Chapter 19

THORACIC INJURIES

LAWRENCE L. YEE, M.D.[*]; ALEXANDER S. RUBIN, M.D.[†]; AND RONALD F. BELLAMY, M.D.[‡]

[*]*Formerly, Major, Medical Corps, U.S. Army; Staff Anesthesiologist, Walter Reed Army Medical Center, Washington, D. C. 20307-5001; currently, Staff Anesthesiologist, O'Connor Hospital, 2105 Forest Avenue, San Jose, California 95128*

[†]*Formerly, Major, Medical Corps, U.S. Army; Staff Anesthesiologist, Walter Reed Army Medical Center, Washington, D. C. 20307-5001; currently, Staff Anesthesiologist, Frederick Memorial Hospital, Frederick, Maryland 21702*

[‡]*Colonel, Medical Corps, U.S. Army; Managing Editor and Officer in Charge, Textbook of Military Medicine, Borden Institute, Walter Reed Army Medical Center, Washington, D. C. 20307-5001*

INTRODUCTION

The casualty suffering thoracic trauma usually poses a lesser challenge to the military trauma anesthesiologist than might be supposed. As we discussed in Chapter 1, Combat Trauma Overview, the reasons for this are simple: (1) most casualties who are wounded in the chest die on the battlefield, and (2) only a minority of those who reach a medical treatment facility alive require a formal thoracotomy. Most survivors require only the insertion of a chest tube. Thus, thoracic trauma in combat casualties has some of the all-or-none characteristics of an airplane crash: the victim is either killed outright or has a injury that is treatable by simple interventions. Nevertheless, there are exceptions (eg, the casualty who suddenly exsanguinates when a mediastinal hematoma ruptures, the casualty whose tracheobronchial tree is filling with blood, the casualty who suddenly develops massive arterial air embolism when mechanical ventilation is started) that can push even the most competent anesthesiologists to their limits.

TYPES OF INJURY

Thoracic trauma can be the result of one or more of the four basic mechanisms of injury: penetrating (ballistic), blunt, blast, and thermal. Thermal injury resulting from the inhalation of hot, poisonous gases is an important cause of combat mortality and morbidity in the navy and among soldiers who crew armored fighting vehicles. Inhalation injury is discussed in Chapter 22, Burn Injuries; therefore, it will not be further discussed in this chapter.

Penetrating missiles have been far and away the major source of thoracic trauma in combat casualties, as seen in Table 19-1, which presents data from two countries and two wars. The data from the U.S. Army in the Vietnam War covers a 14-month period (September 1968–November 1969) at the 24th Evacuation Hospital.[1] During this interval, the hospital served as a specialty center for neurological, head and neck, and thoracic casualties. Data from the Rambam Medical Center covers the period 6 to 29 June 1982 (during the military operation in Lebanon that the Israelis call Peace in Galilee).[2]

Because penetrating trauma is the cause of the overwhelming majority of combat casualties with thoracic injuries, and because it is generally agreed that missile wounds are distributed randomly, the overall prevalence of thoracic trauma should approximate the fraction of the body surface area that overlies the thorax: about 16 in 100. Most casualties with thoracic trauma are killed; therefore, the actual percentage of the casualty population who will require treatment for thoracic trauma will be somewhat smaller and usually ranges between 5% and 10%.[3]

Penetrating Missiles

The nature of the medical problems caused by penetrating missiles, whether they are bullets from small arms or fragments from explosive munitions, is due to three factors:

1. the function of the organ or organs struck,
2. the physical characteristics of the missile, and
3. the biophysics of the interaction between the missile and the tissue that is struck.

It should be readily apparent that the first factor—the nature of the organ hit—is of paramount importance in determining the medical outcome of a ballistic injury. A missile wound of the brainstem is likely to be much more serious than a hit made by a similar missile to the little toe. Unfortunately, the thoracic cavity has more than its share of organs—the heart, great vessels, and lungs—the injury of which will be immediately catastrophic.

The second factor—the physical characteristics of the missile—is usually assessed in terms of kinetic energy (calculated as $\frac{1}{2}MV^2$, where M represents the projectile's mass and V its velocity). Tissue destruction caused by a ballistic injury is related in a general sense to the kinetic energy transferred to the target tissue such that

$$\frac{1}{2}M(V_{ent} - V_{ext})^2$$

where V_{ent} and V_{ext} represent the velocity of the missile when it enters and exits the body, respectively. When the missile does not exit but stops within the body, the kinetic energy transferred equals $\frac{1}{2}MV_{ent}^2$. Fragments usually do not exit from the body, while bullets, unless they deform or fragment, frequently cause perforating wounds. Bullets that fragment (ie, rounds fired from the M16 series of assault rifles) or deform (ie, many

TABLE 19-1

ETIOLOGY OF THORACIC TRAUMA

	24th Evacuation Hospital US Army, Vietnam[1]	Rambam Medical Center Haifa, Israel[2]
Total Casualties	7,500	938
Chest Trauma	900*	64
Intrathoracic Injury	547	63
Fragment	443	41
Bullet	76	7†
Blunt	28	6‡
Blast		6§

*Including superficial missile wounds as well as blunt trauma to the chest wall but without any intrathoracic component

†There were, in addition, two casualties with both bullet and fragment wounds

‡There was a seventh casualty with both fragment and blunt thoracic trauma

§All six casualties with pulmonary blast injury also had fragment wounds of the thorax

Data sources: (1) McNamara JJ, Messersmith JK, Dunn RA, Molot MD, Stremple JF. Thoracic injuries in combat casualties in Vietnam. *Ann Thor Surg*. 1970;10:389–401. (2) Rosenblatt M, Lemer M, Best LA, Peleg H. Thoracic wounds in Israeli battle casualties during the 1982 evacuation of wounded from Lebanon. *J Trauma*. 1985;25:350–354.

commonly available bullets used by civilian hunters and law-enforcement agencies) typically are associated with much greater energy transfer than occurs with intact bullets that cause perforating wounds.

A second important physical characteristic is the shape of the projectile. Streamlined bullets can pass through tissue with little reduction in velocity, while irregular fragments from explosive munitions slow rapidly and, therefore, can transfer a much greater fraction of their kinetic energy—with a corresponding increase in the potential for tissue damage.

The third factor determining the medical consequences of ballistic injury has to do with the interaction of the projectile and the tissue along its pathway. Obviously, the longer the wound tract, the more damage that is likely to occur. This is true because (*a*) the bullet passes through more tissue, and (*b*) it is more likely that the bullet will destabilize (either *yaw* [ie, the long axis of the bullet deviates from the direction of flight] or *tumble* [ie, the bullet flips end over end]), greatly increasing energy transfer. The density of the tissue is another determinant of the projectile–target interaction. The more dense the tissue, the more complete the energy transfer. Thus, projectile energy transfer is maximal in bone but minimal in the least-dense body tissue, the lung. (A more complete discussion of the biophysics of wound ballistics can be found in the Textbook of Military Medicine volume *Conventional Warfare: Ballistic, Blast, and Burn Injuries*.)[4]

Wound ballistics as it applies to the thorax is best summarized in the official history of thoracic surgery in World War II:

> [H]igh-velocity missiles that traverse the pulmonary tissue therefore often cause surprisingly little pulmonary damage. The high immediate lethality of high-velocity wounds of the chest is apparently directly related to the percentage chance of damage to vital structures, particularly the heart and great vessels. If the high-velocity missile does not inflict a mortal wound, then it often traverses the chest with considerably less damage to the thoracic contents than is caused by a low-velocity shell fragment.[5(p59)]

Blunt and Blast Injuries

In the field, nonpenetrating injuries to the chest are caused either by forceful contact with a blunt object or by blast overpressure. Blunt chest trauma such as may occur in motor vehicle accidents results from gross compression of the thorax, which may cause rib fractures and a variable degree of displacement of intrathoracic viscera such as the heart. In extreme circumstances, the heart may be so displaced that stretch-and-shear strain affecting the proximal descending aorta may cause it to rupture. With blunt trauma, but not with blast injury, the more serious the injury, the greater the likelihood of rib fractures. Fractures of the upper five ribs are usually associated with the most serious, and often occult, injuries.

The exact mechanisms that produce visceral injury within the thoracic cavity following blunt and blast injury are poorly understood, but it is no longer thought that acceleration–deceleration of the body is an important mechanism *per se*. Extensive experimental studies using living animals and human cadavers indicate that the probability of injury can best be understood in terms of (*a*) the magnitude of compression of the thorax and (*b*) the velocity at which the compression occurs (Figure 19-1).[6,7]

When compression is applied slowly (< 1 m/s; eg, in vigorous closed-chest cardiopulmonary resuscita-

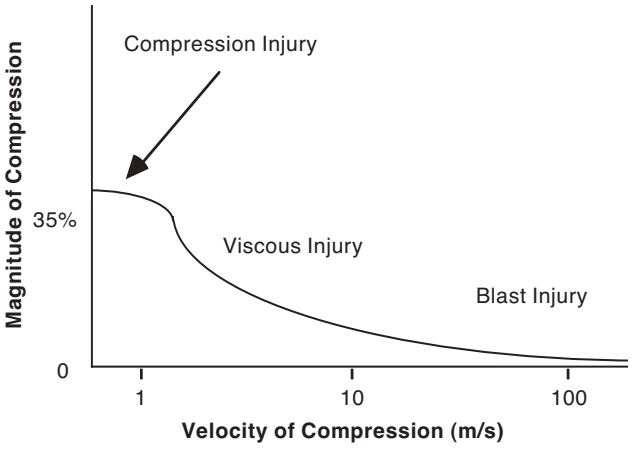


Fig. 19-1. In this depiction of the relation between the amount and the speed of compression, the curved line indicates a constant probability of the occurrence of a thoracic injury of a specified lethality. The magnitude of compression is given as the percentage of the anteroposterior diameter of the thorax. Data sources: (1) Viano DC, Lau IA. A viscous tolerance criterion for soft tissue injury assessment. *J Biomechanics.* 1988;21(5):387–399. (2) Viano DC. Live fire testing: Assessing blunt impact and acceleration injury vulnerabilities. *Milit Med*. 1991;156:589–595.

tion), a decrease in the thoracic anteroposterior diameter of at least one third must occur before there is a high risk of damage to internal viscera. However, when compression occurs at a faster velocity (> 3 m/s), the same injury can occur with much less compression. When compression is applied very slowly, the viscera have time to adjust to the distortion produced by their displacement. With more rapid compression, however, this compensation is impaired because it involves a time-dependent variable: namely, the internal displacement of layer upon layer of tissue as they slide over one another. The sliding is resisted by the inherent viscosity of the tissue much as a the viscosity of a flowing liquid causes hydraulic resistance. When the sliding cannot keep up with the velocity of compression, shearing forces arise that cause tissue laceration, rupture, or fractures. Such trauma, which results from a mechanical process that is determined by both the velocity of the compression and the viscoelasticity of the tissue, is known as a *viscous* injury.

Blast injury may be thought of as occupying the opposite end of the compression–velocity continuum depicted in Figure 19-1. With blast injury, the compression causes a tiny displacement (measured in a fraction of a millimeter) that occurs at a speed approaching that of sound. Blast overpressure is especially likely to injure very light, feebly supported structures such as the alveolar capillaries and tympanic membranes.

