The manifestations of thoracic polytrauma are diverse, depending on both the mechanism of injury and the organ system or systems affected. Blunt trauma refers to closed, nonpenetrating physical trauma caused by impact injury or other compressive and shear forces. Common examples include deceleration injuries (motor vehicle accidents, falls) and blunt force injuries (physical attacks, crush injuries). Complications include abrasions, contusions, organ laceration or rupture, and bone fractures [1–4]. In contrast, penetrating trauma occurs when an object pierces the skin and enters the body. Injury severity is determined by the pathway and momentum of the object. Low-velocity items, such as knives, are propelled by hand and damage only areas that are in direct contact. Higher-velocity projectiles, including bullets and other shrapnel, create pressure waves that force out adjacent tissue as the projectile enters the body. This damages regions in direct contact while also causing cavitation injury to a large surrounding area [1, 5, 6].

This article discusses the utility of chest radiography in the evaluation of thoracic polytrauma. The pathophysiology, imaging manifestations, and management recommendations will be discussed for injuries to the chest wall, diaphragm, pleura, lungs, mediastinum, heart, aorta, and great vessels.

**OBJECTIVE.** Chest radiography is the first-line imaging examination for assessment of thoracic polytrauma, serving to evaluate the extent of injury and facilitate early triage to observation, further imaging, or immediate surgical intervention. The objective of this article is to review the spectrum of injuries that occur in the chest and upper abdomen after blunt and penetrating trauma. Pathophysiology, imaging findings, and management recommendations will be discussed for injuries to the chest wall, diaphragm, pleura, lungs, mediastinum, heart, aorta, and great vessels.

**CONCLUSION.** Chest radiography plays an important role in the initial evaluation of blunt and penetrating chest trauma, providing rapid imaging information to supplement the history and physical examination. In the emergency department, familiarity with the spectrum of injuries that can occur in the chest and upper abdomen is important for accurate interpretation of chest radiographs as well as establishment of appropriate recommendations for management and follow-up.
pleura, lungs, mediastinum, heart, aorta, and great vessels will be reviewed. Several classic trauma-related signs in chest radiology will also be defined and illustrated.

**Chest Wall**

**Soft Tissues**

Subcutaneous emphysema refers to the presence of air in the extrathoracic soft tissues. This condition can result from chest wall infection, blunt trauma with damage to the respiratory or gastrointestinal systems, and penetrating injuries that introduce external air into the soft tissues. Chest radiography shows air in the subcutaneous tissues, which may create radiolucent striations outlining the individual fibers of the pectoralis major muscles (“ginkgo leaf” sign) (Fig. 1). Air can spread via fascial planes to the rest of the chest wall and abdomen and even to the head, neck, and extremities. The condition is usually self-limiting, but severe cases may compress the trachea and require intervention. Sources of persistent air leakage will require corrective surgery.

Subcutaneous hematomas are produced by accumulation of blood in the soft tissues. This condition may result from damage to thoracic vessels, muscles, or ribs during blunt or penetrating chest trauma. On chest radiographs, nonspecific opacities are visualized in the soft tissues (Fig. 2). Localization to the chest wall may not be possible without lateral radiographs. Most hematomas resolve spontaneously, but persistent bleeding may be seen with severe trauma, coagulopathies, and vascular malformations. Foreign bodies, such as knife blades and bullet shrapnel, also can become lodged in the soft tissues after penetrating trauma (Fig. 3). Operative removal is indicated when surgically feasible [7–10].

**Bones**

Blunt chest trauma can result in a variety of skeletal injuries depending on the mechanism involved. Forceful shoulder injuries can produce scapular fractures, which are shown on chest radiographs and scapular views (Fig. 4). Scapulothoracic dissociation, or flail shoulder, occurs when strong forces pull the shoulder girdle away from the thorax. This can predispose to muscle, vascular, and nerve injury. Scapular dislocation, edema, and hematoma formation are noted on chest radiographs.

