUCTION

ECHELONS OF CARE FOR CASUALTIES WHO HAVE PRIMARY BLAST INJURY
- First Echelon of Care
- Second Echelon of Care

PRIMARY BLAST INJURY TO THE RESPIRATORY SYSTEM
- Initial Physical Examination and Triage
- Initiation of Life Support
- Evacuation
- Definitive Physical Examination
- Diagnostic Screening Procedures
- Stabilization and Life Support
- Treatment

AIR EMBOLISM IN PRIMARY BLAST INJURY
- **Initial Physical Examination and Triage**
- Initiation of Life Support
- Evacuation
- Stabilization and Life Support
- Definitive Physical Examination
- Diagnostic Screening Procedures
- Treatment of Air Emboli

PRIMARY BLAST INJURY TO THE GASTROINTESTINAL TRACT
- Initial Physical Examination and Triage
- Initiation of Life Support
- **Evacuation**
- Definitive Physical Examination
- Diagnostic Screening Procedures
- Treatment of Gastrointestinal Injury

PRIMARY BLAST INJURY TO THE AUDITORY SYSTEM
- Initial Physical Examination, Triage, and Evacuation
- Definitive Physical Examination
- Diagnostic Screening Procedures
- Treatment

SUMMARY

*Lieutenant Colonel, United States Army; Chief, Pulmonary and Critical Care Medicine Service, Walter Reed Army Medical Center, Washington, D.C. 20307 5001, and Consultant to the in Pulmonary Medicine and Respiratory Therapy

**Colonel, United States Army; Chairman, Division of Otolaryngology, Uniformed Services University of the Health Sciences, Bethesda, Maryland 203074799

INTRODUCTION

Explosions are ubiquitous in modern warfare. Although fragmentation and thermal effects cause by far the most combat injuries, the detonation of explosive munitions can create pressure waves that are powerful enough to injure the internal organs of casualties who are directly exposed to them. This injury—called primary blast injury (PBI)—may debilitate or kill the casualty by causing severe damage to the gas-containing organs of the body while leaving no external trace of injury. The damage caused by PBI is a kind of *barotrauma,* an injury caused by a local pressure differential. It results from the interaction between the passing blast wave and the body tissues, which creates an imbalance between the ambient pressure and the pressure within the affected cavity of the body. The symptoms and treatment of PBI will depend upon which organ has been affected.

Victims of an open-air blast will usually also have penetrating or nonpenetrating secondary blast injuries from fragments or objects that have been hurled through the air from the force of the blast. These wounds do not differ from classic ballistic wounds that are caused by bullets or fragments from conventional explosive munitions.

Tertiary blast injury refers to the blunt trauma that can occur when the victim is bodily lifted and thrown against a nearby structure by the force of either the blast wave itself or the venting of the blast wind or combustion gases through a constricted opening. Tertiary blast injuries may complicate primary and secondary blast injuries, especially when nuclear and larger conventional weapons are used.

The true incidence of significant PBI is unknown, perhaps in part because it is difficult to diagnose a problem that one is not prepared to recognize. Medical officers who wait for a patient who exhibits classic,

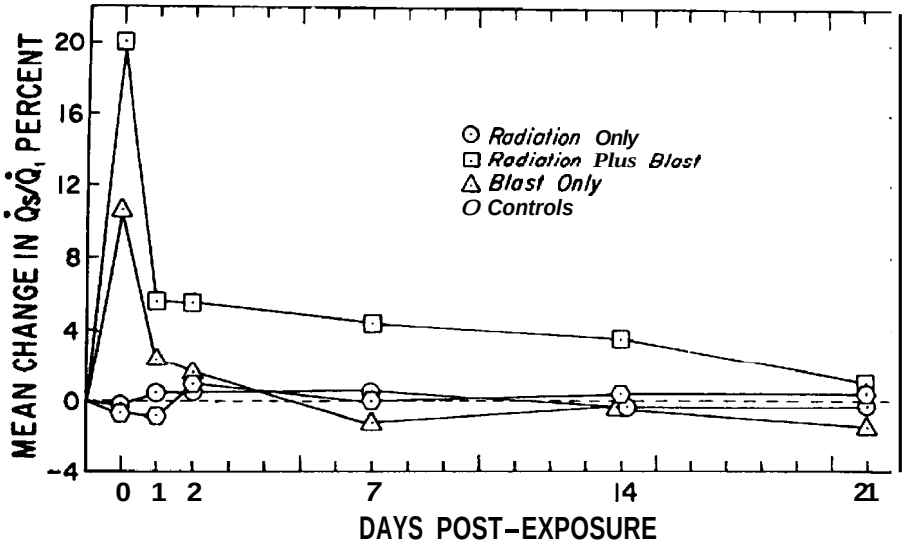


Fig. 9-1. Researchers studied the effect on the ability of sheep lungs to transfer oxygen over time following radiation only (ooo), blast only (A A A), or radiation plus blast (□□□). Controls are indicated on the graph by hexagons. The animals that were subjected to both insults showed both the greatest decrement in oxygenation and the longest-lasting effects, even though the radiation alone had no detectable effect on pulmonary gas exchange. The development and resolution of lung injury is measured by venous admixture (shunt fraction or Qs/Qt) following injury.
Source: Reference 1

pure PBT—that is, who has neurological deficits and respiratory failure without so much as a scratch—will miss most of the PBI cases that they can expect to encounter in a trauma-care environment. An explosion might result in a mix of traumatic amputations of limbs and penetrating fragment injuries, as well as PBI to the casualties' abdomens, lungs, or ears. The thermal pulse from a detonation may burn exposed skin, or secondary fires may be started by the detonation and more serious burns may be suffered. Smoke and fumes from fires contain toxic chemicals and may cause inhalation injury. Victims may also be crushed in the collapse of a building.

Given such dramatic combined injuries, the primary blast component may be hidden but nevertheless significant, and the physician must be aware of the possibility of such occult injury lest it further complicate the patient's care. In addition, the effects of combined injuries may be synergistic rather than additive. For example, radiation injury will combine with blast or burn injury to cause much more severe and long-lasting damage than would be expected from each of the injuries individually (Figure 9-1).[1]

The classic case of pure PBI is usually seen in a casualty who has been exposed to an underwater detonation. Water transmits blast waves more efficiently than air does; that is, the blast waves' effects do not diminish over distance in water as much as they do over distance in air. An explosion in water has a lethal area that is approximately nine times greater than the lethal area of an airburst using the same amount of explosive.[2] At the same time, water greatly reduces the effective range of any fragments that might be propelled from the detonation site.

In contrast, a casualty who presents with pure PBI from an open-air explosion is likely to have been very close to the explosion but not close enough to have been dismembered by fragments from the exploding device. Pure PBI, particularly to the ear, is more likely when the detonation occurs in a closed space (such as an armored fighting vehicle or a room in a building, in which the blast may reverberate off the walls), or when a special enhanced-blast munition is used.

Pure PBI may be severe when the exposures are repeated, even if the individual blasts are of relatively low intensity. Experimental animals that received repeated blast exposures (commonly called *multiple blast*) had the same types of primary blast lesions that were produced in animals exposed to single blasts.[3] However, repeated exposures to blast *(a)* increased the severity of lesions over those produced by a single blast of the same magnitude, and *(b)* decreased the threshold for injury. These additive effects may be due to the fatigue factor (see Chapter Seven), which describes the lower stress requirement for tissue failure after repeated exposures, and may be important to operators of artillery weapons, for whom the effects of repeated low-level blasts have significant occupational-health implications.

This chapter will focus specifically on PBI. The first section will briefly outline the procedural aspects of the blast casualty's movement through the military health-care system, with particular attention to triage, stabilization, and evacuation. The remaining sections will focus on the most serious manifestations of PBI: *(a)* damage to the respiratory system, *(b)* the production of air emboli, *(c)* gastrointestinal injury, and *(d)* damage to the auditory system. These effects will be discussed in the context of their management at the first level of care at which an evaluative or treatment measure may be performed, although the reader should understand that, under certain conditions, the evaluations may have to be repeated or conducted in greater detail at a higher echelon of care.

ECHELONS OF CARE FOR CASUALTIES WHO HAVE PRIMARY BLAST INJURY

The medical officer is most likely to see PBI when many casualties (some with combined injuries) have survived a civilian blast catastrophe, a terrorist bombing, or a military action. Such a mass-casualty incident will almost certainly cause confusion and chaos. Some of the routine procedures that medical personnel are trained to perform may be impossible to carry out in an ideal sequence under such conditions, and so a certain amount of procedural overlap is built into the evacuation and evaluation system to ensure that important (but perhaps latent) aspects of injury are not overlooked.

The medical evacuation system is designed to move casualties from the site of injury to a definitive-care facility as rapidly as possible. Because the casualties' physical status and the conditions of the battle may both fluctuate, the means of transportation and the health-care destinations may vary as well. Triage (the sorting of casualties according to the severity of their injuries) is done at every level of care.

First Echelon of Care

The first echelon of care occurs on the battlefield, and is usually provided to a casualty by a buddy, a trained combat lifesaver (that is, a member of the unit

who has had supplementary first-aid training), a medic, or medics and battalion surgeons at the battalion aid station. First-echelon treatment is limited to essential emergency care, and can range from minimal first-aid interventions that allow a slightly injured soldier to return to duty right away, to crucial stabilizing measures (such as establishing an airway, controlling a hemorrhage, and administering intravenous fluids) that are intended to keep the casualty alive during evacuation to the appropriate care facility. When evaluating blast casualties, medical personnel need to ascertain *(a)* what, if anything, can be done immediately to save the casualty's life and limbs on the battlefield, *(b)* whether the **casualty** needs to be evacuated, and *(c)* how the casualty should be transported to the next level of care.

Initial Physical Examination and Triage. Blast casualties may have PBI in several anatomical sites and in any degree of severity. They should be evaluated according to normal triage standards. In the military, four triage categories are generally used: *(a) immediate,* which includes casualties who have severe, life-threatening injuries but are likely to survive if they receive the appropriate lifesaving treatment, *(b) delayed,* which includes casualties who can tolerate a delay prior to surgery or other treatment without suffering further damage, *(c) minimal,* which includes casualties who have superficial injuries that can be treated by first-aid procedures, and *(d) expectant,* which includes casualties who are not expected to survive no matter how much medical treatment they receive, or who would not benefit from the limited medical resources available.

Although the care of blast casualties at this echelon will usually center on secondary blast injuries (such as fractures, penetrating wounds, lacerations, and burns), the medical officer needs to be particularly alert for the more subtle signs of PBI. If circumstances permit, medical personnel should carefully examine the casualty for signs of contusion or penetrating wounds.[3] Sometimes, a sentinel (or associated) injury, which may be as dramatic as a traumatic amputation or as relatively minor as a temporary hearing loss, will indicate that the casualty may also have significant PBI. Because some of the most serious manifestations of PBI have few or no overt signs, taking certain preventive measures at this stage may save the life of a blast casualty who is apparently less severely injured.

Triage decisions and the amount of time that can be devoted to them depend upon the nature of the blast incident itself. *Overtriage* may occur when casualties who should be admitted to the MTF for further observation or treatment are instead discharged to duty. *Undertriage* may occur when casualties who have relatively minor injuries are admitted to the MTF for observation, a level of caution that might not have a great effect on a large civilian medical facility, but one that would severely strain available medical resources and result in a significant loss of fighting strength were it to occur in a military mass-casualty situation. During wartime, the military physician or medic may have to return a soldier to combat when *(a)* the underlying severity of the blast injury is not objectively evaluable (that is, radiographic and laboratory screens are unavailable), and *(b)* the risk that a blast lesion would develop later is only problematic (that is, soldiers with only mild symptoms consistent with PBI may be returned to combat despite the theoretical consideration that such activity may worsen the PBI).

Initiation of Life Support. Medical personnel must ensure that the casualty is hemodynamically stable and that the airway is patent. In the PBI casualty, the life-threatening injuries that require immediate stabilization are usually caused by respiratory damage or by blood loss from gastrointestinal hemorrhage.

Respiratory support and mechanical ventilation will be discussed in the section of this chapter that deals with PBI to the respiratory system.

Volume replacement will be discussed in the section that deals with PBI to the gastrointestinal tract, although the reader should understand that a casualty can be hypotensive for many reasons other than blood loss from an abdominal hemorrhage.

The casualty will be stabilized and life-support measures will be continued at the second echelon of care, if necessary.

Establishing a Medical Record. The medic is also responsible for beginning the blast casualty's medical evacuation record, although the realities of the battlefield will determine how complete it will be, or even whether it is done at all. Unlike civilian terrorist bombings, in which the undivided attention of a medical team can be focused on blast casualties, an explosion in combat is unlikely to be an isolated event relatively near an urban medical facility. Even if the medic—who may be working alone—has the opportunity to glean blast-related information, the small size of the medical-record card limits the amount of information that can be conveyed. This limitation is particularly unfortunate in blast incidents, because the casualty's buddies may have noticed important details that the casualty (even if conscious) may have missed. Nevertheless, as soon as possible after the casualty is stabilized and if circumstances permit, medical personnel ideally should try to determine the following:

- What type of ordnance was used and how large was the explosion?

- Where was the casualty located with respect to the blast?
- Did the blast occur inside an enclosed space such as a room or vehicle?
- What was the casualty's activity after exposure?
- Were there fires or fumes that might lead to an inhalation injury?
- What was the orientation of the casualty's head and body to the blast?

Evacuation. PBI may evacuation of these casualties from the battlefield. The casualty's body position, for example, can affect the severity of some primary blast effects, such as potentially lethal air embolism.[4,5,6]

Because physical exertion after blast exposure can exacerbate the severity of PBI, victims of an explosion should minimize physical activity and, if they are experiencing respiratory distress, should be carried from the battlefield by litter rather than be allowed to leave under their own power.[4,7,8] In 11, for example, some blast casualties initially appeared to be well, but died after vigorous exercise following their blast exposure **Their** comrades, who were initially more severely injured and too ill to move, remained sedentary and survived.[8] In experimental studies, rats were either kept quiet or forced to swim to exhaustion afterbeing exposed to blast.[9] The blast alone killed 30% of the sedentary animals, but those rats that were forced to swim to exhaustion 1hour after exposure had a 70%mortality. When the swimming was delayed for 4 hours, the rats' mortality was 40%. Although the exhausting exercise seemed to increase the rats' lung injury and mortality, **a** period of rest bcforc cxcrtion appeared to significantly alter their susceptibility to further injury.

Certain manifestations of PBI—particularly those that involve the respiratory, circulatory, and gastrointestinal systems—are known to be more dangerous when the casualty is evacuated by air. Medical personnel should be aware that even a short helicopter flight might jeopardize the stability of a blast casualty, and should take the precautions that are detailed in the system-specific sections of this chapter. The aircraft should fly at the lowest practical altitude. If possible, blast casualties should avoid long-distance, high-altitude flights for several days.

Second Echelon of Care

At the second echelon, which is the medical company of the brigade or division, the casualty will most likely be seen by a military physician or a physician's assistant, who will glean more details of the casualty's blast-exposure history, if **possible.** If circumstances permit, routine laboratory tests will be done at this stage.

Stabilization and Life Support. Stabilization measures, such as volume replacement, will be continued or initiated if needed. Personnel at this level of care can monitor the casualty's oxygenation and replace blood volume with intravenous solutions, activities that may have been beyond the scope of the medic. Second-echelon facilities may also have the equipment to vcntilation.

Definitive Physical Examination. The blast casualty will receive a thorough physical examination, and medical personnel should look for certain sentinel signs, such as a ruptured tympanic membrane, hypopharyngeal contusions, hemoptysis in the absence of external chest trauma, or subcutaneous emphysema, Aspects of the examination that focus on particular anatomical areas will be discussed in those sections of the chapter.

Diagnostic Screening. **As** soon as the blast casualty is hemodynamically stable, medical personnel should take a chest roentgenogram, regardless of the casualty's symptoms. Failure to do *so* can **be** disastrous. In one case, a soldier who was injured by a mine explosion had a bilateral tympanic-membrane rupture and abdominal pain.[10] He was rushed to surgery, during which military physicians found diffuse intestinal petechiae and a subcapsular splenic hematoma. The surgeons had failed to obtain a chest roentgenogram before operating, however, and the patient—who also had an unsuspected pulmonary contusion from the blast—rapidly deteriorated into a state of respiratory insufficiency.

If the casualty has complications but is stable enough both to cooperate and to be transported to an MTF that has radiologic facilities, medical personnel may order a computed axial tomography (CAT) scan of the chest, abdomen, or head.

Serial hemoglobin determinations are important guides to blood replacement in all casualties who have severe bleeding, including those with hemorrhage into the lungs or gastrointestinal tract from PBI.

Most studies add little to the evaluation of blast-injured patients. Researchers have used animals to evaluate potential PBI markers, including a multichannel blood-profile chemistry analysis." Both sedentary and exercise-stressedanimals were exposed to blast intensities that ranged from trivial to LD, (that is, a lethal dose, or fatal injury, for 1%of cases), and their blood was drawn prior to and 90 minutes after exposure. Unfortunately, none of the putative markers proved to be useful as early indica**tors** of either the presence or the degree of blast injury

in any organ system

However, serial monitoring of hematological and biochemical parameters may be useful in following the complicated medical course of any seriously injured patient. In one report from Israel, for example, four out of five patients with PBI to the lung had significant hypokalemia within a few hours of injury.[12] The authors speculated that stress-induced catecholamine release was responsible and were concerned that the electrolyte disturbance might cause or worsen arrhythmias. Three of the patients went on to develop a disseminated intravascular coagulation syndrome with low platelet counts and prolonged coagulation times. The coagulopathies responded to replacement therapy and did not complicate the clinical course.

Treatment. Some manifestations of PBI will resolve on their own, or will require only a continuation of stabilization measures until the casualty is out of danger. Other manifestations will require immediate surgical intervention, or may call for sophisticated equipment that would not be available at the lower echelons of care. In addition, some manifestations of PBI may have long-term sequelae. The following sections will discuss the treatment of PBI as it appears in the most vulnerable systems of the body.

Unless otherwise specified, diagnostic and therapeutic interventions to be discussed require the resources found in a third-echelon MTF or in a level-one civilian trauma service.

PRIMARY **BLAST** INJURY TO THE RESPIRATORY SYSTEM

The lungs are the vital organs that are most vulnerable to PBI. Damage to the lungs may include *(a)* pulmonary contusions, with or without lacerations, *(b)* pneumothorax, *(c)* traumatic lung cysts, *(d)* interstitial emphysema, *(e)* pneumomediastinum, or *(f)* subcutaneous emphysema. The term *blast lung* is commonly used clinically to refer to PBI to the respiratory tract with pulmonary contusion and respiratory insufficiency, with or without extravasation of air (Table 9-1). Casualties who have pulmonary PBI will experience dyspnea, but those who do not have extrapulmonary air will not usually experience chest pain.