OVERVIEW OF COMBAT THORACIC TRAUMA

As seen in reports from three recent wars (the experience of the U.S. Army's 24th Evacuation Hospital in Vietnam[1]; Rambam Medical Center in Haifa, Israel[2]; and the Basrah, Iraq, Teaching Hospital in a 6-month period in 1981 during the Iran–Iraq War[3]), the most common thoracic clinical problems were combined hemothorax and pneumothorax, hemothorax, pneumothorax, pulmonary contusion, cardiac tamponade, pulmonary hematoma, rib or sternal fractures or both, perforation of the diaphragm, and laceration of the heart or great vessels (Table 19-2). These data clearly show that the most common medical treatment problems—collections of blood and air within the pleural space—are the result of pulmonary lacerations. It is important to recognize that the thoracic injury is unlikely to be the only injury present: 85% of the casualties in the 24th Evacuation Hospital report[1] and 80% of the casualties reported from Rambam[2] had one or more additional injuries involving another part of the body.

The specific nature of the medical interventions required to treat combat casualties with thoracic injuries is shown in Table 19-3. It is apparent that only a minority of thoracic casualties required a formal thoracotomy.

When treating a casualty with a thoracic injury, the military trauma anesthesiologist is much more likely to provide anesthesia for a major operation in some other body part than for an operation on the thorax. Published series, with the exception of the work of one surgeon in Lebanon (which is discussed below), indicate that a formal thoracotomy is needed in only about 10% to 20% of casualties with thoracic injuries who reach a military hospital alive.[8] In most thoracic casualties, the interventions required for the treatment of the thoracic injury consist of inserting a chest tube and providing local care to the wounds of entrance and exit. However, military trauma anesthesiologists should be aware that some thoracic surgeons perform what is really

TABLE 19-2

DIAGNOSIS OF THORACIC CASUALTIES

Diagnosis	24th Evac. Hospital US Army, Vietnam[1]	Rambam Medical Center Haifa, Israel[2]	Basrah Teaching Hospital, Iraq[3]
Combined hemo- and pneumothorax	230	15	
Hemothorax	199	9	81
Pneumothorax	100	13	37
Pulmonary contusion	99	5	20
Cardiac tamponade	7	1	4
Pulmonary hematoma		15	
Rib/sternal fractures		14	12*
Diaphragmatic perforation			17
Heart or great-vessel laceration			4

*Listed as flail chest

Data sources: (1) McNamara JJ, Messersmith JK, Dunn RA, Molot MD, Stremple JF. Thoracic injuries in combat casualties in Vietnam. Ann Thor Surg. 1970;10:389–401. (2) Rosenblatt M, Lemer M, Best LA, Peleg H. Thoracic wounds in Israeli battle casualties during the 1982 evacuation of wounded from Lebanon. J Trauma. 1985;25:350–354. (3) Suleman ND, Rasoul HA. War injuries of the chest. *Injury*. 1985;16:382–384.

a mini-thoracotomy when they carry out local wound management. Under general anesthesia, one or both wounds are extended so that it is possible to inspect the pleural space and lung and, if necessary, to repair accessible intrathoracic injuries.

Perhaps medical officers will need to reconsider the consensus that only a minority of thoracic casualties require a formal thoracotomy. The most compelling evidence stems from the experience of one civilian surgeon who treated 1,992 thoracic casualties of the civil war in Lebanon (1969–1982).[9] Of the casualties this surgeon treated, 282 had a cardiac injury, 1,251 had a noncardiac thoracic injury as the major treatment problem, and 456 had one or more injuries in addition to thoracic trauma. Of the noncardiac thoracic casualties, a formal thoracotomy

TABLE 19-3

THERAPEUTIC INTERVENTIONS IN THORACIC TRAUMA

Procedure	24th Evac. Hospital US Army, Vietnam[1]	Rambam Medical Center Haifa, Israel[2]	Basrah Teaching Hospital, Iraq[3]
Tube thoracostomy	448	38	113
Thoracotomy	78	6	11
Debridement of thoracic wounds		30	
Thoracentesis or no treatment	2	26	31
Major operation outside the thorax	~ 400	35	

Data sources: (1) McNamara JJ, Messersmith JK, Dunn RA, Molot MD, Stremple JF. Thoracic injuries in combat casualties in Vietnam. *Ann Thor Surg*. 1970;10:389–401. (2) Rosenblatt M, Lemer M, Best LA, Peleg H. Thoracic wounds in Israeli battle casualties during the 1982 evacuation of wounded from Lebanon. *J Trauma.* 1985;25:350–354. (3) Suleman ND, Rasoul HA. War injuries of the chest. *Injury*. 1985;16:382–384.

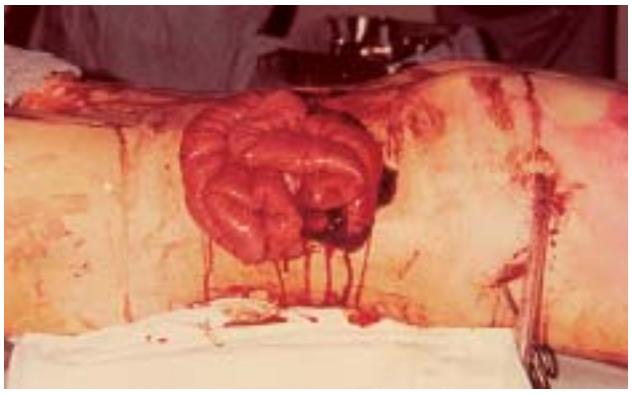


Fig. 19-2. A shell fragment entered this casualty's chest at the inferior-posterior portion of the left hemithorax at the site of the hemostat. It passed through the left lung, diaphragm, spleen, and small bowel before exiting through the left upper quadrant. The small bowel has eviscerated through the wound of exit. Notice that the chest tube is vented to the atmosphere. Suction applied to the tube would have caused ambient air to be sucked through the hole in the diaphragm into the thorax. Photograph: Wound Data and Munitions Effectiveness Team slide collection.

was performed in 818 (54.5% of the total procedures), and of these, an anatomical pulmonary resection was performed in 310. Although the U.S. Army Medical Department's experience during the Persian Gulf War was quite limited, anecdotal observations[10] from that war suggest that a greater fraction of thoracic casualties was treated by thoracotomy than during the Vietnam War. It is likely that in future wars, thoracotomy will increasingly be established as the standard of care for thoracic casualties.

Military trauma anesthesiologists need also to be aware of a variant of thoracic trauma: the thoracoabdominal injury (Figure 19-2). In about 10% of the casualties seen during World War II, the wounding missile was found to have passed from the chest through the diaphragm into the abdomen.[11] The vast experience of surgeons in World War II indicates that in most such casualties, the abdominal component was the more life threatening. This is probably because the casualties who had more-serious thoracic components died before they could be evacuated from the battlefield. The anesthetic management of the casualty with a thoracoabdominal wound will be similar to that of any abdominal casualty but with this important exception: unimpaired drainage of air and blood from the chest must be assured prior to the induction of anesthesia. Once the abdomen is opened, the hole in the diaphragm establishes a direct connection between the atmosphere and the left pleural space. Collapse of the left lung is prevented by positive-pressure ventilation through an endotracheal tube and a functioning chest tube.

GENERAL PRINCIPLES OF MANAGEMENT

Indications for Thoracotomy

Generally accepted indications for immediate or early thoracotomy are as follows[12]:

- an opacified hemothorax on the initial radiograph;
- initial drainage of 1,500 mL of blood, followed by 500 mL or more during the next hour;
- drainage of 200 to 300 mL of blood per hour for more than 4 hours;
- massive air leak with continuous bubbling throughout the respiratory cycle;
- radiographic evidence of massive pulmonary contusion or hematoma, with clinical and laboratory evidence of a life-threatening shunt or airway compromise secondary to bleeding into the airway; and
- physical signs of pericardial tamponade or suspicion of tamponade or shock, and radiographic evidence of a missile in proximity to the heart.

Most casualties with penetrating thoracic injuries will require some type of local wound management, which may or may not require general anesthesia. Casualties with large chest defects, such as would be associated with a sucking chest wound, will certainly require operative closure using general endotracheal anesthesia.

Preoperative Evaluation and Preparation

The U.S. Army Medical Department was one of the first supporters of the American College of

EXHIBIT 19-1

INITIAL MANAGEMENT OF TRAUMA VICTIMS

In the American College of Surgeons' Advanced Trauma Life Support (ATLS) approach, the initial management of the trauma victim is carried out in the following sequence, with the definitive-care phase being done later:

Primary Survey
- *A*irway management with cervical spine control
- *B*reathing and ventilation
- Circulation with hemorrhage control
- Neurological status
- Exposure

Resuscitation, including
- Inserting airways, chest tubes, and large-bore intravenous lines
- Infusing crystalloid fluid
- Obtaining blood samples
- Inserting urinary and gastric catheters
- Establishing vital-sign monitors
- Obtaining appropriate radiographic studies

Secondary Survey
- History
- Physical examination, including the following areas:
  - Head
  - Maxillae and face
  - Cervical spine and neck
  - Chest
  - Abdomen
  - Perineum/rectum/vagina
  - Musculoskeletal system
  - Neurological systems
- Additional laboratory and radiographic studies as needed

Source: Committee on Trauma, American College of Surgeons. Initial assessment and management. In: *Advanced Trauma Life Support Program for Physicians: Instructor Manual.* 5th ed. Chicago, Ill: American College of Surgeons; 1993: 17–37.

Surgeons' Advanced Trauma Life Support (ATLS) approach to the initial management of the trauma victim and has long emphasized such training—especially for medical personnel assigned to the field echelons. Military anesthesiologists serving at third- or fourth-echelon hospitals should, therefore, expect to find that the acute lifesaving interventions—the ATLS ABCs (airway, breathing, and circulation)—have already been performed before the casualty arrives at their hospital (Exhibit 19-1). Nevertheless, there are exceptions. In fact, during much of the Vietnam War, given the atrophy of the unit and division echelons of care, casualties commonly arrived at third-echelon hospitals without having received any care.[13] An equivalent situation—the lack of a mature field medical system—will likely exist in the future whenever the U.S. military becomes involved in peacekeeping or nation-building missions. As a member of the medical unit, the anesthesia provider should be involved in the resuscitation the moment the patient arrives to the receiving area.

After the initial management has been performed, the military anesthesiologist will be involved in the definitive care phase of management. Because the definitive-care phase may involve an emergency thoracotomy, the anesthesia provider should perform a specific preanesthesia evaluation only if time permits. In the conscious patient, a brief history and physical examination should be taken (Exhibit 19-2); laboratory investigation should include recent arterial blood-gas and hematocrit levels.

Because time is of the essence when dealing with trauma patients, it is important to have a resuscitation area and an operating room prepared to manage a trauma victim. The anesthesia machine and monitors should be ready for immediate use. Intravenous tubing and fluid warmers should be available for use, requiring only priming with the intravenous fluid when the patient arrives. Syringes should be labeled and cardiac drugs immediately available. A functioning defibrillator should also be ready.