Clavicle fractures are common in trauma patients and are generally of minor clinical significance (Fig. 4). Sternoclavicular dislocations or fractures occur after severe shoulder trauma and may be identified on angled chest radiographs (Fig. 5). Posterior dislocations may injure the mediastinal organs and great vessels. These injuries require closed or surgical reduction.

Fractures to the upper ribs are rare and suggest severe downward trauma with damage to the great vessels and brachial plexus. Lower rib fractures may also involve upper abdominal organs such as the liver, spleen, and kidneys, and CT should be ordered if suspicion for injury is high. Fractured rib ends can lacerate the pleura or lung, leading to the formation of pulmonary hematomas, hemothorax, or pneumothorax. Most fractures can be visualized on chest radiographs, and a radiodense fracture callus develops after several weeks.
Chest Radiography in Thoracic Polytrauma

Fig. 6—Fractures of the ribs. A, 21-year-old man with remote history of bilateral rib fractures who was injured in motor vehicle crash. Frontal chest radiograph shows radiodense fracture callus (asterisk), indicative of healed fracture. B, 30-year-old man injured in motor vehicle crash. Frontal chest radiograph shows left-sided fractures in posterior segments of at least seven adjacent ribs (arrows), creating flail chest physiology.

Fig. 7—25-year-old man injured in motor vehicle collision. Lateral chest radiograph shows fracture and displacement of sternum (double-headed arrow).

(Fig. 6A). Flail chest occurs when at least five contiguous single fractures or three adjacent segmental rib fractures are present, resulting in paradoxical motion during the respiratory cycle (Fig. 6B). Posterior flail segments are supported by overlying muscles and scapulae, and therefore may not cause serious complications. Anterior and lateral flail segments, which are free-moving, can severely impair respiratory function and predispose to atelectasis and infection. Positive-pressure ventilation or surgical fixation may be required for stabilization.

Sternal injuries are seen in anterior chest trauma. Most fractures occur in the upper or mid body of the sternum and are seen in conjunction with retrosternal hematoma and myocardial contusion. These injuries are difficult to identify on frontal chest radiographs and often require lateral or sternal views for enhanced visualization (Fig. 7). Surgical fixation is unnecessary, and healing occurs over several weeks.

Spinal fractures can result from compression or whiplash injury and are associated with damage to neurologic and vascular structures. Optimal evaluation requires dedicated frontal and lateral spine radiographs. Immobilization and surgical fixation are necessary to prevent further damage. Infection of the intervertebral disks (diskitis) can produce disk space narrowing and erosion with adjacent abscess formation. Immobilization with antibiotic treatment is required [7–10] (Fig. 8).

Diaphragm

Diaphragmatic rupture may be secondary to blunt or penetrating injury. Hemidiaphragmatic elevation may be seen, and upper abdominal organs, including the stomach (collar sign), liver (“cottage loaf” sign), spleen, small bowel, and colon, may herniate into the thoracic cavity. Associated findings include basilar lung opacities, irregular diaphragmatic contours, and lower rib fractures. In addition, pneumoperitoneum can result from open thoracoabdominal communication or perforated abdominal viscera, with air seen accumulating beneath the diaphragm superolaterally on erect radiographs or anteromedially on supine radiographs (cuptula sign). However, other conditions such as basilar lung atelectasis, subpulmonic effusion, subphrenic abscess, colonic interposition (Chilaiditi syndrome), diaphragmatic evagination, congenital diaphragmatic hernia, and phrenic nerve injury can have a similar appearance on chest radiographs, and CT is required for diagnosis. Surgical repair is necessary to prevent late complications such as bowel incarceration or strangulation, thoracic organ compression, and diaphragmatic paralysis. Splenosis is a rare complication of left-sided thoracoabdominal trauma in which thoracic autotransplantation of splenic tissue results in the formation of left-sided chest wall masses. Heat-damaged RBC scanning is diagnostic, and no interventions are necessary [7, 8, 11, 12] (Fig. 9).

