CHAPTER 38

Thoracic Trauma

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Many causes of early deaths (within the first 30 to 180 minutes) resulting from thoracic trauma are preventable and include tension pneumothorax, cardiac tamponade, airway obstruction, and uncontrolled hemorrhage.

Approximately 75% of patients with thoracic trauma require only simple tube thoracostomy and volume resuscitation, and the initial care and disposition of these patients is usually performed by emergency clinicians. Care of severe thoracic trauma is multidisciplinary in nature, involving trauma surgeons, cardiothoracic surgeons, and intensivists. Improved understanding of underlying physiologic mechanisms, newer imaging modalities, minimally invasive approaches, and pharmacologic therapies have contributed to decreasing morbidity and mortality in patients with thoracic injuries.

Injury location and type dictates both assessment and management. This chapter is organized into sections for chest wall, pulmonary, tracheobronchial, diaphragmatic, cardiovascular, and esophageal injuries.

CHEST WALL INJURY

RIB FRACTURE

Principles

Background and Importance

The susceptibility to rib fracture increases with age. These injuries can be exquisitely painful, but their importance lies not in the fracture itself, which generally is self-limiting and will heal, but rather associated complications, particularly pneumothorax, hemothorax, pulmonary contusions, and post-traumatic pneumonia. Rib fractures in children are discussed in Chapter 165.

Anatomy and Physiology

An intact chest wall, protected by its rib cage, is necessary for normal ventilation. Outward expansion of the thorax by the respiratory muscles with descent of the diaphragm creates negative intrathoracic pressure. This allows passive air entry into the lungs during inspiration. Chest trauma, particularly blunt trauma, can severely disturb the physiology of respiration. Fortunately, most individuals have substantial respiratory reserve and can tolerate significant chest wall injury with adequate support.

Flail chest results when three or more adjacent ribs are fractured at two points, allowing a free segment of the chest wall to move in paradoxical motion (Fig. 38.1), with the flail segment moving inward with inspiration and outward with expiration. It can also occur with costochondral separation or vertical sternal fracture in combination with rib fractures. Underlying pulmonary contusion is considered to be the major cause of respiratory insufficiency with flail chest, and it is therefore one of the most serious chest wall injuries (Fig. 38.2). In addition, flail chest can be associated with a variety of other injuries, including hemopneumothorax, liver or spleen lacerations, and mediastinal injury.

Pathophysiology

Ribs usually break at the point of impact or at the posterior angle or posterolateral area, which is structurally the weakest area. The 4th through 9th ribs are most commonly involved. Ribs 1 to 3 are short and relatively protected, and ribs 9 to 12 are longer and more mobile at the anterior end. This confers the relative resistance to fracture of the "high" and "low" ribs. Fractures occur more easily in older adults than younger adults or in children, due to the progressive inelasticity of the chest wall that develops through aging.

The true danger of rib fracture involves not the rib itself but the potential for penetrating injury to the pleura, lung, liver, or spleen. Fractures of ribs 9 to 11 are also associated with intra-abdominal injury. Right-sided rib fractures are associated with hepatic injury and left-sided rib fractures with splenic injury. Injury severity is indicated by the number of rib fractures. The presence of two or more rib fractures at any level is associated with a higher incidence of internal injuries than with a single, isolated fracture. Patients older than 65 years old with multiple rib fractures have a greater incidence of pneumonia and a higher mortality compared their younger counterparts.

Clinical Features

Rib fracture is often a clinical diagnosis, with severe point tenderness, bony crepitus, ecchymosis, and muscle spasm over the rib being the most common findings. Also, bimanual compression of the thoracic cage remote from the site of injury (barrel compression test) usually produces pain at the site of fracture. Injury to the parenchyma may be detected by assessing the respiratory rate, oxyhemoglobin saturation, respiratory effort, effectiveness of ventilation, and pulmonary sounds.

Flail chest is characterized by paradoxical motion of a portion of the chest wall during respiration, and is usually obvious on physical examination. Unless the patient is unconscious, there will be severe pain, splinting, tenderness, and crepitus. Paradoxical chest wall motion is a product of negative intrathoracic pressure, and is obscured if the patient has been intubated and is receiving positive-pressure ventilation. For such patients, the diagnosis is usually evident on examination of the integrity of the chest wall (compression, crepitus).

Differential Diagnoses

Patients with suspected rib fracture have always sustained trauma, thus focusing the evaluation. Patients with significant, potentially multisystem trauma require a thorough trauma evaluation (see Chapter 33). Rib fracture, costochondral separation, and rib contusion may present in similar fashion, and it is not critical to identify a single, isolated rib fracture. Patients with multiple suspected fractures are at higher risk for intrathoracic injury and also for later decompensation and complications. Important diagnoses to consider include chest wall or clavicle fracture (especially sternal fracture), pulmonary injuries (pulmonary contusion/

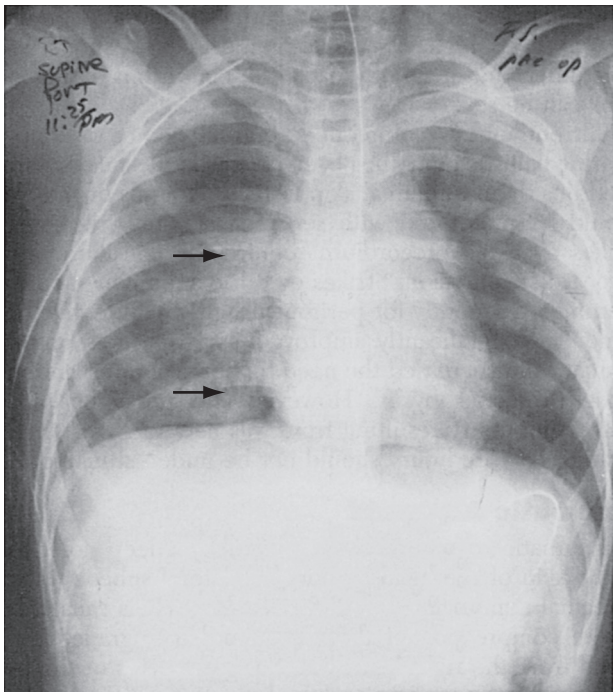

Fig. 38.1. Bilateral alveolar infiltrates *(arrows)* suggesting pulmonary contusion. Pneumopericardium and pneumomediastinum are also present.

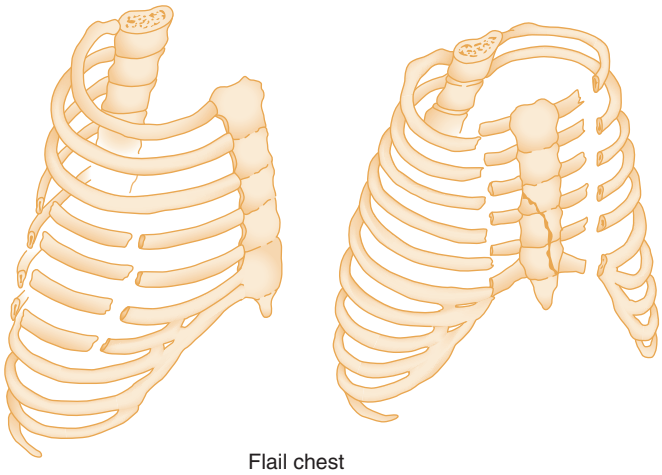

Fig. 38.2. Flail chest. Fracture of several adjacent ribs in two places with lateral flail or central flail segments.

laceration, pneumothorax, or hemothorax), tracheobronchial injury, diaphragmatic injury, cardiovascular injury (cardiac contusion or aortic injury), or esophageal injury. This broad differential diagnosis holds true for most patients with thoracic trauma, given the close proximity of the organs involved and the similar mechanisms behind most of the injuries in this chapter.

Diagnostic Testing

Many patients with relatively minor thoracic trauma are evaluated and managed exclusively based on physical findings, and they do not require imaging. When the injury is significant enough to raise concern for underlying pulmonary injury, imaging is required. Because rib fractures are managed expectantly, imaging should be reserved for patients in whom multiple rib fractures, underlying pulmonary injury, or comorbid pulmonary status (eg,

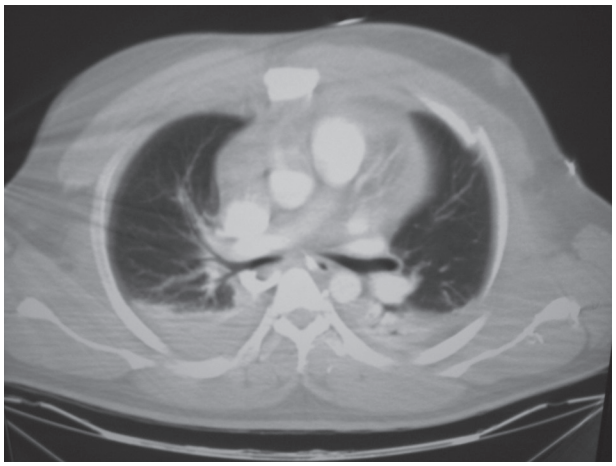

Fig. 38.3. Multiple rib fractures seen on chest computed tomography (CT) scan. (Also note presence of bilateral hemothoraces.)

BOX 38.1

NEXUS-Chest Computed Tomography Criteria for Chest Computed Tomography After Blunt Trauma

Abnormal chest x-ray
Rapid deceleration mechanism (defined as fall >20 feet or motor vehicle collision >40 mph)
Distracting painful injury
Chest wall tenderness
Sternal tenderness
Thoracic spine tenderness
Scapular tenderness

From Rodriguez RM, Langdorf MI, Nishijima D, et al: Derivation and validation of two decision instruments for selective chest CT in blunt trauma: a multicenter prospective observational study (NEXUS Chest CT). PLoS Med 12(10):e1001883, 2015.

chronic obstructive pulmonary disease [COPD]) is a concern. A plain chest radiograph will identify only about 50% of single-rib fractures; its greatest value is in identifying or excluding significant intrathoracic and mediastinal injuries. Rib series and expiratory, oblique, and cone-down views should not be used routinely. If additional imaging is required beyond a standard upright posteroanterior and lateral chest x-ray, a computed tomography (CT) scan should be obtained (Fig. 38.3). A CT scan is not indicated to confirm suspected isolated rib fracture, but it will identify multiple-level fractures and associated pulmonary injury, such as pneumothorax or hemothorax, with much greater accuracy than additional chest x-ray views.

In an attempt to limit unnecessary use of diagnostic ionizing radiation, the National Emergency X-Radiography Utilization Study (NEXUS) group derived and validated a decision instrument to guide the use of chest CT in patients with blunt trauma.[1] The NEXUS-Chest CT derivation and validation studies, performed at eight trauma centers in the United States, enrolled 11,477 patients. In the validation phase, their decision instrument had a sensitivity of 95.4%, a negative predictive value of 93.9%, and a specificity of 25.5% for all thoracic injuries. The results were even more impressive for major clinical injuries, with both sensitivity and negative predictive value approaching 100% (99.2% and 99.8%, respectively). We recommend that CT scan not be obtained for patients sustaining blunt trauma who do not meet any of the seven NEXUS-Chest CT criteria (Box 38.1).[2]

Management

Respiratory decompensation is the primary indication for endotracheal intubation and mechanical ventilation for patients with multiple rib fractures. Obvious problems, such as hemopneumothorax or severe pain, should be corrected before intubation and ventilation are presumed necessary. In the awake and cooperative patient, noninvasive continuous positive airway pressure (CPAP) by mask may obviate the need for intubation.[3] In general, the most conservative methods for maintaining adequate oxygenation and preventing complications should be used. Adequate analgesia is of paramount importance in patient recovery and may contribute to the return of normal respiratory mechanics.

Treatment of acute rib fractures is based on adequate pain relief and the maintenance of pulmonary function. Outdated management techniques involving stabilization of the flail segment by positioning the person with the injured side down or placing a sandbag on the affected segments have been discredited. These interventions actually inhibit expansion of the chest and produce increased atelectasis of the injured lung. Instead, oxygen should be administered, cardiac and oximetry monitors applied, and the patient observed for signs of an associated injury, such as tension pneumothorax.

Otherwise well patients with single rib fractures are managed with opioid and non-opioid analgesia. These injuries can be severely painful, and regular opioid medication, particularly at bedtime, is usually necessary for up to a week. Thereafter, simple analgesia with acetaminophen or a nonsteroidal anti-inflammatory analgesic generally will suffice. Continuing daily activities and deep breathing should be stressed to ensure ventilation and prevent atelectasis. It is helpful to advise patients to wait 30 to 45 minutes after taking their pain medications before performing deep breathing exercises, ideally with an incentive spirometer.

The greater the number of fractured ribs is, the higher the mortality and morbidity rates. Hospitalization should be considered for patients with three or more fractured ribs, despite the lack of other identified injuries, to receive pulmonary therapy, repeated evaluation, and appropriate effective analgesia. Elderly patients with six or more fractured ribs should be treated in intensive care units owing to high morbidity and mortality.

Multiple rib fractures in trauma patients are associated with significant morbidity and mortality. Intercostal nerve blocks with a long-acting anesthetic, such as bupivacaine with epinephrine, may relieve symptoms up to 12 hours. Other alternatives for hospitalized patients include patient-controlled analgesia, parenteral opioids, and thoracic epidural analgesia.

For flail segments, consultation with a trauma or thoracic surgeon is essential to plan for surgical intervention. Early operative internal fixation of the flail segment results in a speedier recovery, decreased complications, and better cosmetic and functional results, and it is cost-effective.[3] Indications for open fixation for flail chest include patients who are unable to be weaned from the ventilator secondary to the mechanics of flail chest, persistent pain, severe chest wall instability, and a progressive decline in pulmonary function.[4]

The patient with flail chest should be treated in the emergency department (ED) as if pulmonary contusion exists regardless of whether mechanical ventilation is used.

Disposition

Most rib fractures heal uneventfully within 3 to 6 weeks, and patients should expect a gradual decrease in their discomfort during this period. However, in addition to the complications of hemopneumothorax, atelectasis, pulmonary contusion, and pneumonia, rib fractures can result in post-traumatic neuroma, empyema, nonunion, or costochondral separation.[5] These rare complications are painful and heal slowly (if at all). Patients with blunt trauma and multiple rib fractures should be observed for 12 to 24 hours to ensure that occult vascular or intrapulmonary injuries are not present and then considered for discharge, with the full understanding that the rib fractures themselves will require a prolonged recovery period and close follow-up.

The outcome of flail chest is a function of associated injuries. Because many different physiologic mechanisms have been implicated in flail chest, there is no consensus about hospital treatment. The cornerstones of therapy include pulmonary physiotherapy, effective analgesia, selective use of endotracheal intubation and mechanical ventilation, and close observation for respiratory compromise.

STERNAL FRACTURE

Principles

Background and Importance

Sternal fractures and dislocations are caused primarily by anterior blunt chest trauma (eg, motor vehicle collisions [MVCs] or bicycle accidents when the chest strikes the steering wheel or handlebars). Risk factors for sternal fracture from blunt trauma include types of vehicular passenger restraint systems and patient age. Restrained passengers are more likely than unrestrained passengers to sustain sternal fracture, likely related to the central location of the shoulder portion of the restraint. Cardiac complications, such as myocardial contusion, occur rarely, and there is no association between sternal fracture and aortic rupture. Although sternal fractures may occur in the context of major blunt chest trauma, the presence of a sternal fracture itself does not imply other major life-threatening conditions.

Pathophysiology

During rapid deceleration from a frontal impact, the forward thrust of the body against the fixed seat belt across the sternum can result in a fracture. The location of the sternal fracture varies depending on the position of the belt, patient size, the magnitude of the impact, and the vector of the forces.

Clinical Features

Patients with sternal fractures typically present with a history compatible with the injury, and anterior chest pain, point tenderness over the sternum, ecchymosis, soft tissue swelling, or palpable deformity.

Diagnostic Testing

When sternal fracture is suspected after a relatively minor mechanism of injury (eg, ground level fall or punch to the chest), posteroanterior and lateral chest radiography is sufficient to establish the diagnosis and for evaluation of the pulmonary structures. However, when more significant traumatic signs or symptoms are present (as per the NEXUS-Chest CT criteria)[1] or when plain radiography shows a displaced fracture or possible evidence of intrathoracic injury, we recommend obtaining a chest CT scan. Results will guide management of the sternal fracture and any associated mediastinal or other intrathoracic injuries.[6] Notably, there are also specific ultrasound views during, or in addition to, evaluation by extended focused assessment with sonography in trauma (E-FAST) that may be more sensitive than plain radiography for sternal fracture.[7]

Management

Treatment consists of providing adequate analgesia, as for rib fractures. In the absence of associated injuries, most patients with isolated sternal fractures who can achieve adequate pain control with oral medications can be safely discharged home. A small subset of patients with more severe sternal fractures may have severe pain and develop respiratory compromise or nonunion. These patients are best referred for operative fixation.