Pulmonary contusions impair gas exchange at the alveolar level. The degree of respiratory insufficiency will depend on the degree of the hemorrhage.[13,14] These contusions develop, stabilize, and resolve relatively rapidly (Figure 9-2). In humans, roentgenographic evidence of lung contusion may appear only hours after exposure; these contusions may resolve in about 1 week.[13,15,16] In animal studies, rats had significant resolution of blast-induced pulmonary hemorrhages after only 24 hours, although there were small residual increases in lung weight after a week (Figure 9-3).[17] The symptoms of significant pulmonary contusion are likely to include *(a)* cough, *(b)* hemoptysis, or *(c)* dyspnea, resulting from widespread alveolar disruption with hemorrhage or pneumothorax or both.

The blood in a pulmonary contusion usually stays within the lung, but if the contusion is complicated by parenchymal laceration, bleeding may occur not only within the parenchyma but also into the pleural space, creating a hemothorax.

Pneumothorax, the most serious form of intrathoracic barotrauma, is the presence of air in the pleural cavity. This extrapleural air interferes with the normal expansion of the lung that occurs when the downward movement of the diaphragm creates negative pressure in the chest cavity. In pneumothorax, the negative pressure acts instead upon the extrapleural air, leaving the lung in its collapsed (expiration) position and thus soon compromising gas exchange. The symptoms of pneumothorax may include *(a)* dyspnea, *(b)* chest pain on one or both sides without signs of external injury, and *(c)* cough. Even more dramatically, the pressure of the air that is trapped in the pleural cavity during expiration can increase so much that it displaces the mediastinal contents, thus decreasing the casualty's venous return to the point of cardiovascular collapse. This condition is called a tension pneumothorax. The casualty will be hypotensive and may exhibit other symptoms of cardiovascular distress, including tachycardia and diaphoresis. A tension pneumothorax can be immediately life threatening.

A blast casualty may have a hemopneumothorax if both blood and air are in the pleural space, and might experience not only the respiratory-distress symptoms of pneumothorax, but also hemoptysis and cardiovascular collapse.

Air may be forced from the alveoli and airways into the interstitium of the lung either as traumatic lung cysts or as interstitial emphysema.[13] These injuries have no overt signs or symptoms, and there is no information available regarding their resolution. The large parenchymal air cysts may also form in casualties who are receiving mechanical ventilation and in whom they have a high risk of rupturing, causing pneumothorax. Interstitial emphysema occurs when air dissects from the airway along bronchial walls.

TABLE *9-1*

SYMPTOMS, CLINICAL SIGNS, AND FINDINGS OF PRIMARY BLAST INJURY OF THE RESPIRATORY SYSTEM

Findings	Signs	Symptoms
Nonspecific findings (Common to all primary **blast** injury)	Cyanosis Tachypnea	Chest pain Dyspnea
Parenchymal Lung Injury (Contusion)	Crackles (rales) sounds Dullness to percussion Tachypnea	Hemoptysis
Pulmonary Barotrauma (Pneumothorax) (Pneumomediastinum)	Diminished breath sounds* Increased resonance* Retrnsternal crunch Subcutaneous crepitus Tracheal deviation** or mediastinal shift**	Cardiovascular collapse
Pulmonary Laceration (Hemopneumothorax)	Same as pulmonary barotrauma and dullness to percussion*	Hemoptysis Cardiovascular collapse

* On **side of collapse**
**Awayfrom the side of collapse

Pneumomediastinum and subcutaneous emphysema can occur when interstitial emphysema decompresses into the mediastinurn or the subcutaneous tissue space. These events usually will not cause symptoms and will be detected on radiographs or by the presence of subcutaneous crepitus on physical examination. Neither pneumomediastinum nor subcutaneous emphysema by themselves pose a significant hazard.

Respiratory failure may occur 24-48 hours after blast exposure, but if it does occur that late, it is unlikely to be caused solely by PBI.[18,19] Instead, a combination of blast effects, inhalation injury, massive tissue injury, and transfusion therapy may result in a condition called adult respiratory distress syndrome (ARDS).[18,20,21,22] **A** discussion of ARDS is beyond the scope of this chapter, and will be found in the TMM textbook *Anesthesia and Critical Care.*

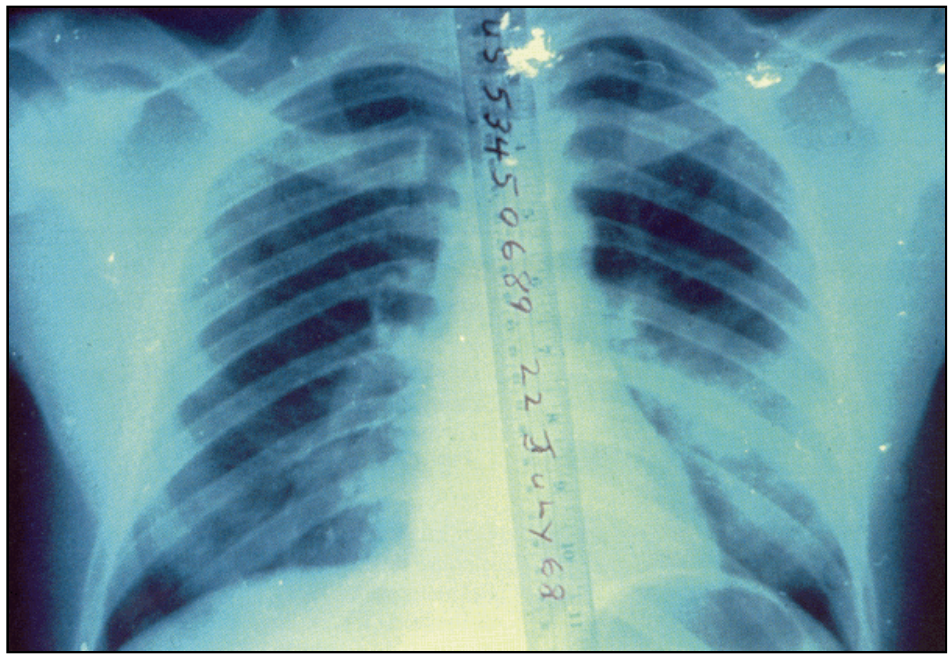
Table 9-2 gives the incidence of respiratory symptoms and findings compiled from two reports of underwater- and air-blast casualties.[13,16]

Initial Yhysical hxamination and Triage

In general, a casualty who has pulmonary PBI will exhibit signs that may include tachypnea, hemoptysis, tachycardia, cyanosis, or an inability to carry on a conversation.[7,13,23,24] Comprehensive emergency care is crucial. The first-echelon medical personnel will not have the opportunity or resources to examine the blast casualty definitively for PBI to the lungs.

Casualties who have *(a)* asphyxia, *(b)* simple or tension pneumothorax, *(c)* cyanosis and extreme dyspnea, *(d)* upper-airway compromise, or *(e)* hypotension from any cause should be placed in the immediate triage category. They should receive emergency stabilization measures and be transported directly to the appropriate echelon of care as soon as possible.

Casualties who *(a)* exhibit lesser degrees of respiratory distress (such as a respiratory rate below 30 breaths per minute), *(b)* are able to carry on a conversation, and *(c)* are hemodynamically stable are in the



Pig. 9-2. This chest roentgenogram of a soldier who was injured by a bomb blast shows bilateral infiltrates from pulmonary contusions. The patient survived without sequelae.
Source: Wound Data and Munitions Effectiveness Team

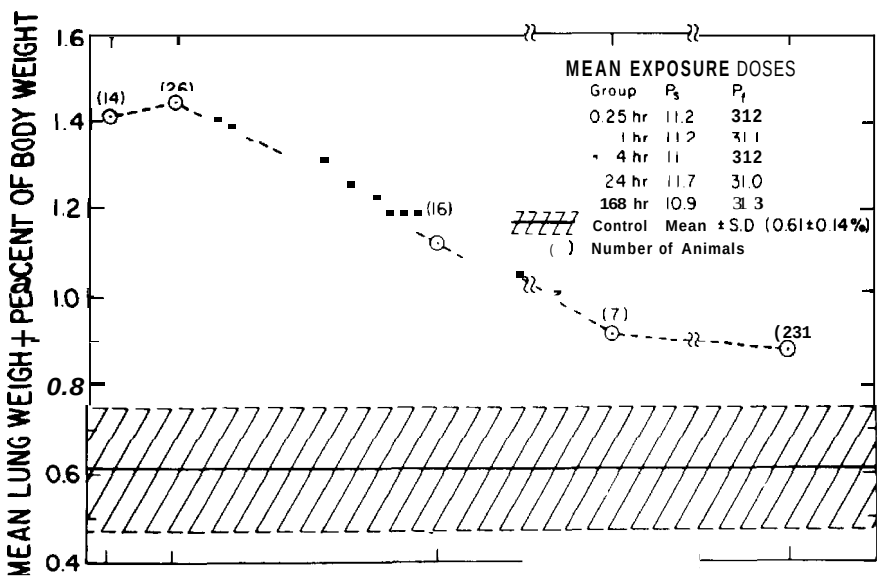


Fig. 9-3. The development and resolution of pulmonary injury in rodents that were exposed to sublethal blast can be measured by their lung weights. Most of the increase in lung weight is secondary to parenchymal hemorrhage.
Source: Reference 18

TABLE 9-2

DISTRIBUTION OF PULMONARY SYMPTOMS AND CLINICAL FINDINGS IN SURVIVORS OF UNDERWATER AND AIR BLASTS,

Clinical Symptoms and Findings	Percentage**
Pulmonary symptoms	
Hemoptysis	55
Dyspnea	38
Chest Pain	22
Pulmonary findings	
Parenchymal infiltrates	84
Crackles (rales)	40
Hemothorax	27
Pneumomediastinum	27
Pneumothorax	4

*Hospitalized survivors of major underwater blast (N=43) and air blast (N=2)

**See Table 9-5 for abdominal injury data from the same incidents. The percent total mortality from pulmonary and abdominal blast injuries was 16%.

delayed triage category, and should be evacuated as soon as the emergency cases have been stabilized and evacuated.

Initiation of Life Support

Establishing an Airway. For blast casualties who are in pulinonary distress, the most **crucial** emergency measure is the establishment of a patent airway. Inserting a simple oral or nasal airway may suffice, but casualties who have extreme respiratory embarrassment should be intubated endotracheally to handle massive hemoptysis and in anticipation of mechanical ventilatory support.

Inserting Chest Tubes. For either a tension pneumothorax or a simple pneumothorax that has no accompanying contusion or evidence of cardiovascular invulvement, an immediate **tube** thoracostomy **is** the definitive treatment. The extrapleural air will be evacuated through the tube, allowing the lung to reinflate. This emergency measure may save the casualty's life.

If the casualty has a hemopneumothorax, a foamy mixture of both blood and air will be evacuated through the tube, indicating that a pulmonary laceration exists. The amount of blood that is evacuated should be monitored, but hemothorax from blast is rarely severe enough to lead to hypotension.

Hypotension and Volume Replacement. Hypotension in blast casualties can be ascribed to several different causes, including *(a)* blood loss from secondary blast injuries or other wounds, *(b)* blood loss from a gastrointestinal hemorrhage or solid-organ rupture, *(c)* the sequelae of air embolism, or *(d)* vagal reflexes. A hypotensive casualty must quickly receive sufficient volume replacement to bring the pulse and blood pressure back within normal limits. However, blast casualties who have pulmonary injuries have an increased **risk** of pulinonary edema when they receive excessive volume replacement.

Because the transudation of hypooncotic fluid is more likely in an injured lung, medical personnel should consider replacing the casualty's lost fluids with blood or a colloid solution rather than with a crystalloid solution.[15,19,22,25,26] Researchers found that the rapid infusion of large volumes of crystalloid solution in dogs that had unilateral lung contusions resulted in a greater impairment of gas exchange and an increase in the weight of the uninjured lung, as compared to infusions of smaller volumes.[27] A German study found less lung damage in pigs that were given furosemide prior to blast exposure, implying that low pulmonary vascular pressures may offer some protection from intrapulmonary hemorrhage or edema after blast.[28] Chinese researchers reported less lung hemorrhage in hypovolemic dogs that had been acutely depleted of 40% of their blood volume before being exposed to a blast, although they did not mention mortality or the nature of the resuscitation protocol after injury.[29]

In casualties with combined burn and relatively mild pulmonary blast injuries, fluids **can** be replenished according to standard infusion formulae for burn casualties. In Chinese experiments, dogs were given second-degree burns over 40% of their bodies and then were exposed to a large but sublethal blast.[30] To simulate evacuation time from the battlefield, resuscitation began 8 hours after injury. The animals were resuscitated by fluid infusions that were based on a formula of 0.5 ml of colloid and 1.0–1.5 ml of crystalloid per kilogram of body weight per percent of burned body surface area per day. The

injuries did not become any worse than what had been expected from the blast effects alone.

Evacuation

Because changes in atmospheric pressure can seriously affect casualties who have suffered respiratory barotrauma, blast casualties have special needs during aeromedical evacuation.

Oxygen Adequacy. Oxygenation problems at air level will be worse at higher altitudes. Both the casualty's level of arterial oxygen (pO_2) and a hematocrit below 30% are other indicators of evacuation risk. If the pO_2 level is below 60 mm Hg, the casualty's amount of dissolved oxygen may be too low to allow safe evacuation. Medical personnel should be aware, however, that casualties may have dangerously low levels of arterial pO_2 even at sea level without showing tachypnea, cyanosis, or other clinical signs of hypoxia. This dangerous situation can become even more so at 35,000 feet, whcrc thc aircraft cabin is pressurized to the equivalent of 8,000 feet and the casualty's alveolar air pO_2 may be one-third less than it was at sea level.

If the casualty develops a respiratory emergency during the evacuation, medical personnel should opt for endotracheal intubation, which is safer, quicker, and more easily tolerated by the casualty than a tracheostomy would be. If an endotracheal tube is used promptly, a later tracheostomy may be unnecessary.

Chest-Tube Cure. Normally, patients whu have chest tubes should not be evacuated by air with the tubes in place, nor should they be evacuated within **72 hours** after remnval of the tube.

A chest roentgenogram must demonstrate the absence of pneumothorax just before the casualty is transported.[31] However, blast casualties who have pneumothorax must sometimes be evacuated quickly from the battlefield. Because these casualties have a high risk of tension pneumothorax with subsequent cardiovascular collapse, they should receive a tube thoracostomy before being transported — especially if evacuation is by air, regardless of the altitude and distance of the flight. The chest tubes may bc lcft in position during evacuation but should be equipped with functioning valves (such as the Heimlich valve). The aircraft should be pressurized to ground level if such casualties will be aboard.

Casualties who require mechanical-ventilatory assistance should not be evacuated by air.

Definitive Physical Examination

Because pulmonary barotrauma may immcdiately threaten the casualty's ability to breathe, first-echelon medical personnel will have automatically addressed many of the more serious manifestations of PBI simply by stabilizing the casualty. Second-echelon medical personnel will be able to give the blast casualty a thorough physical examination and should be able to make a directive diagnosis. Examiners should be particularly alert to sentinel injuries that may indicate more serious covert trauma, as well as to those casualties who may be relatively asymptomatic but are at risk for late-developing signs of PBI.

Hypopharynx. The medical officer should examine the casualty's hypopharynx for petechiae or ecchymoses around the larynx, vocal cords, or other hypopharyngeal structures. These small hemorrhages may be associated with significant PBI to the lung.[2,32]

Lungs. Of the organs in the thorax and the abdomen, the lungs are the most vulnerable to PBI, and the examining physician should focus attention on them. The signs of pulmonary PBI are virtually identical to those of pulmonary occur after blunt chest trauma in motor-vehicle accidents, except that they rarely include rib fractures or aortic and cardiac injuries.[7,13,33,34]

The contused lung will present as a unilateral or bilateral alveolar-filling defect, similar to a pneumonia (Figure 9-2). The medical officer may find dullness to percussion in the presence of crackles or rales. Tachypnea is a common finding; in one study, the average respiratory rate of four patients who had blast lung was 30 breaths per minute.[17] The casualty may also be cyanotic.

If thecontused lung islacerated, then a hemothorax may develop, exhibiting in brcnth sounds and dullness to percussion.

A casualty who has developed a pneumothorax that has not yet been treated may exhibit some of the following signs: *(a)* tracheal deviation from the midline, *(b)* increased resonance on the side of collapse when the chest is percussed, *(c)* diminished breath sounds on the side of the collapsed lung, *(d)* a retrosternal crunching from a pneumomediastinum, and *(e)* subcutaneous crepitation, which the examiner willnoteas a crackling sensation beneath the casualty's skin when palpated. If the pneumothorax has progressed into a tension pneumothorax, a shift of the cervical trachea from the midline will indicate that the mediastinal contents have shifted away from the side of collapse.

Occasionally, the blast casualty's abdominal complaints may distract the examining physician's attention from the presence of pulmonary PBI. For example, only three of twenty-seven survivors who had gastrointestinal injury from an underwater detonation pre-

scntcd with overt respiratory distress, but nineteen were ultimately found to have significant pulmonary compromise.[16] In World War II, naval surgeons suggested that lung damage might be the source of discomfort in an underwater-blast casualty whose pain was limited to the upper-abdominal area.[35]

Diagnostic Screening Procedures

Radiographsare the most useful diagnostic screens for casualties with pulmonary PDI, and some objective measure of respiratory function (such as an arterial blood gas or oximetry) is important. Routine laboratory studies, hnwever, are unlikely to he helpful in either diagnosing or gauging the severity of PBI.

Roentgenography. Because it often reveals a more significant injury than was clinically suspected, a roentgenogram should be taken of any blast casualty regardless of symptoms.[13,36] Routine roentgenographic examinations should include images of both the chest and abdomen, and should be examined carefully for evidence of barotrauma, which will complicate further aeromedical evacuation, mechanical ventilation, and any surgery that requires general anesthesia.

The progression of pulmonary contusion can be tracked by radiographic studies. In uncomplicated PBI, this injury develops quickly. For example, roentgenographic abnormalities were evident in eleven of twelve cases within 9 hours in one series, and within 4 hours of exposure in all fivepatients of another series.[13,15] In the absence of complications, the roentgenogram should be stable after a day and should improve gradually over the course of about a week.[13,15,16] Radiographic progression after 48 hours suggests another process, such as infection or posttraumatic respiratory distress syndrome.[15]

When pneumothorax is suspected, a chest roentgenogram should be taken immediately. If the casualty exhibits cardiovascular compromise and a tension pneumothorax is suspected, however, an emergency tube thoracostomy has a higher priority than a chest roentgenogram.

The chest film will obviously give the greatest information on lung injury, but it may also show *(a)* free air under the diaphragm (called *pneumoperitoneum*) from the rupture of hollow viscera or *(b)* a long lucent strip to the left of the trachea that may be the result of air extravasated from the esophagus.[13] Interstitial emphysema appears on a roentgenogram as long linear peribronchial lucencies.

The presence of extensive subcutaneous emphysema may make it difficult for the physician to appreciate underlying parenchymal injury, just as pulmonary contusion may make it difficult to identify interstitial air.[26] Conversely, extrapleural air may be difficult to recognize in roentgenograms against the background of parenchymal hemorrhage and subcutaneous and interstitial emphysema (Figure 9-4).[37]

Computed Axial Tomography. A CAT scan may reveal lung injuries that were not apparent on the plain roentgenographic film.[37] It is the most accurate technique for evaluating the lung parenchyma and pleural space (Figures 9-5, 9-6, and 9-7)—even more so than the magnetic resonance imager (MRI)—and is much more likely to be available for combat-casualty care.