Plura

After chest trauma, air may enter the pleural cavity from the outside environment (open pneumothorax) or from within the body (closed pneumothorax). Open or communicating pneumothorax, also called “sucking chest wound,” develops when the skin and pleura are injured by penetrating trauma. Immediate closure and chest tube placement are indicated. Closed, or simple, pneumothorax develops after blunt trauma, usually due to pleural laceration by fractured ribs. Conservative management is recommended, and tube thoracostomy should be performed only if the patient is symptomatic. Diagnosis of pneumothorax requires visualization of the “visceral pleural line sign,” which represents separation of the visceral and parietal pleura. On supine radiographs, anterocaudal movement of pleural air may produce hyperlucent lung bases, a deep and radiolucent costophrenic sulcus (deep sulcus sign), and outlining of the anterior and posterior portions of the hemidiaphragm (double diaphragm sign). Tension pneumothorax occurs when a pneumothorax permits entry into but not exit of air from the thoracic cavity. Increasing intrathoracic pressure leads to collapse of the ipsilateral lung, followed by compression of the contralateral lung and mediastinum. Associated findings include unilateral hyperlucent lung, widened intercostal spaces, hemidiaphragmatic depression, and tracheal deviation. However, diagnosis is primarily clinical because of the emergent nature of the condition and the lack of specificity of early imaging findings. Suspected tension pneumothorax should be immediately decompressed with large-bore needle thoracostomy before radiographs are

AJR:192, March 2009
obtained. A postprocedure radiograph is obtained to verify subsequent tube placement and to assess the effectiveness of therapy (Fig. 10).

Simple hemothorax can result from vascular rupture or laceration in blunt and penetrating trauma. On chest radiographs, the appearance is similar to serous pleural effusion (hydrothorax), with layering of fluid and blunting of the costophrenic angles. Rarely, effusions can be subpulmonic, loculated, or lamellar. Small hemothoraces usually resolve spontaneously, and drainage is rarely required. However, a large hemothorax can fill the entire pleural space and present radiographically as an opacified hemithorax. Chronic hemothorax can be complicated by infection (empyema or pyothorax) with chest wall erosion (empyema necessitatis) or fibrosis (fibrothorax) requiring decortication. Tension hemothorax can result from massive intrathoracic bleeding causing ipsilateral lung compression and mediastinal displacement. Emergent exploratory thoracotomy is indicated to identify and repair the site of bleeding (Fig. 11).

Chylothorax results from damage to the thoracic duct, with chylous fluid recovered through thoracentesis. Left-sided chylothorax is seen in ruptures of the upper thoracic duct, whereas right-sided chylothorax is produced by lower-level injuries in which the thoracic duct has crossed the midline (Fig. 12). CT offers enhanced contrast resolution that is useful for distinguishing chylothorax, hydrothorax, pyothorax, and hemothorax as well as other causes of radiographic density [7, 8, 10].

Lungs

Lung protrusion or herniation can occur through trauma-induced weakening or tears of the cervical, intercostal, and diaphragmatic fascia. Chest radiography identifies regions of lung extending beyond the thoracic cage (Fig. 13). Conservative management is advisable unless respiratory distress, incarceration, or strangulation occurs.