NONPENETRATING BALLISTIC INJURY

Principles

Background and Importance

Many law enforcement officers, emergency medical services personnel, and private security guards wear lightweight synthetic body armor for protection against gunshot injury. In addition, there have been a number of reports of armed robbers wearing such vests in anticipation of exchanging gunfire with police or security personnel. These vests are "bullet resistant" rather than "bulletproof," depending on the weapon being used against them. They are composed of many different combinations of synthetic fibers such as Kevlar, and so wearers who are shot often suffer nonpenetrating ballistic injuries rather than gunshot wounds.

Another type of nonpenetrating ballistic injury is caused by rubber bullets and beanbag shotgun shells. Rubber bullets have been used for many years by police agencies throughout the world for crowd dispersal and for nonlethal use of force. Beanbag shotgun shells are nylon bags filled with pellets, which are fired from a standard shotgun. Both of these projectiles have the potential to cause serious injury despite their classification of "nonmetal" or "less-than-lethal" use of force.

Pathophysiology

Bullet-resistant vests are usually capable of stopping penetration by the low-velocity missiles of most handguns, but the kinetic energy of the missile can be transmitted through the layers of protective cloth or armor and produce significant injury without penetration. The heart, liver, spleen, lung, and spinal cord are vulnerable to nonpenetrating ballistic injury that may occur despite innocent-appearing skin lesions.

Clinical Features

Patients who have been shot with "less-than-lethal projectiles" or with standard bullets while wearing bullet-resistant vests usually have erythema, ecchymosis, and marked tenderness to palpation over the affected area. There may be a projectile, such as a beanbag, still located in the wound. The area of tenderness and surrounding structures should be carefully palpated to identify any subcutaneous emphysema, crepitus, or bony step-offs.

Diagnostic Testing

Most patients with nonpenetrating ballistic injury do not require testing beyond a thorough physical examination. Those in whom there is concern for retained foreign body or underlying injury may need ultrasound examination, chest x-ray, or CT scan (as they might with other blunt trauma). However, patients with only superficial ecchymoses without clinical signs or symptoms of rib fracture, pneumothorax, hemothorax, or intrapleural/peritoneal penetration frequently require no additional testing.

Management

In patients in whom underlying injury has been excluded or it is of low clinical probability, management of nonpenetrating ballistic injury focuses on wound care, either of the ecchymotic area or of the superficial abrasion/laceration. Underlying injuries, when present, should be managed as noted elsewhere in this chapter.

Disposition

It is recommended that patients with all but the most superficial nonpenetrating ballistic injuries to the chest be observed closely for 4 to 6 hours to detect internal injuries that may manifest in a delayed manner.

PULMONARY INJURIES

PULMONARY CONTUSION AND LACERATION

Principles

Background and Importance

Pulmonary contusion is reported to be present up to 75% of patients with significant blunt chest trauma, most often from MVCs with rapid deceleration.[8] Pulmonary contusion can also be caused by high-velocity missile wounds and the high-energy shock waves of an explosion in air or water.

In addition to contusions, the lungs can also sustain lacerations. Although they are most often lacerated from penetrating injury, they may also be injured by the inward projection of a fractured rib or avulsion of a pleural adhesion.[9,10]

Pathophysiology

Pulmonary contusion is caused by an impact to the lung parenchyma followed by alveolar edema and hemorrhage but without an accompanying pulmonary laceration. The early diagnosis of pulmonary contusion is important if treatment is to be successful. Since its onset may be insidious, it should be suspected from a history of a high mechanism of injury (eg, a fall from height, an MVC, and other forms of significant trauma) rather than the initial chest radiograph.

Clinical Features

The clinical manifestations include dyspnea, tachypnea, cyanosis, tachycardia, hypotension, and chest wall bruising. There are no specific signs for pulmonary contusion or laceration, but hemoptysis may be seen. Moist rales or absent breath sounds may be heard on auscultation. Palpation of the chest wall commonly reveals fractured ribs. If flail chest is discovered, pulmonary contusion is commonly present.

Surprisingly, many of the worst contusions occur in patients without rib fractures. It has been theorized that the more elastic chest wall in younger individuals transmits increased force to the thorax. Although isolated pulmonary contusions can exist, they are associated with extrathoracic injuries in the majority of patients.

Diagnostic Testing

Laboratory

Hypoxemia frequently occurs with pulmonary contusions, and is often detected by a decreasing pulse oximetry reading. In patients

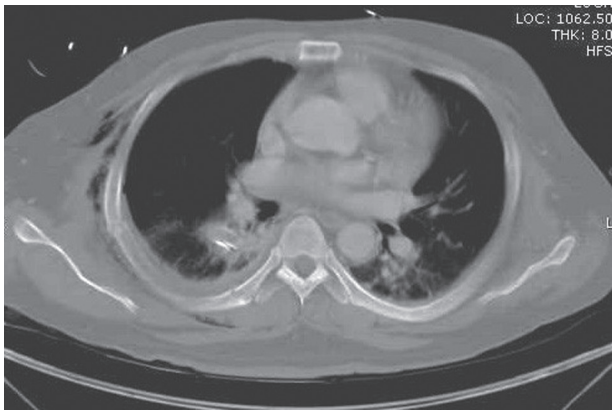

Fig. 38.4. Chest computed tomography (CT) scan showing bilateral alveolar infiltrates suggesting pulmonary contusion. There are also multiple rib fractures and subcutaneous emphysema.

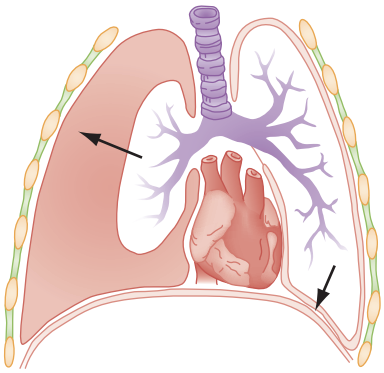

Fig. 38.5. Closed pneumothorax. Simple pneumothorax is present in the right lung with air in the pleural cavity *(left arrow)* and collapse of the right lung *(right arrow)*.

with thoracic injury and hypoxemia in whom other, more severe injuries (eg, pneumothorax) have been excluded, a pulmonary contusion should be suspected. In these patients, an arterial blood gases may be helpful in making the diagnosis, because a widening alveolar-arterial oxygen difference indicates a decreasing pulmonary diffusion capacity of the patient's contused lung, and it is one of the earliest and most accurate means of assessing the current status, progress, and prognosis.

Radiology

Typical radiographic findings can begin to appear within minutes of injury and range from patchy, irregular, alveolar infiltrate to frank consolidation (Fig. 38.4). Although these changes may be present on the initial examination, they are almost always present within 6 hours. The rapidity of changes on chest x-ray visualization usually correlates with the severity of the contusion or laceration. Pulmonary contusion should be differentiated from acute respiratory distress syndrome (ARDS), with which it is often confused because the radiographic appearance of the two conditions may be similar. Contusion usually manifests within minutes of the initial injury, is usually localized to a segment or a lobe, is often apparent on the initial chest study, and tends to last 48 to 72 hours. ARDS is diffuse, and its development is usually delayed, with onset typically between 24 and 72 hours after injury.

The increased frequency of CT scans for blunt trauma patients has resulted in a corresponding increase in the diagnosis of pulmonary contusions and lacerations. CT scans have been shown to detect at least twice as many pulmonary contusions as plain radiographs, and one recent study found that isolated pulmonary contusions seen on CT only had a mortality of only 2.6% (compared to 4.7% for all patients with pulmonary contusion).[11]

Chest CT scan is particularly valuable to identify a pulmonary contusion in the acute phase after injury because plain chest x-ray films have a low sensitivity. Although CT scan may not be necessary to make the diagnosis of a pulmonary contusion that is evident on plain chest radiography, it may be helpful to further define the extent of the contusion and to identify other thoracic injuries. Occult pulmonary contusions are those that are initially visible only on CT scan, not plain radiographs, and usually involve less than 20% of the lung volume. These occult pulmonary contusions are not associated with a worse clinical outcome as compared with blunt trauma patients without pulmonary contusion.[11,12]

Management

Treatment for pulmonary contusion is primarily supportive.[12] As with flail chest, intubation and mechanical ventilation should be avoided if possible, because they are associated with an increase in morbidity, including pneumonia, sepsis, pneumothorax, hypercoagulability, and longer hospitalization.[13] In the rare case in which one lung has been severely contused *and is causing significant hypoxemia,* consideration should be given to intubating and ventilating each lung separately with a dual-lumen endotracheal tube and two ventilators. This allows for the difference in compliance between the injured and the normal lung and prevents hyperexpansion of one lung and gradual collapse of the other.

Management of patients with pulmonary contusions should include the restriction of intravenous (IV) fluids (to maintain intravascular volume within strict limits) and comprehensive supportive care consisting of vigorous tracheobronchial toilet, suctioning, and pain relief. These maneuvers may preclude the need for ventilator support and allow a more selective approach to both flail chest and pulmonary contusion.

Patients sustaining the force necessary to inflict a pulmonary contusion may also have pulmonary lacerations. Most of these are minor and rarely life-threatening, and they can usually be treated with continuous oxygen therapy, observation, or tube thoracostomy. Severe lacerations are associated with hemopneumothorax, multiple displaced rib fractures, and hemoptysis. Often, these life-threatening lacerations require thoracotomy with resection or tractotomy to control bleeding.

PNEUMOTHORAX

Principles

Background and Importance

Pneumothorax, which is the accumulation of air in the pleural space, is a common complication of chest trauma. It is reported to be present in 15% to 50% of patients who sustain significant chest trauma and is invariably present in those with transpleural penetrating injuries.

Pathophysiology

Pneumothorax can be divided into three types depending on whether air has direct access to the pleural cavity: (1) simple, (2) communicating, and (3) tension.

Simple Pneumothorax. A pneumothorax is considered simple (Fig. 38.5) when there is no communication with the atmosphere or any shift of the mediastinum or hemidiaphragm resulting from the accumulation of air. Traumatic simple pneumothorax is most often caused by a fractured rib that is driven inward, lacerating the pleura. It may also occur without a fracture

when the impact is delivered at full inspiration with the glottis closed, leading to a tremendous increase in intra-alveolar pressure and the subsequent rupture of the alveoli. A penetrating injury, such as a gunshot or stab wound, may also produce a simple pneumothorax if there is no free communication with the atmosphere (Fig. 38.6).

Communicating Pneumothorax. A communicating pneumothorax (Fig. 38.7) is associated with a defect in the chest wall and most commonly occurs in combat injuries. In the civilian sector, this injury is typically secondary to gunshot wounds. Air can sometimes be heard flowing sonorously in and out of the defect, prompting the term "sucking chest wound." The loss of chest wall integrity causes the involved lung to paradoxically collapse on inspiration and expand slightly on expiration, forcing air in and out of the wound. This results in a large functional dead space for the normal lung and, together with the loss of ventilation of the involved lung, produces a severe ventilatory disturbance.

Tension Pneumothorax. The progressive accumulation of air under pressure within the pleural cavity, with shift of the mediastinum to the opposite hemithorax and compression of the contralateral lung and great vessels, is the constellations of findings in tension pneumothorax (Figs. 38.8 and 38.9). It occurs when the injury acts like a one-way valve, prevents free bilateral communication with the atmosphere, and leads to a progressive increase of intrapleural pressure. Air enters on inspiration but cannot exit with expiration. The resulting shift of mediastinal contents compresses the vena cava and distorts the cavoatrial junction, leading to decreased diastolic filling of the heart and subsequent decreased cardiac output. These changes result in the rapid onset of hypoxia, acidosis, and shock.

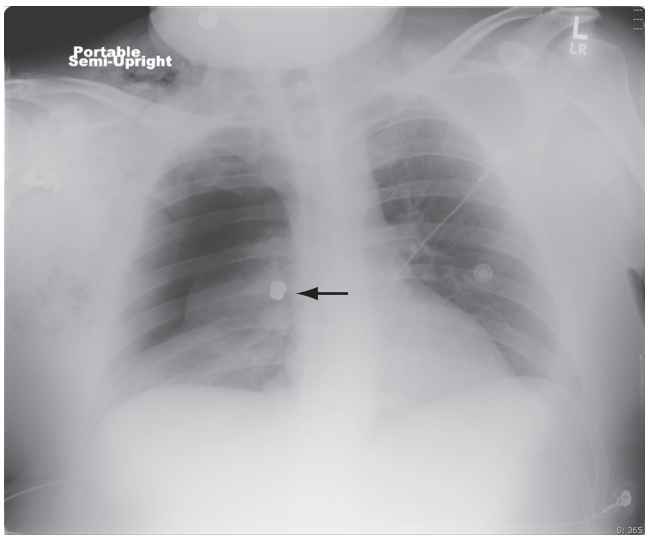

Fig. 38.6. Chest radiograph showing a moderate-sized right pneumothorax. A bullet *(arrow)* can be seen near the right mediastinum.

Clinical Features

Shortness of breath and chest pain are the most common presenting complaints of pneumothorax. The patient's appearance is highly variable, ranging from acutely ill with cyanosis and tachypnea to misleadingly healthy. The signs and symptoms are not always correlated with the degree of pneumothorax. The physical examination may reveal decreased or absent breath sounds and hyper-resonance over the involved side as well as subcutaneous emphysema, but small pneumothoraces may not be detectable on physical examination.

Patients with tension pneumothorax become acutely ill within minutes and develop severe cardiovascular and respiratory distress. They are dyspneic, agitated, restless, cyanotic, tachycardic, and hypotensive and display decreasing mental activity. The cardinal signs of tension pneumothorax are tachycardia, hypotension, oxyhemoglobin desaturation, jugular venous distention (JVD), and absent breath sounds on the ipsilateral side. However, JVD may not reliably be present with massive blood loss.

Diagnostic Testing

Because intrapleural air tends to collect at the apex of the lung, the initial chest radiograph should be an upright full inspiratory film if the patient's condition permits. An upright film will often reveal small pleural effusions that are not visible on supine films, and it also allows better visualization of the mediastinum. Although the chest radiograph has traditionally been the preferred initial study for diagnosing a simple pneumothorax, several studies have found that ultrasound has greater sensitivity for pneumothorax than chest radiography.[14,15] During the focused assessment with sonography in trauma (FAST) examination, pneumothorax will be detected well before chest radiography is performed. This may be particularly critical in the hypotensive

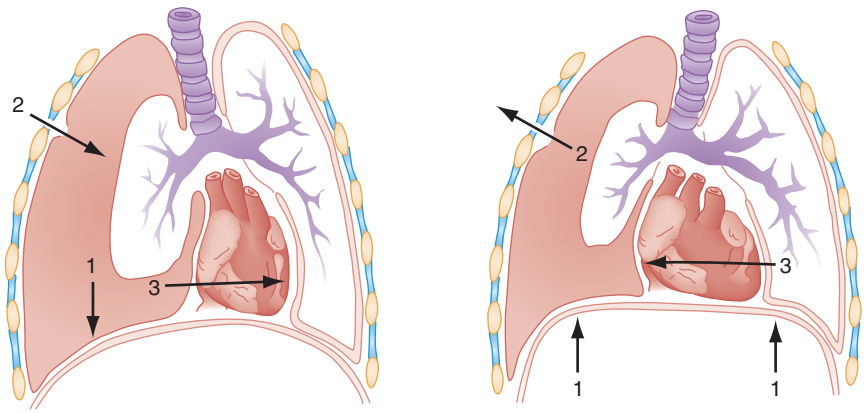

Fig. 38.7. Inspiration *(left):* The diaphragm contracts causing negative intrathoracic pressure *(arrow 1)* that draws air through the sucking chest wound in the pleural cavity *(arrow 2)* and causing the mediastinal structures to shift to the patient's left *(arrow 3).* Expiration *(right):* The diaphragm recoils *(arrow 1)* causing air to exit the chest *(arrow 2)* and allowing the mediastinum to shift back to normal position *(arrow 3).* The collapsed lung paradoxically shrinks on inspiration and expands on expiration.