A CAT scan can also be used to quantitate the extent of injury based on the amount of parenchyma involved. In one study, for example, all blunt-trauma patients whose CAT scans revealed that more than 28% of their lungs were involved with hemorrhage required ventilatory support.[14] Those who had 45% involvement required mechanical ventilation for an average of 7 days. No patient with less than 18% involvement required a ventilator.

Stabilization and Life **Support**

Oxygen monitoring and assisted-ventilation measures that were beyond the scope of first-echelon medical personnel can be initiated at a higher echelon.

Restoring Oxygen Adequacy. Even though symptoms of circulatory, respiratory, or neurological dysfunction may not appear immediately, the blast casualty is in a state of relative distress and will need increased oxygen. The adequacy of oxygenation should be evaluated on clinical grounds (Figure 9-8) and with the direct measurement of arterial oxygen saturation by means of either *(a)* pulse oximetry or *(b)* arterial blood gases.

Pulse oximetry (available at the third echelon) is a technique whereby the percentage of oxygenated hemoglobin is noninvasively monitored by infrared reflectometry of the vascular bed in the ear or in a finger. Ideally, pulse oximetry should be continuously monitored in a seriously injured casualty.

An analysis of arterial blood gases reveals the levels of oxygen and carbon dioxide in the blood, and thus gives the medical officer important information on respiratory sufficiency and acid-base status. In general, a blast casualty with uncomplicated PBI can be expected to have a level of arterial pCO_2 that is normal (35–40 mm Hg) or low (hypocarbia). Hypercarbia suggests that something other than PBI may be limiting spontaneous ventilation. For example, the casualty may have muscular or mechanical problems, such as *(a)* flail chest, *(b)* muscle weakness from chemical agents or metabolic derangements, *(c)* airway compromise, or *(d)* diaphragm rupture. An-

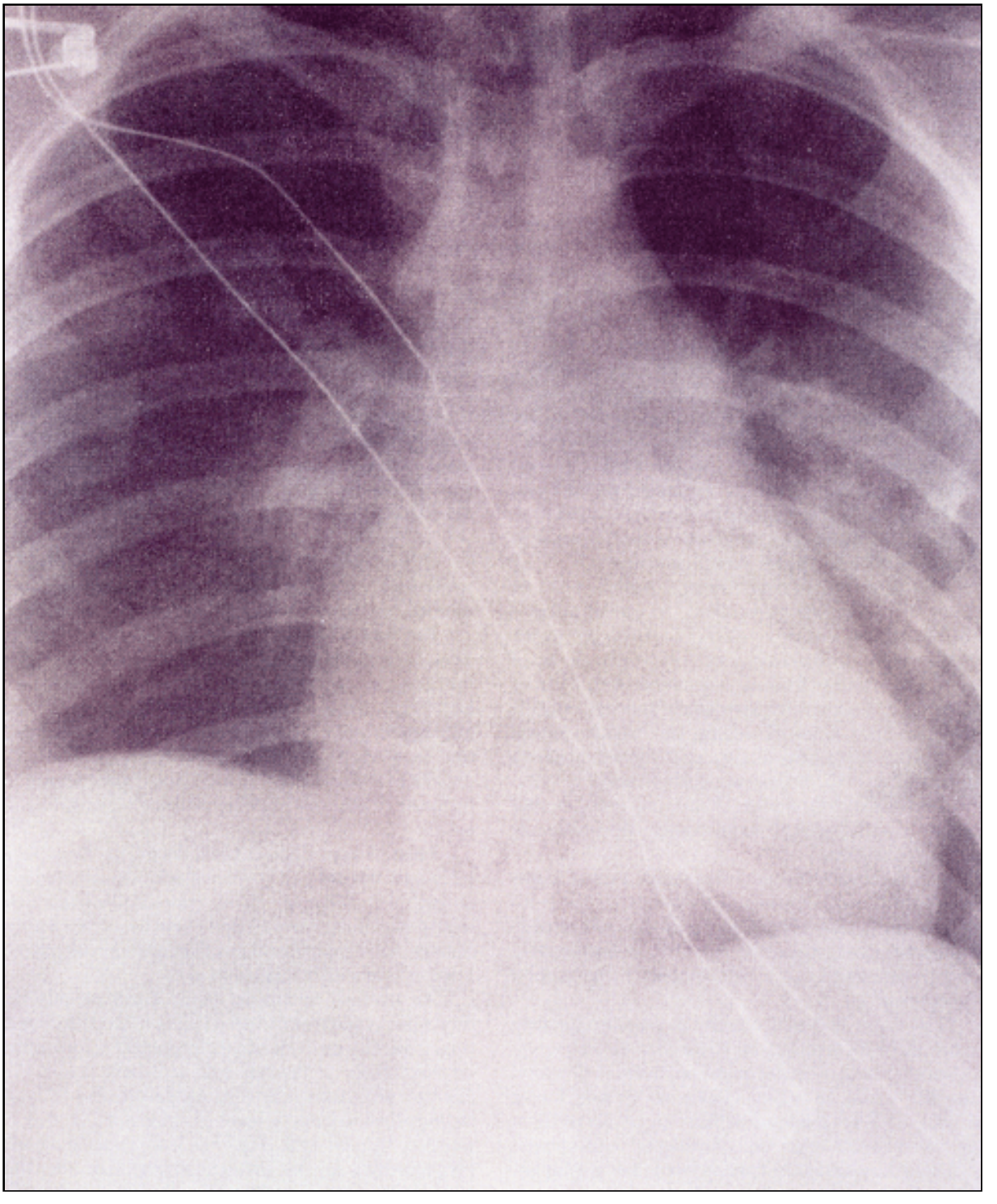


Fig. **9-4. A** chest roentgenogram of a patient in the supine position who had blunt thoracoabdominal trauma shows a patchy left midlung infiltrate consistent with pulmonary contusion but shows no evidence of barotrauma.
Source: With permission from reference **36**

Fig. 9-5. The CAT scan of the same **lungs** shown in Figure 9-4 not only reveals **the** air-space consolidation of pulmonary hemorrhage, but also shows unsuspected bilateral pneumothoraces. Arrows indicate the visceral pleural surfaces.
Source: With permission from reference 36

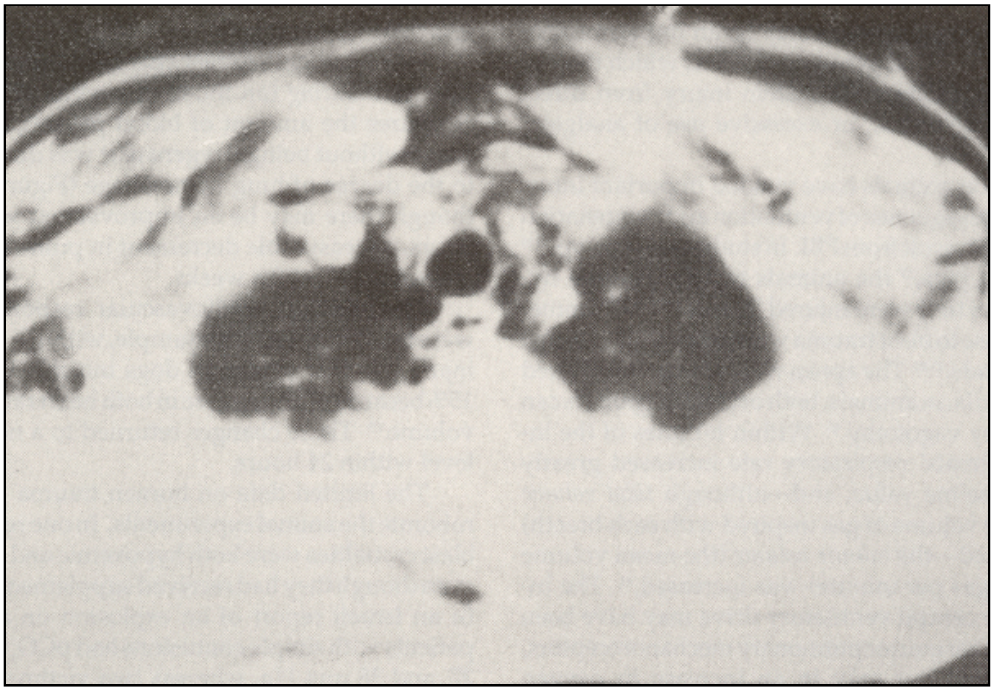


Fig. 9-6. This CAT scan of a blast casualty's thorax shows extensive subcutaneous emphysema that may obscure intrathoracic findings in a more routine roentgenographic evaluation.
Source: With permission from reference *25*

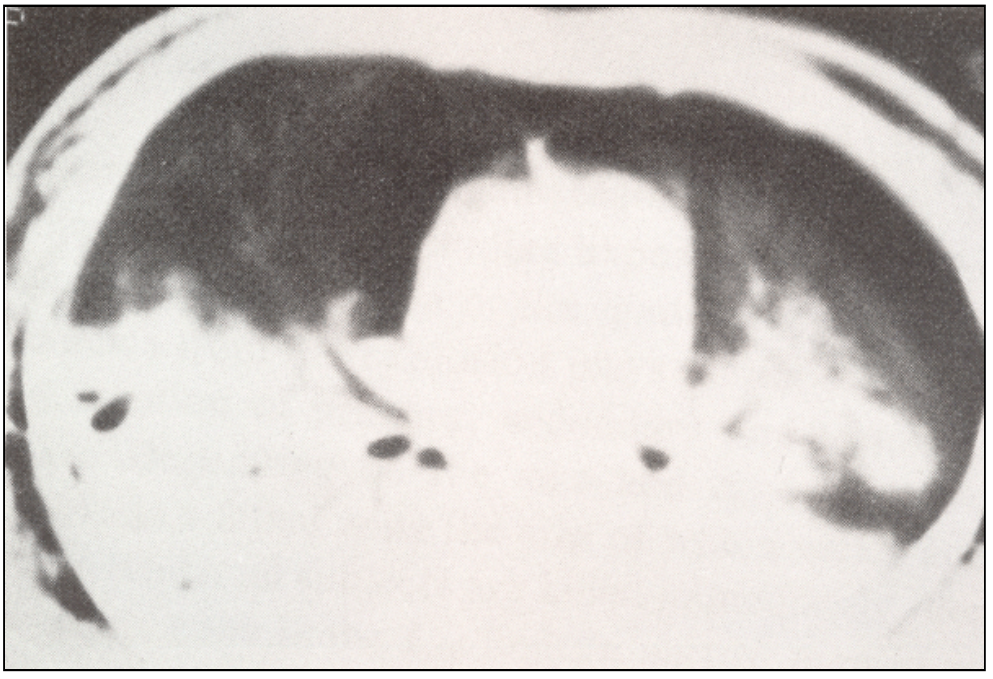


Fig. 9-7 This image reveals bilateral pneumothoraces, pneumomediastinum, and extensive parenchymal consolidation consistent with pulmonary contusion and hemorrhage.
Source: With **permission from reference** *25*

other cause of hypercarbia may be an impairment of the casualty's central ventilatory drive, which may be **due to a central nervous system injury from direct** trauma, air embolism, or excessive use of analgesic narcotics.

Studies on animals have yielded important information on the sequence of ventilation and oxygenation problems that result from PBI. In studies with sheep,[38,39] dogs,[29,38] and pigs,[33] the animals rapidly became hypoxemic after they were injured by blast. Their immediate response to blast trauma was apnea, which lasted **30–120** seconds.[33,40] The apnea was often accompanied **by bradycardia; in animals, both of thcsc rcsponscs can** be ablated by vagotomy.[40] Within minutes of the injury, the animals' respiratory rate increased greatly over the baseline value, and — although *tidal volume* (the average volume of gas inspired with each breath) was decreased — the *minute volume* (the mean volume of inspired gas per minute) was increased.[41] The increase in the central ventilatory drive may have been caused by either pain or pulmonary mechanoreceptors, and it resulted not only in an increase in oxygen consumption, but also in a decrease in the level of arterial pCO_2.[40] For example, twenty-seven of twenty-nine sheep that had pure PBI but were not in hypovolemic shock had arterial pCO_2 levels that were less than 40 mm Hg.[38]

These animal studies also found that the magni**tude of *shunting* (an index of oxygen efficiency that** measures the amount of blood passing through the lungs without being oxygenated) was directly related to the degree of lung hemorrhage (Figure 9-9).[39] **Al**though there may be an improvement within **24-48** hours, a measurable decrement in pulmonary gas exchange may last for weeks.

The animals' cardiovascular responses to blast were less dramatic. For example, within a few hours of injury, cardiac output in dogs increased only about **15% because of an increase in both heart rate and stroke** volume.[29] These changes returned to a near-baseline level within 24 hours.

The limited data on human trauma victims corroborate the animal experiments. In one report, all five blast casualties were both hypoxemic and hypocarbic, even though they had received supplemental oxygen.[15] In an Israeli report of an explosion on a bus, three patients with simple contusions had pCO_2 values of **26, 27,** and **38** mm Hg, whereas two victims with chest-wall injuries were hypercarbic and had pCO_2 values of 46 and 63 mm Hg.[12]

Mechanical Ventilation. Some casualties who have pulmonary contusions will require endotracheal

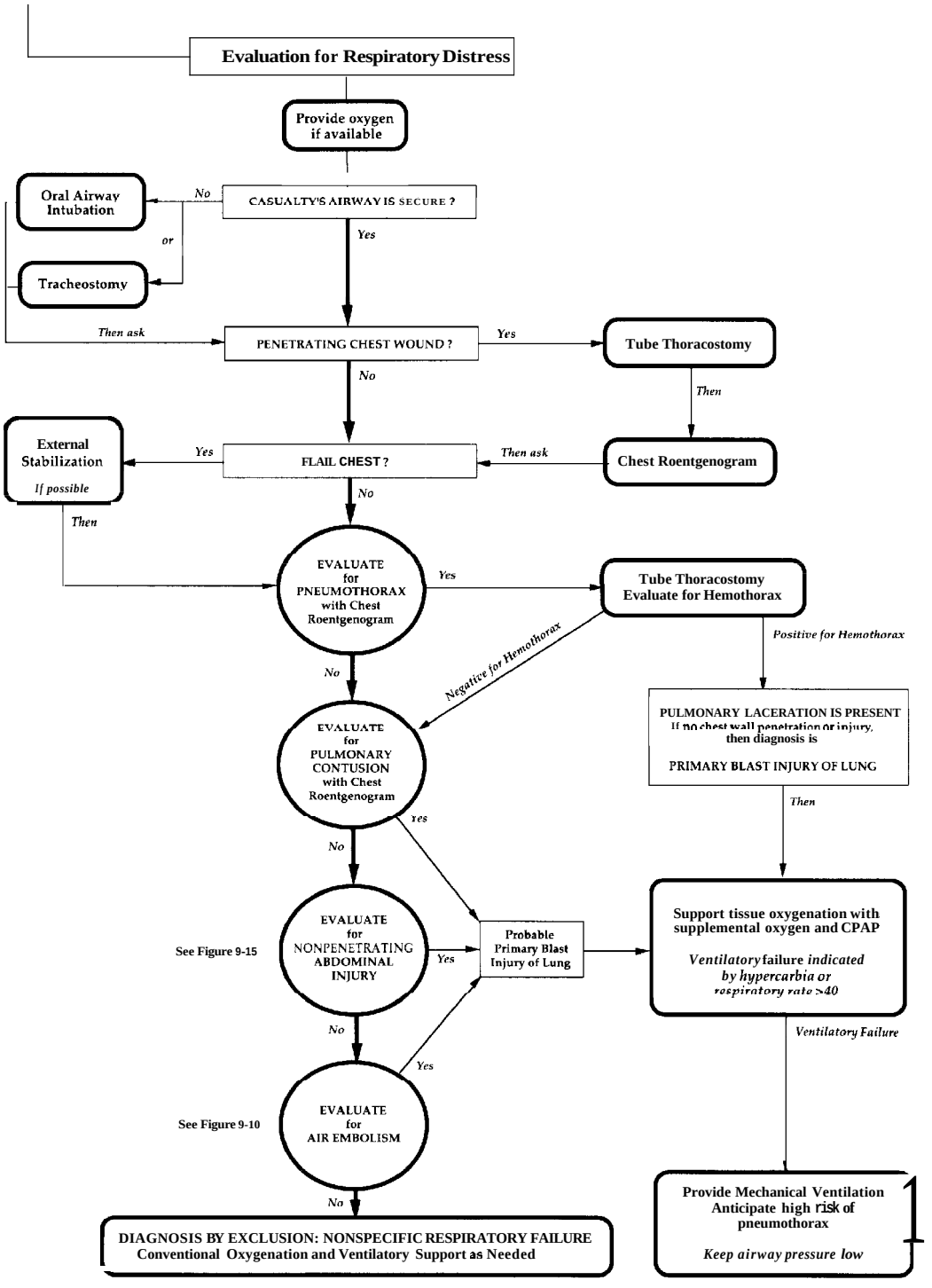
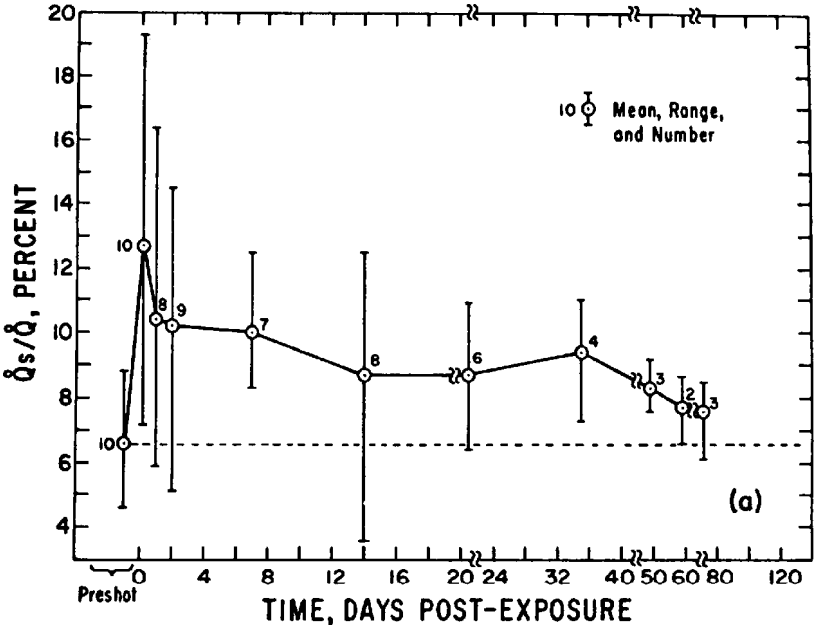
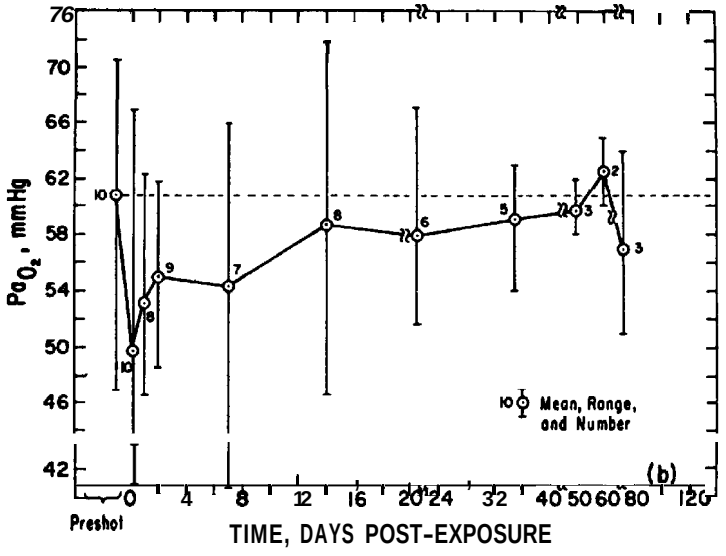


Fig. 9 **8. Algorithm for** of **a** blast **casualty's**





Effects of Blast Pressure on Venous-Arterial Shunt ($\dot{Q}s/\dot{Q}$) and Arterial Oxygen Teneion (Pa_{O_2}) in the Sheep.