The essential step that must be taken in preparing the casualty with thoracic injuries for general anesthesia, especially when the operation also involves another part of the body, is to assure that the potential for the development of an intraoperative hemothorax or pneumothorax is minimized by the placement of a functioning chest tube. Finally, it cannot be stressed too greatly

that the military trauma anesthesiologist managing the casualty with a penetrating thoracic injury will constantly strive to keep the airway free of secretions.

Monitoring

In addition to the usual noninvasive monitors (electrocardiograph, pulse oximeter, end-tidal CO_2 monitor, automatic blood pressure cuff, precordial stethoscope), arterial, central venous pressure, and urinary output monitoring are essential. However, precious time should not be spent attempting to place these monitors in unstable, exsanguinating patients.

Induction of Anesthesia

Unconscious, moribund patients are intubated immediately without anesthesia, and surgery is performed without anesthesia until the patient's vital signs or state of consciousness indicate a need for anesthesia. The anesthesiologist is performing a resuscitation in this scenario, rather than an anesthetic. Concerns about possible intraoperative recall may be overstated: providing an ideal, complete anesthetic to a trauma patient at the risk of hemodynamic instability and possible cardiac arrest is inappropriate. Scopolamine may be administered as an amnestic agent, but valuable time should not be spent to locate or administer this drug when other, more-important lifesaving tasks need to be performed. Midazolam may be used but its hypotensive effect, particularly in the presence of hypovolemia or when given concurrently with narcotics, may preclude its use in the trauma patient. Patients in a state of shock are particularly susceptible to the adverse effects of thiobarbiturates, which should be administered only with extreme caution. To allow the surgeon to arrest hemorrhage, ketamine has been advocated as the drug of choice for induction of anesthesia in hypovolemic, hemorrhaging patients who require anesthesia (for further discussion, see Chapter 10, Intravenous Anesthesia). However, the direct myocardial-depressant effects of ketamine may be unmasked in the patient who is severely physically stressed and is relying on maximal intrinsic catecholamine secretion to maintain adequate vital signs. In most cases, though, the anesthesiologist will have had sufficient time to replace intravascular fluid deficits; therefore, thiobarbiturates or other induction agents can safely be used. Trauma is frequently associated with decreased gastric emptying, which places these patients at greater risk of aspiration pneumonitis; a rapid-sequence induction after preoxygenation is usually indicated. An awake intubation must certainly be considered in patients with obvious or suspected airway abnormalities.

EXHIBIT 19-2

HISTORY AND PHYSICAL EXAMINATION OF THE CONSCIOUS PATIENT

Brief history:

1. Review of systems
2. Past medical history
3. Past surgical history
4. Previous anesthesia difficulties
5. Family history of anesthetic complications
6. Current medications
7. Allergies
8. Time of last oral intake

Brief physical examination:

1. Global assessment
2. Evaluation of the airway
3. Respiratory and cardiovascular examinations
4. Vital signs

Maintenance of Anesthesia

Successful resuscitation of patients with major chest trauma requires teamwork; blood and fluid replacement must be prompt and given in appropriate quantities. If patients do not respond to volume replacement, then ongoing hemorrhage, tension pneumothorax, or pericardial tamponade must be suspected. Loss of infused fluid through an undiagnosed hole in the superior or inferior vena cava may explain why some patients fail to respond to fluid therapy. After the surgeon has entered the chest cavity, compression of the aorta above the site of bleeding may provide the valuable time needed to restore blood volume. Although patients in shock require little, if any, anesthesia, their consciousness rapidly returns when the blood volume

is restored. At this point, anesthesia can be maintained with oxygen, narcotics, neuromuscular blocking drugs, amnestics, and small quantities of inhalational agents, if required. It is prudent to avoid nitrous oxide in patients with chest trauma because the gas tends to collect in the dependent areas of the lung. The risk is that nitrous oxide will expand in the closed chest and create a pneumothorax. (Nitrous oxide is not available in deployable hospitals.)

Special considerations with the use of neuromuscular blocking agents in the trauma patient deserve mention. Succinylcholine, the rapid-acting depolarizing relaxant often used for rapid-sequence inductions, is known to cause profound hyperkalemia in patients who have extensive muscle damage, particularly if the drug is given 24 to 36 hours after the traumatic insult. Alternatives include the nondepolarizing relaxants, such as vecuronium given at 0.28 mg/kg, for conditions requiring rapid intubation. This agent is useful during the maintenance of anesthesia because it has minimal hemodynamic effects. The vagolytic actions of pancuronium may be undesirable because the tachycardia may lead to further confusion in distinguishing drug effect from hypovolemia or inadequate levels of anesthesia. *d*-Tubocurarine and atracurium should probably be avoided because of their histamine-mediated hypotensive effects. It is important to remember that all neuromuscular blocking agents are subject to aberrant behavior in the presence of changing acid–base status, altered metabolism and excretion, hypothermia, and certain antibiotics. For this reason, it is mandatory to monitor the status of the neuromuscular blockade.

Hypothermia is a major problem in traumatized patients undergoing exploratory surgery, and every effort should be made to maintain a normal core body temperature. Heated humidifiers, low gas flows, warmed intravenous and irrigating fluids, and a warmed operating room can help maintain normothermia. Certainly, heating lamps, warming blankets, and plastic to wrap exposed body surfaces can also be used.

Positioning for Operation

The position of patients with thoracic injuries (whose major injury does not involve the thorax) will obviously depend on the location of that injury; the usual position will be with the trunk supine. The position of casualties who are to have an actual thoracic procedure will depend on the indication for the operation. Most thoracic surgeons use an anterolateral incision for the casualty who is *agonal* (ie, a casualty who exhibited one or more signs of life [motion, spontaneous respiration, palpable pulse] shortly before arrival at the hospital, but who is now lifeless) or in profound shock, although a few prefer a median sternotomy for the operative approach. Thus, the desired position for an emergency operation is supine. Because the needed operation is best done through a posterolateral thoracotomy incision with the patient in the right lateral decubitus position, there are two exceptions to this rule:

1. the unusual casualty who is known to have an injury to the proximal left subclavian artery, and
2. the casualty who has sustained blunt trauma to the chest and has radiographic evidence of a left hemothorax and a widened superior mediastinum, and who, therefore, may have a proximal descending aortic disruption.

Patients who are undergoing thoracic surgery in which the indications are less emergent (eg, control of nonexsanguinating hemorrhage, a large air leak, repair of a chest-wall defect, or simply to debride the wounds of entrance or exit) are placed in the decubitus position so that a posterolateral incision can be made. Care must be taken to ensure that there is no pressure on the brachial plexus of the dependent arm, and that the thorax and abdominal wall are free to move without restricting respirations. Endotracheal tube position and the position of all monitors must be checked for dislodgement following patient positioning. Patients with concurrent orthopedic injuries may be difficult to position because of pain and the potential for dislocation or worsening of the injury.

Postoperative Care

Most patients presenting for surgery secondary to battlefield chest trauma require postoperative care in an intensive care unit until an adequate level of physiological homeostasis can be achieved. The military trauma anesthesiologist is expected to play a significant role in the postoperative care of these patients, especially in the realm of acute pain and ventilatory management.[14]

DIAGNOSIS AND MANAGEMENT OF SPECIFIC INJURIES

Rib Fractures

The ribs are the most commonly injured component of the thoracic cage, and the incidence of serious complications increases with the number of ribs fractured. This increase is a reflection of the greater traumatic forces associated with multiple rib fractures. As a general rule, a young, otherwise healthy, active-duty recruit with a flexible chest wall is less likely to sustain a rib fracture than is a geriatric patient with thoracic trauma. With this in mind, the presence of multiple rib fractures in young patients implies a sizable transfer of force. Multiple rib fractures most commonly involve the seventh through tenth ribs and thus are often associated with hepatic or splenic lacerations. Acute gastric dilation commonly accompanies left-sided fractures and may increase the likelihood of acid aspiration. Fractures of the upper ribs (first through third) should be regarded as harbingers of serious blunt trauma to the chest. The bony framework of the upper limb and its muscular attachments provide a barrier to injury in this area. Therefore, severe maxillofacial, cervical, neurological, pulmonary, and vascular injuries may accompany upper-rib fractures.[15]

Rib fractures, even in the absence of underlying cardiopulmonary trauma, may contribute to serious pulmonary complications. The associated pain and reflex muscle splinting can exacerbate alveolar hypoventilation, resulting in retention of secretions, atelectasis, and respiratory failure. Taping or binding the ribs will cause even more splinting and further compromise. A tidal volume of less than 5 mL/kg or a forced vital capacity of less than 10 mL/kg indicates severe splinting.[16] Adequate analgesia is essential in the management of rib fractures. The use of intercostal nerve blocks is attractive because it avoids the respiratory depression associated with systemic narcotic administration. Comparable analgesia of longer duration and with less potential for systemic toxicity may be achieved with continuous thoracic epidural analgesia. Intrapleural injection of local anesthetics is another form of analgesia for rib fractures. The efficacy of this technique, however, is controversial at this time.

Flail Chest

Flail chest may be defined as an abnormal movement of the chest wall occurring as a result of fractures of three or more ribs in two places on the same side. This produces a segment of the chest wall that responds to changes in pleural pressure, as opposed to the muscular action of the chest wall, resulting in a paradoxical respiratory pattern. This injury is most commonly associated with blunt trauma to the chest wall and involves the anterolateral aspect of the chest. The posterior wall is rarely involved because it is heavily fortified with muscle. The flail chest injury is rarely isolated and is usually an ominous sign, indicating serious underlying injuries to intrathoracic or intraabdominal organs or both. This condition is often difficult to diagnose and may go unnoticed for extended periods, often overshadowed by more-overt injuries. Chest radiography may not reveal fractures unless the films are overpenetrated and oblique views are taken. Flail chest may not be apparent on simple inspection, as chest-wall muscular spasm may be able to splint the segment until the muscles become fatigued. Palpation is more likely to be diagnostic than inspection, with the presence of crepitus and abnormal respiratory motion aiding in the diagnosis. Serial blood-gas measurements can help establish the diagnosis, because the clinical hallmark of flail chest is *hypoxemia*.

Flail chest can cause hypoxemia by two mechanisms: (1) intrapulmonary shunting due to perfusion of the poorly ventilated, contused lung; and (2) atelectasis caused by compromised ventilatory mechanics. The pendelluft phenomenon, which is a to-and-fro movement of air from the damaged to the normal lung, was once thought to make a significant contribution to hypoxemia. However, it has been shown that there is an increase in ventilation and improved gas exchange on the injured side.[17]

The hypoxia observed in flail chest is now recognized to be caused by abnormalities in the contused lung underlying the flail segment.[18] Thus, the modern treatment of flail chest focuses on preventing the arterial desaturation (that results from the shunting) from reaching life-threatening proportions. Stabilization of the flail segment, maintenance of adequate ventilation, oxygen enrichment, physical therapy, and effective pain relief should all be tried; however, a period of intubation and ventilation may be necessary. The criteria for intubation, which should only serve as a guide, include:

- partial pressure of arterial oxygen (Pa_{O_2}) < 70 mm Hg with oxygen enrichment,

- partial pressure of arterial carbon dioxide ($Paco_2$) > 50 mm Hg,
- pH < 7.25,
- tachypnea > 30/min,
- vital capacity < 15 mL/kg, and
- negative inspiratory force < –20 cm water.