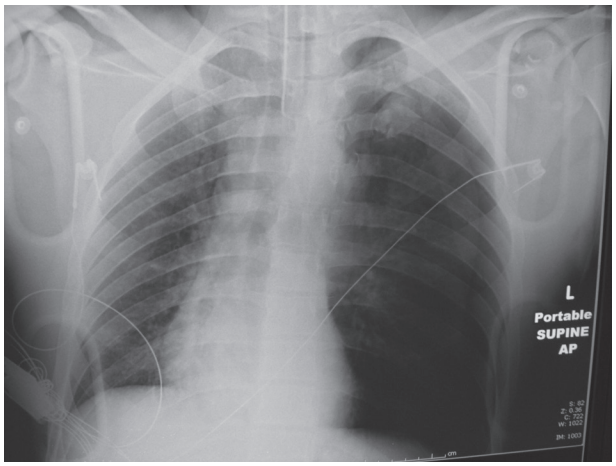

Fig. 38.8. Tension pneumothorax seen in intubated patient.

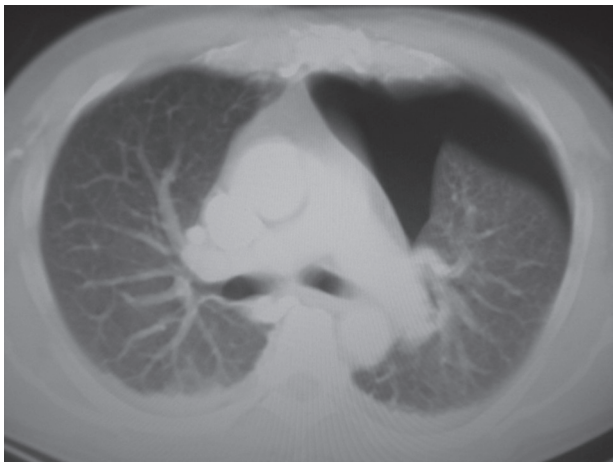

Fig. 38.10. Occult pneumothorax. Large left-sided pneumothorax visible on chest computed tomography (CT) scan, which was not visible on chest x-ray film.

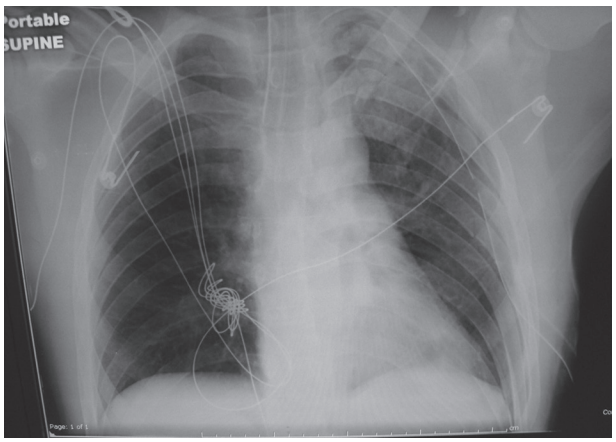

Fig. 38.9. Resolution of the tension pneumothorax shown in Figure 38.7 with placement of a left-sided tube thoracostomy.

BOX 38.2

Indications for Tube Thoracostomy

Traumatic cause of pneumothorax (except asymptomatic, apical pneumothorax)
Moderate to large pneumothorax
Respiratory symptoms regardless of size of pneumothorax
Increasing size of pneumothorax after initial conservative therapy
Recurrence of pneumothorax after removal of an initial chest tube
Patient requires ventilator support
Patient requires general anesthesia
Associated hemothorax
Bilateral pneumothorax regardless of size
Tension pneumothorax

From Dougall AM, et al: Chest trauma—current morbidity and mortality. J Trauma 17:547, 1977.

poly-trauma patient with oxyhemoglobin desaturation for whom tension pneumothorax is one of myriad considerations as the cause of the unstable state. Suspicion of tension pneumothorax on the basis of clinical findings is indication for immediate tube thoracostomy. Treatment should not be delayed pending a confirmatory chest radiograph. Ultrasound can confirm the presence of a pneumothorax within the first minutes of the patient's arrival, and identification of a pneumothorax in a patient with pulmonary and hemodynamic compromise is also considered confirmatory empirical evidence of tension pneumothorax requiring thoracostomy. Even without ultrasound confirmation, clinical suspicion of tension pneumothorax is justification for tube thoracostomy, because delay can be hazardous.

Occult Pneumothorax

A pneumothorax that is absent on the initial chest radiograph but is identified on subsequent chest or abdominal CT is called an *occult pneumothorax.* Occult pneumothorax is being diagnosed more frequently given the increased use of CT (Fig. 38.10).[16-18]

Management

An asymptomatic patient who suffers a low-velocity penetrating trauma (typically a stab wound) and who has negative initial imaging can be safely observed, typically for 6 hours.[16] If the initial imaging was a CT scan, the patient may be safely discharged after the period of observation. If the initial imaging was via x-ray, a delayed chest x-ray should be performed prior to discharge.

Simple Pneumothorax

Treatment of a simple pneumothorax depends on its cause and size. Small isolated apical pneumothoraces due to stab wounds may be observed without intervention. However, this conservative method seldom has application in multisystem trauma, and a chest tube should be inserted immediately upon any sign of deterioration.

Similarly, small occult pneumothoraces found only on CT scan in hemodynamically stable patients without symptoms can be managed with observation and do not need treatment, even if the patient is placed on positive-pressure ventilation.[17-19]

Any moderate to large pneumothorax should be treated with a chest tube. The indications for tube thoracostomy (chest tube) are listed in Box 38.2. The preferred site for insertion is the fourth or fifth intercostal space at the midaxillary line. This lateral placement of the tube is preferred not only because it is more efficient, but also because it does not produce an easily visible cosmetic defect, as does the anterior site at the second interspace at the midclavicular line. With multisystem trauma in which hemothorax is likely, a large chest tube (36-F to 40-F in adults and 16-F to 32-F in children) should be used. Conversely, spontaneous pneumothoraces or those due to minor or isolated injuries can be treated with smaller chest tubes.

Care is taken to be certain the vent holes along the side of the tube are all inside the chest cavity. A radiopaque line along the side of the tube with interruptions at these drainage holes helps greatly in radiographically interpreting tube position. The tube should be attached to a water seal drainage system that allows reexpansion of the pneumothorax. If there is significant air leak or a large hemothorax, the tube may be connected to a source of constant vacuum at 20 to 30 cm H_2O for more rapid reexpansion.

Tube thoracostomy does have some potentially serious complications, including the formation of a hemothorax, pulmonary edema, bronchopleural fistula, pleural leaks, empyema, subcutaneous emphysema, infection, intercostal artery laceration, contralateral pneumothorax, and parenchymal injury.[20,21] A recent meta-analysis was unable to find sufficient evidence for or against the use of empirical antibiotics with all tube thoracostomy placements to prevent empyema or pneumonia, especially those needed for spontaneous pneumothoraces. However, for patients with multisystem trauma or hemothorax, the data was suggestive of a benefit. We recommend routine intravenous antibiotic administration in these patients, specifically cefazolin 1 to 2 g given prior to—or within 1 hour of—chest tube insertion. Vancomycin (1 g) or clindamycin (600 mg) are appropriate alternatives in patients in whom cephalosporins are inappropriate.[22]

Communicating Pneumothorax

For a patient with a communicating pneumothorax in the out-of-hospital setting, the defect should be covered immediately, which helps convert the condition to a closed pneumothorax, eliminating the major physiologic abnormality. Either a partially occlusive dressing or a commercial vented chest seal can be applied; care should be taken to continually assess for conversion of the injury to a tension pneumothorax, especially in patients who are intubated and undergoing positive-pressure ventilation.[23-25] The wound should never be packed, because the negative pressure during inspiration can suck the dressing into the chest cavity. These considerations are not as important once the patient is in the ED, where formal tube thoracostomy can be performed. Positive-pressure ventilation can then be initiated, if needed, without the fear of producing a tension pneumothorax, and the patient can be prepared for definitive surgical repair.

Tension Pneumothorax

When the diagnosis of tension pneumothorax is suspected clinically, the pressure should be relieved immediately with needle thoracostomy, which is performed by inserting a large-bore (14-gauge or larger) catheter, at least 5 cm in length, through the fourth or fifth interspace laterally or the second or third interspace anteriorly on the involved side. Recent studies have suggested that some catheters may not be of sufficient length to penetrate the pleural space.[26,27] So, we recommend the lateral approach if it is accessible. This method can be easily performed in the field, allowing vital signs to improve during transport or preparation for a tube thoracostomy.[28]

In the ED, it may be just as expeditious to insert a chest tube (or even perform a "finger thoracostomy," without actually inserting the chest tube) as it is to perform a needle thoracostomy, depending on the availability of equipment. Regardless, even if a needle thoracostomy is performed on a patient with suspected tension pneumothorax in the ED, a chest tube should emergently follow.

The intubated patient in the ED who is receiving positive-pressure ventilation and external cardiac compressions is at particular risk for developing tension pneumothorax. Fractured ribs from cardiopulmonary resuscitation (CPR) can penetrate lung parenchyma and cause pneumothorax. Positive-pressure ventilation then increases intrapleural pressure and produces a tension pneumothorax. The earliest sign of this complication is an increase in resistance to ventilation. If the patient has vital signs, the blood pressure will fall and the central venous pressure (CVP) will rise. Misplacement of an endotracheal tube does not result in tension pneumothorax but, rather, asymmetry of breath sounds. If tension pneumothorax is suggested, the clinician should proceed with empirical emergent therapy.

HEMOTHORAX

Principles

Background and Importance

Hemothorax, which is the accumulation of blood in the pleural space after blunt or penetrating chest trauma, is a common complication that may produce hypovolemic shock and dangerously reduce vital capacity. It is commonly associated with pneumothorax and extrathoracic injuries.

Pathophysiology

Hemorrhage from injured lung parenchyma is the most common cause of hemothorax, but this tends to be self-limited unless there is a major laceration. Specific vessels are less often the source of hemorrhage, with intercostal and internal mammary arteries causing hemothorax more often than hilar or great vessels. Bleeding from the intercostal arteries may be brisk, however, because they branch directly from the aorta.

Clinical Features

Depending on the rate and quantity of hemorrhage, varying degrees of hypovolemic shock will be manifested. Patients may present in respiratory distress and be tachycardic and hypoxemic. Breath sounds may be diminished. The diagnosis should also be remembered as a potential complication of central line placement, and considered—along with pneumothorax—in patients who present with these symptoms after the procedure.

Diagnostic Testing

The *upright* chest radiograph remains the primary diagnostic study in the acute evaluation of hemothorax. A hemothorax is noted as meniscus of fluid blunting the costophrenic angle and tracking up the pleural margins of the chest wall when viewed on the upright chest x-ray film. Blunting of the costophrenic angles on upright chest radiograph requires at least 200 to 300 mL of fluid. The supine view chest film is less accurate but, unfortunately, is often the only film available because of the patient's unstable condition. In the supine patient, blood layers posteriorly, creating a diffuse haziness that can be rather subtle (or appear to be a pulmonary contusion), depending on the volume of the hemothorax (Fig. 38.11). With massive hemothorax, the large volume of blood can create a tension hemothorax, with signs and symptoms of both obstructive and hemorrhagic shock (Fig. 38.12).

As is the case with pneumothoraces, ultrasound has greater sensitivity than supine chest radiography in the detection of hemothoraces.[29] Given this, early bedside ultrasonography should routinely be performed in patients with thoracic trauma, regardless of findings on supine radiography. Additional imaging via chest CT (Fig. 38.13) should be performed if indicated by the NEXUS-Chest CT criteria as discussed earlier,[1] because it may detect hemothorax or other associated injuries (Fig. 38.14).

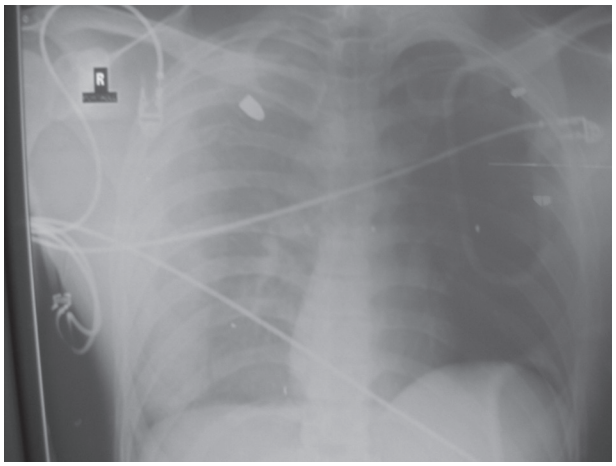

Fig. 38.11. Hemothorax secondary to gunshot wound. Note haziness over right hemithorax with bullet seen in right upper lobe.

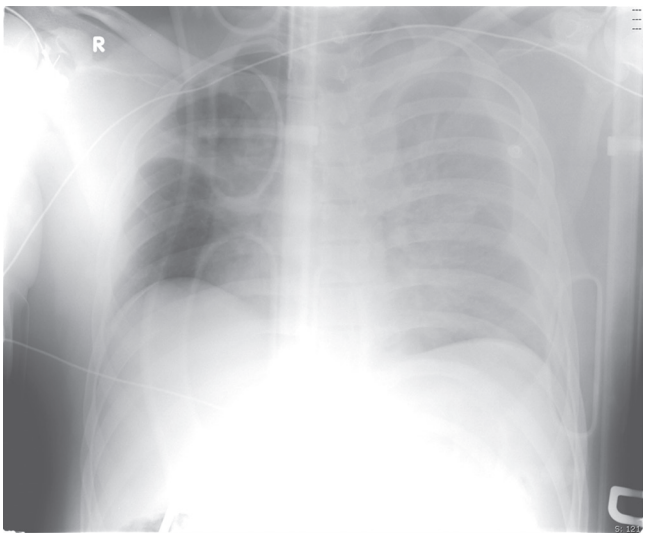

Fig. 38.12. Tension hemothorax.

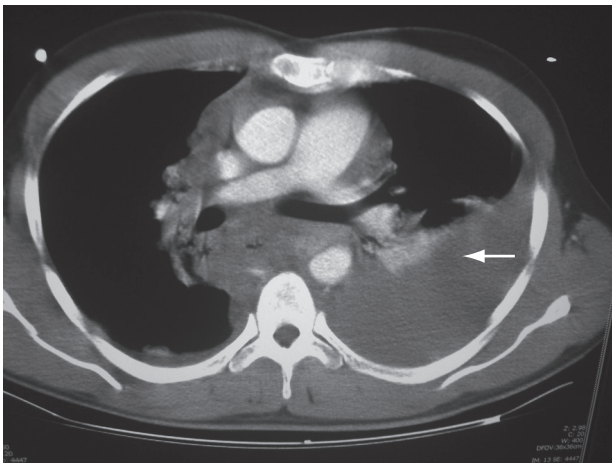

Fig. 38.13. Left-sided hemothorax visible on chest computed tomography (CT) scan *(arrow)*.

Management

Treatment of hemothorax consists of restoring the circulating blood volume, controlling the airway as necessary, and evacuating the accumulated blood. Tube thoracostomy allows constant monitoring of the blood loss, and serial chest radiographs help monitor lung reexpansion. A large-bore tube (36-F to 40-F) should be inserted in the fifth interspace at the anterior axillary line and connected to underwater seal drainage and suction (20 to 30 mL H_2O).

Although small hemothoraces may be observed in stable patients, a moderate hemothorax or any hemothorax in an unstable or symptomatic patient requires tube thoracostomy. Severe or persistent hemorrhage requires thoracostomy or open thoracotomy. Studies are required to better delineate the size of a hemothorax detected on CT scan that requires tube thoracostomy drainage.

Autotransfusion has been successfully used in tube thoracostomy. Autotransfusion also eliminates the risk of incompatibility reactions and transmission of certain diseases, such as hepatitis C. Because the majority of blood loss occurs immediately after tube thoracostomy placement, autotransfusion apparatus should be immediately available in the ED.

Close monitoring of the initial and ongoing rate of blood loss should be performed. Immediate drainage of more than 1500 mL of blood from the pleural cavity is considered an indication for urgent thoracotomy, as is a continued output of at least 200 mL/hr for 3 hours.[17,30] General considerations for urgent thoracotomy are outlined in Box 38.3.

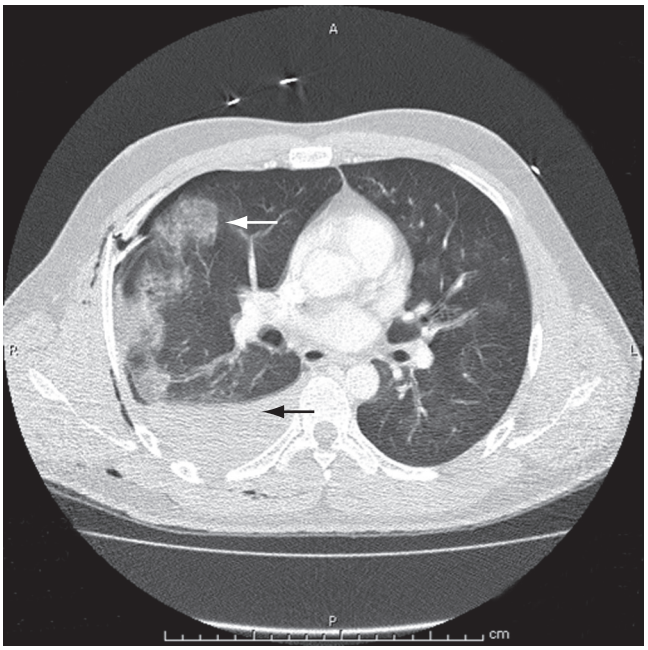

Fig. 38.14. Rib fracture resulting in pneumothorax, with associated hemothorax *(bottom arrow)* and pulmonary contusion *(top arrow)* seen on chest computed tomography (CT) scan.