Lobar atelectasis or collapse may result from foreign body obstruction, aspiration, or bronchial rupture. Any lobe can be involved, and classic chest radiographic signs have been described for upper and middle lobe (“juxtaphrenic peak” sign or “Katten” sign), left upper lobe (luftsichel sign), left lower lobe (“flat waist” sign, “ivory heart” sign), and right lower lobe (superior triangle sign) collapse. Pneumothorax ex vacuo is a rare complication of acute lobar collapse that increases the negative intrapleural pressure around the collapsed lobe. This selectively draws gas into the space surrounding the collapsed lobe without affecting the visceral and parietal pleura of adjacent lobes. The pneumothorax resolves spontaneously after relief of the bronchial obstruction with reexpansion of the corresponding lobe. Identification of this condition is important for directing treatment toward the affected bronchus rather than inserting a chest tube into the pleural space (Fig. 14).
Chest Radiography in Thoracic Polytrauma

Pulmonary contusions occur when injury to the lungs results in leakage of blood and edema into the interstitial and alveolar spaces. On chest radiographs, contusions appear as geographic areas of peripheral air-space opacity or ground-glass opacification, usually adjacent to bony structures. Lesions are evident within 6 hours after trauma and generally resolve within 5–7 days. Pulmonary lacerations are more severe injuries involving disruption of the lung architecture. Organ ruptures and foreign body trauma may introduce air (pneumatocele), blood (hematoma), and infection (abscess) into the lung parenchyma. On chest radiographs, localized air collections are seen within areas of air-space opacity. Injuries take weeks or months to resolve, and chronic scarring may develop (Fig. 15).

Acute respiratory distress syndrome (ARDS) can develop secondary to a variety of insults, including trauma, infection, shock, aspiration, transfusion, and drugs. After 12–48 hours, damage to the alveolar–capillary barrier allows influx of fluid into the alveolar space, which manifests radiographically as diffuse bilateral patchy lung opacities (Fig. 16). The imaging differential diagnosis includes atelectasis, aspiration, fat embolism, alveolar pulmonary edema, pneumonia, and hemorrhage. Therapy involves treatment of the underlying condition and supportive care over weeks to months [7, 8, 10].

Mediastinum

Pneumomediastinum, or mediastinal emphysema, refers to the presence of air in the mediastinal structures, which can result from penetrating injury or blunt pharyngeal, tracheobronchial, or esophageal injury. Air freely tracks throughout the mediastinum and communicating spaces via vascular sheaths and readily ruptures through fascial planes to affect adjacent anatomic compartments. Several chest radiographic signs have been described, including air superior to the diaphragm (continuous diaphragm sign, continuous left hemidiaphragm sign, extrapleural air sign), surrounding the right pulmonary artery (“ring-around-the-artery” sign), lateral to the descending aorta (“Naclerio’s V” sign), and superior to the brachiocephalic veins (“V” sign at confluence of brachiocephalic veins) (Fig. 17). In children, elevation of the thymic lobe (thymic sail sign) can...
be seen. Identification and repair of the affected organs are indicated.

Mediastinal bleeding (mediastinal hematoma) can result from vascular injury. Large hematomas can produce radiographic irregularity and enlargement of the mediastinum. Proposed criteria for mediastinal widening include a width greater than 8 cm and a mediastinal to chest width ratio greater than 0.25 (Fig. 18A).

Esophageal rupture and foreign body injury can lead to mediastinal infection (mediastinitis). Radiography may show edema, hemorrhage, and gas production in the mediastinal and cervical soft tissues, as well as pleural effusions and lower-lobe consolidation [7, 8, 13, 14] (Fig. 18B).

Trachea and Bronchi

Tracheobronchial injury includes lacerations due to penetrating trauma and ruptures from blunt airway injury, particularly when the glottis is closed. Generally, concomitant injury to the chest wall, lungs, and great vessels is also present. Transverse tears usually occur between cartilaginous tracheal rings, whereas longitudinal tears are seen in the posterior tracheal membrane. This results in massive pneumomediastinum and possible airway edema, hemorrhage, and pneumothorax. On
chested radiographs, endotracheal tube overdistension may be seen, with herniation through the ruptured tracheal wall. In bronchial transections, the involved lung may be visualized falling inferiorly away from the hilum on erect radiographs and posterolaterally in the supine position (fallen lung sign) (Fig. 19). Surgical repair is required to maintain airway continuity and to prevent complications such as tracheobronchial stricture [7, 8, 13, 14].