Fig. 9-9. These graphs illustrate the magnitude and resolution of arterial oxygenation and shunting in sheep that were exposed to a large but sublethal blast. In the top graph, Qs/Qt is the shunt fraction or venous admixture. In the bottom graph, the preblast PaO_2 is consistent with normal function at the test-site altitude of 1,640 m.
Source: Reference 33

intubation and mechanical ventilation. Ventilatory assistance with a modern positive-pressure ventilator can *(a)* make the work of breathing less strenuous for the casualty, *(b)* correct hypercarbia, and *(c)* improve oxygenation.

Mechanical ventilation is not without risk for the blast casualty. For example, casualties who receive positive-pressure ventilatory support are at a much higher risk for developing tension pneumothorax than spontaneously breathing casualties are. This is a disastrous complication, and some authors have suggested that bilateral prophylactic chest tubes be placed in all blast victims who receive mechanical ventilation.[22,42] In addition, mechanical ventilatory support tends to encourage the production of air emboli from injured lungs.

In spite of these risks, mechanical ventilation should not be withheld in cases of respiratory insufficiency. Instead, the medical officer must take prudent steps to minimize the risks of air embolism and additional barotrauma. For cxamplc, the casualty who is hypoxemic but not hypercarbic can be treated with supplemental oxygen or the application of continuous positive airway pressure with an endotracheal tube or a face mask.[25,43,44] When positive-pressure ventilatory support is used, the medical officer should choose parameters for tidal volume, respiratory rate, and inspiratory-flow rate that will minimize the peak airway pressure during machine-delivered breaths. High-frequency jet ventilation may also be useful in lowering airway pressures.[12] Medical personnel should make every effort to keep the airway pressure low and to promote good bronchial hygiene by *(a)* using bronchodilators, *(b)* suctioning, and *(c)* changing the casualty's body position frequently.[45] Ideally, the casualty should be constantly monitored, especially in the intensive-care ward and the operating room.

Extracorporeal membrane oxygenation, a blood-perfusion system that actually takes the place of the injured lung for a time, was used in one heroic but unsuccessful attempt to save an Israeli blast casualty who suffered from both respiratory failure and air emboli.[19] However, this method is unlikely to be useful in a mass-casualty situation because *(a)* it requires massive logistical support, and *(b)* the patient must receive anticoagulants.

Treatment

The treatment of pulmonary blast injury focuses on correcting the effects of barotrauma and supporting gas exchange.

Pulmonary Contusion. Although many simple pulmonary contusions will begin to resolve within 24–48 hours, some patients will need assistance from mechanical ventilation for several days until they have adequate gas exchange and can resume spontaneous respiration.

Most hemorrhages in the airway will subside within a day or two as well. However, if brisk hemoptysis persists or if refractory lobar atelectasis is noted, fiberoptic bronchoscopy should be used to inspect the major airways. For example, in a study of fifty-three patients who had blunt chest injuries, twenty-eight had abnormal bronchoscopies.[46] The findings commonly included unappreciated bronchial fractures and lacerations. Continued hemoptysis without such a proximal lesion may indicate a persistent hemorrhage from a pulmonary contusion or laceration.

Pulmonary lacerations may require both ventilatory support and pleural drainage. A small hemothorax will generally resolve on its own, but surgeons will drain large collections of blood in the pleural space to prevent late complications of empyema and fibrothorax. If the casualty continues to lose blood from a hemothorax, the medical officer should consider surgical exploration.

Pneumothorax. A tube thoracostomy is the definitive treatment for pneumothorax. If the casualty has evidence of an accompanying pulmonary contusion, supplemental oxygen may also be required.

Pneumomediastinum and Subcutaneous Emphysema. By themselves, these injuries are not particularly hazardous to the blast casualty. However, they should prompt the medical officer to be alert to the casualty's increased risk of pneumothorax.

AIR EMBOLISM IN PRIMARY BLAST INJURY

Air emboli may be liberated from the lacerated lung into the arterial circulation, where they may cause occlusions—often with disastrous results. Any organ may be affected by a local vascular obstruction, but the casualty may suffer *(a)* a cerebrovascular accident when cerebral vessels are occluded, or *(b)* a myocardial infarction when coronary vessels are occluded. Air emboli cause most of those deaths that occur within an hour of the blast incident.

Initial Physical Examination and Triage

Air emboli may be produced very soon after blast injury, and their clinical prcscntation dcpcnds upon

which vascular bed has been compromised (Table 9-3). Medical personnel should note the following signs and symptoms:

- Does the casualty *(a)* complain of headaches or *(b)* exhibit seizures, changes in mental status, transient blindness, vestibular disturbances, focal neurological deficits, or coma? These symptoms and signs indicate that the central nervous system has been affected (Figure 9-10).[7,15,19,45]
- Is the casualty exhibiting hypotension or other obvious indicators of cardiac distress, such as dysrhythmia, hypotension, or frank ischemic changes?[15,16,19]
- In addition to other symptoms, does the casualty exhibit signs of pulmonary contusion?

The signs of air embolism develop so early that major manifestations are likely to be evident at the initial first-echelon triage, and so medical personnel should evaluate the blast casualty according to present (rather than potential) indications. Only casualties who exhibit clinical evidence of pulmonary contusion will he at risk for air embolism.

TABLE 9-3

CLINICAL SIGNS AND SYMPTOMS OF ARTERIAL AIR EMBOLISM

Signs
Air in retinal vessels
Arrythmias or cardiac ischemia
Focal neurological deficits
Livedo reticularis
Tongue blanching
Symptoms
Blindness
Chest pain
Focal neurological deficits
Loss of consciousness
Vestibular disturbances

Initiation of Life Support

The blast casualty should receive life support measures according to the signs he or she exhibits. Following standard procedures, medical personnel should respond to a blast casualty's respiratory difficulties by making sure that the casualty has a patent airway. Chest tubes should be inserted as warranted to relieve pneumothorax.

As soon as it is available, oxygen should be administered in order to (a) support gas exchange in the injured lung, and *(b)* help the tissues to absorb air emboli, a process that occurs faster when the bubbles contain a higher-than-normal proportion of oxygen rather than a predominance of nitrogen. Tissue oxygenation (and hence oxygen reserve) will also be increased.

Air emboli can cause myocardial infarction, and casualties may be hypotensive from this catastrophe as well as from any blood loss they might have suffered. They will require rapid but cautious volume replacement. Medical personnel should be aware that casualties who have extensive pulmonary contusion are at risk for further impairment of lung function if intravenous fluid resuscitation is excessive.[16,26] On the other hand, because lower vascular pressures favor the movement of air from the alveoli into the pulmonary vessels, casualties who do not receive volume replacement will have intravascular volume depletion that may predispose them to air embolism.[5]

Evacuation

As soon as they find the casualty at the blast site, medical personnel can begin to limit air-embolism damage by *(a)* positioning the casualty's body appropriately, and *(b)* ensuring that the casualty is evacuated by litter.

Air emboli in the arterial circulation tend to flow upward in the body and to travel to organs that require a large blood flow.[4,45] Thus, the position of the casualty's body may affect the site of embolism damage. Unless the casualty has a right-lung injury that is obviously more severe than an injury to the left lung, he or she should be kept recumbent in the left-lateral decubitus position with the head down.[4,5,6] An upright posture will direct bubbles to the brain, and the Trendelenburg position may predispose the coronary arteries to air-embolism damage.[45]

If one lung is more severely affected than the other, however, the damaged lung should be in the dependent position.' Throughout the dependent lung, alveolar pressures will be lower than vascular pressures. Although this may worsen gas exchange, it will also decrease the risk that air emboli will enter the pulmonary veins.

Stabilization and Life Support

Medical officers who utilize mechanical ventilation must be aware that it can increase the risk of lethal air embolism for blast casualties. Must air emboli

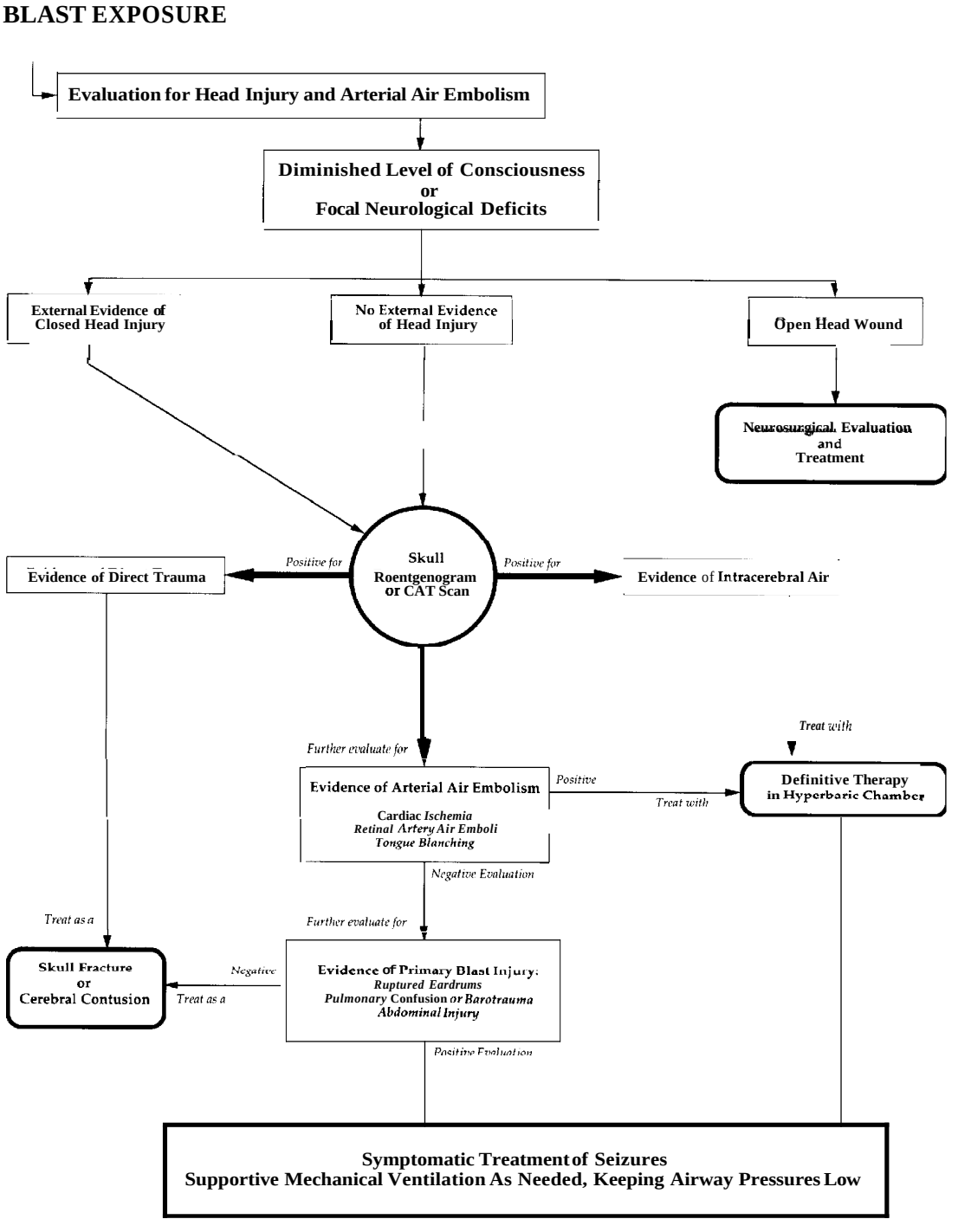


Fig. 9 10. Algorithm for the evaluation of neurological abnormalities in a blast casualty

probably occur within the first 2 hours after the blast exposure, particularly if mechanical ventilation is initiated soon after the injury. In a series of experiments, for example, three groups of dogs received intermittent positive-pressure respiration *(a)* immediately, *(b)* 4 hours after a severe blast injury, or *(c)* not at all (Figure 9-11).[47] Those animals that were ventilated immediately after injury had an 80% mortality, with arterial air emboli found at autopsy in three of ten dogs. The dogs in the second group were treated for 4 hours in a hyperbaric chamber and were then mechanically ventilated for 1 hour. This delayed group had only a 50% mortality and no emboli were detected at autopsy. The control group that received no intervention had a 60% mortality, with one of ten animals exhibiting air emboli at autopsy.

When the lungs are stressed by mechanical ventilation, air emboli can continue to be produced for hours or even days after blast exposure. For example, an autopsy on an Israeli soldier who had received revealed cerebral vessel air emboli 60 hours after the blast.[19] Emboli can occur in animals hours after blast exposure once positive-pressure ventilation is initiated (Figure 9-11).[47] In an experiment with dogs that were subjected to a penetrating wound in the chest, for example, the production of air emboli was more extensive if the animals were hypovolemic or if high gas pressures were used to expand their lungs.' Even if mechanical ventilation is initiated later, the high airway pressures associated with pusitive-pressure ventilation may predispose the injured lung to the later reopening of alveolovenous fistulae.[25] For example, two patients with nontraumatic ARDS developed repeated evidence of systemic emboli, which included cerebral infarction, myocardial injury, and cutaneous manifestations.[45]

The age of the casualty may affect the production of air emboli as a result of mechanical ventilation. For example, arterial air embolism is a well-recognized complication of mechanical ventilation in the infant,[48,49] perhaps because of the decreased adherence of peribronchial tissue planes in children.[45] It is less common in older patients on ventilators, despite the common occurrence of other forms of barotrauma in adults.

Definitive Physical Examination

Air embolism is recognized primarily from its effects on specific organs. For example, the physician who examines a blast casualty would recognize the cardiac, cerebral, or other distress that resulted from the air embolism, rather than the embolism itself. One exception might be the presence of air emboli, which have been described as "streaming bubbles or pale silvery sections representing columns of air or, indirectly or later, as pallor of the retina" (Figure 9-12).[19]

Severe or progressive neurological deficits in a casualty who has evidence of PBI should prompt early consideration of hyperbaric therapy (Figure 9-10).

Emboli to the superficial vasculature may cause facial blanching (with later reactive edema), tongue blanching, or *livedo reficularis* (a bluish network-patterned discoloration of the skin).[19,45]

Diagnostic Screening Procedures

Laboratory or roentgenographic studies will not be very helpful in diagnosing arterial air emboli. For example, the chest roentgenogram may show some evidence of PBI. But even though pulmonary damage is a sensitive indicator of air embolism (that is, it is always present when emboli are created), it is not predictive of embolism. Similarly, electrocardiographic monitoring may reveal ischemic changes or rhythm disturbances, but both are nonspecific. A CAT scan of the head may show intracerebral air,[50] but such a study is time consuming and should not be done if the diagnosis is clinically sound and definitive hyperbaric treatment is available.

When medical officers evaluate a blast casualty who has impaired consciousness or a focal neurological deficit, one of the most important differentiations they should make is between cerebral vessel air embolization and closed head injury with cerebral contusion (Figure 9-10). In terrorist bombings, for example, both closed and open blunt traumas to the head are common and are much more likely to be the cause of alterations of consciousness than PBI is. However, if the blast occurred under water or as the result of special military ordnance, then air embolism becomes a more likely diagnosis.[19,51,52,53]

Treatment of Air Emboli

Air emboli that escape from the lacerated lung into the arterial circulation may greatest effect on mortality from PBI. They are treated either definitively with hyperbaric therapy or in a less specific supportive manner for clinical manifestations.

Hyperbaric-Chamber Treatment. Treatment in a hyperbaric chamber is the definitive therapy for arterial air embolism.[4,19,54,55] An increase in ambient pressure will decrease the size of the emboli and promote their rapid absorption. The higher partial pressure of oxygen that occurs even without oxygen enrichment of the atmosphere may also play a role in improving

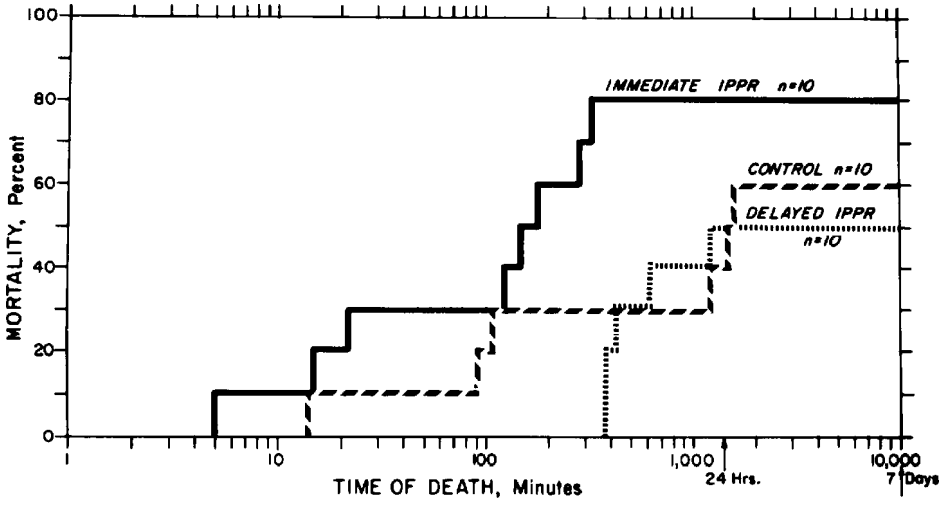


Fig. 9-11. The graph illustrates the mortality following blast injury for beagles that **received** *(a)* **immediate intermittent** positive-pressure respiration (IPPR) (——), *(b)* IPPR after a 4-hour delay in a hyperbaric chamber (▮▮▮▮), or *(c)* no specific treatment at all (– – –). Mortality was greatest with artificial ventilation and was both delayed and lessened by hyperbaric therapy.
Source: Reference 46

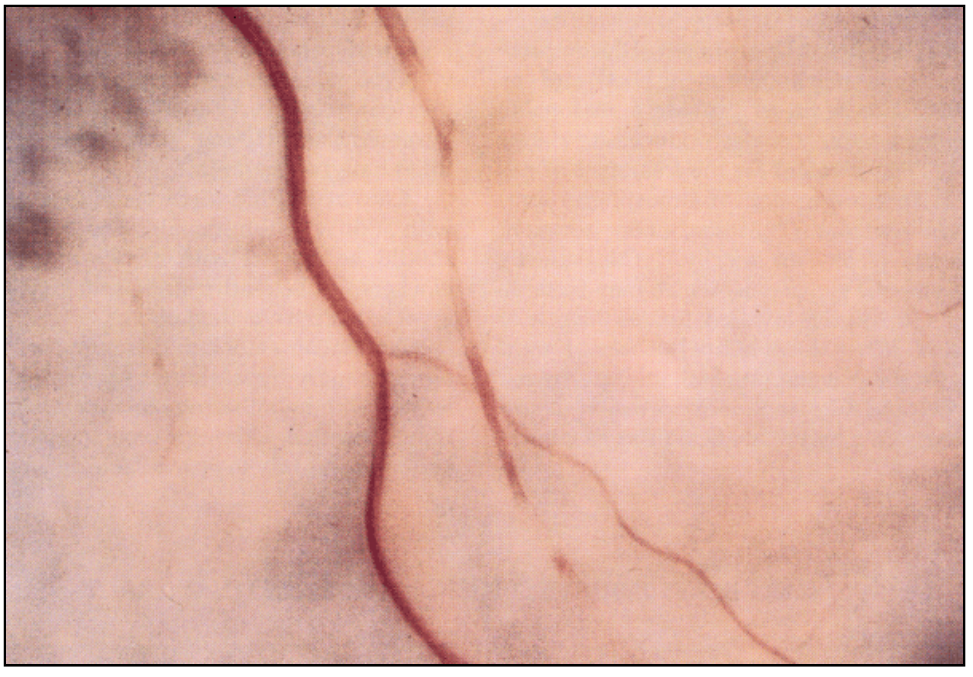


Fig. 9-12. This photograph of the fundus of a sheep that was exposed to intense blast shows air in the retinal artery.
Source. Reference 19

tissue oxygenation by increasing *(a)* the amount of oxygen that is carried dissolved in blood and *(b)* the gradient for diffusion from blood to tissue. The immediate reduction in bubble size, which is dictated by Boyle's law, may be responsible for the rapid response to therapy that has been observed in some cases.[19]

A series of animal experiments demonstrated the effectiveness of hyperbaric therapy in treating PBI (Figure 9-13).[4,56] When hyperbaric therapy was maintained for 29 hours, the blast mortality was reduced from 60% to 0%. The same result was reported for *(a)* hyperoxic therapy at relatively low pressures (15 psi above ambient pressure) and *(b)* a normoxic environment at higher pressures (60 psi above ambient pressure). When pressurization was limited to just 4 hours, deaths were delayed but not prevented. Supplemental oxygen did not improve survival in these studies, but could delay mortality.