TRACHEOBRONCHIAL INJURY

PRINCIPLES

Background and Importance

Tracheobronchial injuries may occur with either blunt or penetrating injuries of the neck or chest. MVCs are the most frequent mechanism causing tracheobronchial injury, accounting for more than half of all cases.

Although there has been an increase in the occurrence of tracheobronchial disruption, it is still a relatively rare injury, occurring in fewer than 3% of patients with significant chest injury. Its

BOX 38.3

Indications for Thoracotomy

Initial thoracostomy tube drainage is more than 20 mL of blood per kilogram.
Persistent bleeding at a rate greater than 7 mL/kg/hr is present.
Increasing hemothorax seen on chest x-ray films.
Patient remains hypotensive despite adequate blood replacement, and other sites of blood loss have been ruled out.
Patient decompensates after initial response to resuscitation.

associated mortality rate is reported to be approximately 10%, although mortality rates are significantly affected by associated injuries and the timing of diagnosis and surgical repair.

Pathophysiology

Tracheobronchial injuries caused by knife wounds develop almost exclusively from wounds in the cervical trachea (see Chapter 37), whereas gunshot wounds may damage the tracheobronchial tree at any point. Intrathoracic injury to the tracheobronchial tree occurs most commonly from blunt trauma. These injuries may result from direct blows, shearing stresses, or burst injury. A direct blow to the neck may crush the cervical trachea against the vertebral bodies and transect the tracheal rings or cricoid cartilage. Shear forces on the trachea will produce injury at the carina and the cricoid cartilage, which are its relatively fixed points.

Sudden deceleration of the thoracic cage pulls the lungs away from the mediastinum, producing traction on the trachea at the carina. When the elasticity of the tracheobronchial tree is exceeded, it ruptures. It has also been suggested that if the glottis is closed at the time of impact, the sudden increase in intrabronchial pressure will rupture the tracheobronchial tree. Regardless of the mechanism, more than 80% of these injuries occur within 2 cm of the carina.

CLINICAL FEATURES

Massive air leak through a chest tube, hemoptysis, and dramatic or increasing subcutaneous emphysema should suggest the diagnosis of major airway damage. Subcutaneous emphysema is typically the most common physical finding. Auscultation of the heart may reveal Hamman's crunch if air tracks into the mediastinum. Hamman's crunch is a crunching, rasping sound that is synchronous with the pulse and is best heard over the precordium. It is the result of the heart beating against air-filled tissues. Patients with tracheobronchial disruption have one of two distinct clinical pictures. In the first group of patients, the wound opens into the pleural space, producing a large pneumothorax. A chest tube fails to evacuate the space and reexpand the lung, and there is continuous bubbling of air (persistent leak) in the underwater seal device (Fig. 38.15).

In the second group of patients, there is complete transection of the tracheobronchial tree but little or no communication with the pleural space. A pneumothorax is usually not present. The peribronchial tissues support the airway enough to maintain respiration, but within 3 weeks, granulation tissue will obstruct the lumen and produce atelectasis. These patients are relatively free of symptoms at the time of injury; but weeks later, they have unexplained atelectasis or pneumonia. Radiographic signs in either group of patients are pneumomediastinum, extensive subcutaneous emphysema (Fig. 38.16), pneumothorax, fracture of the upper ribs (first through fifth), air surrounding the bronchus, and obstruction in the course of an air-filled bronchus.

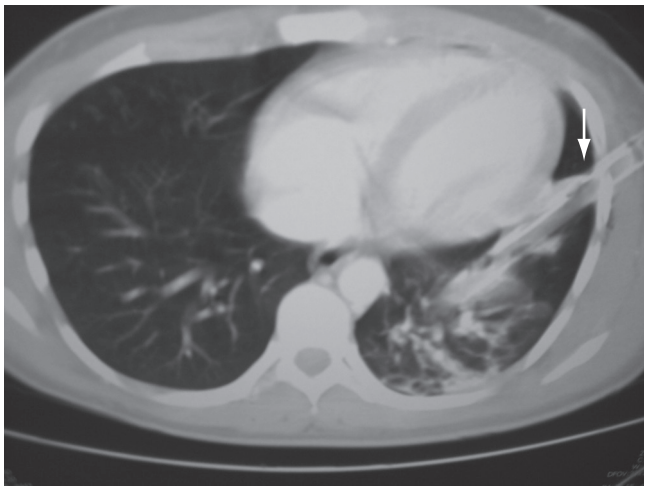

Fig. 38.15. Penetration of lung parenchyma by tube thoracostomy *(arrow),* with large residual pneumothorax, visible on computed tomography (CT) scan.

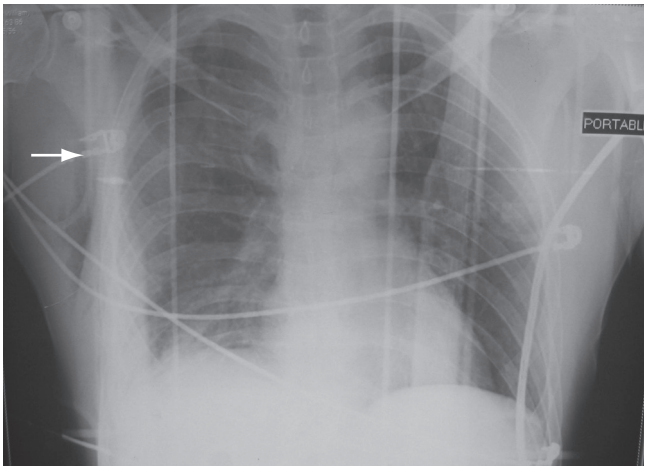

Fig. 38.16. Multiple rib fractures with extensive subcutaneous emphysema *(arrow),* with no pneumothorax seen.

DIAGNOSTIC TESTING

When tracheobronchial injury is suspected, bronchoscopy should be performed. Flexible endoscopic bronchoscopy is the most preferred and reliable means of establishing the diagnosis and determining the site and extent of the injury. However, CT scan has been shown to have high sensitivity in detecting tracheobronchial injury. Bronchopleural fistula (a communication between a bronchus and the lung parenchyma) can occur as a complication of tracheobronchial disruption and in some cases has been treated successfully via the fiberoptic bronchoscope. A mediastinal fluid collection, evidence of mediastinitis, or both may be noted on chest CT.

MANAGEMENT

If diagnostic bronchoscopy is performed, endotracheal intubation can be performed over the bronchoscope to ensure that the tube passes safely beyond the site of injury. Blind intubation should not be attempted. If intubating using a video laryngoscope (or

conventional laryngoscope), the tube is advanced slowly and gently to avoid creating a false passage or converting a partial tracheal tear to a complete tear.

The standard treatment for tracheobronchial injury has been thoracotomy with intraoperative tracheostomy and surgical repair of the disrupted airway. However, conservative medical treatment of such injuries can be considered for patients with tracheal tears less than 2 cm and without esophageal prolapse, mediastinitis, or massive air leakage.[30]

DIAPHRAGMATIC INJURY

PRINCIPLES

Background and Importance

Diaphragmatic rupture is present in 1% to 6% of major thoracic injuries. Diaphragmatic rupture occurs most commonly after blunt thoracoabdominal trauma, such as occurs in MVCs or falls from heights, but can also occur from penetrating trauma.

Pathophysiology

Diaphragmatic hernia is a herniation of abdominal structures into the thoracic cavity through a defect on the diaphragm, with a potential risk of strangulation of abdominal viscera, especially the small bowel. Signs and symptoms may not occur during the initial admission and may be delayed for as long as months to years, with a significant mortality rate.

Three-quarters of cases of diaphragmatic rupture secondary to blunt trauma occur on the left side, the remainder on the right, presumably due to the protective effect of the liver on the right side. Only about 5% of cases are bilateral. With penetrating injury, diaphragmatic injury occurs at the site of the penetration but may be occult.

In cases of blunt injury, the raised pressure within the abdominal cavity causes the diaphragmatic tear, and a pressure difference of generally 5 to 10 mm Hg forces the abdominal organs through the diaphragmatic defect. Because blunt trauma can cause multiple organ injuries, these coexisting injuries can mask the more silent diaphragmatic injuries, and diaphragmatic rupture may be initially overlooked. Over time, negative intrathoracic pressure generated by inspiration tends to draw abdominal contents into the thorax. This effect is lost with the use of intubation and positive-pressure ventilation.

DIAGNOSTIC TESTING

Accurate diagnosis of traumatic diaphragmatic hernia is essential because prompt surgical repair is the treatment of choice, but plain chest radiography alone is poorly sensitive for diaphragm rupture.

In patients with blunt trauma, CT examination of the abdomen and chest can be very useful for the evaluation of diaphragmatic injury, although injury site and type affect its sensitivity. Nevertheless, CT can demonstrate findings consistent with diaphragmatic injury, including diaphragmatic discontinuity, intrathoracic herniation of abdominal contents, and waist-like constriction of abdominal viscera (the "collar sign").

In patients with penetrating left thoracoabdominal trauma, the incidence of herniation of abdominal contents is sufficiently high that thoracoscopy or laparoscopy is recommended for the diagnosis and repair of a diaphragmatic injury. Even in patients with right-sided penetrating lesions (which do not typically result in herniation because of the protective effect of the dome of the liver), evaluation of both sides of the diaphragm with laparoscopy or thoracoscopy is recommended.

MANAGEMENT

Diaphragmatic injuries may be markers of severity and predictors of serious associated injuries in trauma and should be surgically repaired. The treatment of choice is surgery. CT scan should identify the site and extent of herniation, herniated organs, complications, and damage to associated organs. Although they are not without complications, laparoscopy and thoracoscopy may be used for diaphragmatic hernia repair.

The incidence of diaphragmatic involvement after penetrating left-sided thoracoabdominal injury is high, making nonoperative, expectant management of these patients potentially unsafe.

CARDIOVASCULAR TRAUMA

BLUNT CARDIAC TRAUMA

Blunt cardiac injury usually results from high-speed MVCs in which the chest wall strikes the steering wheel. Other causes, such as falls from heights, crushing injuries, blast injuries, and direct blows, are less common. The diagnosis of a blunt injury to the heart remains elusive because of the usual concomitant serious injuries to other body organs and, more important, because there is no gold standard for making the diagnosis.

The importance of detecting blunt myocardial injury lies in the recognition of associated potentially fatal complications. Life-threatening dysrhythmias, conduction abnormalities, congestive heart failure, cardiogenic shock, hemopericardium with tamponade, cardiac rupture, valvular rupture, intraventricular thrombi, thromboembolic phenomena, coronary artery occlusion, ventricular aneurysms, and constrictive pericarditis have all been reported as complications.

Blunt cardiac trauma may be viewed as part of a continuous spectrum (ie, myocardial concussion, contusion, infarction, and rupture). Myocardial concussion occurs when a blunt injury to the interior chest produces a "stun" response in the myocardium. No permanent cellular injury occurs, but transient clinical effects may result. Myocardial contusion is the least severe form of injury that can be demonstrated pathologically. Cellular injury occurs with extravasation of red blood cells into the muscle wall, along with localized myocardial cellular necrosis. Permanent myocardial damage is rare. Infarction typically occurs with traumatic occlusion or disruption of a coronary artery. Cardiac rupture is obviously the most severe form of blunt cardiac injury.

MYOCARDIAL CONCUSSION

Principles

Background and Importance

The terms *myocardial concussion* or *commotio cordis* are used to describe an acute form of blunt cardiac trauma that is usually produced by a sharp, direct blow to the midanterior chest that stuns the myocardium and results in brief dysrhythmia, hypotension, and loss of consciousness. It is a rare event and primarily occurs in adolescents, especially those playing sports involving hard spherical objects (eg, baseballs, hockey pucks, and so on).

Pathophysiology

Animal models of commotio cordis have determined that it is much more likely to occur if the impact occurs during early ventricular repolarization.[31] Additionally, flat objects and softer balls (eg, tennis or soccer) are less likely to cause commotio cordis than smaller balls.[32] Once this dysrhythmia occurs, it can result in a non-perfusing rhythm, such as asystole or ventricular

fibrillation, and irreversible cardiac arrest. There are, however, a number of documented cases of successful resuscitation with both rapid provision of CPR and the use of an automated external defibrillator (AED).[33]

Clinical Features

Commotio cordis has a characteristic mechanism (blunt chest trauma) and presentation (sudden collapse). Notably, the disease itself is defined by a lack of structural cardiac damage, and so more severe trauma (such as that necessary to cause cardiac contusion or rupture, as described later) is incongruent with the diagnosis.

Diagnostic Testing

Laboratory Tests and Electrocardiogram

Patients who present with the characteristic mechanism and presentation above and who have shockable rhythms on electrocardiogram (ECG) (or who were defibrillated by an AED) can be presumed to have commotio cordis if there is no evidence of structural heart damage on echocardiography or CT. Laboratory evaluation of serum electrolyte and cardiac biomarker levels may identify additional contributors to their presentation, but these will typically be normal.

Management

The initial treatment of patients with commotio cordis should follow standard advanced cardiac life support (ACLS) algorithms, ideally with initiation of bystander CPR and early defibrillation (especially at sporting events). Barring any other more severe cardiac injuries (discussed later), commotio cordis does not require any specific interventions.

Disposition

In patients who survive the dysrhythmia of commotio cordis and are not found to have more severe traumatic cardiac injury, a short period of observation is appropriate. Although there is a paucity of evidence as to the duration of this observation period, we recommend 6 to 12 hours of telemetry monitoring. After this, patients may be discharged, although they should not return to play until additional outpatient cardiac testing (eg, stress testing, cardiac magnetic resonance imaging [MRI], and pharmacologic testing for primary conductive disorders, if indicated) is performed.[33]

MYOCARDIAL CONTUSION

Principles

Background and Importance

Myocardial contusion is a very poorly understood and nebulous condition. Decades of research and widely varied clinical practice have failed to produce a consensus regarding its diagnosis, complication rate, and proper disposition.

Pathophysiology

Several mechanisms have been postulated by which the heart may be injured in cases of blunt trauma. A direct blow to the chest transmits energy through the ribs to the spine. When a large force is applied to the chest wall, the sternum is displaced posteriorly and the heart is compressed between the sternum and vertebrae or an elevated diaphragm. Either can presumably result in cardiac injury. Increased intrathoracic pressure from a direct blow to the chest may contribute to the injury. In addition, compression of the abdomen and pelvis may displace abdominal viscera upward and result in cardiac injury.

Clinical Features

Myocardial contusion manifests clinically as a spectrum of injuries of varying severity. Although the majority of patients with myocardial contusion have external signs of thoracic trauma (eg, contusions, abrasions, palpable crepitus, rib fractures, or visible flail segments), the absence of identifiable thoracic injury decreases the likelihood of myocardial contusion but does not exclude it. Virtually every known intrathoracic and chest wall injury has been associated with myocardial contusion. The most sensitive but least specific sign of myocardial contusion is sinus tachycardia, which is present in approximately 70% of patients with documented myocardial contusions and is a very common vital sign in trauma patients. A reduction in cardiac output, which can be clinically insignificant or manifest as pronounced cardiogenic shock, may occur in patients with significant cardiac contusion.

Diagnostic Testing

Unfortunately, there is not an agreed upon gold standard diagnostic definition for myocardial contusion. Clinical evidence is often nonspecific, especially in the setting of multiple traumas. Many tests and definitions have been proposed over the years, but none has emerged as definitive.

Laboratory Tests and Electrocardiogram

Electrocardiogram. Because of its anterior position in the thorax and proximity to the sternum, the right ventricle is far more likely to be injured than the left ventricle. The standard 12-lead ECG is relatively insensitive to right ventricular damage, as demonstrated by pathologic evidence of cellular damage in patients with normal ECGs. A cardiac contusion usually results in moderate right ventricular damage with only minor electrical changes, which can easily be missed on ECG. Right-sided ECGs (the addition of V_4R) have not been found to be of any benefit.