Both external stabilization of the flail segment by traction and internal fixation with intramedullary pinning of the fractured ribs have been described.[19,20] Continuous electrocardiographic monitoring is essential because flail chest can often be associated with cardiac contusions, from which arrhythmias may arise. Frequent arterial blood-gas analyses, along with other laboratory studies, justify placing an arterial line. Pulmonary artery catheterization, which may be available in deployable hospitals, may help with the evaluation of cardiac performance and guide fluid management. This is especially necessary because the injured lung underlying the flail segment is sensitive to both underresuscitation of shock and fluid overload, the latter condition leading to pulmonary edema in the injured lung tissue and a further impairment of gas exchange.[21] In intubated, ventilated patients, excessive airway pressure can also cause an iatrogenic injury by mechanically disrupting damaged alveolar capillary membranes in the lung underlying the flail segment. This may enlarge the volume of the contusion, but a more serious problem is the increased likelihood of systemic air embolism.[22]

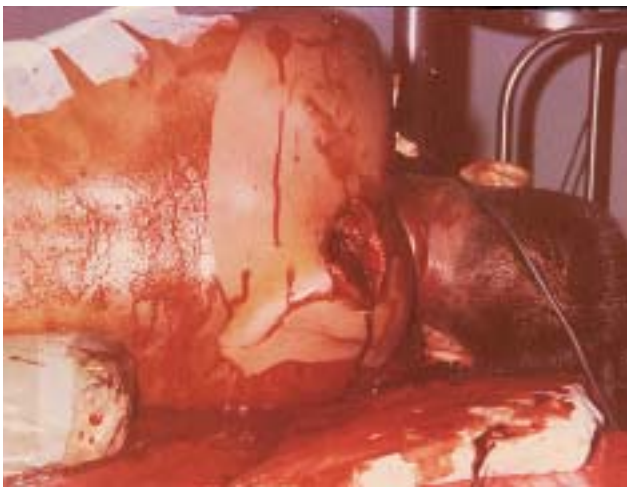


Fig. 19-3. This casualty had a sucking chest wound. A large fragment entered his back; frothy material can be seen in the base of the wound. Photograph: Wound Data and Munitions Effectiveness Team slide collection.

Sucking Chest Wound

An open, sucking, or blowing chest wound is a serious injury that is usually found at the site where a yawing or tumbling bullet has exited or where a large fragment from an explosive munition has entered. The most serious component of the wound is usually the injury to the underlying lung. The characteristic appearance is of bloody foam being alternately sucked into and blown from the wound (Figure 19-3). In this condition, intrapleural pressure equalizes with the atmosphere, resulting in a diminished movement of air on the affected side. The immediate treatment of this condition is to occlude the defect but provide for egress of air from the chest cavity by inserting a chest tube with a one-way valve. Sucking chest wounds are not especially common among combat casualties. In the Israeli series, they accounted for only seven (11%) of the total of the thoracic casualties.[2] The reason for their low prevalence is that the intrathoracic component of the injury is much more likely to be life-threatening than is the hole itself. Once the intrathoracic component has been treated, surgical treatment is directed toward achieving an air-tight closure of the chest wall defect.

Acute Traumatic Hemothorax

Hemothorax can occur with both penetrating and nonpenetrating thoracic trauma and is usually caused by blood emanating from injuries of the heart, great vessels, pulmonary parenchyma, or chest wall.[23] The diagnosis of hemothorax is made on the basis of history and clinical examination of the chest. When a significant quantity of blood has accumulated in the chest cavity, the trachea may be deviated, the chest wall is dull to percussion, and breath sounds are diminished on auscultation. A chest radiograph and an electrocardiogram should be obtained for all patients except those presenting in extremis. Approximately 500 mL of blood must accumulate in the chest cavity before it is radiologically detectable.

Because hypovolemia from blood loss is the most common problem in patients who present with significant chest injury, the immediate treatment should be directed toward restoring blood volume. Two large-bore intravenous cannulae should be inserted as soon as possible. Central venous pressure moni-

toring may be useful in both diagnosing and managing patients who have mechanical interference with ventilation, as the central venous pressure is low in the presence of severe hypovolemia but elevated in the presence of a chest cavity full of blood. Oxygen therapy is mandatory. Tube thoracostomy should be performed as soon as possible because it is not only therapeutic, it also provides an assessment of cumulative and ongoing blood loss, facilitates reexpansion of the lung, and simplifies autotransfusion when major bleeding is encountered. In most cases, the source of the bleeding is the pulmonary vessels, which normally have low perfusion pressures. On the other hand, bleeding from systemic vessels or the heart is usually more persistent and voluminous.

For casualties who have an indication for operative intervention but who are stable (ie, blood pressure > 100 mm Hg, pulse rate < 100, good peripheral perfusion and ventilation), the thoracotomy is performed in the operating room. Immediate thoracotomy in the admitting area of the hospital may be indicated in patients who become agonal on arrival. The best results have been obtained in casualties who have penetrating thoracic injuries, and the poorest results in those sustaining blunt trauma—especially when the abdomen is involved. Regardless of the etiology, survival in this group of casualties is poor. In one recent series,[24] survival was 6% in those with blunt trauma and 27% for casualties with penetrating wounds of the chest.

The reasons for performing a thoracotomy are to

- decompress a tension pneumothorax or pericardial tamponade, if present;
- perform open-chest cardiac massage; and
- stop bleeding if it arises within the thorax or, by occluding the descending aorta, to slow bleeding if it arises from the abdomen.

After the airway has been intubated, if necessary via cricothyroidotomy, an anterolateral incision is made on the side of injury, the pericardial sac opened, and cardiac compressions begun. Bleeding sites are controlled by direct pressure or by application of a clamp or Rommel tourniquet around the hilum of the lung. The military trauma anesthesiologist should anticipate the need to correct severe metabolic acidosis and to manage a variety of malignant cardiac arrhythmias. The patient is then transported to the operating room for definitive treatment.

The management of agonal casualties is very labor and resource intensive. The likely poor outcome, even with the best of care, has led some authorities[25] to argue that emergency thoracotomy for agonal victims of truncal trauma has no place in combat casualty care. The military trauma anesthesiologist, in consultation with the hospital thoracic or trauma surgeon and the hospital commander, should develop an agreed-upon triage policy for such casualties. By necessity, any such policy will have to take account of the existing tactical situation.

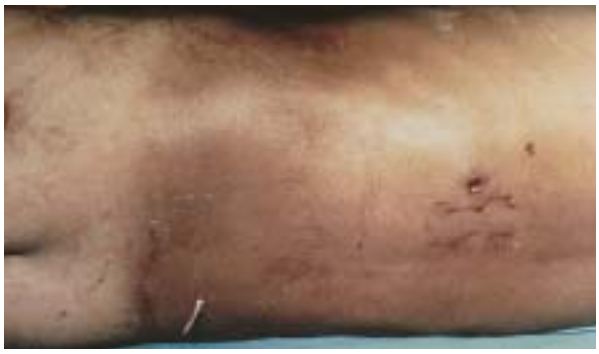
Cardiac Tamponade

Although cardiac tamponade is found in casualties who require emergency thoracotomy, its occurrence is not limited to this group. In some casualties, the presentation of cardiac tamponade may be quite subtle (Figure 19-4). Acute, traumatic pericardial tamponade is caused by the collection of blood or other fluid (eg, serous exudate) within an intact pericardial sac. This sac is composed of a fibrous, poorly compliant membrane. Rapid accumulation of 100 to 200 mL of fluid in this closed space restricts filling of the cardiac chambers during diastole, which produces a fixed, low, cardiac output. Positive-pressure ventilation will lower cardiac output even further, as will any cardiac depressant.

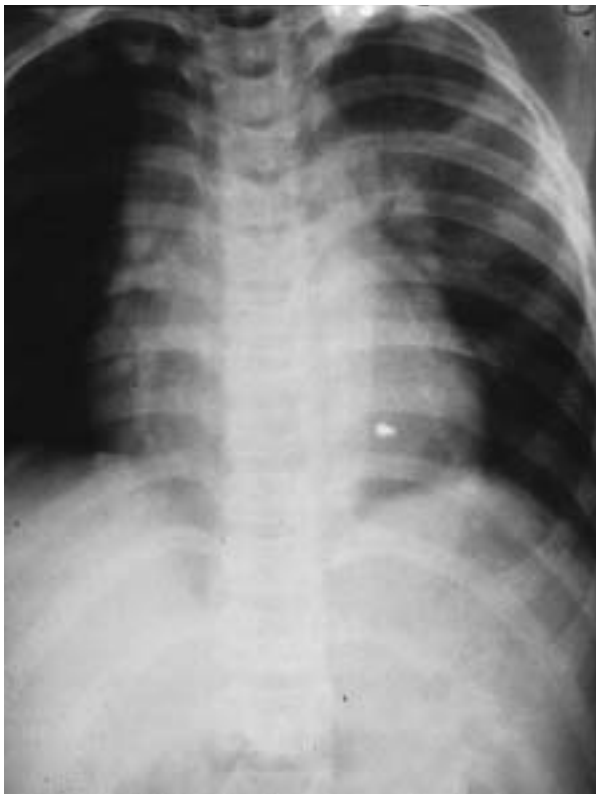
The diagnosis of cardiac tamponade requires a high degree of clinical suspicion. It should always be suspected in patients with wounds in the vicinity of the neck, precordium, or upper abdomen, and

Fig. 19-4. (**a**) This casualty was wounded when a grenade fragment penetrated his back. He was initially neither hypotensive nor in much distress but while being observed developed tachycardia, dyspnea, and distended neck veins. (**b**) The chest radiograph shows a fragment within the silhouette of a globular-shaped heart. A thoracotomy was performed because of the casualty's deteriorating clinical state and the appearance of the radiograph. (**c**) The pericardium has been opened and several blood clots have been extruded. (**d**) The removed clots. (**e**) A small defect, which was not then bleeding, in the free wall of the left ventricle. (**f**) The fragment after its removal from the myocardium. The tip of the hemostat serves as a reference for size. The left ventricular wall had not been perforated, possibly because of the fragment's small size. Other fragments lacerated the stomach and spleen; these were found during a subsequent laparotomy. Photographs: Swan Vietnam Surgical Slide Collection.