The data on the effectiveness of hyperbaric therapy for human arterial embolism are scanty. Most such interventiuna took place hours after blast exposure and had little effect on outcome.[19,50] As demonstrated by animal studies, hyperbaric treatment must begin as soon as possible after blast exposure. After ten of thirteen swimmers died from a single submersion-blast event, Israeli military physicians advocated the development of helicopter-transportable monoplace compression chambers so that therapy could begin at the site of injury.[19]

Protocols for hyperbaric therapy have been developed by the U.S. Navy for the treatment of decompression sickness (commonly known as the *bends)* and gas embolism.[57,58] They seem to provide a reasonable guidclinc for thc trcatmcnt of blast air embolism.[4,6,55] The protocol for the treatment of gas embolism calls for compression to 6 atmospheres (atm) with an air environment. (The initial air environment reduces the risk of oxygen toxicity.) The patient's tolerance of embolic symptoms, which may recur as the chamber's pressure is lowered, guides the rate of decompression. When the pressure has been brought down to 2.8 atm, the air environment should be changed to 100% oxygen and the rate of decompression should again be determined by the medical officer's clinical judgment.

Hyperoric hyperbaric therapy is not without potential complications.[19,57,58] The elevated pressure may cause pain in ears or sinuses. More importantly, extremely high oxygen concentrations can cause both acute and delayed effects. Symptoms of acute oxygen toxicity include retrosternal burning, muscle fasciculations, paresthesias, and dizziness that may progress to seizures. In the lungs, the very high oxygen concentration may cause serious (but delayed) oxidation damage to the alveolocapillary membrane that may aggravate pulmonary injury. Thc high concentration of oxygen can also pose some risk for medical attendants, so the prescribed protocol should be strictly followed.

Most hyperbaric chambers are relatively large and can accommodate the patient and medical attendants. Smaller monoplace chambers, which can be pressurized to about 3 atm, are also available. They offer extremely limited access to the patient and make no provision for sophisticated medical care or mechanical ventilation.[55]

The locations of hyperbaric chambers in the United States can be obtained from the Undersea Hyperbaric Medical Society.[59] In overseas locales, medical societies in host countries or naval medical-liaison groups should be able to provide similar information.

Nonspecific Treatments for Air-Embolism Sequelae. The sequelae of air embolism should be treated as if they had arisen from any impairment in the vascular supply. Such treatments are generally nonspecific and supportive.

Cerebral insults may respond to nonspecific therapies that reduce cerebral edema, such as intravenous dexamethasone (10 mg bolus followed by 4 mg four times daily) or mannitol.[55]

Cardiac-rhythm disturbances should be treated with antidysrhythmics. Significant cardiac ischemia should be treated with nitrates, calcium channel blockers, or beta-adrenergic antagonists to reduce myocardial oxygen demand.

Arterial air emboli that are produced during cardiopulmonary-bypass surgery have been successfully treated with hypothermia, corticosteroids, and barbiturate sedation, although such an elaborate support system is unlikely to be available on a battlefield.''

PRIMARY BLAST INJURY TO THE GASTROINTESTINAL TRACT

An injury to the gastrointestinal tract is often overshadowed by the more immediately life-threatening pulmonary contusions and lacerations, as well as by the air emboli that result from them. However, gastrointestinal damage may be the most dramatic injury at the time of presentation. It may also determine the

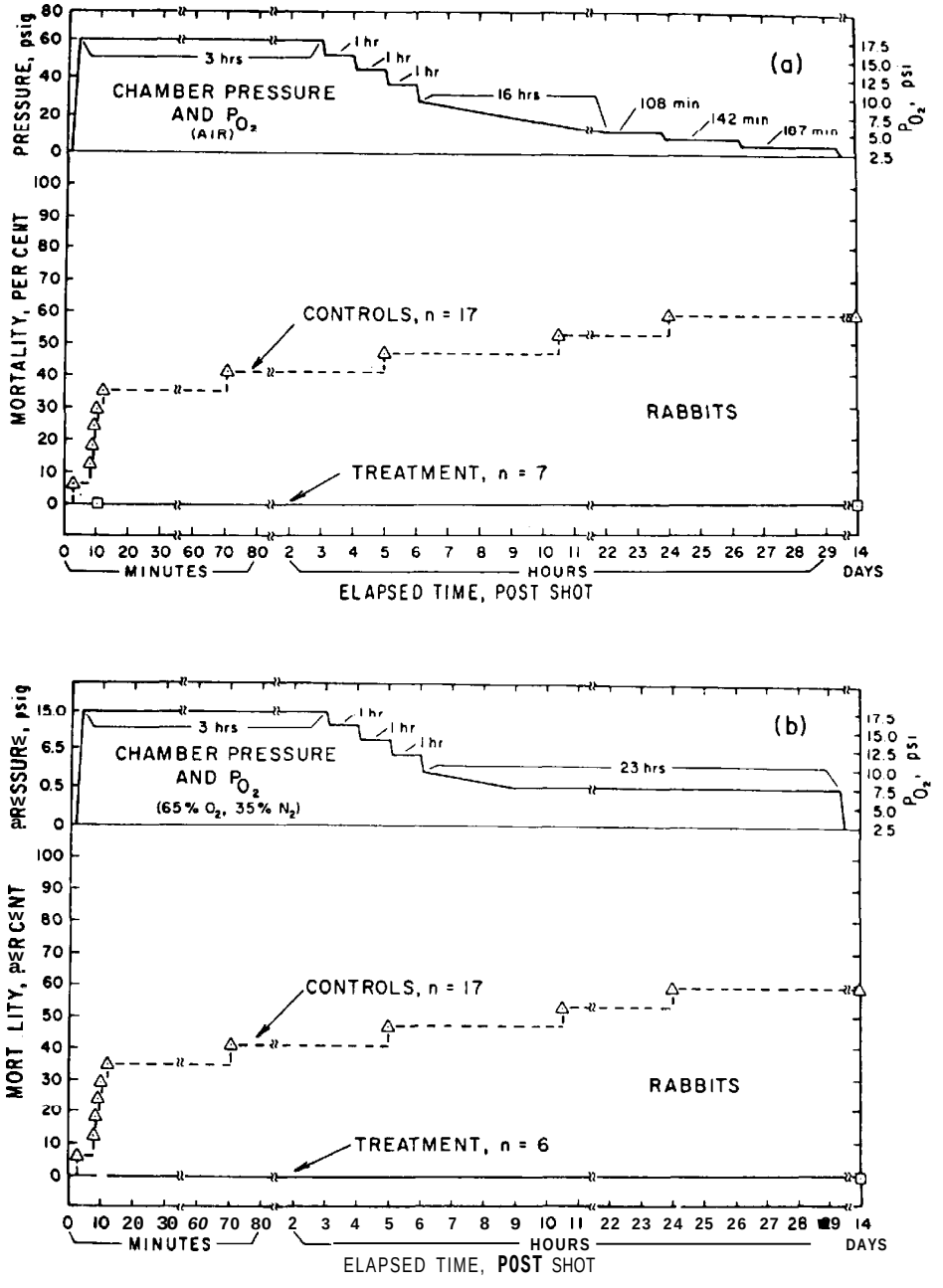


Fig. 9-13. These graphs illustrate the mortality of blast-injured rabbits that were treated with two long-duration hyperbaric protocols. Mortality was 0% for both higher-pressure normoxic therapy (a) and lower-pressure hyperoxic treatment (b).
Source: Reference 56

morbidity of those casualties who survive the first few hours after an exposure to blast. Many of these abdominal injuries will require surgical intervention.

Like injuries to the lungs, the most common primary blast lesions of the gastrointestinal tract are found in the air-containing organs. These lesions commonly include *(a)* hematomas and perforations of the bowel, *(b)* hematomas and tears of the mesentery, and *(c)* ruptures of the hollow abdominal viscera.

Retroperitoneal hemorrhage and damage to the solid abdominal organs are much less common; they are more likely to be secondary blast injuries from fragments, or tertiary blast injuries from bodily displacement.[42] Injury in the liver, spleen, or kidneys is the most common of these injuries; very rarely, the force of the blast will fracture one of these solid organs.[16,23,61]

Blast casualties with gastrointestinal PBI may experience symptoms that include abdominal pain, nausea, testicular pain, an electric-shock sensation, tenesmus, or a temporary loss of motor control in the legs.[16,23,35,62]

Casualties who have pure PBI to the gastrointestinal tract are most likely to have been injured in an underwater blast (Figure 9-14).

Initial Physical Examination and Triage

The physical examination of a casualty with abdominal blast injuries will reveal signs that are similar to those found in blunt abdominal trauma from any cause, except that injury to the solid viscera will be much less common (Table 9-4).

Casualties with abdominal PBI may vomit; a few may even exhibit hematemesis. They may also have signs of peritoneal irritation such as guarding (voluntary or involuntary) or rebound tenderness. Bowel sounds may be absent. Bright-red rectal bleeding may occur later.

Patients who have unimpressive abdominal complaints may temporarily improve, only to develop an abdominal crisis days or even weeks later.[61,63] A soldier who has suffered a significant blast injury and has abdominal complaints should be observed for at least 1 week before being returned to full duty.

Any casualty who is in shock or is hypotensive should receive volume replacement. A blast casualty in shock may also require emergency exploratory laparotomy to control internal bleeding, and should be placed in the immediate triage category if he or she is unresponsive to the initial volume replacement.

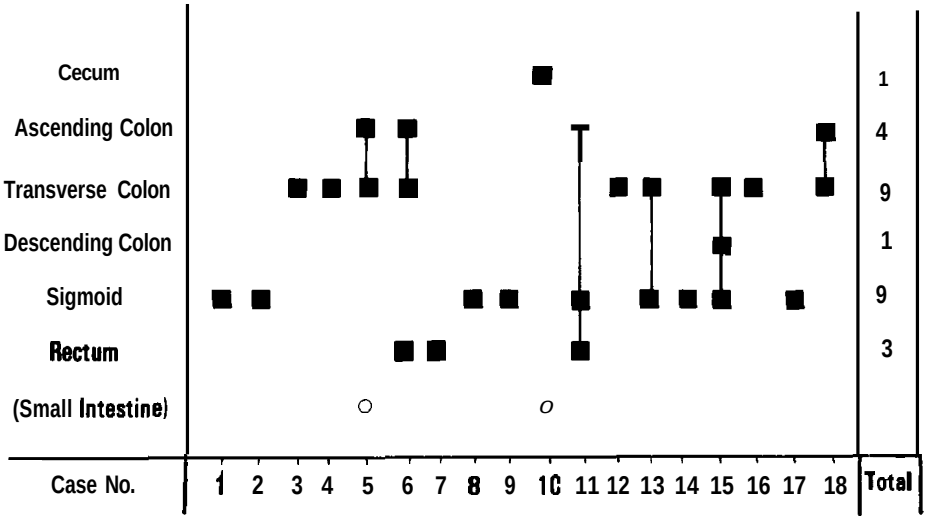


Fig. 9-14. This graph shows the locations of gastrointestinal injuries found at laparotomy in survivors of a large underwater explosion. Three of thirty-two casualties had rectal injuries.
Source: Redrawn with permission from reference 17

TABLE 9-4

CLINICAL SIGNS AND SYMPTOMS OF PRIMARY BLAST INJURY OF THE ABDOMEN

Signs
Absent bowel sounds
Bright red blood from rectum
Hypotension
Involuntary guarding
Rebound tenderness
Symptoms
Abdominal pain
Nausea and vomiting
Orthostasis or syncope
Testicular pain
Tenesmus

If their blood-pressure levels are within normal ranges, blast casualties should be placed in the delayed triage category.

Initiation of Life Support

Some casualties with PBI may be hypotensive because of blood loss from an abdominal hemorrhage or solid organ fracture. Secondary blast injuries or other wounds may also cause hypotension.

The hypotension associated with PBI may be multifactorial, with contributions from (a) loss of intravascular volume, most commonly because of an abdominal hemorrhage or solid-organ blast fracture, *(b)* bradycardia with or without vasodilation, or *(c)* myocardial ischemia caused by arterial air emboli.[16,26] If the casualty has a vagally associated bradycardia, then medical personnel can administer 0.5–1.0 mg of atropine parenterally.

Initial volume resuscitation should be vigorously pursued according to standard guidelines to maintain an adequate pulse and blood pressure, but the casualty should not be overhydrated. Volume resuscitation should be guided by evidence of adequate tissue perfusion, as assessed clinically by *(a)* blood pressure, *(b)* urinary output, and *(c)* mental status.

In a complicated patient, invasive monitoring of central-venous or pulmonary-artery pressures may be invaluable in guiding volume replacement.

Evacuation

Increased gas in the gastrointestinal tract, which can exacerbate abdominal injuries, can be minimized by nasogastric decompression. This procedure should be performed before the casualty is evacuated.

Definitive Physical Examination

The physical examination plays an important role in evaluating intraabdominal injury. Unequivocal signs of peritoneal irritation, particularly if the casualty is hypotensive, require prompt surgical intervention. Most casualties who will need surgery will exhibit guarding and rebound tenderness, but these signs are not specific and may be found with lesser degrees of injury.[64]

Casualties who have tenesmus or bright-red rectal bleeding should receive a flexible sigmoidoscopy at the first echelon where it is available, which will allow the physician to note rectal tears or perforations.[16]

Table 9-5 summarizes the incidence of abdominal

TABLE 9-5

DISTRIBUTION OF ABDOMINAL SYMPTOMS, SIGNS, AND CLINICAL FINDINGS IN SURVIVORS OF UNDERWATER AND AIR BLAST*

Symptoms, Signs, and Findings	Percentage**
Abdominal symptoms	
Abdominal pain	73
Nausea and vomiting	39
Tenesmus	28
Abdominal signs and findings	
Perforated bowel	67
Isolated bowel hematoma	2
Solid visceral injury	2

*Hospitalized survivors of major underwater blast (N=43) and air blast (N=2)

**See Table 9-2 for pulmonary injury data from the same incidents. The percent total mortality from pulmonary and abdominal blast injuries was 16%.

symptoms and signs in two series of patients who were injured predominantly by underwater blast.[13,16]

The patient who is unconscious or who has a less clear-cut examination will present a diagnostic challenge (Figure 9-15). Military medical officers may find more information on diagnostic evaluations in the extensive civilian literature on blunt abdominal trauma.

Diagnostic Screening Procedures

Serial hemoglobin determinations are essential if the patient might have suffered an intraabdominal blood loss. However, routinely available serum-chemistry and enzyme studies (such as serum transaminases, lactate dehydrogenase, creatine kinase, and alkaline phosphatase) failed to correlate with either the presence or the degree of gut involvement in animals that were injured by blast."

Although routine laboratory screens may not be helpful in evaluating the stable patient who has PBI to the abdomen, *diagnostic peritoneal lavage* (DPL) is a useful procedure.

If abdominal-trauma patients are unstable, medical officers should forego detailed radiologic studies in favor of obtaining only a chest roentgenogram, and should instead perform prompt laparotomy.[65]

Diagnostic Peritoneal Lavage. DPL is an important diagnostic test when abdominal trauma is suspected in blast casualties. For example, in casualties from Northern Ireland's civil conflict, most of whom had penetrating wounds, abdominal lavage was seldom necessary "except in closed injuries due to bomb blast."[20] A recent report of a jejunal transection and duodenal hematoma from a rocket-motor explosion noted that DPL was positive and useful in guiding therapy.[66]

The Nelson-Lazarus DPL technique is relatively simple and can be done in any emergency room or triage ward.[67] First, the casualty's bladder should be drained with a Foley catheter, the gastrointestinal tract should be decompressed with a nasogastric tube, and the abdominal skin should be prepped and draped. An area in the midline that is 2–3 cm below the umbilicus is infiltrated with xylocaine to provide anesthesia and epinephrine to reduce the chance of a false positive tap. Next, an 18-gauge needle is introduced into the peritoneal cavity at a 45° angle towards the pelvis through a small stab wound. A J-wire guide is placed through the needle and a #8 French lavage catheter is passed over the wire into the peritoneal space. If more than 10 cc of nonclotting blood are aspirated, the DPL is positive and urgent laparotomy is indicated. If the aspirate is negative, a liter of normal saline is introduced through the catheter and allowed to drain out by gravity return. More than 100,000 erythrocytes or 500 leukocytes per cc of effluent are considered a positive lavage. If the effluent contains any bacteria, bile, or vegetable fibers, it is also considered positive."[67] In equivocal cases, the catheter may be left in place for several hours so that serial lavages can be done.[68]

A positive DPL is not absolutely specific for major abdominal injury. In a review of blunt-trauma injuries, 6%–25% of laparotomies done for a positive DPL were unnecessary because surgically correctable abnormalities were found. On the other hand, the sensitivity of DPL is said to be as high as 100% for blunt abdominal trauma.[68,69,70] The complication rate in experienced hands is less than 1% if error occurs, inadvertent bowel aspiration is the most common.[71]

A major drawback of DPL is its insensitivity to retroperitoneal damage and mesenteric hematoma.[64,68,69,71,72] DPL may also fail to detect subcapsular injuries of the liver or spleen.[65,68]

Gastrointestinal Imaging. Radiographic imaging of the blast casualty's abdomen is an important part of the evaluation. The skill with which it is performed and interpreted is crucial.[71]

Noncontrast abdominal roentgenograms may show extraluminal air in about 50% of perforations from blunt trauma.[64] They may also show secondary signs of intraabdominal injury, such as ileus or lumbar scoliosis." Because less than 800 cc of intraperitoneal fluid cannot be reliably detected on noncontrast roentgenograms, these studies are inherently limited in detecting free peritoneal bleeding.[65]

Of the noninvasive imaging procedures, CAT scans and sonograms seem to have the most promise.[65] The CAT scan is likely to be most useful in a patient who is stable enough to permit detailed evaluation." Signs of serious injury that may appear on a CAT scan include (a) extraluminal gas or contrast (Figure 9-16), *(b)* hernoperitoneum (Figure 9-17), *(c)* a hypodense accumulation of peritoneal fluid, possibly including bowel contents, and *(d)* bowel-wall hematomas greater than 3 mm in diameter, especially when associated with large fluid collections."