The ECG for patients with myocardial contusion often shows evidence of dysrhythmia, conduction disturbance, or ischemia. Dysrhythmias or ECG changes also can be caused by significant hypoxia as a result of pulmonary injuries or blood loss, which resolve once the hypoxemia or blood loss has been corrected.

A few cases of delayed life-threatening dysrhythmia have been reported up to 12 hours after injury, and patients may develop less lethal dysrhythmias up to 72 hours after injury. The onset of ECG changes may be delayed up to 48 hours after injury, but all ECG changes usually resolve in 4 to 60 days. The presence of ECG abnormalities is neither specific enough to confirm the diagnosis of myocardial contusion nor reliable enough to predict subsequent complications, but a newly abnormal ECG (arrhythmia/heart block or ischemic changes) warrants admission for continuous ECG monitoring.[34]

Cardiac Biomarkers. Because creatine kinase (CK) is nonspecifically increased in trauma patients owing to associated skeletal muscle injury, and CK-MB levels have also been found to be falsely elevated in multi-trauma patients, the troponin assay is the preferred cardiac biomarker for testing.

The combination of a normal troponin level and a normal 12-lead ECG has a negative predictive value sufficient to "rule out" myocardial contusion, and these patients do not need any other evaluation or monitoring specific for myocardial contusion.[34]

Imaging

Although echocardiography provides a means to directly visualize cardiac structures and chambers and can be very useful to rule out structurally significant myocardial injuries (eg, wall motion or valvular abnormalities), it should not be routinely used as a primary screening modality for blunt cardiac injury. Rather, echocardiography should be reserved for patients in whom myocardial contusion is suspected (based on ECG or troponin level) and who have unexplained hypotension or arrhythmias.[34]

Management

Treatment of a suspected myocardial contusion is similar to that of a myocardial infarction (MI): saline lock (if IV fluids are not otherwise indicated), cardiac monitoring, administration of oxygen if hypoxic, and analgesic agents. Dysrhythmias are typically transient and do not require treatment. Serious dysrhythmias, such as ventricular tachycardia or atrial flutter should be treated with appropriate medications as per current ACLS guidelines. No data exist to support prophylactic dysrhythmia suppression. Measures should be taken to treat and prevent any conditions that increase myocardial irritability (eg, metabolic acidosis). Thrombolytic agents and aspirin are contraindicated in the setting of acute trauma. In rare instances, there may be an acute MI associated with trauma, which can arise from lacerations or blunt injury to the coronary arteries. These cases should be managed by percutaneous coronary intervention (PCI), with cardiothoracic surgery for definitive repair as indicated.

In the setting of depressed cardiac output caused by suspected or confirmed myocardial contusion, judicious fluid administration to augment preload is warranted (eg, 200 to 250 mL boluses every 15 minutes to a maximum of 1 to 2 L). A dobutamine infusion may be useful once preload has been optimized. While intra-aortic balloon counterpulsation has been used successfully in refractory cardiogenic shock, the priority is to ascertain that the decreased cardiac output is not the result of other undiagnosed traumatic injuries, particularly aortic rupture.

The prognosis of a patient with myocardial contusion depends on the character and magnitude of the initial trauma, the size and location of the contusion, the preexisting condition of the coronary arteries, and, most importantly, with the number of associated serious injuries. Recovery without complications is the usual course.

Disposition

Patients with suspected myocardial contusion who have a normal troponin level and a normal ECG do not have the diagnosis. Myocardial contusions resulting in ECG changes or troponin elevations necessitate telemetry observation or in-hospital monitoring, depending on the patients' other injuries. Markedly abnormal ECGs, troponin elevations, or hypotension warrant echocardiography and cardiology consultation.

MYOCARDIAL RUPTURE

Principles

Background and Importance

High-speed MVCs are responsible for most cases of traumatic myocardial rupture, which is almost always fatal. Approximately one-third of these patients have multiple chamber rupture, and one-fourth have an associated ascending aortic rupture. Approximately 20% of patients survive at least 30 minutes, theoretically long enough to get them to the cardiac surgery operating room if the injury is recognized immediately and the center is capable.

Anatomy and Physiology

Myocardial rupture refers to an acute traumatic perforation of the ventricles or atria, but it may also include a pericardial rupture or laceration or rupture of the interventricular septum, interatrial septum, chordae, papillary muscles, valves, and lacerated coronary arteries. A delayed rupture of the heart may also occur weeks after nonpenetrating trauma, probably as a result of necrosis of a contused or infarcted area of myocardium.

The chambers most commonly involved in cardiac rupture are the ventricles, with right ventricular rupture being most common. Ruptures of the atria are less common, with right atrial rupture being more common than left. Multiple chamber involvement occurs in 20% of patients. Twenty percent of nonsurvivors have concomitant aortic rupture.

Pathophysiology

A rupture occurs during closure of the outflow tract when there is ventricular compression of blood-filled chambers by a pressure sufficient to tear the chamber wall, septum, or valve. This is the most likely mechanism for ventricular rupture when injury occurs in diastole or early systole concomitant with maximal ventricular distention. The atria are most susceptible to rupture by sudden compression in late systole when these chambers are maximally distended with venous blood and the atrial ventricular valves are closed. Other proposed mechanisms of rupture include: (1) deceleration shearing stresses acting on the "fixed" attachment of the inferior and superior vena cava at the right atrium; (2) upward displacement of blood and abdominal viscera from blunt abdominal injury that causes a sudden increase in intracardiac pressure; (3) direct compression of the heart between the sternum and vertebral bodies; (4) laceration from a fractured rib or sternum; and (5) complications of a myocardial contusion, necrosis, and subsequent cardiac rupture.

Because of the mechanisms involved in cardiac rupture, associated multisystem injuries are common. More than 70% of reported survivors of myocardial rupture have other major associated injuries, including pulmonary contusions, liver and spleen lacerations, closed head injuries, and major fractures.

The immediate ability of the patient to survive cardiac rupture depends on the integrity of the pericardium. Two-thirds of patients with cardiac rupture have an intact pericardium and are protected from immediate exsanguination. These patients may survive for a brief period but will then develop significant hemopericardium and pericardial tamponade. One-third of patients with cardiac rupture have associated pericardial tears and succumb promptly to exsanguination.

Clinical Features

The clinical presentation of a patient who has sustained a myocardial rupture is usually that of cardiac tamponade or severe hemorrhage. Rarely, a patient is seen with a large hemothorax, hypotension, and hypovolemia, obscuring the diagnosis by mimicking a serious pulmonary or other intrathoracic injury. A patient with an intact pericardial sac and developing tamponade displays physical findings of tamponade, usually with subsequent clinical deterioration. Initial inspection of the torso may reveal little more than a bruised area over the sternum or no external physical evidence. More often, however, signs of significant chest trauma or other associated injuries will be present, indicating a mechanism of injury that could result in myocardial rupture. Auscultation may reveal a harsh murmur, known as a *bruit de moulin,*

which has been classically described as sounding like a splashing mill wheel. This is caused by pneumopericardium.

Diagnostic Testing

Early use of ED ultrasound may facilitate the early diagnosis of cardiac rupture and pericardial tamponade. The combination of shock and JVD in a patient with blunt chest trauma should immediately suggest pericardial tamponade or tension pneumothorax, both conditions rapidly assessable by bedside ultrasound. In patients with coexistent hemorrhage from other injuries, JVD may be absent. Other considerations include myocardial contusion, superior vena cava obstruction, and ruptured tricuspid valve. Sonographic visualization of pericardial fluid in this setting should be followed by emergent thoracotomy (Fig. 38.17).

A chest radiograph may be helpful in patients suspected of having sustained trauma severe enough to cause myocardial rupture. Although this study usually does not help diagnose cases of myocardial rupture, it notes the presence of other intrathoracic injuries (eg, hemothorax, pneumothorax, and signs of possible aortic dissection). An increase in the size of the cardiac silhouette more commonly reflects preexisting disease or valvular incompetence with chamber enlargement caused by increased filling pressures. ECG changes may occur with myocardial injury, but these are often nonspecific. Bedside echocardiography in the ED should be performed in any case of suspected cardiac rupture, pericardial tamponade, a previously undiagnosed murmur, or shock unexplained by other causes (eg, exsanguination).

Management

When nonhospital medical personnel evaluate a patient who has sustained blunt chest trauma, they should concentrate on rapid transport and observe for any signs of pericardial tamponade. If examination is consistent with tension pneumothorax, this should be treated with needle decompression.

In the ED, treatment of patients with a myocardial rupture is directed toward immediate decompression of cardiac tamponade and control of hemorrhage. Pericardiocentesis may be effective in cases of a small rupture, but it is usually performed as a diagnostic or temporizing therapeutic procedure until surgical correction can be undertaken. Emergency thoracotomy and pericardiotomy may be required in the ED if the patient has rapidly deteriorating vital signs or a cardiac arrest. After emergency thoracotomy and pericardiotomy, the myocardial rupture should be controlled until the patient can be transported to the operating room for definitive repair. Hemorrhage from a ruptured atrium can often be controlled by finger occlusion or application of a vascular clamp. Insertion of a Foley catheter through the defect, followed by inflation of the balloon and traction on the catheter, may also control the bleeding. Ventricular rupture can usually be controlled by direct digital pressure or by suturing with nonabsorbable vascular sutures.

Cardiopulmonary bypass is required in only 10% of successful repairs of myocardial rupture. Therefore, for patients with suspected myocardial rupture, it is appropriate to undertake emergency thoracotomy in institutions that have qualified surgeons but no immediate access to cardiopulmonary bypass.

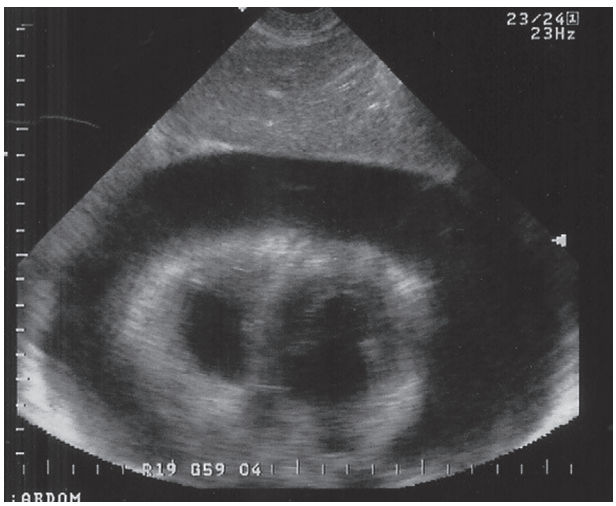

Fig. 38.17. Echocardiogram demonstrating pericardial fluid with cardiac tamponade.

PENETRATING CARDIAC TRAUMA

Penetrating cardiac injuries are one of the leading causes of death in the setting of urban violence, with patients who survive to hospital arrival having a mortality rate of almost 80%. Improvements in emergency medical services, along with an emphasis on rapid transport, are responsible for an increasing number of cardiac injury patients arriving in impending or full cardiopulmonary arrest at busy urban trauma centers.[35] The proportion of gunshot wounds versus stab wounds varies widely in reported case series, depending on the location of the trauma center.

The right ventricle is affected more often (43%) than the left ventricle (34%) owing to its anterior anatomic location. The left or right atrium is affected in 20% of cases. One-third of penetrating cardiac wounds affect multiple chambers, and survival is much worse in these cases.[36] In 5% of cases, a coronary artery is lacerated, although these injuries usually involve a distal segment of the artery and rarely produce significant acute MI when they are ligated. More proximal coronary artery lacerations require coronary bypass. Rarely, the interventricular septum, a valve, papillary muscle, or chordae tendineae are lacerated, producing an acute shunt or valvular insufficiency. These lesions are poorly tolerated and can quickly produce massive pulmonary edema and cardiogenic shock.[37]

Two conditions may occur after penetrating heart injury: (1) exsanguinating hemorrhage if the cardiac lesion communicates freely with the pleural cavity, or (2) cardiac tamponade if the hemorrhage is contained within the pericardium. Patients with exsanguinating wounds frequently die before they reach medical attention, or they have rapidly progressive hemorrhagic shock on presentation, culminating in cardiac arrest. This presentation is most typically seen in patients sustaining gunshot wounds to the heart. Cardiac tamponade is a life-threatening condition but appears to offer some degree of protection and increased survival in patients with penetrating cardiac wounds. These patients often require immediate resuscitation by emergency department thoracotomy (EDT) if they meet the criteria listed in Box 38.4.

BOX 38.4

Indications for Emergency Department Thoracotomy

PENETRATING TRAUMATIC CARDIAC ARREST

Cardiac arrest at any point with initial signs of life in the field
Systolic blood pressure below 50 mm Hg after fluid resuscitation
Severe shock with clinical signs of cardiac tamponade

BLUNT TRAUMA

Cardiac arrest in the emergency department (ED)

ACUTE PERICARDIAL TAMPONADE

Principles

Background and Importance

The reported incidence of acute pericardial tamponade is approximately 2% in patients with penetrating trauma to the chest and upper abdomen; it is rarely seen after blunt chest trauma. It occurs more commonly with stab wounds than with gunshot wounds, and 60% to 80% of patients with stab wounds involving the heart develop tamponade. Patients with acute pericardial tamponade can deteriorate in minutes, but many can be saved if proper steps are taken.

Pathophysiology

The primary feature of a pericardial tamponade is an increase in intrapericardial pressure and volume. As the volume of the pericardial fluid encroaches on the capacity of the atria and ventricles to fill adequately, ventricular filling is mechanically limited, and thus the stroke volume is reduced. This results in decreased cardiac output and ultimately diminished arterial systolic blood pressure and decreased pulse pressure. As little as 60 to 100 mL of pericardial blood may produce the clinical picture of tamponade. Concomitantly, CVP rises because of the mechanical backup of blood into the vena cava.

Several compensatory mechanisms then occur. The heart rate and total peripheral resistance rise in an attempt to maintain adequate cardiac output and blood pressure. A less effective compensatory response, resulting in a greater rise in CVP, is an increase in venomotor tone caused by contractions of the smooth muscles within the wall of the vena cava.

The diagnosis of pericardial tamponade should be suspected in any patient who has sustained a penetrating wound or blunt trauma to the thorax or upper abdomen. One is never certain of the trajectory of the bullet or the length, force, and direction of a knife thrust on initial evaluation. Obviously, wounds directly over the precordium and epigastrium are more likely to produce a cardiac injury resulting in tamponade than those in the posterior or lateral thorax. Nevertheless, it is assumed that a penetrating wound, particularly a gunshot wound, anywhere in the thorax or upper abdomen may have injured the heart. Rapid bedside echocardiography, performed as part of the standard FAST examination, easily detects a pericardial effusion causing cardiac tamponade.

Clinical Features

Patients with cardiac tamponade may initially appear deceptively stable if the rate of bleeding into the pericardial space is slow or if the pericardial wound allows intermittent decompression. Other patients may complain primarily of difficulty breathing, which suggests pulmonary rather than cardiac pathology.

The physical findings of pericardial tamponade—hypotension, distended neck veins, and, rarely, distant or muffled heart tones (known as *Beck's triad*)—may be difficult to identify clinically, especially in the midst of a major resuscitation with concomitant hypovolemia, when the neck veins may be flat. Although the most reliable signs of pericardial tamponade are an elevated CVP (>15 cm H_2O) in association with hypotension and tachycardia, bedside echocardiography performed as part of the FAST examination rapidly diagnoses pericardial tamponade (by identifying pericardial fluid with concomitant tamponade physiology) and has largely replaced the use of CVP measurements to make the diagnosis. Echocardiography also distinguishes pericardial tamponade versus tension pneumothorax when the triad of elevated CVP, hypotension, and tachycardia is present.

Acute pericardial tamponade may be seen with three distinct clinical pictures. If the hemorrhage is confined to the pericardial space, the patient is initially normotensive but will have a tachycardia and elevated CVP. If untreated, most of these patients go on to develop hypotension. If significant hemorrhage has occurred outside the pericardial sac, either through a tear in the pericardium or from associated trauma, the clinical picture is that of hypovolemic shock with hypotension, tachycardia, and a low CVP. If the CVP rises to a level of 15 to 20 cm H_2O with volume replacement but hypotension and tachycardia persist, pericardial tamponade should be considered. The third clinical picture is that of an intermittently decompressing tamponade due to intermittent hemorrhage from the intrapericardial space, partially relieving the tamponade. The clinical picture may wax and wane depending on the intrapericardial pressure and volume and total blood loss. In general, this condition is compatible with a longer survival than are the first two clinical presentations.

Pulsus paradoxus is defined as an excessive drop in systolic blood pressure during the inspiratory phase of the normal respiratory cycle. This sign may be an additional clue to the presence of pericardial tamponade, but it is often difficult to measure during an intensive resuscitation or in the presence of shock.