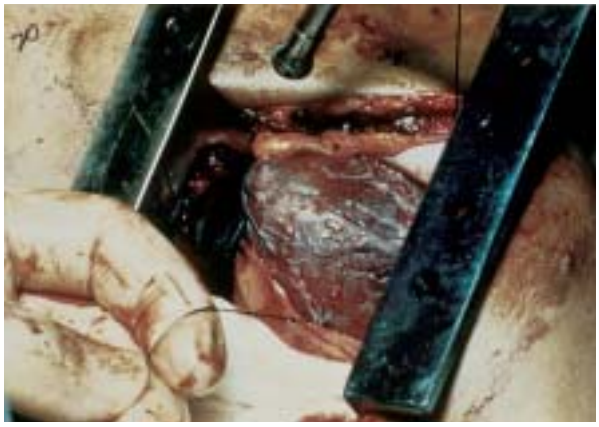
a



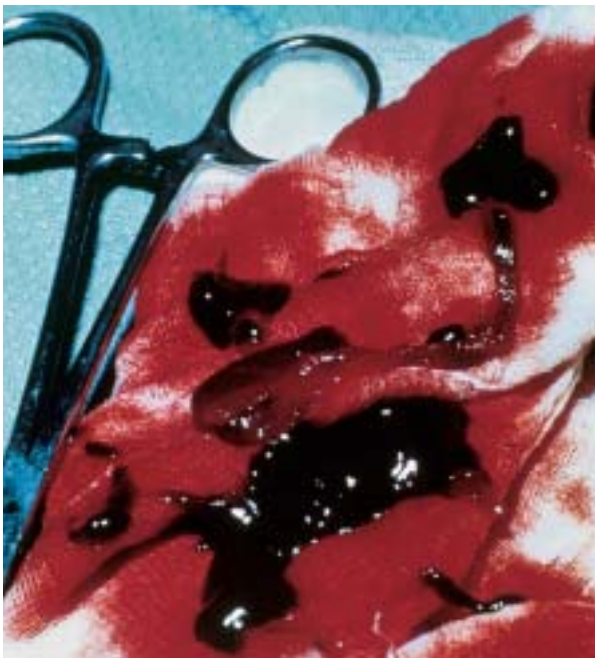
b



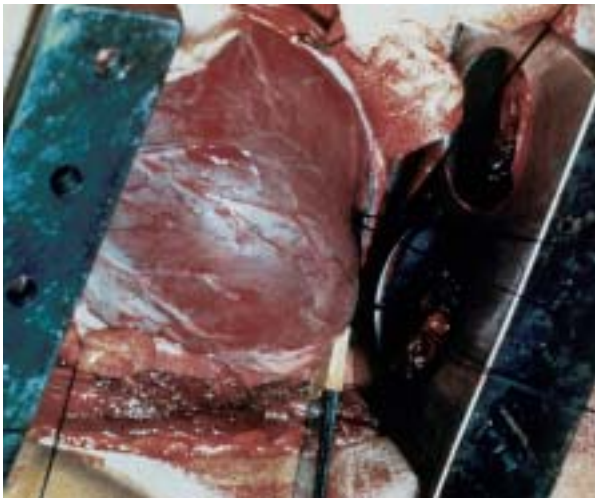
c



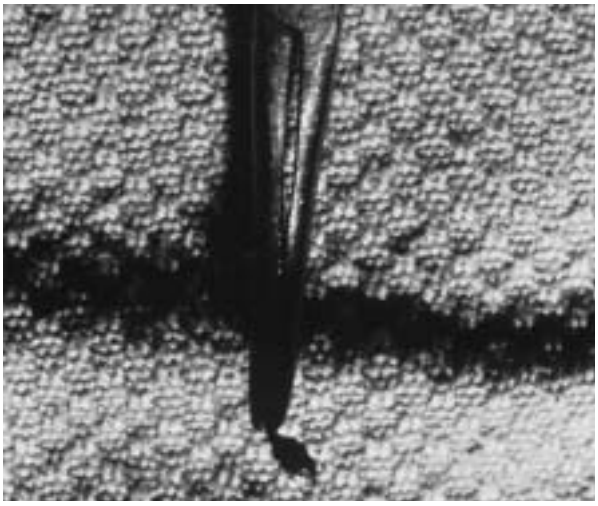
d



e



f



especially in patients who are in shock. The classic signs, although present in only 41% of patients with penetrating cardiac wounds,[26] include Beck's triad: hypotension, distended neck veins, and distant heart sounds. Other signs include pulsus paradoxus, which is a decline in systolic blood pressure of 10 mm Hg or more on inspiration. This is of limited value in the field, however, as continuous arterial pressure monitoring may not be possible.[27] Paradoxical filling of neck veins on inspiration (Kussmaul's sign) may be too subtle to detect in uncooperative, struggling patients. If significant blood loss has occurred, distension of neck veins may not occur. Equalization of pressures across the heart, measured by a pulmonary artery catheter, is very suggestive of tamponade. The chest radiograph may reveal a large, globular heart shadow, and the electrocardiogram will be either normal or show nonspecific ischemic changes. Occasionally, the electrocardiogram will show electrical alternans, which is usually diagnostic of tamponade.

Again, it should be emphasized that heavy reliance is placed on clinical impression in diagnosing patients on the battlefield with cardiac tamponade. *Shock and a wound in the anterior chest indicate cardiac tamponade until proven otherwise*. Elevation of the central venous pressure is strong confirmatory evidence of the diagnosis, and treatment should be started immediately. The definitive treatment of this condition is surgery as soon as possible. Pericardiocentesis may be used to relieve the tamponade in rapidly deteriorating patients. However, this is of limited value because blood rapidly reaccumulates in the pericardial sac.

The patient should be prepared and draped for surgery, and an arterial line should be inserted prior to the induction of anesthesia. *The administration of a general anesthetic to a patient with a significant tamponade is potentially lethal*.[28,29] Almost any maneuver, other than administration of 100% oxygen, including positive-pressure ventilation, causes deterioration.[30] Inhalational agents or thiopental cause further cardiac depression and impairment of diastolic filling. Peripheral vasodilation associated with these agents further impairs filling. Anesthetic goals include maintaining adequate preload with copious volume infusion (central venous pressure at least 15 cm H_2O), and avoiding vagotonic medications (eg, narcotics). It is important to maintain sinus tachycardia in the face of limited stroke volume to maximize cardiac output. The combination of ketamine and pancuronium is ideal for this purpose. The recommended dose of ketamine for patients with diminished cardiac output is 0.25 to 0.50 mg/kg. Despite careful anesthetic management, the patient's condition may deteriorate before the tamponade is relieved. If deterioration occurs, isoproterenol has been advocated,[31] infused at a rate of 0.1 μg/kg/min, and titrated to effect. When the pericardium is decompressed, the patient's vital signs usually improve instantaneously.

Cardiac Contusions

Myocardial contusion is the most common cardiac injury due to nonpenetrating trauma. Depending on the sophistication of the diagnostic armamentarium and the definition of the condition, cardiac contusions have been reported in approximately 15% to 70% of trauma patients who sustain blunt chest trauma. Rapid deceleration occurring during motor vehicle accidents is the most common cause of cardiac contusions. The heart is subjected to marked anteroposterior compression resulting in rupture of the intramyocardial vessels, which bleed into the myocardium. Subsequently, myocytes may die and undergo fragmentation. Iatrogenic trauma caused by closed-chest massage during cardiopulmonary resuscitation may also be responsible for myocardial contusion. The right ventricle is injured more frequently than the left during cardiopulmonary resuscitation, due to its proximity to the chest wall. However, the valves in the left side of the heart, especially the aortic valve, are more susceptible to injury than those in the right side of the heart.[16]

Symptoms of myocardial contusion may be similar to those of myocardial infarction or pericarditis, because anginal chest pain is the most common presenting complaint. However, contusion pain is not relieved by nitroglycerin. Many trauma patients with myocardial contusion report few or no symptoms from their cardiac injuries. Ventricular arrhythmias are the most common clinical sign of cardiac contusion. Conduction disturbances are more commonly associated with injuries to the right atrium and ventricle.[32] Other signs may be a current of injury on the electrocardiogram or a pericardial friction rub; there may be no change at all in cardiac examination. The diagnosis of cardiac contusion can be a real dilemma for the clinician, as most routine and sophisticated tests have proven unreliable.

Casualties presenting with myocardial contusion should be treated in the same manner as those presenting with myocardial infarctions. Many casualties with this condition need immediate sur-

gery to treat their other injuries. Because the right ventricle is particularly vulnerable to contusion owing to its proximity to the anterior chest wall, right ventricular dysfunction and failure may complicate perioperative management. The right ventricle is sensitive to afterload; therefore, conditions that may increase pulmonary vascular resistance (ie, hypoxia, hypercarbia) should be avoided. Adequate preload is usually necessary in the setting of right ventricular dysfunction; intravascular volume resuscitation is therefore mandatory.

Cardiac Rupture

Rupture of the heart is a common cause of immediate death in victims of blunt trauma. In nonpenetrating injuries, severe compression may so raise cardiac chamber pressure and wall stress that the myocardium ruptures. Thus, the mechanism is similar to the process that gives rise to myocardial contusion. Puncture of the heart by ribs broken by blunt trauma has also been described. In combat casualties, penetrating injuries are a more frequent cause of cardiac perforations than is blunt trauma. Although right ventricular perforations may appear to predominate, a comparable incidence of left ventricular injuries has been described.[33] Because of the higher immediate mortality associated with ventricular trauma, patients with atrial perforations are more frequently encountered in the operating room.

The clinical presentation of cardiac rupture or perforation is determined by the status of the pericardium. With an open pericardial wound, blood extravasating from the heart drains freely into the pleural space, producing a rapidly fatal hemothorax. If the pericardial defect is small, or if it is occluded (as may happen on rare occasions by clotted blood or by the adjacent lung), the patient may not exsanguinate but live long enough to develop the signs of cardiac tamponade. The anesthetic management of cardiac rupture is similar to that for pericardial tamponade, except that exsanguination makes emergency thoracotomy more common.

Cardiopulmonary bypass, although usually not available in combat zone hospitals, may be required to repair some cardiac defects. Although traumatic ventricular septal defects may close spontaneously, most of them require surgical closure through the left ventricle, utilizing cardiopulmonary bypass.

Vasodilators often produce some degree of clinical and hemodynamic improvement. However, it should be emphasized that vasodilators are contraindicated in severely hypovolemic patients.

Injuries to the Coronary Arteries

The incidence of coronary artery laceration following penetrating cardiac injuries has been reported[34] to be about 4%. Division of a coronary artery invariably leads to hemorrhage and tamponade, and may cause myocardial infarction. Because the right coronary artery is protected by the sternum, most injuries involve the left coronary artery or its branches. Coronary artery occlusion after blunt chest trauma has been reported.[35] In these cases, myocardial infarctions evolved after the traumatic injury in patients without preexisting coronary atherosclerosis.

Several mechanisms may be involved in the pathogenesis of myocardial infarction after blunt chest trauma. In perhaps the simplest scenario, the forces accompanying an episode of blunt trauma could dislodge a previously adherent intramural plaque, resulting in intraluminal obstruction of the involved coronary artery.[36] Patients with preexisting atherosclerotic heart disease also may be at increased risk for infarction because of trauma-induced hemorrhage into a plaque, which is known to be richly vascularized with fragile, thin-walled capillaries.[37] A third possible mechanism of ischemic injury is trauma-induced coronary vasospasm. Although spasm also occurs in normal vessels, angiographically visualized spasm often occurs at a site of atherosclerotic narrowing.[38] Other etiologies of traumatic infarction include coronary thrombosis due to vascular trauma, direct transection of the coronary arteries, coronary embolization, and dissecting aneurysm.[39]

In the absence of cardiac tamponade, the anesthetic management of these casualties should be similar to that for patients with coronary artery disease with unstable angina. A variety of techniques are consistent with the hemodynamic goals of avoiding tachycardia and extremes of blood pressure. In young soldiers with good ventricular function, a combined technique with high-dose narcotics and an inhalational anesthetic agent is ideal: intraoperative sympathetic reflexes are reliably blunted, while myocardial contractility and oxygen consumption are appropriately depressed. Although there may be no ideal induction technique for the patient with ischemic heart disease and a full stomach, a moderate dose of narcotic (eg, 10–15 μg/kg

fentanyl) combined with etomidate (0.1–0.2 mg/kg) and succinylcholine results in a stable induction, with adequate blunting of autonomic responses to laryngoscopy. In the setting of severe skeletal muscle injury, particularly if more than 24 to 36 hours have elapsed since the injury, nondepolarizing relaxants may be preferable because of the possibility of succinylcholine-induced hyperkalemia.