Abdominal CAT scans should be done with both an oral contrast bolus and an intravenous contrast infusion.[65,73] All electrocardiogram leads and other external paraphernalia that might cause artifact should be removed. After the stomach is emptied through a nasogastric tube, 2,300 ml of 1% radiocontrast material should be instilled and the gastric tube should be withdrawn into the distal esophagus. The entire abdomen should be scanned, and 1-cm cuts should be made through the chest as well. If a CAT scan or sonogram is to be done, it must precede any attempt at DPL because the lavage may leave both fluid and air behind

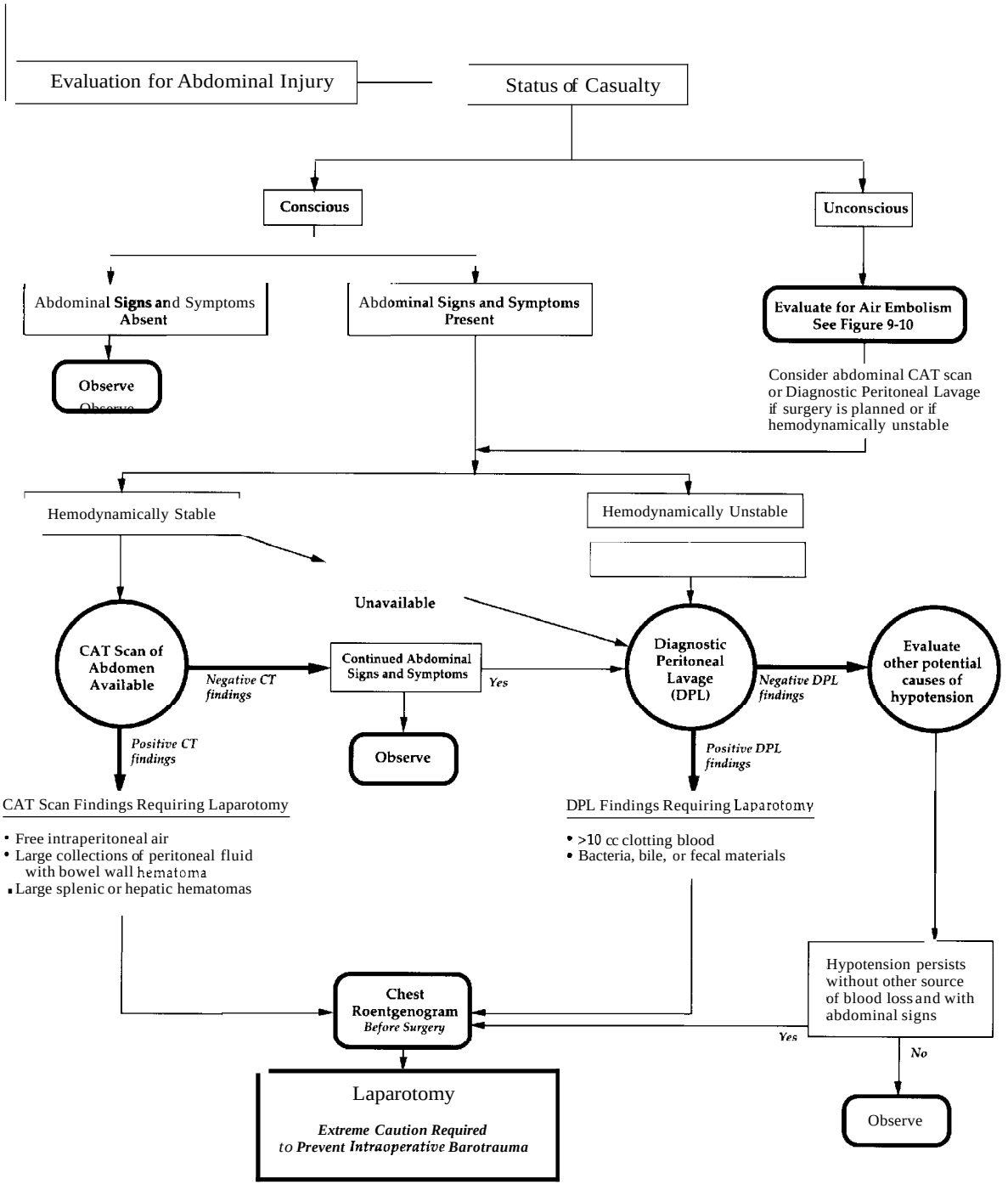


Fig. 9-15. Algorithm for the evaluation of a blast casually's gastrointestinal injury

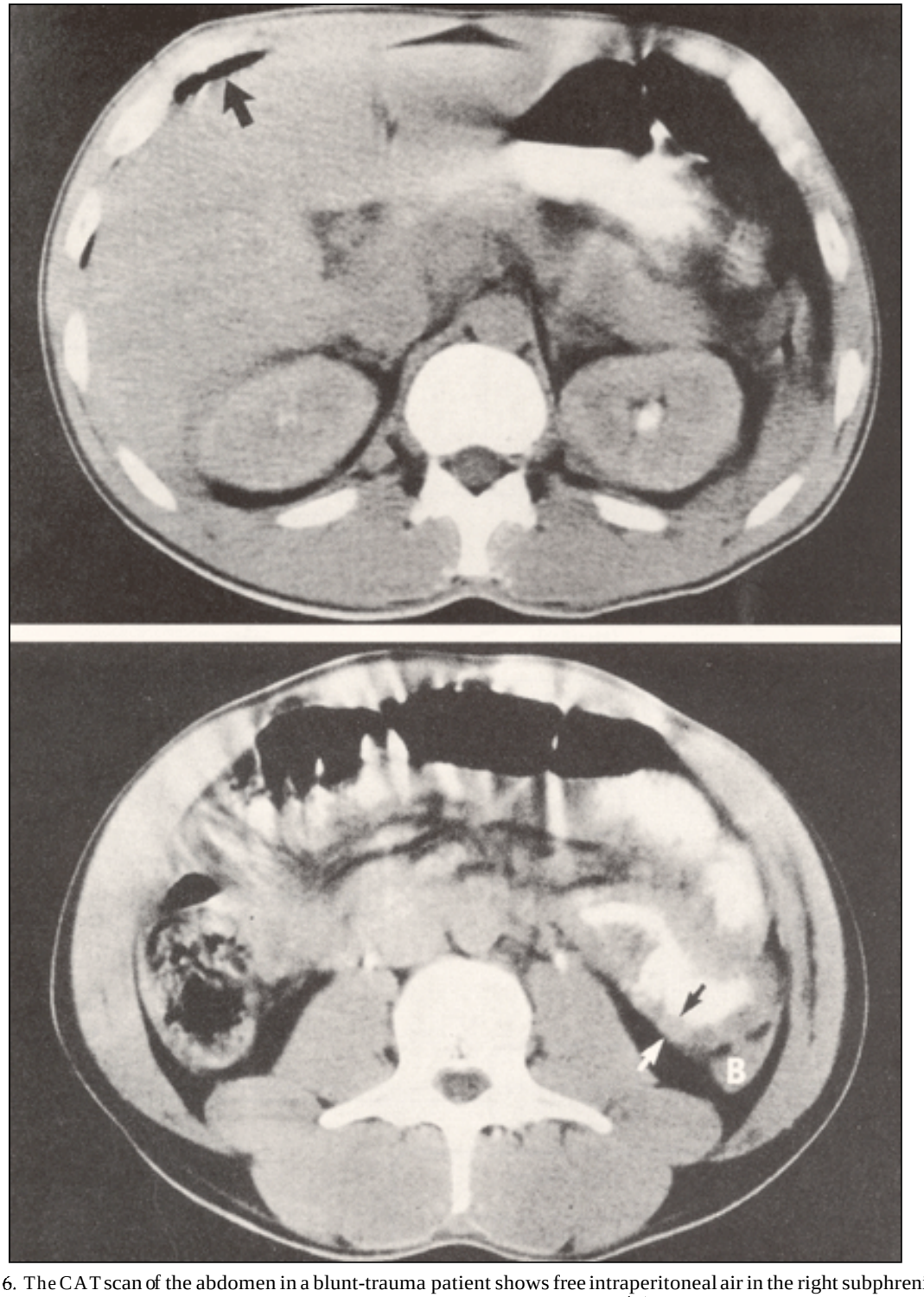


Fig. 9-16. The CAT scan of the abdomen in a blunt-trauma patient shows free intraperitoneal air in the right subphrenic space (arrow in upper image). The lower image shows a thickened jejunal bowel wall (10 mm between arrows); normal walls are 3 mm or less. Free blood is in the adjacent gutter (B).
Source: With permission from reference **64**

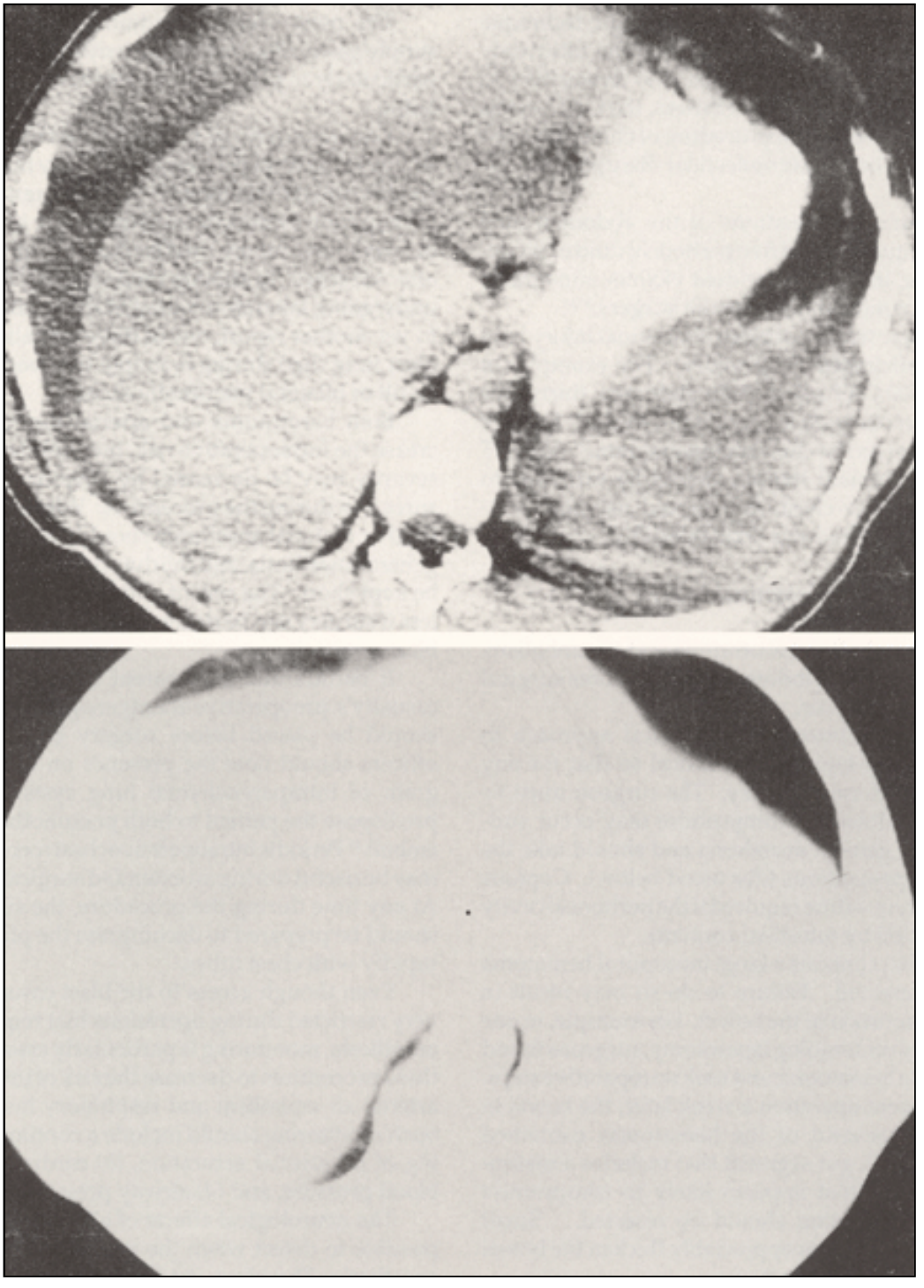


Fig. 9-17. This **CAT** scan of the upper abdomen of a blunt-trauma casualty was viewed with two windows. The soft-tissue window (upper image) reveals a lacerated spleen and accompanying hernoperitoneum. The lung window (lower image) is the same cut and shows a left pneumothorax. Note the difference between lung parenchyma in the right hemithorax and air in the left chest.
Source: With permission from reference 36

in the peritoneal cavity, thereby causing a false-positive imaging study.[64,73]

Most investigators have concluded that CAT scans are less sensitive than DPL for blunt abdominal injury. According to some studies, the radiologic technique identified only 25%–74% of patients who ultimately required laparotomy.[69,70] It was also insensitive to hollow-viscus injury.[71] Nevertheless, the CAT scan is the probably the imaging procedure of choice for blast casualties in whom gastrointestinal disruption is suspected.[73]

Ultrasound examinations of the abdomen may reveal small quantities of free peritoneal fluid or blood. For example, in civilian accident victims, sonograms detect 84% of injuries that require surgery.[65,68]

Radiologic studies of blunt abdominal injury may reveal unanticipated lung injury. For example, in more than 500 CAT scans of the abdomen, thirty-five pneumothoraces were evident, ten of which had not been apparent on the chest roentgenogram.[37] All CAT scans for abdominal trauma should be extended to include the lower chest, and should use both soft-tissue and lung windows (Figure 9-17).

Treatment of Gastrointestinal Injury

Unlike respiratory barotrauma and air embolism, significant PBI to the abdomen often requires surgical exploration and repair.

Surgical Treatment. The surgical approach to abdominal blast injuries is identical to that for any other blunt abdominal injury. The surgeon must be aware that pulmonary compromise may occur suddenly during general anesthesia and should appropriate precautions (discussed below). Copious saline irrigation will be required if the peritoneal cavity has been soiled by intestinal contents.

Bowel that contains a large intramural hematoma should be resected. Failure to do so may result in delayed perforation of the bowel. For example, in one blast casualty undergoing laparotomy, surgeons found only multiple hematomas and took no reparative steps. At a subsequent operation a week later, the bowel in proximity to several of the hematomas contained perforations.[16] A gut segment that includes a mesenteric hematoma that appears likely to compromise bowel-wall circulation should be resected. Small hematomas that do not appreciably thicken the bowel wall should be left alone.[66]

A disruption of the large bowel should lead to exteriorization of the colon. Simple perforations of the small intestine should be closed primarily; more extensively damaged intestine should be resected and reanastornosed. If surgery is greatly delayed, the devitalized small bowel should be resected and exteriorized.[3,20,42,66,74]

Lacerations of solid organs should be oversewn or resected as indicated by the injury's complexity.

The most important adjunct to surgical care is simple gastric decompression with a nasogastric tube, particularly in patients who receive mechanical ventilation.[61]

Complications of ***General Anesthesia.*** Blast casualties tend to suffer a high morbidity rate when they receive general anesthesia.[15,16,19,55] For example, three of twenty-four victims of an underwater blast who underwent laparotomy died intraoperatively or shortly after surgery.[16] In another review, one of nine patients undergoing laparotomy died in the operating room.[13] In yet another report, one of three casualties who had blast lung died during a simple tracheostomy as the result of bilateral pneumothoraces.[15]

Both barotrauma and arterial air embolism increase the intraoperative risk, but their incidence and severity may be decreased by keeping airway pressures low during the procedures. Whenever possible, local or regional anesthesia should be used instead of general anesthesia, and the duration of surgery should be kept short. The risk of added injury will be much less if surgery can be delayed for 24 48 hours after the blast event.

If barotrauma is apparent or suspected on the casualty's preoperative roentgenograms, chest tubes should be placed before surgery begins. Medical officers should note the presence on the roentgenogram of intraparenchymal lung cysts, which may predispose the patient to both pneumothorax and air emboli.[45] Periodic intraoperative chest roentgenograms may be useful during an extended surgical procedure. At any time during the procedure, the surgical team should be prepared to decompress the pleural spaces rapidly with chest tubes.

Even though access to the blast casualty may be very restricted during operations that require general anesthesia, monitoring is particularly essential during these procedures to decrease the risk of intraoperative arterial air embolism and respiratory insufficiency.[15] Such monitoring should include a continuous recording of *(a)* arterial saturation, *(b)* cardiac rhythm, *(c)* blood pressure, and *(d)* airway pressure.

The neurological effects of air emboli will be impossible to detect when the patient is under general anesthesia. Therefore, medical personnel should note any cardiac-rhythm or ischemic-electrocardiographic changes that may herald an embolic catastrophe. The anesthesiologist should periodically examine the patient's fundi for evidence of air emboli in the retinal artery (Figure 9-12).[5] If Doppler ultrasound probes are

available, medical officers should monitor carotid-artery blood flow for evidence of embolic bubbles."

Drug Therapy. The role of histamine-2 antagonists and prophylactic antibiotics in the treatment of abdominal blast injury is unknown. However, if surgery must be delayed for tactical, logistic, or technical reasons, the patient should receive antibiotic coverage for anaerobes, Gram-negative aerobes, and enterococci.

PRIMARY BLAST INJURY TO THE AUDITORY SYSTEM

The modern military environment depends heavily upon communication. The soldier must acquire and process a great deal of aural information and act upon it quickly and decisively. However, the nature of the combat environment may hinder the soldier from communicating effectively. High levels of background noise, the complex content of the messages, and the increasingly common possibility that neither the speaker nor the listener may be fluent in English make communication difficult enough for soldiers with normal hearing. A hearing-impaired soldier on guard duty or assigned to radio communication may be more of a liability than an asset.