Diagnostic Testing

Radiology

Ultrasound. Ultrasound enables rapid, accurate, and noninvasive diagnosis of pericardial tamponade. This study can be performed at the bedside in the ED during the initial resuscitation of the patient as part of the FAST examination.[38] Although the sonographic definition of tamponade is the simultaneous presence of pericardial fluid and diastolic collapse of the right ventricle or atrium, the presence of pericardial fluid in a patient with chest trauma is highly suggestive of pericardial hemorrhage (see Fig. 38.17). An indirect sonographic sign of tamponade is the demonstration of a dilated inferior vena cava in a hypotensive patient. EDs in which cardiac ultrasonography is performed with subcostal and long parasternal views have reported a sensitivity and specificity of nearly 100% for the detection of pericardial effusion. Because ultrasound is noninvasive and extremely accurate, its immediate availability in the initial phase of major trauma resuscitation can be very helpful in detecting pericardial fluid before the patient deteriorates hemodynamically.

Radiography. The radiographic evaluation of the cardiac silhouette in acute pericardial tamponade generally is not helpful, unless a traumatic pneumopericardium is present. Because small volumes of hemopericardium lead to tamponade in the acute setting, the heart will typically appear normal. This is in contrast to the "water-bottle" appearance of the heart with chronic pericardial effusion. Usually the latter condition is tolerated for a long period.

Electrocardiography

Many ECG changes of pericardial tamponade have been described in the literature, but few are diagnostic, and each is more likely to be seen with chronic rather than acute tamponade. For example, electrical alternans (in which the morphology and amplitude of the P, QRS, and ST-T waves in any single lead alternate in every other beat [Fig. 38.18]) has been reported to be a highly specific marker of pericardial tamponade. The postulated cause is the mechanical oscillation of the heart in the pericardial fluid, which is called the *swinging heart phenomenon.* Echocardiographic

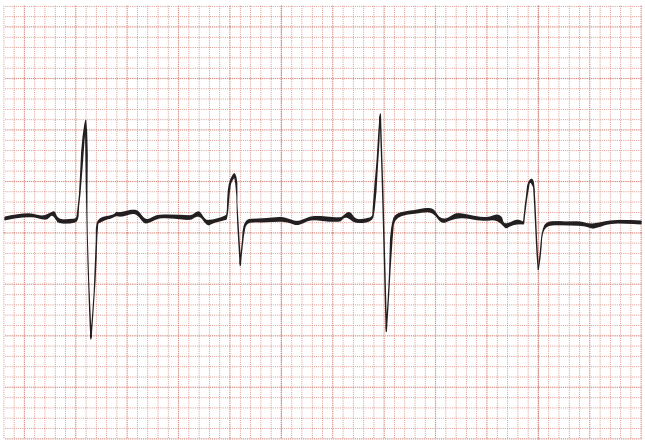

Fig. 38.18. Lewis (S5) lead electrocardiogram (ECG) revealing total electrical alternans of the QRS complexes. (From Sotolongo RP, Horton JD: Total electrical alternans in pericardial tamponade. Am Heart J 101:853, 1981.)

studies have revealed that when fluid accumulates to a critical extent, the frequency of cardiac oscillation may abruptly decrease to half the heart rate. The cardiac position will alternate, with the heart returning to its original position with every other beat, and thus electrical alternans may be seen. Electrical alternans, when present, is pathognomonic for tamponade. However, it is much more common in chronic pericardial effusions that evolve into a tamponade, and it is rarely seen in acute pericardial tamponade. Notably, however, low amplitude of the QRS complexes may be seen as a result of the presence of pericardial effusion.

Management

Field treatment for cases of pericardial tamponade is essentially the same as that outlined for any victim of major trauma. The diagnosis of tamponade should be suspected by the location of penetrating wounds or by the patient's poor response to vigorous volume resuscitation. Tension pneumothorax, which is much more common, mimics certain aspects of acute pericardial tamponade. If the patient is in extremis or the clinical condition rapidly deteriorates, consideration should be given to performing a needle thoracostomy, which, if not therapeutic, suggests pericardial tamponade in the appropriate clinical presentation by virtue of "diagnosis of exclusion." Expedient transport to the nearest trauma center should be paramount.

Upon ED arrival, volume expansion with crystalloid solution via two or three large-bore (14- or 16-gauge) catheters should be established immediately. The presence of a pneumothorax or hemothorax, which is often associated with penetrating cardiac trauma, is treated expeditiously with tube thoracostomy. Bedside echocardiography should be performed as quickly as possible to establish the diagnosis of pericardial tamponade, which then should be followed by emergent surgical repair.

There is increasing controversy regarding the role of pericardiocentesis. In the past, it was recommended that pericardiocentesis be performed for both diagnostic and therapeutic reasons. Aspiration of as little as 5 to 10 mL of blood may result in dramatic clinical improvement. However, it should be emphasized that pericardiocentesis is not a benign or invariably successful procedure. Blood in the pericardial space tends to be clotted, and aspiration may not be possible. Possible complications include the production of pericardial tamponade, the laceration of coronary artery or lung, and induction of cardiac dysrhythmias. Whenever possible, pericardiocentesis should be performed under sonographic guidance, because this approach will increase the success rate and decrease the incidence of complications. A pigtail catheter may be introduced into the pericardial space for repeated aspirations while preparations are underway to quickly transport the patient to the operating room for definitive therapy. If pericardiocentesis is unsuccessful or the clinical status deteriorates, and if acute pericardial tamponade remains important in the differential diagnosis, thoracotomy should be performed as quickly as possible.[39] Patients with penetrating cardiac injury invariably require surgical repair. The location (operating room vs. ED) and timing (immediate vs. urgent) depend on the patient's clinical status.

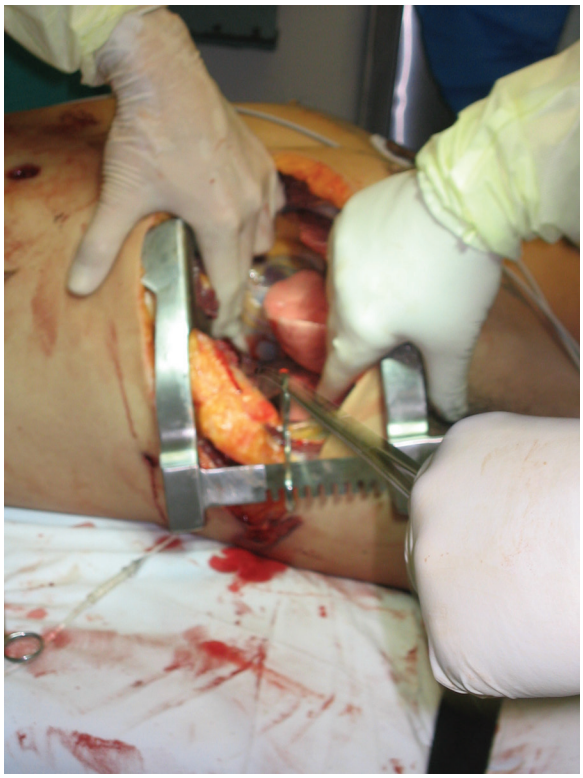

Fig. 38.19. Emergency department thoracotomy (EDT).

Emergency Department Thoracotomy

EDT is a drastic, dramatic, and potentially lifesaving procedure in which emergency clinicians should be proficient. Although the procedure is not described in detail here, a few technical points merit discussion. A left lateral incision is preferred because it is rapidly accomplished; allows the best exposure of the heart, aorta, and left hilum; and facilitates open cardiac massage and internal defibrillation (Fig. 38.19). With right-sided or multiple injuries, it may be necessary to extend the incision across the sternum and right chest wall, creating a "clamshell" incision. The internal mammary arteries need to be ligated if this maneuver restores effective perfusion. After the heart is sufficiently exposed, the pericardium is vertically incised anterior to the phrenic nerve. Release of a tamponade may rapidly restore cardiac output. The heart is then delivered through the pericardium, and penetrating wounds are identified.

There are several alternatives for repairing cardiac wounds. Small wounds can be compressed by digital pressure to control bleeding en route to the operating room. If the injury is quite large, balloon tamponade can be achieved by applying gentle traction on a Foley catheter inserted into the wound with the balloon inflated with saline. This can temporarily stop the hemorrhage to allow suture repair of the injury (cardiorrhaphy) or to gain time while the patient is transferred to the operating room

for a more definitive surgical procedure. Lacerations of the atria can be temporarily controlled with a vascular clamp.

Suture of cardiac wounds over pledgets is the time-honored and effective technique but is technically more difficult and more time-consuming. The use of a monofilament suture, such as 2-0 Prolene, is recommended. Some trauma surgeons recommend stapling cardiac wounds with standard skin staplers because this technique may be much quicker and equally effective in closing these wounds.

Care is taken to avoid ligating coronary arteries during the repair. Direct insertion of a large-bore catheter (eg, a 5-French catheter) into the left atrial appendage provides a route for rapid infusion of fluids. If the heart is empty or the patient fails to respond to rapid fluid administration, the aorta is cross-clamped to divert cardiac output to the brain and heart. Prolonged ischemia and severe acidosis often result in post-resuscitation myocardial depression with ineffective contraction and diminished cardiac output. Thus the cross-clamp should be temporarily released every 30 to 45 minutes to minimize ischemic complications.

Indications for Emergency Department Thoracotomy. Although it is often tempting to perform EDT on all traumatic arrest victims in the ED, there are many cases in which patients have virtually no chance of survival. In addition, EDT is costly; requires the undivided attention of all personnel in the ED, diverting care from other, more salvageable patients in critical condition; and poses a risk to ED personnel for injury from needle sticks and other blood-contaminated exposures.

Evidence-based guidelines from the Eastern Association for the Surgery of Trauma recommend that EDT be performed on patients who lose pulses but who initially presented to the ED with signs of life after *penetrating* thoracic trauma (see Box 38.4). They conditionally recommend EDT for both patients who present to the ED without signs of life after *penetrating* thoracic trauma and patients who present to the ED with signs of life after *blunt* injury, and recommend against EDT in patients who sustain *blunt* injury and present to the ED pulseless.[39] It is worth emphasizing that EDT is a temporizing measure and should only be performed if definitive treatment is a viable option in the setting to which the patient presents.

BLUNT AORTIC INJURY

Principles

Background and Importance

Blunt aortic injury is a life-threatening injury, usually resulting from sudden deceleration, usually from automobile crashes. Other mechanisms of injury include pedestrians struck by automobiles, motorcycle crashes, airplane crashes, and falls from heights. Despite the improvement in and increased use of restraint systems, the overall incidence of blunt aortic injury associated with fatal automobile crashes has remained unchanged over the past decade.

Blunt aortic injury includes a spectrum of lesions, ranging from a small intimal tear to frank rupture, which usually causes rapid lethal hemorrhage. The most common sites of injury are the aortic isthmus and the ascending aorta just proximal to the origin of the brachiocephalic vessels. Sixty percent to 90% of patients with blunt aortic injury die at the site of accident or within hours of hospital admission. However, an increasing number of patients arrive at a treatment facility because of improvements in out-of-hospital care, more appropriate resuscitation in the field, and rapid transportation to a trauma center. The early survival rate of such patients depends on the initial resuscitation and/or the timeliness and correct choice of diagnostic procedures. A rapid and accurate diagnosis is thus mandatory to optimize treatment and maximize odds of survival.

Pathophysiology

The descending thoracic aorta is relatively fixed and immobile because of its tethering by intercostal arteries and the ligamentum arteriosum. With sudden deceleration, the more mobile aortic arch swings forward, producing a shearing force or "whiplash effect" on the aorta at the isthmus. A bending stress at the isthmus, created by sudden lateral oblique chest compression, may also result in rupture by causing flexion of the aortic arch on the left mainstem bronchus and the pulmonary artery. Forces created by the whiplash effect or lateral oblique compression may not be sufficient to provoke aortic tears. It is now postulated that those injuries may be caused by inferior and posterior rotation of anterior thoracic osseous structures (manubrium, first rib, and medial clavicles), pinching and shearing the interposed aorta as they strike the vertebral column.

Rupture of the ascending aorta just distal to the aortic valve likely occurs through a different mechanism. At the time of rapid deceleration and chest compression, the heart is displaced into the left posterior chest, which causes a shearing stress just above the aortic valve. A sudden increase in intra-aortic pressure, "the water hammer effect," may cause an explosive rupture of the aorta at this location. Involvement of the coronary ostia with coronary artery occlusion may occur in association with tears to the ascending arch. The intraluminal pressure tolerance of the aorta may be exceeded in a high-speed MVC.

A total of 80% to 90% of aortic tears occur in the descending aorta at the isthmus, just distal to the left subclavian artery (Fig. 38.20). Less common sites of involvement are the ascending aorta, the distal descending aorta at the level of the diaphragm, the midthoracic descending aorta, and the origin of the left subclavian artery. Although ruptures of the ascending aorta are much less common than those of the descending aorta, they have a 70% to 80% incidence of associated lethal cardiac injuries. This is in contrast to ruptures at the isthmus, which have a 25% incidence of associated cardiac injuries. Lethal cardiac injuries commonly

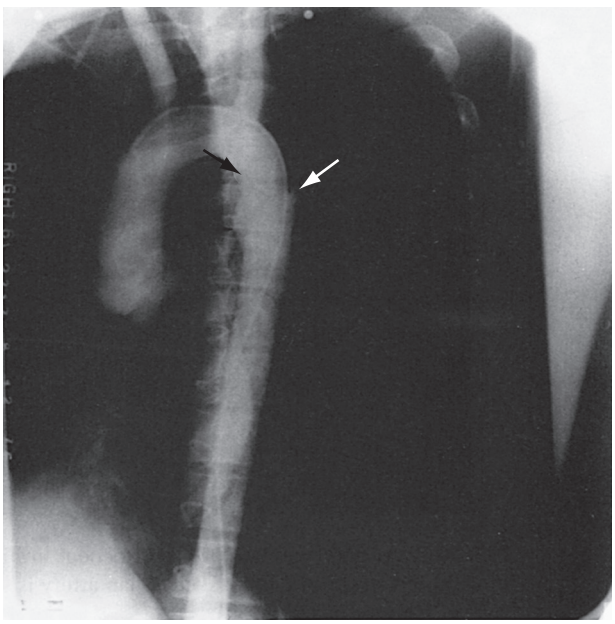

Fig. 38.20. Aortogram shows tear in aorta *(arrows)* at the most common location, at or just distal to takeoff point of left subclavian artery, which is not visualized.

include pericardial tamponade, aortic valve tears, myocardial contusion, or coronary artery injuries. Passenger ejection, pedestrian impact, severe falls, and crush injuries commonly result in ascending thoracic aortic ruptures. Survival long enough to be evaluated in the ED is rare among patients who sustain an ascending aortic rupture.

Aortic rupture may occur from causes other than high-speed MVC deceleration. Rupture has been documented as a complication of external cardiac massage and has been known to occur after fracture-dislocations of the thoracic spine, presumably as a result of direct shearing force. Vertical deceleration injuries resulting from falls can cause a rupture of the ascending aorta by producing an acute lengthening of the ascending aorta. This is the likely mechanism responsible for aortic rupture in the setting of airplane and elevator accidents. Direct kicks by animals, crush injuries, sudden burial by landslide, and air bag deployment have also been reported as causes of aortic rupture. Direct compression of the compliant thorax has been postulated to contribute to aortic rupture in children. Displaced fractures of the sternum, ribs, and clavicle have also been shown to directly lacerate the aorta.

Clinical Features

The possibility of aortic disruption should be considered in every patient who sustains a severe deceleration injury, because approximately 30% of surviving patients with blunt aortic injury will die within the first 24 hours without treatment. This is especially true if the automobile was moving in excess of 45 mph or if there is evidence of severe blunt force to the chest (eg, from a damaged steering wheel). In the case of any moderate- or high-speed MVC, it is imperative that paramedics carefully evaluate the extent of damage to the vehicle, the complaints of the victims, and the physical manifestations of blunt chest trauma. This information should be promptly relayed to the emergency clinician.

Despite the severe nature of the injury, the clinical manifestations of an aortic rupture are often deceptively meager. Associated pulmonary, neurologic, orthopedic, facial, and abdominal injuries are commonly present. Coexisting injuries can mask the signs and symptoms of an aortic injury or divert the physician's attention away from the more lethal aortic rupture. The absence of any external evidence of a chest injury does not eliminate the possibility of an aortic tear. One-third to one-half of patients reported in the literature have no external signs of chest trauma.