The common presentation of the casualty with a coronary artery injury caused by penetrating trauma is pericardial tamponade. Because direct repair of the arterial laceration in a beating heart is usually not feasible, and because the cardiopulmonary bypass apparatus that would allow the construction of an aortocoronary saphenous graph bypass is not likely to be available in field hospitals, the military surgeon has little option but to ligate the injured artery. Given this circumstance, the military trauma anesthesiologist should be prepared to treat potentially life-threatening cardiac arrhythmias and cardiogenic shock.

Injuries to the Great Vessels

The thoracic aorta can be injured as a result of penetrating or blunt trauma to the chest. The resulting hemorrhage is usually devastating, allowing only approximately 15% of patients to reach a field hospital alive.[40]

In the U.S. military, the great majority of aortic ruptures result from motor vehicle accidents and airplane crashes. Abrupt deceleration of the thorax (*a*) causes compression and displacement of the heart and (*b*) creates shearing and bending stresses in the aortic wall, which are greatest at the origin of the subclavian artery and, in the ascending aorta, at the level of the coronary arteries.[41] A minority of these patients (perhaps 10%) survive because of containment and tamponade of the hemorrhage by adjacent mediastinal structures. In the proximal descending aorta, the aortic adventitia may remain intact and temporarily prevent exsanguination. Sudden aortic rupture may develop in these patients at a later time.

Penetrating chest trauma may also involve the great vessels. Depending on whether the vessels are injured at an intrapericardial or an extrapericardial location, the clinical presentation is usually one of acute tamponade or massive hemothorax. These casualties may have sustained one or more penetrating cardiac injuries as well, but severe hypovolemia may account for the frequently atypical hemodynamic findings.

The clinical findings of aortic rupture include the diagnostic triad that is seen in more than 50% of cases[42]:

- increased arterial pressure and pulse amplitude in the upper extremities,
- decreased pressure and pulse amplitude in the lower extremities, and
- a widened superior mediastinum seen on the chest radiograph.

Other clinical and radiographic findings are included in Exhibit 19-3. An active search for these clinical and radiographic findings is important during evaluation because as many as one third of patients with aortic rupture have minimal or no external signs of chest trauma.[43,44]

When dealing with injuries to the major thoracic vessels, the foremost goal of the anesthesiologist is to simply maintain an adequate blood volume, which allows the surgeon the time needed to find the source of bleeding and, if possible, to repair the injured vessel. Ischemia of the abdominal viscera and spinal cord may occur during the period of proximal aortic clamping; the reported incidence of

EXHIBIT 19-3

CLINICAL AND RADIOGRAPHIC FINDINGS OF AORTIC RUPTURE

Clinical:
- Increased arterial pressure and pulse amplitude of upper extremities
- Decreased arterial pressure and pulse amplitude of lower extremities
- Retrosternal or interscapular pain
- Hoarseness
- Systolic flow murmur over the precordium or medial to the left scapula
- Neurological deficits in the lower extremities

Radiographic:
- Widened mediastinum
- Unsharp aortic contours
- Widened paraspinal interfaces
- Opacified pulmonary window
- Broadened paratracheal stripe
- Displacement of the left main-stem bronchus
- Rightward deviation of the esophagus
- Sternal or upper-rib fractures or both
- Left hemothorax

paraplegia in survivors ranges from 1.0% to 11.7%.[41] Prophylactic measures for preserving spinal cord viability include partial cardiopulmonary bypass, heparin-coated shunts, partial bypass of the left side of the heart, draining the cerebrospinal fluid, and inducing systemic hypothermia.

The presumed mechanism of paraplegia in these patients is ischemia of the spinal cord caused by damage to the intercostal vessels that supply the spinal cord. Monitoring of somatosensory evoked potentials may be useful in heralding spinal cord ischemia but is not practicable in field hospitals.[45]

The kidney is the abdominal organ at greatest risk for ischemic injury during the period of aortic occlusion. Preserving distal perfusion with shunts or partial bypass is associated with a lower incidence of postoperative renal dysfunction; however, because the needed equipment is not available in the field, the military trauma anesthesiologist has no other recourse than to use mannitol, renal-dose dopamine, and/or diuretics as part of a strategy for renal protection similar to that used for abdominal aortic aneurysm surgery.[46]

Intraoperative monitoring should include placing a right radial arterial line and a lower-extremity arterial line for monitoring distal aortic pressure. The surgeon's possible clamping of the left subclavian artery would make monitoring the left radial arterial pressure impossible during that time. In all but the most emergent cases, pulmonary arterial catheterization is desirable because proximal aortic occlusion imposes a severe afterload stress on the left ventricle.

A double-lumen endotracheal tube is desirable because it allows the left lung to be collapsed to facilitate surgical exposure. Equally important, however, is the fact that the dependent right lung can be isolated from the parenchymal hemorrhage that may occur in the left lung as a result of pulmonary contusion.

Pneumothorax

Pneumothorax occurs secondary to blunt or penetrating trauma to the chest wall. Less obvious pneumothoraces occur as a result of penetration of the chest wall and lung by sharp objects, such as bullets, knives, or rib fragments. Pneumothoraces of less than 20% are usually not clinically detectable. Patients may complain of chest pain, which is accentuated by deep breathing. Cyanosis may be evident, and with larger pneumothoraces, the trachea can be deviated. Percussion of the chest reveals a tympanitic sound, and breath sounds may be diminished or absent. Conditions that can mimic pneumothorax include hemothorax or atelectasis. Radiological examination is the best diagnostic aid available, and all films should be exposed during expiration, if possible.[47] The presence of rib fractures or surgical emphysema should provide a clue to the diagnosis. In trauma patients, pneumothoraces with a volume greater than 10% should be treated by tube thoracostomy. Temporizing the treatment is inappropriate for the patient who may require intubation and positive-pressure ventilation during surgery for extrathoracic injuries, as the pneumothorax may enlarge under these conditions. Therefore, the prophylactic insertion of a chest tube is indicated.

A tension pneumothorax occurs when air enters the pleural cavity during inspiration but, owing to a ball-valve action, cannot escape during expiration. In conscious patients, rapid deterioration is noted. The cardinal signs of a tension pneumothorax are

- cyanosis,
- marked decrease in pulmonary compliance,
- rapid deterioration of vital signs,
- diminished or absent breath sounds, and
- tracheal deviation.

This condition can be easily confused with pericardial tamponade or shock, secondary to hypovolemia; however, the hypertympanic percussion note over the ipsilateral chest usually makes differentiation possible.

The casualty shown in Figure 19-5 is a particularly dramatic presentation of a tension pneumothorax. However, it is important to stress that a tension pneumothorax is a clinical rather than a radiological diagnosis. Two factors contribute to the hemodynamic deterioration: (1) hypoxia from decreased gas exchange and (2) increased impedance of venous return into the chest due to the mechanical distortion of the mediastinum that is caused by the expanding pneumothorax. During general anesthesia, a dramatic decrease in pulmonary compliance should alert the anesthesiologist to the problem. Nitrous oxide should be discontinued if it is being administered, as it accentuates the size of the pneumothorax.[48] As soon as a hemodynamically significant tension pneumothorax is suspected, the anesthesiologist might need to perform temporizing measures, including the immediate insertion of a large-bore needle into the second intercostal space along the midclavicular line. A "hissing" sound may be created by the escaping,

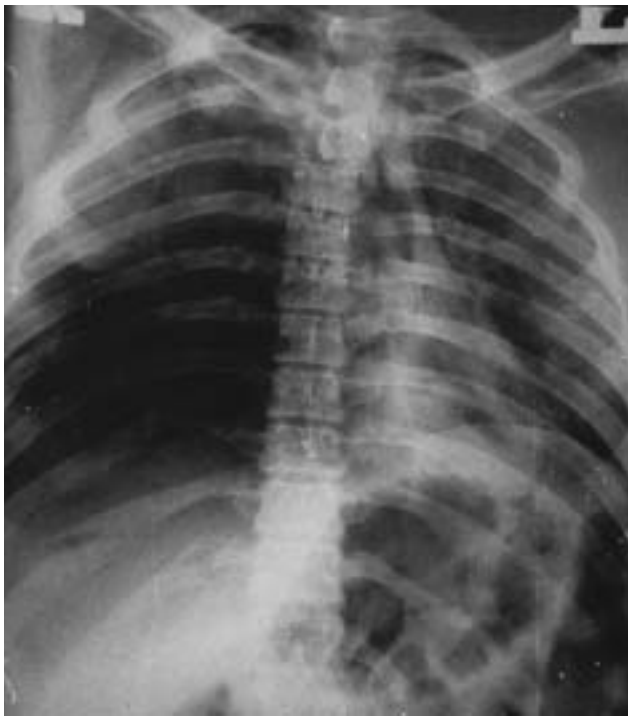


Fig. 19-5. This casualty was killed by a perforating gunshot wound of the right hemithorax. A radiograph was taken before the autopsy was performed. A tension pneumothorax with massive shift of the mediastinum is apparent. Photograph: Wound Data and Munitions Effectiveness Team slide collection.

pressurized air. Intravenous extension tubing can then be attached to the catheter and placed under water seal (ie, submerging it in a liter bottle of sterile water). Care must be taken to keep the water bottle lower than the patient; otherwise, the flow may be reversed, causing a hydrothorax. Temporizing measures should be replaced as soon as possible by the more definitive closed-system tube thoracostomy.

Tracheal and Bronchial Injuries

Tracheal and bronchial injuries are uncommon sequelae of thoracic trauma but are among the most serious encountered in thoracic surgery. Although most penetrating wounds to these structures are caused by fragments, these injuries also occur following blunt trauma associated with high-speed travel. A 2.8% incidence of tracheobronchial rupture has been reported[49] in patients who succumbed to blunt chest trauma. Approximately 80% of tracheobronchial disruptions occur within 2.5 cm of the carina. Three mechanisms may explain this finding[50]:

1. Sudden, forceful compression is the most common insult causing tracheobronchial injuries during blunt thoracic trauma. This anteroposterior compression combined with simultaneous lateral expansion of the lungs can result in severe traction on the pericarinal portion of the trachea.
2. Acute increase in intrabronchial pressure may be caused by reflex glottic closure at the moment of impact.
3. Shearing forces at points of fixation of the intrathoracic airways, caused by rapid deceleration, may account for the preponderance of disruptions near the carina.