The very nature of the auditory system makes it particularly vulnerable to injury during combat. Pressure waves in the air are collected by the pinna of the ear and are focused in the external auditory canal to act upon the tympanic membrane. The resulting pressures on one side of the eardrum cause it to distort; this movement is amplified by the ossicular chain and is transduced into the fluid-filled inner ear where it is converted into electrical impulses by the hair cells. In the normal acoustic range of pressures, this process is an efficient means of collecting and amplifying a weak air-pressure wave into a physiologically detectable variable (that is, sound).[2,17,75] However, the powerful pressure wave caused by a blast does not normally occur in nature, and it may overwhelm the extremely delicate structures within the ear. The tympanic membrane may rupture, the ossicles may fracture or dislocate, and so much energy may be transmitted to the inner ear that permanent damage will be done.

Aural injuries from blast may range from a trivial insult to a major disruption of middle- and inner-ear structures. Under the stress of combat, a soldier who has been stunned by a nearby explosion may initially be unaware of any aural injury.

The most common injury will be hearing loss, with or without tympanic-membrane rupture. The hearing loss may be *(a) conductive, (b) sensorineural,* or *(c)* a combination of the two.

A conductive hearing loss is defined as decreased hearing sensitivity to sounds transmitted through the air with normal bone conduction of sound. This loss is usually caused by a tympanic-membrane rupture, although ossicular damage and serous otitis can also cause a conductive deficit.[76,77] The amount of conductive hearing loss depends on the size and location of the tympanic-membrane perforation. In the past, medical officers considered the mere presence of perforation to mean that the soldier was unfit for duty. However, many soldiers can function well in spite of it, provided they have no other incapacitating injuries from the blast.[78,79,80]

Sensorineural hearing loss results from damage to the inner ear, and may be the most incapacitating aural symptom in the moments immediately after the blast. It is usually in the high frequencies, but because it can also affect speech frequencies, it may compromise the ability of these casualties to communicate and may seriously disorient them with respect to their environment. Initially unaware of the deficit, they may notice the loss only when they see people talking to them but hear little or no sound.[81]

Sensorineural loss usually resolves within the first few hours, but permanent loss may occur in as many as 30% of blast victims.[18,82,83,84]

Blast-related sensorineural losses range widely in severity. Relatively minor injury may cause a *temporary threshold shift* (TTS) as measured by standard audiometric testing. TTS is a change in hearing acuity that ultimately recovers and that is presumed to be secondary to a transient and reversible biochemical or subtle mechanical cochlear dysfunction. More severe injury may cause longer-lasting or permanent hearing losses from *(a)* loss of hair-cell integrity, *(b)* labyrinthine or perilymphatic fistulae, *(c)* labyrinthine- or basilar-membrane ruptures, or *(d)* other defects.

Either conductive or sensorineural hearing loss may be a permanent complication in a significant number of patients. For example, in one study of seventy-seven military blast casualties, hearing loss was tracked for 1–3 years following injury.[84] Residual sensorineural hearing loss was found in 29% of these casualties' ears, 5% showed a persistent conductive loss, and 7% had a mixed hearing loss.

Tympanic-membrane rupture or perforation can be caused by a blast force as low as 2.5 kg/cm^2, and a

blast force of 6.75 kg/cm^2 will rupture half of adult tympanic membranes.[85,86] By comparison, the blast would have to be about ten times stronger to cause severe lung damage.[2] Thus, tympanic-membrane rupture may be the only significant blast consequence for casualties of relatively small-scale terrorist bombings, and in fact does affect as many as one-third of these casualties.

However, the absence of tympanic-membrane damage does not preclude a significant blast exposure, particularly car was not as cxposcd **as** other parts of the body, such as *(a)* during an underwater blast, *(b)* when a helmet shielded the ear, or *(c)* when hearing protection was worn.[2] Although almost two-thirds of blast casualties who have nonauditory PBI that is significant enough to warrant hospital admission will also have tympanic-membrane rupture, one-third of such casualties will have intact tympanic membranes.[18,36]

Other less common symptoms of auditory damage include *(a)* persistent otalgia, *(b)* distracting tinnitus, or *(c)* vestibular dysfunction, which manifests itself as vertigo.

may have numerous etiologies, including tympanic-membrane rupture, ossicular disruption, or concussive injury from other blast effects. The delayed infectious complications of middle-ear barotrauma might also cause otalgia, although it would probably not be incapacitating. Surprisingly, pain is not a common spontaneous complaint of blast casualties, although most **do** report ear discomfort when specifically asked.[81]

Tinnitus is a tone or ringing in the ears that may be caused by a persistent signal (in the abscncc of any sound input) from a damaged acoustic organ. It can be an unpleasant sensation that may distract a soldier from his or her tasks. Tinnitus tends to parallel sensorineural hearing loss, and will generally resolve as the hearing loss does.

Vertigo is the sensation of dizziness or irregular whirling motions that a casualty may feel after blast injury, although it is more commonly a symptom of blunt trauma to the head from secondary or tertiary blast effects rather than **a** symptom of PBI. Rarely, vertigo may result from PBI when both the cochlea and labyrinthine apparatus are disrupted by blast.[81,82] Such a loss of equilibrium can be expected to completely incapacitate a soldier.

Late sequelae of PBI to the auditory system may include *(a)* permanent hearing loss, *(b)* a predisposition to infection secondary to tympanic-membrane perforation, *(c)* persistent tympanic-membrane defects, and *(d)* cholesteatoma.[76,77,82,83] Cholesteatoma is a granulomatous mass lesion of the middle ear that may ultimately destroy middle-ear structures and cause a conductive hearing loss.

Initial Physical Examination, Triage, and Evacuation

The evaluation of a blast casualty with auditory damage may differ dramatically depending on the situation in which it occurs. Ideally, the casualty would be evacuated to a third-echelon facility and would thcrc bc cvaluatcd by an otolaryngologist according to subjective and objective data as well as audiological and microscopic examination. In wartime. these procedures would be luxuries.

In combat, soldiers who suffer isolated blast injury of the ear would be evaluated according to their ability to continue their combat mission. The medical officer should weigh the casualty's current auditory injury, related performance decrement, and risk of suffering further impairment against the unit's need for the casualty's continued participation. Fur example, a soldier who has a small perforation of the tympanic membrane with no subjective hearing deficit could healing within 2 **weeks,** and therefore would not be transferred to an evacuation hospital. On the other hand, a soldier who has severe hearing loss imposed by significant TTS might be nonfunctional in combat.

If the ears of these casualties are not protected from further damage while the initial auditory injury is healing, their hearing impairment may become worse or even permanent. If possible, they should be reassigned to quieter military duties. For example, 433 Israeli soldiers who had hearing loss or tinnitus or both were divided into two groups: *(a)* those who were transferred to a quieter noncombat unit and *(b)* those remaining in the noisy combat units.[80] The hearing of 30.8% of those soldiers who were transferred to less noisy environments improved; the hearing of 4.0% of the soldiers in this group deteriorated. In the group of soldiers who were not transferred, only 8.7% of the injured ears improved, whereas 30.4% deteriorated.

Even if an active combat situation demands that these casualties not be reassigned to quieter environments, they should at least wear hearing protection (either insertional or circumaural) whenever possible after injury. The protectors will decrease the effects not only of subsequent blasts and the sustained impulse noise of weapons, but also the continuous noise of engines, helicopters, and armored-vehicle tracks.

Once the blast casualty is stabilized and any other blast-related injuries (such as PBI to other areas of the body, lacerations, fractures, burns, or penetrating wounds) are assessed, medical officers may have the

opportunity to following signs and symptoms of possible PBI to the ear:

- The size and location of any tympanic-membrane perforation that may be visible upon otoscopic examination
- The hearing level, using any subjective or objective means available
- The presence of any foreign material in the ear
- Any dislocation of ossicles that may be seen behind a torn tympanic membrane
- The presence of tinnitus, otalgia, or bleeding from the ear
- The pressure sensation nf eustachian-tube blockage
- Vertigo

Medical personnel should also note information about any pre-existing hearing loss that the casualty might have had. After the casualty's record has been established, the time course of the hearing loss from this injury and any spontaneous resolution must be noted and, if possible, documented audiometrically.

Casualties who have suffered only auditory PBI should be placed in the delayed or minimal triage categories.

Although significant pain may result from inadequate decompression of the middle ear during a nonpressurized flight, no special precautions need to be taken when evacuating blast casualties who have suffered auditory damage.

Definitive Physical Examination

The signs of blast-related aural injury arc usually clearly visible to the examining physician, and include *(a)* tympanic-membrane perforation, *(b)* foreign material in the ear, *(c)* ossicular damage, and *(d)* inner-ear damage.

If the casualty is experiencing vertigo in the absence of other aural signs, then the medical officer should suspect closed head injury and look for other evidence of neurological compromise. Closed head injury is potentially serious and requires more sophisticated a cursory examination can provide.

Tympanic-Membrane Perforation. By far, the single most common aural injury is tympanic-membrane perforation. Although these ear problems are usually symptomatic (with otalgia, hearing loss, or tinnitus), they may also be silent.[87,88] The ear nearest the explosion is usually more severely damaged, but the blast wave can reflect off nearby walls to damage the contralateral ear.[81] The most susceptible portion of the tympanic membrane is the pars tensa; the least susceptible seems to be the pars flaccida.

The size of perforation appears to be related to the spontaneous healing rate and thus may be a useful criterion for surgical intervention. Table 9-6 lists the incidence of tympanic-membrane perforation and the spontaneous healing rate reported in the various studies.[89] One study found that as many as 90% of casualties who had a perforation that was smaller than one-third of the tympanic membrane healed by themselves, whereas the spontaneous healing rate of casualties who had a perforation larger than one-third of the membrane Inanother study, when perforations involved more than 80% of the tympanir membrane, none nf the membranes healed spontaneously. All perforations that were smaller than 80% of the membrane, however, did heal by themselves.[81] One useful index for physicians who need to estimate the likelihood of spontaneous healing might be the loss of half of the tympanic-membrane area.

The presence of a ruptured tympanic membrane indicates that the cochlea may have been damaged as well. The casualty's hearing acuity should be evaluated as soon as audiometric equipment is available

By itself, the rupture of a tympanic membrane by a blast overpressure is not likely to be the cause of significant acute disability. Fewer than 5% of such cases will involve incapacitating pain or balance disturbance. Barring infection, the vast majority will heal without intervention. Some 10%–20% will ultimately require surgical closure, and a small percentage may be chronically infected or develop cholesteatoma.

Foreign Matter. The medical officer should examinc both thc cxtcrnal auditory canal and thc middlc ear space (if perforation has occurred) for foreign matter, which may include material from the soldier's external environment or material that the soldier placed in the ear as a form of self-protection.

In addition, because the disruption of the tympanic membrane is caused by the positive phase of blast overpressure, squamous epithelium from a ruptured tympanic membrane may have been blown into the middle-ear space. The edges of the tympanic-membrane Perforation may have been inverted under the remaining portion of the membrane, and the inwardly folded perforation edges may provide an avenue for the later ingrowth of squamous epithelium.

Either the inverted edges of the perforation or squamous epithelium that has been driven into the middle ear may result in the formation of cholesteatoma over time, and the medical officer should consider this complication when planning the management of blast-injury perforations.

TABLE 9-6

TYMPANIC MEMBRANE PERFORATION

Blast Incidents	Number of Patients	Number of Perforated Tympanic Membranes	Percentage of Ears with Perforations	Percentage of Ears that Healed Spontaneously
Restaurant in Belfast, Ireland (1972)	101	66	33	83
Two pubs in Birmingham, England (1974)	111	29	13	82
Police station in Shaker Heights, Ohio (1970)	7	11	79	*
Indo-Pakistani conflict (1965)	41	52	63	78
Two explosions: sinking of destroyer by missiles (1967); explosion of truck carrying explosives (1970)	77	82	53	88
Combat-related blast incidents: truck bombing, rocket-propelled grenade, and landmine explosions	37	39	53	**

*All repaired surgically
**Not reported
Source: *Ann. Oto. Rhinol. Laryngol. 98:* 9–12, 1989

Ossicular Damage. The medical officer should carefully evaluate the integrity of the ossicular chain. Although ossicular damage is generally associated with large tympanic-membrane perforations, the ossicles may be fractured or dislocated even if the tympanum is intact.

In a study of ossicular damage in casualties who had tympanic-membrane ruptures during the Vietnam War, the most common abnormality (25%) was medial displacement of the malleus handle with disruption of the incudomalleolar joint and adherence of the tip of the malleus to the promontory.[77] Of the incus and stapes abnormalities (8.4% of cases), incudostapedial joint separation (four cases) was most common, followed by incudostapedial joint separation with fracture of the stapes superstructure (one case). In one case of ossicular damage that was sustained when a shell burst 50 yards away from a soldier, his incudomalleolar joint was dislocated, the incus was lying free in the hypotympanum, and both stapedial crura were also fractured.[86]

Inner-Ear Damage. Inner-ear damage (other than hearing loss) as a result of blast is uncommon, but medical officers should be aware that it can occur even without ossicular damage.[76,82,85,91] Inner-ear damage may include *(a)* perilymphatic fistulae in the oval window,[92] *(b)* dislocated stapes,[93] or *(c)* ruptures of the saccule, utricle, and basilar membrane.[76,82,85,91] Such injuries would be associated with a profound hearing loss and the presence of a clear otorrhea.

An otolaryngologist at a rear-echelon facility would have to make a precise delineation of the location and degree of damage.

Cholesteatoma. Most cases of cholesteatoma are believed to be the result of chronic inflammation and the subsequent development of granulomas around ectopic epithelium that has been displaced by trauma or infection. An otologic surgeon should provide long-

term follow-up surveillance to blast casualties. Although one study of 5,200 terrorist-blast casualties reported only two cases of cholesteatoma, this complication may be clinically silent for years.[76,82,85,91]

Diagnostic Screening Procedures

Evaluation of hearing sensitivity will often start with the recognition of a hearing deficit at the time of the initial interview, but quantification of aural loss will require formal audiometric testing at a higher echelon.

Audiometric measures of hearing threshold with both air and bone conduction can be used to distinguish sensorineural hearing loss (in which both will be decreased) from conductive loss (in which the bone threshold is normal). In one report, for example, thirty-seven soldiers were victims of a truck bombing, rocket-propelled grenade detonations, or landmine explosions.[79] Twenty-six of the thirty-seven soldiers (70.3%) suffered either unilateral (thirteen soldiers) or bilateral (thirteen soldiers) tympanic-membrane perforations. Thus, 52.7% of the seventy-four ears at risk were ruptured. When were given routine audiometric testing, 6.8% of them had pure conductive loss, 44.4% of them had pure sensorineural hearing loss, and 41.9% of them had a mixed hearing loss.

Auditory brainstem response (ABR) audiometry may help the medical officer evaluate blast injuries of the ear. This sophisticated technique uses a machine that measures brain-wave response to sound. ABR audiometry studies have correlated the types of hearing loss after blast injury with the prognosis for recovery.

The field surgeon is unlikely to have audiometric testing equipment available and will have to assess the degree of hearing impairment subjectively. The examiner can make a gross estimate of the casualty's global hearing by standing out of sight and testing the patient's ability to comprehend phrases spoken with varying degrees of loudness, from a conversational tone to a whisper.

Treatment

There is no specific therapy that has been shown to aid recovery from acoustic trauma, although some authorities would administer a course of dextran or corticosteroids in an attempt to limit the damage.[82]

Hearing Loss. If possible, casualties who have severe hearing loss should *(a)* avoid high noise levels, *(b)* be assigned to duties that do not require sensitive hearing, if possible, and *(c)* wear hearing protection.

Casualties who have less severe hearing losses will require individual disposition. They will likely be returned to duty, but should be advised to avoid noise and to use hearing protection to whatever degree they can in their duties.

Tympanic-Membrane Rupture. Basic treatment for a simple tympanic-membrane rupture consists of *(a)* removing debris from the external canal and *(b)* gently irrigating the canal with antiseptic solution.[21,85] In general, this injury does not require immediate attention in a combat setting. Care can be delayed for hours or days without serious adverse consequences.

Although blast-induced ruptures often resolved without surgical intervention, the chances of spontaneous healing and delayed complications were affected by *(a)* size, *(b)* location, and *(c)* time elapsed after [18,81,84,86,94]

Most perforations involving less than one-third of the tympanum will close spontaneously.[77] Blast casualties with this injury may be returned to duty that does not require sensitive hearing or expose the casualty to further auditory damage. If the perforation is small, isolated, and uncomplicated, the casualty should observe *water precautions* (that is, ensure that water or other nonsterile material is not introduced into the canal) to prevent infection during the course of healing. Neither antibiotics nor ear drops are recommended unless the casualty has an infection or drainage.[85] Auditory blast casualties will need to receive otologic examinations at regular intervals until the rupture is healed. As a rule, 1 month is usually required to heal each 10% of the tympanic membrane.[77] In some studies, perforations larger than 30% had significantly lower spontaneous-healing rates (0%–***22%);*** definitive tympanoplastic surgery would be recommended for ruptures of this size.[81,95]

Posterior-superior perforations may later develop retraction pockets on the healing membrane or cholesteatoma.[97] The medical officer should carefully remove visible debris from the middle ear.

If no healing is evident 10–15 days after the injury, then complete spontaneous healing at any later time is unlikely.[82,96] Some clinicians, however, advocate waiting as long as 6 months to make this determination. An observation period of ***2–3*** months seems reasonable as long as there are no infectious complications.

If the rupture does not close spontaneously, the physician can treat it definitively by *(a)* patching it with paper or Gelfilm or *(b)* tympanoplasty with fascial grafting, with or without ossicular reconstruction.

A perforated tympanic membrane can be patched under a local anesthetic. The medical officer should clean the ear and remove any fragments of tympanic membrane from the middle ear. Torn edges should be everted and reapproximated under sterile conditions. A Gelfoam Led should be ear for

support and a patch of Cclfilm or paper should then be applied over the perforation. This method results in consistent healing rates of more than 90%.[86] The possibility of late cholesteatoma may also be lessened by debridement and eversion of torn membrane edges.

Additional measures may include *(a)* cauterizing the edges of the perforation with trichloroacetic acid (especially if the patching is done long after the blast event), *(b)* relaxing incisions to permit better approximation of edges, and *(c)* using fascial grafts instead of paper or

Tympanoplasty is recommended for perforations that have not healed after 3–6 months or in failed attempts at patching. Tympanoplasty with a medially placed fascial graft alone has a success rate that is greater than 90%.[96] The techniques are beyond the scope of this chapter and would be done by an otologic surgeon in a rear-echelon referral facility.