The most common symptom is interscapular or retrosternal pain. It is often found in nontraumatic aortic dissection but is present in only 25% of patients with a traumatic aortic disruption. Other symptoms described in the literature but uncommonly present include dyspnea resulting from tracheal compression and deviation, stridor or hoarseness caused by compression of the laryngeal nerve, dysphagia caused by esophageal compression, and extremity pain caused by ischemia from decreased arterial flow.

Clinical signs are uncommon and nonspecific. Generalized hypertension, when present, may be an important clinical sign. Sympathetic afferent nerve fibers, located in the area of the aortic isthmus, are capable of causing reflex hypertension as a response to a stretching stimulus. The presence of a harsh systolic murmur over the precordium or posterior interscapular area may be heard in up to one-third of patients. The murmur is thought to result from the turbulent flow across the area of transection. A less commonly encountered physical finding is a swelling at the base of the neck caused by the extravasation of blood from the mediastinum, which results in an increased neck circumference or a pulsatile neck mass. Other clinical signs suggestive of aortic rupture include lower extremity pulse deficit and lower extremity paralysis. Initial chest tube placement output in excess of 750 mL is also suggestive of aortic rupture, especially if the hemothorax is left sided. However, the physical examination is neither sensitive nor specific for aortic injury.

Diagnostic Testing

Chest Radiography

Radiography of the chest can be a valuable tool when aortic rupture is suspected. An increase in the width of the superior mediastinum is the most sensitive sign and is found in the majority of aortic ruptures (Fig. 38.21).

However, specificity of this radiologic sign may be as low as 10%; mediastinal widening may be caused by venous bleeding from a clavicle, thoracic spine, or sternal fracture; pulmonary contusions; a previous mediastinal mass; a misplaced CVP catheter; or magnification caused by the anteroposterior and supine position of a portable chest radiograph. Hence the sign is not pathognomonic for aortic rupture. Every effort should be made to obtain a standard upright inspiratory posteroanterior film, if clinically feasible, before a mediastinum is declared abnormal, to avoid false-positive interpretations. However, although mediastinal widening may be indicative of aortic rupture, its absence does not preclude the injury. Up to nearly half of patients with blunt aortic injury may have a normal mediastinum on chest radiography.[40] Given this, we recommend the use of chest CT scanning in patients with suspected aortic rupture, regardless of x-ray findings.[40]

Chest Computed Tomography Scan

Chest CT scanning is the gold standard test for blunt aortic injury and has replaced aortography as the test of choice. CT scans have almost 100% sensitivity and specificity for rapidly detecting aortic injury while requiring only IV contrast administration (Figs. 38.22 and 38.23). A normal aortic contour on CT, even in the presence of a mediastinal hematoma, has been shown to be highly accurate in excluding thoracic aortic disruption (Figs. 38.24 and 38.25).[41]

As a result of the improvements in CT scanning technology, more subtle aortic lesions are now being identified, which has led to the term "minimal aortic injury." A minimal aortic injury is defined as an aortic injury with an intimal flap less than 1 cm, and no or minimal periaortic mediastinal hematoma. Up to 10% of patients with blunt aortic injury diagnosed on CT scan may have

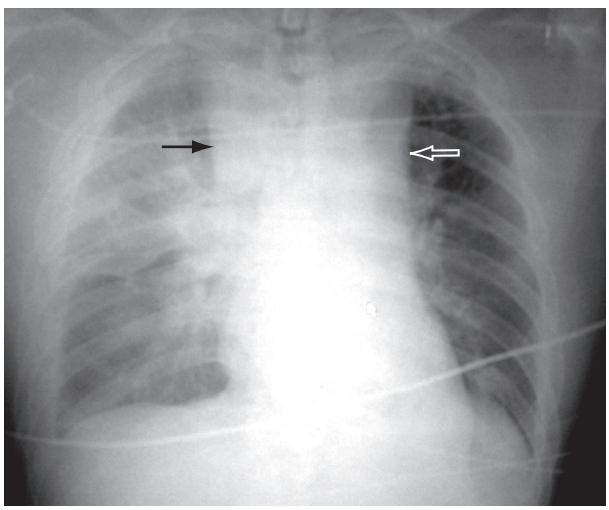

Fig. 38.21. Anteroposterior radiograph of the chest showing wide mediastinum *(arrows).*

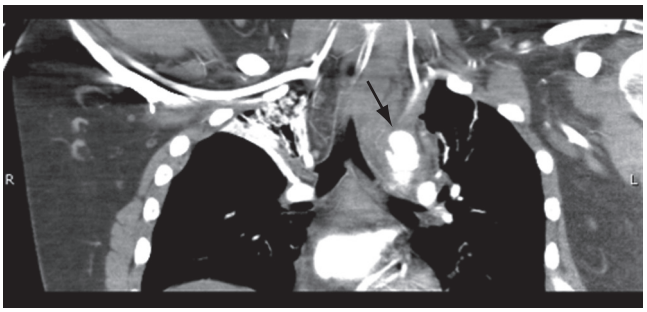

Fig. 38.22. Chest computed tomography (CT) scan showing aortic intimal tear with surrounding mediastinal hematoma *(arrow).*

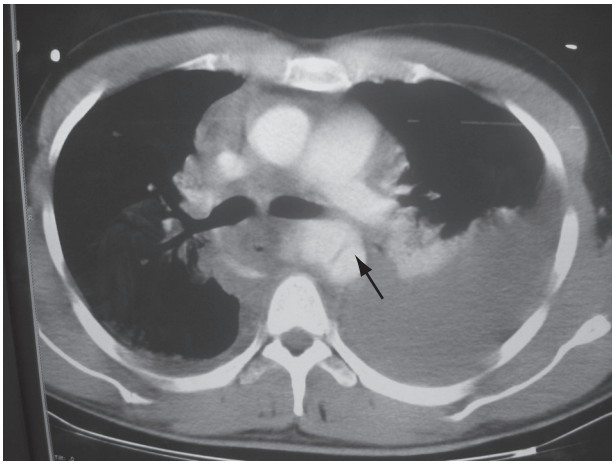

Fig. 38.23. Chest computed tomography (CT) scan demonstrating periaortic hemorrhage and an intimal flap *(arrow).*

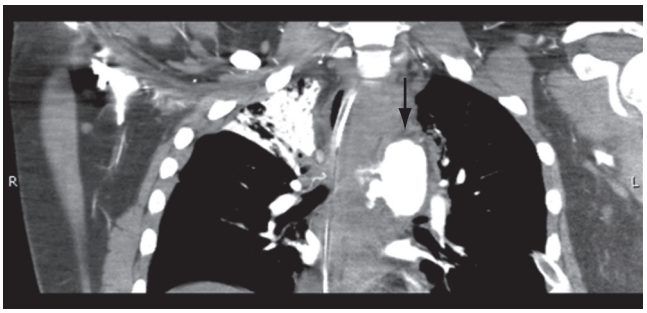

Fig. 38.24. Chest computed tomography (CT) scan showing aortic injury with active extravasation *(arrow).*

a minimal aortic injury.[42] It is felt that minimal aortic injuries carry a relatively low risk of rupture. Although at least two grading systems linking CT findings of blunt aortic injury to outcomes have been developed, neither has been externally validated.[43,44] In lieu of a validated decision instrument, we recommend following these patients with serial CT scans. If the injury is associated with significant thrombus, periaortic hematoma, lumen encroachment, or pseudoaneurysm, the patient can be treated via endograft.

Management

Stabilization and Empirical Therapy

Due to the ever-present risk of sudden rupture and exsanguination, repair of the aortic injury should be performed as soon as the diagnosis is made. Management of the patient with multiple injuries who has documented rupture of the thoracic aorta

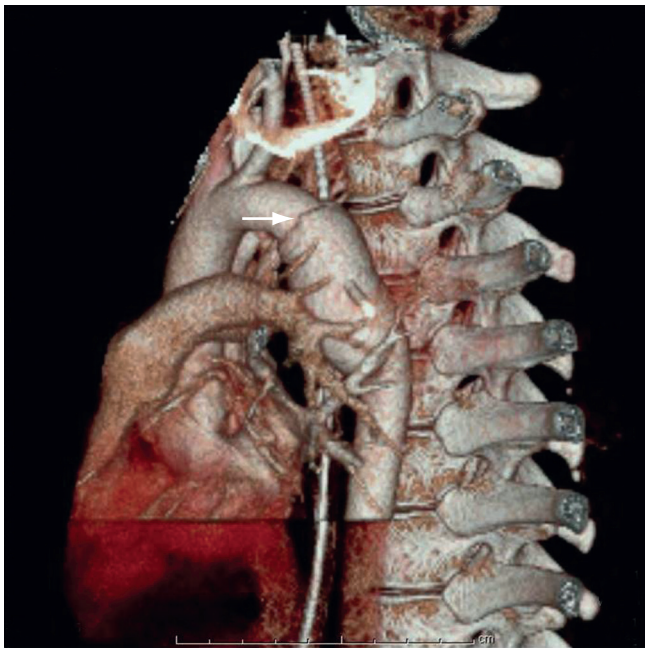

Fig. 38.25. Three-dimensional reconstruction of chest computed tomography (CT) scan showing aortic injury *(arrow).*

depends on the nature of associated injuries. Endovascular or surgical repair of the aortic rupture should be delayed in the presence of life-threatening intracranial or intra-abdominal injury or profuse retroperitoneal hemorrhage.[40] Consideration for delay of the procedure should be made for patients at high risk for infection (eg, those who have extensive body surface burns, contaminated large open wounds, established sepsis, or severe respiratory insufficiency caused by thoracic trauma).

Careful regulation of blood pressure is mandatory until definitive surgical repair can be performed. If operative repair is delayed, the systolic blood pressure should be maintained between 100 and 120 mm Hg. The objective of lowering the blood pressure is to decrease the shearing jet effect of an elevated pulse pressure, thus decreasing the possibility of continued adventitial dissection and subsequent free rupture.

Esmolol, a short-acting titratable beta-blocker, is ideally suited for this purpose because, unlike nitroprusside sodium, it decreases the pulse pressure and minimizes the shearing effect on the intact adventitia of the aorta.[45] Esmolol can be initiated with a bolus of 0.5 mg/kg over 1 minute, followed by an infusion of 0.05 mg/kg/min (titrated upward in 0.05 mg/kg/min increments to a maximum of 0.3 mg/kg/min). If blood pressure is not adequately controlled, nitroprusside sodium can be added as a second agent, beginning at a dose of 0.25 to 0.5 mcg/kg per minute.

Definitive Management

Many surgical techniques have been described since the first successful repair by Passaro and Pace in 1959.[46] The pathologic condition found dictates the type of repair, and a synthetic graft is often required because of extensive tension on the vessel walls or jagged torn ends of the vessel. However, open repair can have associated complications of stroke, paraplegia, and renal failure due to aortic clamp time.

Endovascular Repair. A number of studies indicate that success rates and complication rates are likely better than those of traditional open surgical repairs and that the risk of major surgery and subsequent paraplegia from prolonged aortic clamping is significantly reduced with endovascular repair. Current

guidelines recommend endovascular treatment for patients without contraindications.[40]

ESOPHAGEAL PERFORATION

PRINCIPLES

Background and Importance

The classic description of esophageal perforation resulting from forceful vomiting was published in 1724 by Boerhaave, and from 1724 to 1941 the occurrence of Boerhaave's syndrome was almost uniformly fatal.[47] In 1941 the first successful surgical treatment, a drainage procedure, was reported, and in 1947 the first successful closure of a ruptured esophagus was described. Since then, improved surgical techniques, greater physician awareness leading to a more prompt diagnosis, the availability of more effective antibiotics, and better general supportive measures have reduced the mortality to approximately 20%. Mortality data cited for perforation are affected by several variables, such as location (with perforations of the thoracic segment having the highest mortality rate), mechanism of injury, time elapsed between injury and diagnosis, the presence of preexisting esophageal disease, and general health of the patient.

Pathophysiology

The anatomic feature responsible for the prolonged morbidity and high mortality associated with esophageal perforation is the lack of an esophageal serosal covering that allows perforation at any level direct access to the mediastinum. Perforations in the upper or cervical esophagus enter the retropharyngeal space, where fascial planes extend from the base of the skull to the bifurcation of the trachea. Perforations in the midesophagus and lower esophagus enter directly into the mediastinum. Only the thin mediastinal pleura prevents free access to the entire pleural cavity, and this barrier is commonly overcome by continued drainage and the massive exudative inflammatory reaction induced by chemical and bacterial mediastinitis. When the mediastinal pleura are penetrated, the negative pressure generated by respiratory efforts tends to increase soilage by promoting drainage from the gastrointestinal tract into the mediastinum and pleural space.

When esophageal rupture results from forceful emesis, as in cases of Boerhaave's syndrome, the intrinsic weakness of the left posterior distal esophagus is important. Other areas (including cervical, midthoracic, and infradiaphragmatic sites) have been reported only rarely to rupture secondary to emesis. In addition, the esophagus has three areas of anatomic narrowing: (1) the cricopharyngeal muscle near the esophageal introitus, (2) the level at which the esophagus crosses the left mainstem bronchus and the aortic arch, and (3) the gastroesophageal junction. In the absence of a preexisting esophageal disease (such as, carcinoma), it is unusual for a perforation caused by a foreign body to occur anywhere other than at these three sites. Foreign bodies may cause perforation by direct penetration, pressure, or chemical necrosis.

CLINICAL FEATURES

The most reliable symptom of an esophageal injury is pleuritic pain localized along the course of the esophagus that is exacerbated by swallowing or neck flexion (Fig. 38.26). Pain may be located in the epigastrium, substernal area, or back; usually worsens over time; and may migrate from the upper abdomen to the chest. As the infectious process worsens, dyspnea usually ensues.

The early physical signs of an esophageal perforation are sparse. As air and caustic contaminated material move through the esophageal tear into the mediastinum and pleural space, and before any subcutaneous air is palpable at the root of the neck, the mediastinal air may impart a nasal quality to the voice. Mediastinal air may surround the heart and produce a systolic

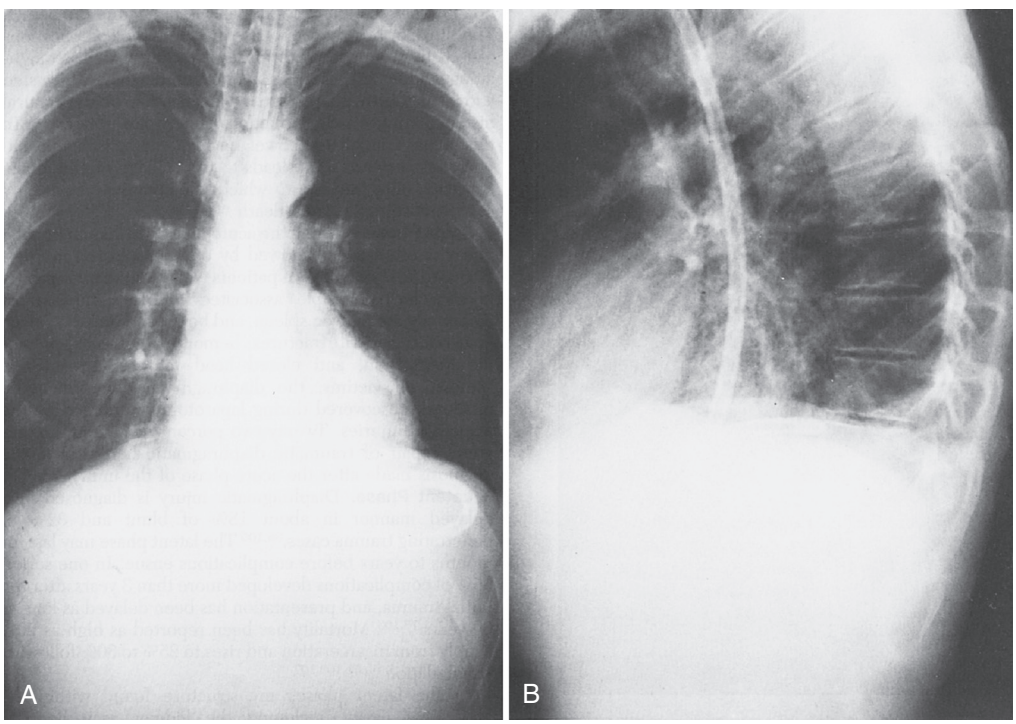

Fig. 38.26. A, Chest radiograph of a 36-year-old man with acute onset of pleuritic chest pain after forceful vomiting. **B,** Chest radiograph shows mediastinal and subcutaneous air typical of ruptured esophagus. Mediastinum is not yet widened, and there is no soilage of the pleural cavity.