Dyspnea, cough, painful hemoptysis, and subcutaneous emphysema are the most common clinical findings in patients with tracheobronchial disruption. However, 10% of patients are nearly asymptomatic,[51] which accounts for the frequent delays in diagnosis. Auscultation of the heart may reveal a crunching sound associated with pericardial air, also known as Hamman's sign. When the site of disruption freely communicates with the mediastinal pleura, pneumothorax results, and tube thoracostomy and suction often fail to reexpand the affected lung. When the pleural space and the site of disruption do not communicate, there is little or no pneumothorax, and mediastinal air may be the only radiographic abnormality. In addition to pneumothorax and pneumomediastinum, indicative radiographic findings include subcutaneous and deep cervical emphysema. With complete transection of a main bronchus, the superior margin of the affected lung drops below the level of the transection. This occurs because the lung is deprived of its normal bronchial tethering in the thoracic cage.

All patients with suspected tracheal or bronchial injury require diagnostic bronchoscopy as soon as they are stable. Blind endotracheal intubation without endoscopic visualization of the tracheobronchial tree is unlikely to be successful and may produce further trauma because the distal trachea is displaced posteriorly. In addition, acute airway obstruction may develop when the endotracheal tube enters a false passage and occludes the tracheal lumen. Both a surgically guided oral intubation of the distal tracheal segment and removal of the initial surgical airway are acceptable means of allowing surgical repair of the tracheal separation.

When the disruption and distal segment can be visualized clearly, a double-lumen tube can be inserted distal to the tracheal tear or, in the case of

bronchial disruption, into the contralateral bronchus. Alternatively, the area of disruption can be visualized directly with a fiberoptic bronchoscope; then the instrument can be used as a guide over which an endotracheal tube can blindly be passed into the distal tracheal segment. This approach is equally acceptable for bronchial disruption, although it is preferable to intubate the uninjured bronchus selectively for ventilation during bronchial repairs. Therefore, the military trauma anesthesiologist must have available a variety of sterile endotracheal tubes that can be passed into the operative field and used to intubate the distal airway prior to and during its anastomosis to the proximal segment. Alternative techniques such as simply insufflating oxygen into the distal airway or employing high-frequency jet ventilation using uncuffed catheters in the contralateral bronchus have also been described.[52]

Parenchymal Lung Lacerations

Extensive laceration of the lung parenchyma—such as might occur when an assault-rifle bullet yaws and tumbles during its passage through the lung—can create one of the most difficult therapeutic problems for the military trauma anesthesiologist. Not only will there be massive leakage of air into the pleural space, which will make difficult the maintenance of an acceptable tidal volume, but the normal anatomical proximity of airways and blood vessels in the lungs potentiates (1) the passing of blood into airways and (2) the passing of air into the circulation. The first condition causes an airway obstruction as the casualty literally drowns in blood. The second causes rapidly fatal systemic air embolization. The simultaneous leakage of air into the pleural space and passage of blood into the tracheobronchial tree can best be managed by having the anesthesiologist pass a standard endotracheal tube into the bronchus of the uninjured lung. (The use of a double lung tube in this situation is too time consuming to be safe.) Once the injured lung has been isolated by the anesthesiologist, the surgeon, after exposing the injured lung, can then cross-clamp the pulmonary hilum. The appropriate surgical management may be to perform an anatomical pulmonary resection—a lobectomy or pneumonectomy—rather than to attempt a repair of the laceration (Figure 19-6).[53]

Systemic air embolism is a catastrophic complication that suddenly converts a previously stable patient into one who is agonal. Increasingly, evidence shows that cerebral and coronary air emboli are common causes of death in victims of penetrating lung injuries.[22] There is frequently a causal relation to the application of positive pressure to the airway. The treatment, insofar as there is one, consists of

- immediate performance of a thoracotomy,
- application of a large clamp or Rommel tourniquet around the hilum of the injured lung to prevent further embolization, and
- exposure of the heart to confirm the diagnosis (air bubbles should be visible in the coronary vessels).

If the diagnosis is confirmed, the surgeon manually occludes the ascending aorta and manually compresses the heart so as to drive the air bubbles through the coronary arteries and into the coronary sinus. The management of the cerebral component is a matter for conjecture.

Traumatic Rupture of the Diaphragm

Traumatic rupture of the diaphragm is a relatively uncommon injury, seen in 2% to 3% of patients with blunt chest trauma.[18] In the context of military operations, it is most frequently seen in casualties who are injured when the vehicle in which they are riding detonates a buried antitank mine. The soldier shown in Figure 19-7 was killed during the Vietnam War when the jeep in which he was riding was destroyed by a mine. The chest radiograph explains the scaphoid appearance of his abdomen: his stomach and much of his gut have herniated into the left hemithorax.

Rupture of the left hemidiaphragm accounts for 70% to 75% of these injuries, presumably because of the shielding effect of the underlying mass of the liver on the right.[54] Traumatic diaphragmatic hernia may be discovered at the casualty's initial evaluation or incidentally at thoracotomy or laparotomy performed for other reasons. The casualty may be asymptomatic or may present with respiratory distress. Patients with chronic rupture usually present with symptoms of intestinal obstruction.

Diaphragmatic hernia is caused by three factors in blunt trauma[55]:

1. The abdominothoracic pressure gradient may exceed the usual maximum value of 100 cm H_2O if the trauma patient gasps against a closed glottis at the instant that a large external force raises the intraabdominal pressure.

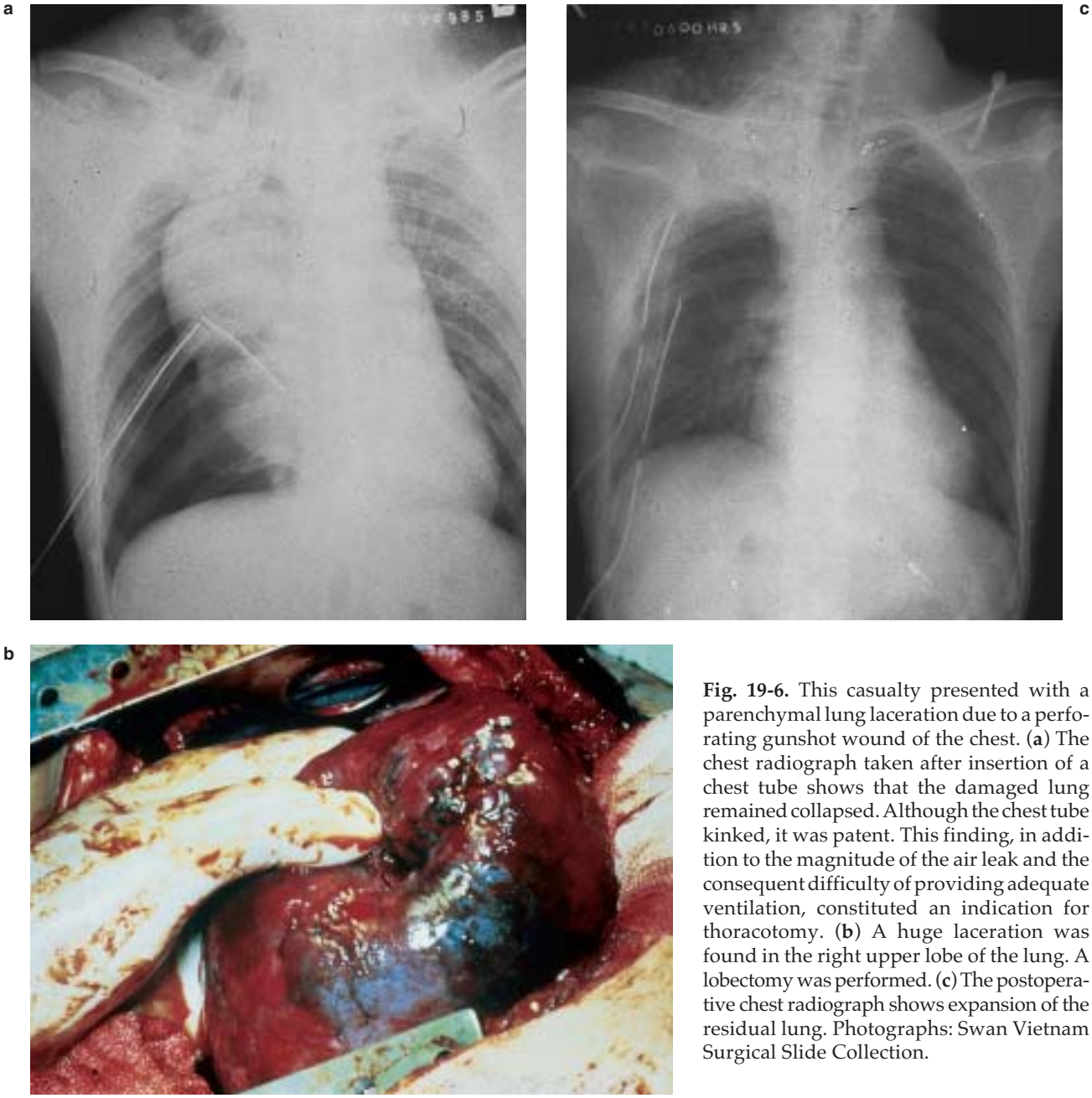


Fig. 19-6. This casualty presented with a parenchymal lung laceration due to a perforating gunshot wound of the chest. (**a**) The chest radiograph taken after insertion of a chest tube shows that the damaged lung remained collapsed. Although the chest tube kinked, it was patent. This finding, in addition to the magnitude of the air leak and the consequent difficulty of providing adequate ventilation, constituted an indication for thoracotomy. (**b**) A huge laceration was found in the right upper lobe of the lung. A lobectomy was performed. (**c**) The postoperative chest radiograph shows expansion of the residual lung. Photographs: Swan Vietnam Surgical Slide Collection.

2. Thoracic compression during blunt trauma distorts the anatomy of the diaphragm and tears it with large shearing forces.
3. The trauma patient may have a congenital weakness of the diaphragm.