Ossicular Damage, Inner-Ear Damage, and Cholesteatoma. The casualty will receive definitive therapy at a rear-echelon facility from an experienced otolar

SUMMARY

Although most of our current understanding of PBI comes from animal experiments and terrorist bombing incidents, the medical officer will need to understand PBI in a more complicated military context. Blast casualties in war will enter a medical system thdt will differ considerably from what civilian casualties would experience. For example, the number of litters that can be allocated on the battlefield—if they can he used at all—may be few, and combat casualties with PBI may have to exert themselves at levels that would be discouraged among civilian casualties, even though to do so might exacerbate their pulmonary injury and the risk of air embolism. Unlike casualties of terrorist bombings, who would most likely be transported to nearby emergency rooms in ambulances, military blast casualties would probably be evacuated by air, and would require special precautions to prevent further barotrauma from air-pressure changes. Military personnel are more likely than civilians to be in an underwater environment that is threatened by blast, and thus are at greater risk of PBI to the gastrointestinal system. Aural injuries from blast might have serious personal implications for civilian casualties, but in a military setting the loss of the ability to communicate could pose a hazard to other personnel as well.

All medical personnel should be aware that excessive volume resuscitation may worsen the casualty's gas exchange, mechanical ventilation support may exacerbate both pulmonary barotrauma and air embolism, and general anesthesia for the blast casualty will be poorly tolerated.

REFERENCES

1. Yelverton, J. T.; Damon, E. G.; Jones, R. K.; Chiffelle, T. L.; and Luft, U. C. 1971. *Effects of irradiation and blast on pulmonary function in sheep* [Technical Report 26301. Washington, DC: Defense Atomic Support Agency.

2. Phillips, Y. Y III. 1986. Primary blast injuries. *Ann. Emerg. Med.* 15:1446–1450.

3. Pyper, P. C., and Graham, W. J. H. 1982. Analysis of terrorist injuries treated at Craigavon Area Hospital, Northern Ireland. *Injury* 14:332–338.

4. White, C. S. 1968. *Rationale of treatment of primary blast injury to the lung* [Technical Progress Report DA-49-146-XZ-3721. Albuquerque, NM Lovelace Foundation for Medical Education and Research.

5. Ponn, R. B.; Zatarain, G.; Gerzberg, L.; Hottinger, C. F.; Haase, W.; and Nelsen, T. 1977. Systemic air embolism in experimental penetrating lung injuries. *J. Thorac. Cardiovasc. Surg.* 74:766–773.

6. Phillips, Y. Y 111, and Graeber, G. M. 1988. Blast injuries. In *Emergency War Surgery,* 2d U.S. ed. of *The Emergency War Surgery NATO Handbook,* edited by T. E. Bowen and R. F. Bellamy, 74–82. Washington, DC: U.S. Department of Defense.

7. Hutton, J. E., Jr. 1986. Blast lung: History, concepts and treatment. *Curr. Concepts in Trauma Care* 9:8–14.

8. Whelan, T. J. 1981. Surgical lessons learned in the care of the wounded. *Med. Bull. of the U.S. Army, Europe* 38:1–9.

9. Yelverton, J. T.; Viney, J. F.; Jojola, B. III; and Jones, R. K. 1971. *The exercise on rats at various times following blast exposure* [Technical Report DASA 2707]. Washington, DC: Headquarters, Defense Nuclear Agency.

10. Van Achterbergh, S. M. 1985. Anaesthesia for a patient injured in a landmine explosion. S. *Afr. Med. J.* 67:858–859

11. Harmon, J. W.; Sampson, J. A.; Graeber, G. M.; Phillips, Y. Y III; and Richmond, D. 1988. Readily available serum chemical markers fail to aid in diagnosis of blast injury. *J. Trauma* 28:S153–S159.

12. Melzer, E.; Hersche, M.; Fischer, D.; and Hershko, C. 1986. Disseminated intravascular coagulation and hypopotassemia associated with blast lung injury. *Chest* 89:690–693.

13. Hirsch, M., and Bazini, J. 1969. Blast injury of the chest. *Clin. Radiol.* 20:362–370.

14. Wagner, R. B., and Jamieson, P. M. 1989. Pulmonary contusion: Evaluation and classification by computed tomograpy. *North Am.* 69:31–40.

15. Caseby, N. G., and Porter, M. F. 1976. Blast injuries to the lungs: Clinical presentation, management and course *Injury* 8:1–12.

16. Huller, T., and Bazini, Y. 1970. Blast injuries of the chest and abdomen. *Arch. Surg.* 100:24–30.

17. White, C. S.; Jones, R. K.; Damon, E. G.; Fletcher, E. R.; and Richmond, D. R. 1971. *The biodynamics of airblast* [Technical Report DNA 2738T]. Washington, DC: Headquarters, Defense Nuclear Agency.

18. Cooper, G. J., Maynard, R. L.; Cross, N. L.; and Hill, J. F. 1983. Casualties from terrorist bombings. *J. Trauma* 23:955–967.

19. Weiler-Ravell, D.; Adatto, R.; and Borman J. B. 1975. Blast injury of the chest: A review of the problem and its treatment. *Isr. J. Med. Sci.* 11:268–274.

20. Roy, D. 1982. Gunshot and bomb blast injuries: A review of experience in Belfast. *J. R. Soc. Med.* 75:542–545.

21. Hadden, W. A.; Rutherford, W. H.; and Merrett, J. D. 1978. The injuries of terrorist bombing: A study of 1,532 consecutive patients. *Br. J. Surg.* 65:525–531.

22. McCaughey, W.; Coppel, D. L.; and Dundee, J. W. 1973. Blast injuries to the lung. *Anaesthesia* 28:2–9

23. Cope, Z. 1953. The general effects of blast. Chapt. 18, part 1 of *Surgery*, edited by Z. Cope, 652–664. London: Her Majesty's Stationery Office.

24. The Joint United Kingdom–United States Maritime and Marine Combat Casualty Care Workshop. 1981. *Ballistic injuries.* Alverstoke, England: Institute of Naval Medicine.

25. Uretzky, G., and Cotev, S. 1980. The use of continuous postive airway pressure in blast injury of the chest. *Crit. Care Med.* 8:486–489.

26. Adler, O. B., and Rosenherger, A. 1988. Blast injuries. *Acta Radiol.* 29:1–5

27. Fulton, R. L., and Peter, E. T. 1973. Physiologic effects of fluid therapy after pulmonary contusion. *Am. J. Surg.* 126:773–777.

28. Wuensche, O., and Scheele, G. 1970. Die anwendung des diureticums Lasix bei druckstossverletzungen (The use of the diuretic Lasix in blast injuries). *Wehrmedizin und Wehrpharmazie* 9:113–117.

29. Wang, Z.-G. 1990. Research on blast injury in China. *Chuang Shang Tsa Chih* 6:222–228.

30. SCITRAN, trans. *Treatment and cure of blast injuries* (in Chinese) [Report HT 061-831. Fort Detrick, MD: Armed Forces Medical Intelligence Command.

31. Bowen, T. E., and Bellamy, R. F., eds. 1988. *Emergency war surgery.* 2d US. ed. of *The emergency war surgery NATO handbook.* Washington, DC: U.S. Department of Defense.

32. Buffe, P.; Cudennec, Y. F.; Baychelier, J. L.; and Grateau, P. 1987. Les lesions laryngées par explosion (Laryngeal lesions caused by explosions). *Ann. Otolaryngol. Chir. Cervicofac.* 104:379–382.

33. Cooper, G. J.; Maynard, R. L.; Aldous, F. A. B.; Evans, V. A.; and Kenward, C. E. 1983. *Nonpenetrating injury to the chest: An experimental study of the biomechanical principles of lung injury, the pathology of pulmonary contusions and their acute physiological effects (U)* [Technical Paper 3441. Porton Down, England: Chemical Defense Establishment.

34. Jonsson, A. 1979. *Experimental investigations on the mechanisms of lung injury in blast and impact exposure.* Ph.D. diss. no. 80, Department of Surgery, Linkoping University, Stockholm, Sweden.

35. Gordon-Taylor, G. 1953. Abdominal injuries due to underwater explosion (immersion blast). In *Surgery,* edited by Z. Cope, 664–672. London: Her Majesty's Stationery Office.

36. Rignault, D. P., and Deligny, M. C. 1989. The 1986 terrorist bombing experience in Paris. *Ann. Surg.* 209:368–373.

37. Wall, S. D.; Federle, M. P.; Jeffrey, R. B.; and Brett, C. M. 1983. CT of unsuspected pneumothorax after blunt abdominal trauma. ***Am.** J. Roentgenol.* 141:919–921.

38. Damon, E. G.; Yelverton, J. T.; Luft, U. **C.;** Mitchell, K., Jr.; and Jones, R. K. 1971. Acute effects of air blast on pulmonary function in dogs and sheep. *Aerospace Med.* 42:1–9.

39. Damon, E. G.; Yelverton, J. T.; Luft, U. C.; and Jones, R. K. 1970. *Recovery of the respiratory system following blast injury* [Technical Report DASA 2580]. Albuquerque, NM: Lovelace Foundation for Medical Education and Research.

40. Clemedson, C.-J. 1957. Respiratory and circulatory vagal reflexes in rabbits exposed to high explosive shock waves. ***Am.** J. Physiol.* 190:467–472.

41. Clemedson, C.-J.; Hultman, H.; and Gionberg, B. 1953. Respiration and pulmonary gas exchange in blast injury. *J. Appl. Physiol.* 6:213–220.

42. Mellor, S. G. 1988. The pathogenesis of blast injury and its management. *Br. J. Hosp. Med.* 39:536–539.

43. Hoff, B. H.; Flemming, D. C.; and Sasse, F. 1979. Use of positive airway pressure without endotracheal intubation. *Crit. Care Med.* 7:559–562.

44. Greenbaum, D. M.; Millen, J. E.; Eross, B.; Snyder, J. V.; Grenvik, A.; and Safar, P. 1976. Continuous positive airway pressure without tracheal intubation in spontaneously breathing patients. *Chest* 69:615–620.

45. Marini, J. J., and Culver, B. H. 1989. Systemic gas embolism complicating mechanical ventilation in the adult respiratory distress syndrome. *Ann. Intern. Med.* 110:699–703.

46. Hara, K. S., and Prakash, U. B. S. 1989. Fiberoptic bronchoscopy in the evaluation of acute chest and upper airway trauma. *Chest* 96:627–630.

47. Damon, E. G.; Henderson, E. A.; and Jones, R. K. 1973. *The effects of intermittent positive pressure respiration on occurrence of air embolism and mortality following primary blast injury* [Technical Report DNA 2989F]. Washington, DC: Headquarters, Defense Nuclear Agency.

48. Kogutt, M. S. 1978. Systemic air embolism secondary to respiratory therapy in the neonate: Six cases including one survivor. *AJR **Am.** J. Roentgenol.* 131:425–429.

49. Brown, Z. A.; Clark, J. M.; and Jung, A. L. 1977. Systemic gas embolus: A discussion of its pathogenesis in the neonate, with a review of the literature. *Am. J. Dis. Child* 131:984–985.

50. Hwang, T.-L.; Fremaux, R.; Sears, E. S.; MacFadyen, B.; Hills, B.; Mader, J. T.; and Peters, B. 1983. Confirmation of cerebral air embolism with computerized tomography. ***Ann.*** *Neurol.* 13:214–215.

51. Hill, J. F. 1979. Blast injury with particular reference to recent terrorist bombing incidents. *Ann. R. Coll. Surg. Engl.* 61:4–11.

52. Frykberg, E. R., and Tepas, J. J. III. 1988. Terrorist bombing: Lessons learned from Belfast to Beirut. *Ann. Surg.* 208:569–576.

53. Mellor, S. G., and Cooper, G. J. 1989. Analysis of 828 servicemen killed or injured by explosion in Northern Ireland 1970-84: The Hostile Action Casualty System. *Br. J. Surg.* 76:1006–1010.

54. Stapczynski, J. S. 1982. Blast injuries. *Ann. Emerg. Med.* 11:687–694.

55. Stapcynski, J.S. 1985. Blast injuries. In *Current Emergency Therapy '85,* edited by R. F. Edlich and D. A Spyker, 293–298. Rockville, M D Aspen Systems Corporation.

56. Damon, E. G., and Jones, R. K. 1971. *Comparative effects of hyperopia and hyperbaric pressure in the treatment of primary* [Technical Report DASA 2708]. Washington, DC: Headquarters, Defense Nuclear Agency.

57. Bond, G. F. 1977. Arterial gas embolism. In *Hyperbaric Oxygen Therapy,* edited by J. C. Davis and T. K. Hunt, 141–152. Bethesda, MD: Undersea Medical Society, Inc.

58. Leitch, D. R., and Green, R. D. 1986. Pulmonary barotrauma in divers and the treatment of cerebral arterial embolism. *Aviat. Space Environ. Med.* 57:931–938.

59. The Undersea Hyperbaric Medical Society is located at 9650 Rockville Pike, Bethesda, Maryland 20814 (telephone 301-571-1818).

60. Spampinato, N.; Stassano, P.; Gagliardi, C.; Tufano, R.; and Iorio, D. 1981. Massive air embolism during cardiopulmonary bypass: Successful treatment with immediate hypothermia and circulatory support. ***Ann.*** *Neurol.* 32:602–603.

61. Harmon, J. W., and Haluszka, M. 1983. Care of blast-injured casualties with gastrointestinal injuries. *Milit. Med.* 148:586–588.

62. Pugh, H. L. 1943. Blast injuries. In *The Surgical Clinics of North America,* edited by W. H. Cole, 1589–1602. Philadelphia: W. B. Saunders Company.

63. Katz, E.; Ofek, B.; Adler, J.; Abramowitz, H. B.; and Krausz, M. M. 1989. Primary blast injury after a bomb explosion in a civilian bus. *Ann. Surg.* 209:484–488.

64. Donohue, J. H.; Federle, M. P.; Griffiths, B. G.; and Tmnkey, D. D. 1987. Computed tomography in the diagnosis of blunt intestinal and mesenteric injuries. *J. Trauma* 27:11–17.

65. Jones, I. K.; Walsh, J. W.; and Maull, K. I. 1983. Diagnostic imaging in blunt trauma of the abdomen. *Surg. Gynecol. Obstet.* 157:389–398.

66. Stigall, K. E., and Dorsey, J. S. 1989. Transection of the first portion of jejunum from blast injury in accidental discharge of a (2.75inch aircraft) rocket from an F15. *Milit. Med.* 154:431–433.

67. Sherman, J. C.; Delaurier, G. A.; Hawkins, M. L.; Brown, L. G.; Treat, R. C.; and Mansberger, A. R., Jr. 1989. Percutaneous peritoneal lavage in blunt trauma patients: A safe and accurate diagnostic method. *J. Trauma* 29:801–805.

68. Grüessner, R.; Mentges, B.; Diiber, Ch.; Riickert, K.; and Rothmund, M. 1989. Sonography versus peritoneal lavage in blunt abdominal trauma. *J. Trauma* 29:242–244.

63. Marx, J. A.; Moore, E. E.; Jorden, R. C.; and Eule, J., Jr. 1385. Limitations of computed tomography in of acute abdominal trauma: A prospective comparison with diagnostic peritoneal lavage. *J. Trauma* 25:933–937.

70. Meyer, D. M.; Thal, E. R.; Weigelt, J. A.; and Redman, H. C. 1989. Evaluation of computed tomography and diagnostic peritoneal lavage in blunt abdominal trauma. *J.*

71. Peitzman, A.; Makaroun, M. S.; Slasky, B. S.; and Ritter, P. 1986. Prospective study of computed tomography in initial management of blunt abdominal trauma. *J.*

72. Federle, M. P. 1983. Computed tomography of blunt abdominal trauma. *Radiol. Clin.* North *Am.* 21:461–475

73. Fischer, R. P.; Miller-Crotchett, P.; and Reed, R. L. II. 1988. Gastrointestinal disruption: The hazard of nonoperative management in adults with blunt abdominal injury. *J. Trauma* 28:1445–1449.

74. Parks, T. G. 1986. Gunshot and bomb blast injuries of the large intestine. Brit. *J. Clin. Pract.* 40:7–10.

75. Phillips, Y. Y III; Mundie, T. G.; Hoyt, R.; and Dodd, K. T. 1989. Middle ear injury in animals exposed *to* complex blast waves inside an armored vehicle. *Ann. Otol. Rhinol. Laryngol. Suppl.* 140:17–22.

76. Kerr, A. G., and Bryne, J. E. T. 1975. Concussive effects of bomb blast on the ear. *J. Laryngol.* Otol. 89:131–143.

77. Sudderth, M. E. 1974. Tympanoplasty in blast-induced perforation. *Arch. Otolaryngol.* 99:157–159.

78. Anderson, J. 1984. An audiometric survey of royal artillery gun crews following "Operation Corporate." *J. R. Army Med. Corps* 130:100–108.

79. Brown, J. R. 1985. Noise-induced hearing loss sustained during land operations in the Falkland Islands campaign. *J. Soc. Occup. Med.* 35:44–54.

80. Melinek, M.; Naggan, L.; and Altman, M. 1976. Acute acoustic trauma—A clinical investigation and prognosis in **433** symptomatic soldiers. *Isr. J. Med. Sci.* 12:562–569.

81. Pahor, A. L. 1981. The ENT problems following the Birmingham bombings. *J.* Laryngol. Otol. 95:399–406.

82. Kerr, A. G., and Bryne, J. E. T. 1975. Blast injuries of the ear. *Br. Med. J.* 1:559–561.

83. Pratt, H.; Goldsher, M.; Netzer, A.; Shenhav, R. 1985. Auditory brainstem evoked potentials in blast injury. *Audiology* 24:297–304.

84. Ziv, M.; Philipsohn, N. C.; Leventon, G.; and Man, A. 1973. Blast injury of the ear: Treatment and evaluation. *Milit. Med.* 8:811–813.

85. Kerr, A. G. 1980. Trauma and the temporal bone: The effects of blast on the ear. *J. Laryngol. Otol.* 94107–110.

86. Messervy, M. 1972. Unilateral ossicular disruption following blast exposure. *Laryngoscope* 82:372–375.

87. Walby, A. P., and Kerr, A. G. 1986. Hearing in patients with blast lung. *J. Laryngol. Otol.* 100:411–415.

88. Brismar, B., and Bergenwald, L. 1982. The terrorist bomb explosion in Bologna, Italy, 1980: An analysis of the effects and injuries sustained. *J.*

89. Chait, R. H.; Casler, J.; and Zajtchuk, J. T. 1989. Blast injury of the ear: Historical perspective. *Ann. Otol. Rhinol. Laryngol.* 98:9–12.

90. Singh, D., and Ahluwalia, K. J. S. 1968. Blast injuries of the ear. *J. Laryngol.* Otol. 82:1017–1028.

91. Kerr, A. G. 1978. Blast injuries to the ear. *Practitioner* 221:677–682.

92. Strong, S. 1975. *Proceedings of the Irish Otolaryngological Society.*

93. Ruedi, L., and Furrer, W. n.d. *Das akustische* Trauma. Basel: S. Karger.

94. Gapany-Gapanavicius, B.; Brama, I.; and Chisin, R. 1977. Early repair of blast ruptures of the tympanic membrane. *J. Laryngol. Otol.* 91:565–573.

95. Ruggles, R. L., and Votypka, R. 1973. Blast injuries to the ear. *Laryngoscope* 83:974–976.

96. Kanimturk, E. 1979. Clinical evaluation of traumatic perforations of the tympanic membrane. *Int. Rev. Army Navy Air* Force *Med. Serv.* 6:523–526.