BOX 38.5

Most Common Causes of Esophageal Perforation

Iatrogenic
Foreign bodies
Caustic burns
Blunt or penetrating trauma
Spontaneous rupture (post emetic or Boerhaave's syndrome)
Postoperative breakdown of anastomosis

crunching sound (Hamman's crunch). As air and fluid move into the pleural space, signs of a hydropneumothorax or an empyema may develop. Eventually the air travels into the subcutaneous tissues, dissecting into the neck, where subcutaneous emphysema may first become evident. This classic sign is present in only approximately 60% of patients, however, and in the absence of a tracheal injury, it may occur in only 30%. The most common causes of an esophageal perforation are shown in Box 38.5.

Iatrogenic

Most esophageal perforations are iatrogenic, most commonly as a complication of instrumentation. The rigid endoscope is the most common offender, particularly when general anesthesia is used. Although use of the flexible endoscope has made this complication less likely, the total number of perforations has increased as more procedures are performed. Injuries tend to occur near the cervical esophagus as the endoscope is inserted. Endoscopic procedures that are too vigorous in the presence of a corrosive burn or carcinoma are also a common cause of iatrogenic esophageal injury. In the ED, nasotracheal or nasogastric intubations are the most common causes of iatrogenic perforation, with the perforation usually occurring in the pyriform sinus.

The use of an esophageal obturator airway was also associated with occasional esophageal trauma, specifically midesophageal perforation. Use of the esophageal obturator airway's successors, the laryngotracheal Combitube and the King airway, do not seem to be associated with trauma more severe than occasional esophageal abrasions or contusions.

Foreign Bodies

Foreign bodies can cause an esophageal injury by direct laceration, by pressure necrosis, or during endoscopic removal. Small perforations tend to seal without sequelae, but pressure necrosis or lacerating injuries provide ample access to the mediastinum. Foreign bodies usually lodge in the cervical esophagus. In children younger than 4 years old, the cricopharyngeal narrowing is the usual point of foreign body impaction. After 4 years old, most objects pass this region and traverse the remaining normal esophagus. In adults, a foreign body impaction, especially in cases of repeated episodes, raises the possibility of a stricture and warrants further investigation.

Caustic Burns

Caustic burns of the esophagus occur from intentional or accidental ingestion of acid or alkali. There are two peaks of incidence: (1) from 1 to 5 years old, which is when ingestion is usually of a small amount of material and accidental, and (2) in the teens and 20s, when larger quantities are ingested during suicide attempts. Symptoms include hematemesis, respiratory distress, vomiting, drooling, or the presence of oropharyngeal lesions on physical examination.

The liquefaction necrosis classically resulting from strong alkali burns (pH >12) is more likely to cause esophageal perforation than the coagulation necrosis resulting from strong acid burns (pH <2). Individuals ingesting alkali substances with a pH less than 11.5 rarely sustain injuries more serious than superficial mucosal burns. Acid ingestions cause damage more frequently in the stomach than in the esophagus.

Endoscopy within the first 6 to 18 hours may be used to determine the extent of the injury and to guide therapy. Although admission after significant ingestion is the rule, some authors suggest that in the setting of accidental ingestion in children and in the absence of symptoms, endoscopy and admission may not be indicated. Esophagoscopy is commonly undertaken to ascertain the presence or absence of esophageal injury. Advancing the esophagoscope beyond the first burn in the esophagus increases the risk of perforation and is a common iatrogenic cause of esophageal perforation.

Penetrating and Blunt Trauma

Because of its well-protected position posteriorly, esophageal trauma is rare and usually not an isolated injury. Cervical esophageal injuries are the most common because of a lack of protection by the bony thorax, and the trachea is the most common associated site of injury. In some cases, the esophageal injury may be overlooked initially because of the dramatic presentation of a patient with a tracheal injury.

Typical symptoms seen in cervical esophageal injuries include neck pain, dysphagia, cough, voice changes, and hematemesis. Physical findings may include neck tenderness, resistance to flexion, crepitus, or stridor. In one large series, the most common life-threatening problem in the ED was compromise of the airway. Most of these cases were handled with rapid sequence intubation, but a significant number (12%) of patients required cricothyroidotomy.

If the patient's condition is stable, a preoperative esophagogram with a water-soluble agent should precede any endoscopy. Although chest and neck radiograph and CT also may be used to diagnose this injury, emergent bedside flexible endoscopy seems to be the test of choice to confirm negative findings on esophagography (especially in the setting of penetrating trauma). Operative repair is indicated in most of these injuries (>90%) and should be done as quickly as possible to avoid the development of fistulae, mediastinitis, or abscess formation.

Spontaneous Rupture

Spontaneous esophageal rupture, post-emetic rupture, and *Boerhaave's syndrome* are synonymous terms. This esophageal injury is associated with a poor prognosis because the forces required to rupture the esophagus result in almost instantaneous and massive mediastinal contamination. The distal esophagus is the usual site of injury, with a longitudinal tear occurring in the left posterolateral aspect. More than 80% of these injuries occur in middle-aged men who have ingested alcohol and large meals. Increases in intra-abdominal pressure resulting from blunt trauma, seizures, childbirth, laughing, straining at stool, and heavy lifting have all been reported to cause this injury.

DIAGNOSTIC TESTING

The diagnosis of esophageal perforation is aided by consideration of clinical circumstances. In patients with classic Boerhaave's syndrome, emesis is followed by severe chest pain, subcutaneous emphysema, and cardiopulmonary collapse. Development

BOX 38.6

Clinical Conditions That May Mimic Esophageal Perforation

Spontaneous pneumomediastinum
Aortic aneurysm (thoracic)
Pulmonary embolus
Perforated peptic ulcer
Myocardial infarction (MI)
Pancreatitis
Mesenteric thrombosis
Cholecystitis
Pneumonia

of these signs and symptoms after instrumentation of the esophagus or removal of an esophageal foreign body is relatively straightforward. One-third of cases of perforated esophagus are atypical, however. A careful history and physical examination supplemented by appropriate imaging studies enable the clinician to diagnose a subtle case at an early stage. In considering any of the diagnoses listed in Box 38.6, a perforated esophagus should also be considered.

Radiology

The radiographic examination usually suggests the diagnosis of an esophageal perforation. The classic chest radiograph findings are mediastinal air (with or without subcutaneous emphysema), left-sided pleural effusion, pneumothorax, and widened mediastinum. Lateral views of the cervical spine may reveal air or fluid in the retropharyngeal area that is characteristic of a cervical esophageal perforation but also is found when perforations in the lower parts of the esophagus release air or fluid that dissects superiorly (Fig. 38.27). Water soluble diatrizoate meglumine (Gastrografin) is preferred for visualization in cases of suspected esophageal perforation. It does not obscure visualization during subsequent endoscopy, and it produces less mediastinal soiling than barium. Then, if no leak is found, a barium study may be undertaken to better define the mucosal detail.

Endoscopy

Endoscopy, similar to contrast studies, is not an infallible aid in establishing the presence or absence of an esophageal perforation. The size and location of the perforation and the skill of the endoscopist are important factors in the low incidence of false-negative studies. If the accuracy of the endoscopy is in doubt, an esophagogram should be performed. Helical CT with dilute oral contrast has been reported as a safer, faster, and less manpower-intensive diagnostic examination. Some of the abnormalities that may be seen on CT scan include extraluminal air, periesophageal fluid, esophageal thickening, and extraluminal contrast. These CT findings may be the first clue to the correct diagnosis of esophageal perforation.

MANAGEMENT

Early diagnosis can best be accomplished if one is aware of the pathophysiology and clinical settings in which esophageal perforations occur. Time is crucial in minimizing the mortality and morbidity of this condition. If the diagnosis is strongly suggested or confirmed, management should include broad-spectrum antibiotics (covering oral flora), volume replacement, and airway maintenance.[48] An emergency surgical consultation should be

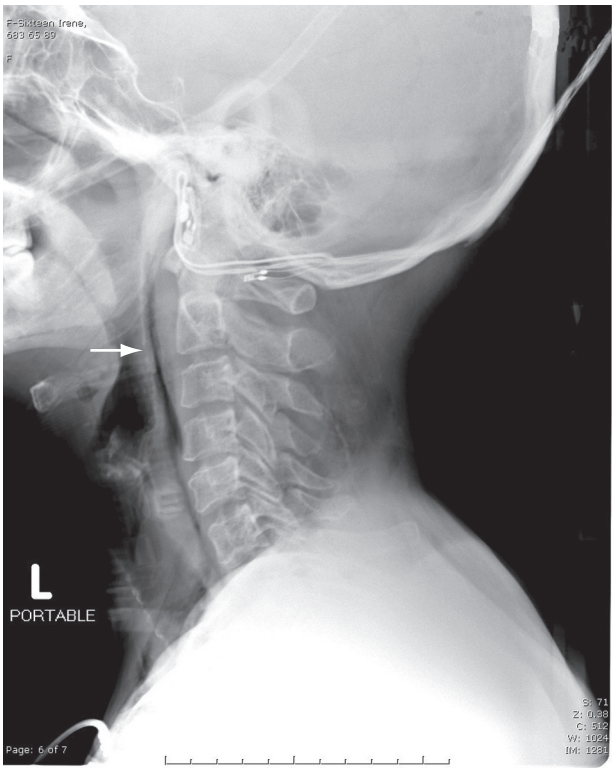

Fig. 38.27. Lateral view of the cervical spine revealing air in the retropharyngeal area *(arrow)*.

obtained because prognosis worsens as time passes, with mortality almost doubling in the first 12 hours.

Although operative therapy is standard, nonoperative therapy is an option for patients with well-contained perforations, with minimal mediastinal involvement, and without evidence of sepsis. In such cases, the patient is placed on *nil per os* (nothing by mouth) (NPO) status for at least 72 hours, broad-spectrum antibiotics are initiated, and usually total parenteral nutrition treatment is begun. The use of nasogastric tubes should be discouraged, because they may increase gastroesophageal reflux and worsen the contamination of the mediastinum.

KEY CONCEPTS

- Even relatively minor chest wall injuries, such as rib fractures, may result in serious complications in elderly patients and patients with preexisting pulmonary disease if adequate analgesia and close follow-up care are not provided.
- Unless there are abnormalities on the electrocardiogram (ECG) or an elevated serum troponin level, there is no need to pursue the diagnosis of myocardial contusion with more sophisticated tests.
- Many patients with myocardial rupture or traumatic aortic rupture survive to reach the hospital and can be salvaged with rapid diagnosis and intervention.
- Pericardial tamponade can be diagnosed accurately before hemodynamic decompensation occurs by standard cardiac ultrasound performed by emergency clinicians.
- Chest computed tomography (CT) scan is the test of choice for blunt aortic injury even in the presence of normal chest radiographs.
- The NEXUS-Chest CT criteria can be used to determine the need for chest CT in patients with blunt trauma.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

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CHAPTER 38: QUESTIONS & ANSWERS

38.1. An 18-year-old man presents after a motor vehicle collision (MVC) in which he was ejected from the vehicle. The paramedics have been administering bag-valve-mask ventilation en route because of respiratory distress and now report increased resistance with ventilations. The patient has decreased breath sounds on the left. His blood pressure is 80/40 mm Hg, and his pulse is 145 beats per minute. His respirations are agonal, with a rate of 5 breaths per minute. Which of the following is the most appropriate next step in the management of this patient?

A. Anteroposterior chest radiograph
B. Emergency department thoracotomy (EDT)
C. Endotracheal intubation
D. Needle decompression
E. Tube thoracostomy

Answer: D. This clinical scenario depicts a patient with a tension pneumothorax. He has decreased blood pressure, decreased breath sounds, and, most important, an increased resistance to ventilation, which is the earliest sign of the development of a tension pneumothorax. Immediate decompression with a large-bore needle is the correct initial management in this condition.

38.2. Which of the following is the most sensitive electrocardiogram (ECG) manifestation of myocardial contusion?
A. Biphasic T wave
B. Left bundle branch block
C. Right bundle branch block
D. Sinus tachycardia
E. Transient ST segment elevation

Answer: D. Sinus tachycardia is present in approximately 70% of patients with documented myocardial contusion and is the most sensitive sign for this condition. It is, however, also the least specific.

38.3. A patient presenting with blunt thoracic trauma complains of shortness of breath and chest pain. On physical examination, he is tachypneic with chest wall bruising and moist rales on the right side on auscultation. Which of the following is the least likely finding?
A. Consolidation within 6 hours of injury
B. Diffuse patchy alveolar infiltrates on chest radiograph in 24 hours
C. Low partial arterial pressure of oxygen (Pao_2) on arterial blood gas sampling
D. Patchy alveolar infiltrate on chest radiograph within minutes of injury
E. Rib fractures

Answer: B. This patient has physical findings consistent with pulmonary contusion. All answers are correct except B. Delayed onset of diffuse alveolar infiltrates is more consistent with acute respiratory disease syndrome (ARDS). The development of ARDS is diffuse and usually delayed, with onset typically between 24 and 72 hours after injury.

38.4. A 55-year-old man complains of chest wall pain after a high-speed motor vehicle collision (MVC). He has ecchymosis of the left lateral chest wall. You notice that there is outward movement of the left lateral chest wall on expiration. Which of the following statements regarding this patient's problem is *not true*?
A. Chest radiograph likely demonstrates parenchymal contusions.
B. Intubation splints the chest wall internally.
C. Multiple rib fractures are likely.
D. Positioning of patient with injured side down improves symptoms.
E. The cornerstone of treatment is pulmonary physiotherapy.

Answer: D. This patient has a flail chest. Out-of-hospital or emergency department (ED) stabilization of the flail segment, by positioning the person with the injured side down or placing a sandbag on the affected segments, has been abandoned. Endotracheal intubation and positive-pressure ventilation will internally splint the chest wall, making the flail segment difficult to detect on physical examination. The cornerstones of therapy include pulmonary physiotherapy, effective analgesia, selective use of endotracheal intubation and mechanical ventilation, and close observation for respiratory compromise.

38.5. A 50-year-old woman is brought in by emergency medical services on a backboard after a motor vehicle collision (MVC), complaining of shortness of breath. She has decreased breath sounds on the right side of the chest. A chest tube is placed, with a return of 200 mL of blood in the first hour, 200 mL in the second hour, and 350 mL in the third hour. What is the next step in the management of this patient?
A. Check coagulation profile
B. Conservative management and transfusion as needed
C. Emergency thoracotomy
D. External fixation of rib fractures
E. Insertion of a second thoracostomy tube

Answer: C. Immediate drainage of more than 1500 mL of blood from the pleural cavity is usually considered an indication for urgent thoracotomy. Perhaps even more predictive of the need for thoracotomy is a continued output of at least 200 mL/hr for 3 hours.

38.6. A 37-year-old man presents with chest pain after a motor vehicle collision (MVC). He states that his chest hit the steering wheel. On initial evaluation, the patient is without fractures of the ribs or sternum, but there is a small chest wall contusion. The initial chest radiograph is negative, and the electrocardiogram (ECG) shows nonspecific ST-T wave changes. What is the next step in the emergency management of this patient?
A. Admit to telemetry for 23-hour observation
B. Discharge home after repeat ECG and troponin at 6 hours
C. Discharge home after repeat ECG in 1 hour
D. Discharge home if echocardiogram is negative
E. Discharge home if initial troponin is negative

Answer: B. In patients who have minor injuries and are otherwise asymptomatic, elevated troponin levels and minor ECG abnormalities do not necessarily indicate a clinically significant myocardial contusion. Very few of these patients will develop complications. However, normal troponin level (4 to 6 hours after injury), along with normal (or unchanged) ECGs, correlate with minimal risk of cardiac complications. Echocardiography is rarely required in this low-risk subset of patients who have minor injuries and are otherwise asymptomatic.

38.7. A 30-year-old woman presents intubated by emergency medical services on a backboard with C spine immobilization. She was found unresponsive and hypotensive after a front-end collision. She was the driver of the vehicle and unbelted. Despite fluid resuscitation, the patient continues to be tachycardic and hypotensive. On physical examination, you note jugular venous distention (JVD) and a harsh murmur that sounds like a splashing mill wheel. An electrocardiogram (ECG) reveals electrical alternans. Which of the following statements is true regarding the patient's underlying problem?
A. Echocardiogram will reveal diastolic collapse of the right ventricle and fluid in the pericardium.
B. Focused assessment with sonography in trauma (FAST) will demonstrate echogenicity in Morrison's pouch.
C. Patchy consolidation will be seen on chest radiograph.
D. Pericardiocentesis is not indicated.
E. She will have a negative focused abdominal sonogram.

Answer: A. Early use of emergency department (ED) ultrasonography may facilitate the early diagnosis of cardiac rupture and pericardial tamponade. The combination of shock and JVD (or an elevated central venous pressure [CVP]) in a patient with blunt chest trauma should immediately suggest pericardial tamponade.