The diagnosis of diaphragmatic trauma requires a high degree of clinical suspicion by the military anesthesiologist. Trauma to the diaphragm should be suspected in casualties who have (*a*) injuries below the fifth intercostal space and (*b*) injuries associated with high-energy impact (eg, fractures of the clavicles, upper ribs, scapulae, sternum, pelvis, or thoracolumbar spine). This condition must be suspected when unexplained changes in pulmonary compliance occur intraoperatively in patients who have sustained serious chest injury. Patients with significant migration of viscera into the chest cavity also appear to be at greater risk from aspiration pneumonitis. Postoperative difficulties in

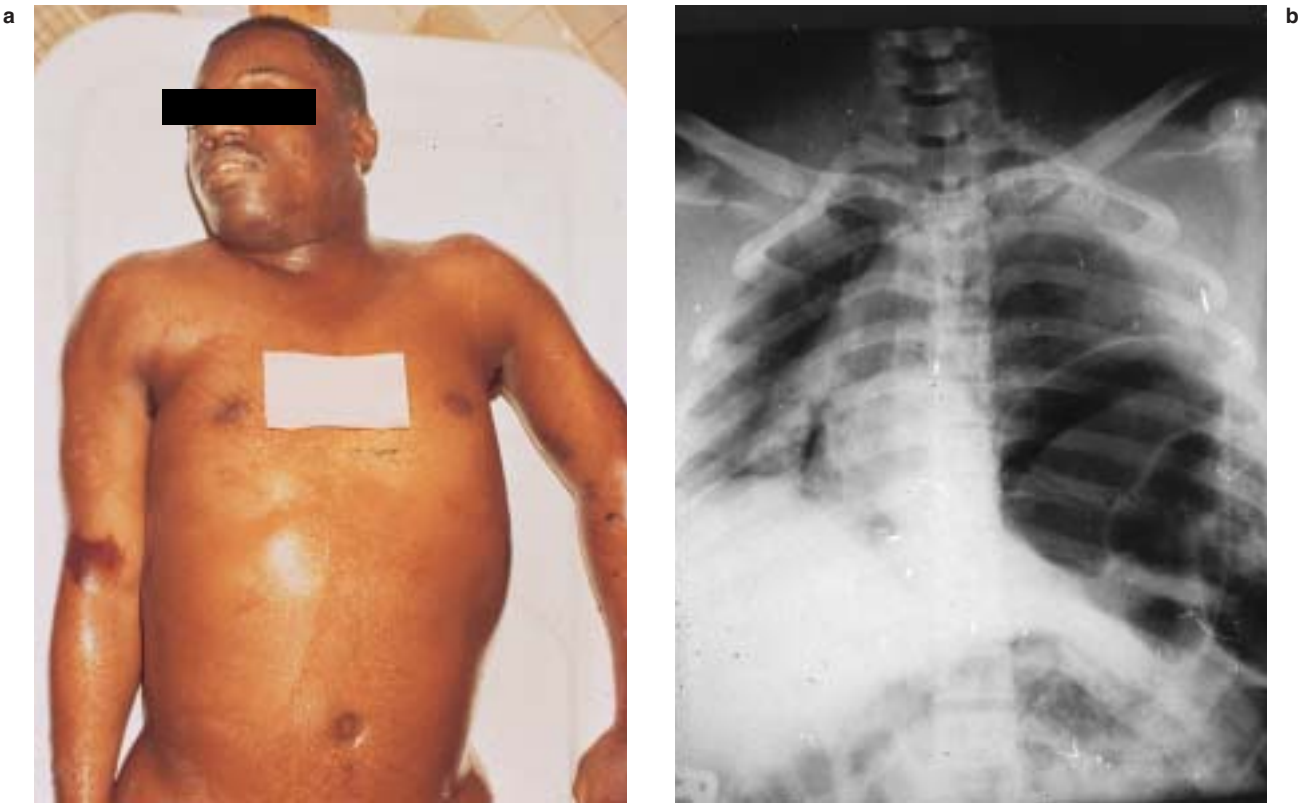


Fig. 19-7. (**a**) The external appearance of this dead soldier is notable for his scaphoid abdomen and his distended chest. (**b**) The chest radiograph shows several transverse lines in the left hemithorax, which are caused by the walls of hollow viscera that have herniated into the chest. The left diaphragm is not apparent. Massive displacement of the mediastinum into the right hemithorax has occurred. Photographs: Wound Data and Munitions Effectiveness Team slide collection.

weaning a patient with thoracic trauma from mechanical ventilation should arouse suspicion of a missed diaphragmatic hernia.

Operative positioning for the repair of a traumatic diaphragmatic hernia depends on the side of the injury and its chronicity. The more common left-side hernia is best repaired through a laparotomy when the diagnosis is made soon after the injury, and when there is no evidence of a significant intrathoracic injury. Chronic left-side hernias are best treated through a low posterolateral incision. Right-side hernias are nearly always repaired through a thoracotomy.

SUMMARY

Thoracic injuries in combat constitute 10% of the surgical workload in wartime. Most of these injuries are caused by penetrating missiles. In caring for soldiers with such injuries, the military trauma anesthesiologist will encounter a paradoxical situation: more often than not, it will be not the thoracic injury itself but an injury to another body part that will be the center of professional attention. The thoracic injury can usually be managed by such simple interventions as inserting a chest tube and debriding wounds.

In most cases in which thoracotomy is indicated, it will be performed for control of (*a*) bleeding from lacerated lung parenchyma or from the chest wall and (*b*) air leaks from lacerated lung parenchyma. Hemorrhage from a laceration, perforation, or rupture of the heart or great vessels will rarely be encountered: such injuries are so rapidly fatal that few casualties will survive long enough to reach a medical treatment facility. If such a casualty does arrive, the military trauma anesthesiologist may be confronted with the need to resuscitate an agonal patient; from the practical standpoint, this means being able to orchestrate an emergency thoracotomy.

Among the more severe intraoperative problems likely to be seen by the military trauma anesthesiologist are (*a*) an air leak that is so massive that tidal volume is inadequate, (*b*) airway obstruction due to accumulation of bloody secretions, and (*c*) systemic air embolism. These problems are best managed by selective intubation of the bronchus of the uninjured lung.

Pulmonary contusion and flail chest are the two treatment problems most likely to be seen in association with blunt trauma. Severe cases will require intubation and mechanical ventilation. As a general rule, the military trauma anesthesiologist will be most successful when treating a casualty with a thoracic injury if he or she prioritizes the many tasks at hand in terms of the elementary ATLS principles of airway, breathing, and circulation management.

REFERENCES

1. McNamara JJ, Messersmith JK, Dunn RA, Molot MD, Stremple JF. Thoracic injuries in combat casualties in Vietnam. *Ann Thor Surg*. 1970;10:389–401.

2. Rosenblatt M, Lemer J, Best LA, Peleg H. Thoracic wounds in Israeli battle casualties during the 1982 evacuation of wounded from Lebanon. *J Trauma*. 1985;25:350–354.

3. Beebe GW, DeBakey ME. *Battle Casualties*. Springfield, Ill: Charles C Thomas; 1952: 167, 181.

4. Bellamy RF, Zajtchuk R. The physics and biophysics of wound ballistics. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistic, Blast, and Burn Injuries*. Part 1, vol 5. In: Zajtchuk R, Jenkins DP, Bellamy RF, eds. *Textbook of Military Medicine*. Washington, DC: US Department of the Army, Office of The Surgeon General, and Borden Institute; 1991: Chap 4.

5. Berry FB. General considerations of thoracic wounds. In: Berry FB, ed. *Thoracic Surgery*. In: Coates JB Jr, ed. *Surgery in World War II*. Vol 1. Washington, DC: US Army Medical Department, Office of The Surgeon General; 1963: 59.

6. Viano DC, Lau IA. A viscous tolerance criterion for soft tissue injury assessment. *J Biomechanics*. 1988;21(5):387–399.

7. Viano DC. Live fire testing: Assessing blunt impact and acceleration injury vulnerabilities. *Milit Med*. 1991;156:589–595.

8. Suleman ND, Rasoul HA. War injuries of the chest. *Injury*. 1985;16:382–384.

9. Zakharia AT. Cardiovascular and thoracic battle injuries in the Lebanon War. *J Thorac Cardiovasc Surg*. 1985;89:723–733.

10. Thompson L. Former Lieutenant Colonel, Medical Corps, US Army. Personal communication, June 1991.

11. Brewer LA. Thoracoabdominal wounds. In: Berry FB, ed. *Thoracic Surgery*. In: Coates JB Jr, ed. *Surgery in World War II*. Vol 2. Washington, DC: US Army Medical Department, Office of The Surgeon General; 1963: 101–136.

12. Bowen TE, Bellamy RF, eds. *Emergency War Surgery NATO Handbook*. 2nd rev US ed. Washington, DC: US Department of Defense, Government Printing Office; 1988: 346–347.

13. Neal S. *Medical Support of the US Army in Vietnam 1965–1970*. Washington, DC: Department of the Army; 1973: 97–98.

14. Finucane BT. Thoracic trauma. In: Kaplan JA, ed. *Thoracic Anesthesia*. 2nd ed. New York, NY: Churchill Livingstone; 1991: 463–484.

15. Kirsh MM. Acute thoracic injuries. In: Siegel J, ed. *Trauma, Emergency Surgery and Critical Care*. New York, NY: Churchill Livingstone; 1987: 863.

16. Stene JK, Grande CM. *Trauma Anesthesia*. Baltimore, Md: Williams & Wilkins; 1991: 221, 226.

17. Duff JH, Goldstein M, McLean APH, et al. Flail chest: A clinical review and physiological study. *J Trauma*. 1968;8:63–74.

18. Symbas PN. *Cardiothoracic Trauma*. Philadelphia, Pa: WB Saunders; 1989: 344, 376–377.

19. Jones TB, Richardson EP. Traction on the sternum in the treatment of multiple fractured ribs. *Surg Gynecol Obstet*. 1926;42:283.

20. Moore BP. Operative stabilization of nonpenetrating chest injuries. *J Thorac Cardiovasc Surg*. 1975;70:619–630.

21. Fulton RL, Peters ET. Physiological effects of fluid therapy after pulmonary contusion. *Am J Surg*. 1973;126:773–778.

22. Meier GH, Symbas PN, Wood WJ. Systemic air embolism following penetrating lung injury. *Ann Thorac Surg*. 1979;27:161–169.

23. Symbas PN. Acute traumatic hemothorax. *Ann Thorac Surg*. 1978;26:195–196.

24. Kavolius J, Golocovsky M, Champion HR. Predictors of outcome in patients who have sustained trauma and who undergo emergency thoracotomy. *Arch Surg*. 1993;128:1158–1162.

25. DiGiacomo JC, Odom JW, Swan KG, Salant M. Resuscitative thoracotomy and combat casualty care. *Milit Med*. 1991;156(8):406.

26. Yao ST, Vanecko RM, Printen K, et al. Penetrating wounds of the heart: A review of 80 cases. *Ann Surg*. 1968;168:67–78.

27. Trinkle JK, Marcos J, Grover FL, et al. Management of the wounded heart. *Ann Thorac Surg*. 1973;17:230–236.

28. Cassell P, Cullum P. The management of cardiac tamponade: Drainage of pericardial effusions. *Br J Surg*. 1967;54:620–626.

29. Proudfit WL, Effler DB. Diagnosis and treatment of cardiac pericarditis by pericardial biopsy. *JAMA*. 1956;161:188–192.

30. Guntheroth WG, Morgan BC, Mullins GL. Effect of respiration on venous return and stroke volume in cardiac tamponade: Mechanism of pulsus paradoxus. *Circ Res*. 1967;20:381–390.

31. Fowler NO, Holmes JC. Hemodynamic effects of isoproterenol and norepinephrine in acute cardiac tamponade. *J Clin Invest*. 1969;48:502.

32. Moseley RE, Vernick JJ, Doty DB. Response to blunt chest injury: A new experimental model. *J Trauma*. 1970;10:673–683.

33. Symbas PN. Cardiac trauma. *Am Heart J*. 1976;92:387–396.

34. Rea WJ, Sugg WL, Wilson LC, et al. Coronary artery lacerations—an analysis of 22 patients. *Ann Thorac Surg*. 1969;7:518–528.

35. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. *Circulation*. 1958;18:371–393.

36. Roberts WC, Maron BJ. Sudden death while playing professional football. *Am Heart J*. 1981;102:1062–1063.

37. Moritz AR. Trauma, stress and coronary thrombosis. *JAMA*. 1954;156:1306–1309.

38. Hillis LD, Braunwald E. Coronary artery spasm. *N Engl J Med*. 1978;299:695–702.