**Esophagus**

Esophageal injury may be caused by violent vomiting (Boerhaave’s syndrome), penetrating injury, or compressive bone forces in blunt trauma. The esophagus runs to the left of the trachea at the level of the thoracic inlet, moves to the right at the level of the carina, and crosses back to the left as it enters the stomach. Most esophageal tears are located in the cervical and upper thoracic regions and present with left- and right-sided pleural effusions, respectively. Occasionally, gastroesophageal junction lesions are seen, typically in

---

**Fig. 14—Lobar collapse injuries.**

A, 21-year-old patient with asthma with left upper lobe collapse. Frontal radiograph shows compensatory hyperexpansion of superior segment of left lower lobe creating paraaortic crescent of hyperlucency (luschtichel sign) (asterisks).

B, 36-year-old patient with history of interstitial lung disease and new left upper lobe collapse. Frontal radiograph shows tenting of ipsilateral hemidiaphragm with visualization of inferior accessory fissure ("juxtaphrenic peak" or "Katten" sign) (arrow).

C, 51-year-old mechanically ventilated patient with history of smoking and bronchogenic carcinoma presenting with right upper and left lower lobe collapse. Frontal radiograph shows dense opacification of heart silhouette ("ivory heart" sign) and loss of concavity of left heart border ("flat waist" sign) (arrow).

D, 31-year-old patient after abdominal surgery with right lower lobe collapse. Frontal radiograph shows triangular opacity (arrow) representing traction on superior mediastinum (superior triangle sign).

E, 48-year-old patient in intensive care unit with acute bronchial obstruction from mucus plugging. Frontal radiograph shows pneumothorax ex vacuo developing around collapsed right upper lobe (arrows), which resolved after bronchoscopy.

**Fig. 15—Pulmonary parenchymal injuries.**

A, 48-year-old woman 1 hour after motor vehicle collision. Frontal chest radiograph shows diffuse bilateral opacities, suggestive of pulmonary contusions.

B, 37-year-old man 1 week after blunt chest trauma. Frontal chest radiograph shows diffuse bilateral opacities and right-sided cavitary lung lesion (asterisk), reflecting sequela of prior lung laceration.
conjunction with left-sided effusions. Other radiographic findings include pneumomediastinum, widened paraspinal lines, and retrocardiac lung opacification (Fig. 20A). CT or upper gastrointestinal studies can show oral contrast extravasation and esophageal thickening. Corrective surgery should be performed immediately because of the risks of edema, infection, and fistulization.

Hiatal hernias can form after blunt or penetrating trauma, with the stomach prolapsing through the diaphragmatic esophageal hiatus. Chest radiographs show a retrocardiac structure filled with gas and/or fluid, representing the intrathoracic stomach (Fig. 20B). No intervention is necessary unless incarceration and strangulation occur [7, 8, 13, 14].

Heart
Pericardium
Pericardial tears can result from severe blunt injury or penetrating trauma. On chest radiographs, irregular convexities of the heart border may be observed (“snow cone” sign) along with cardiac injury, pneumomediastinum, and pneumothorax. Large pleuropertical or diaphragmatic pericardial ruptures can result in cardiac herniation, with marked shift of the cardiac silhouette. This condition predisposes to cardiac volvulus with obstruction of the great vessels and requires immediate surgical repair (Fig. 21).

Organ and vascular ruptures may introduce fluid (pericardial effusion) or air (pneumopericardium) into the pericardial cavity. Pericardial effusions can contain transudative (hydropericardium), exudative (pyopericardium), lymphatic (chylopericardium), or hemorrhagic (hemopericardium) fluid. In addition, organ rupture and foreign body injury can result in pericardial inflammation and infection (pericarditis). Radiographic signs of effusion, which are very rare, include global enlargement of the cardiac silhouette (“water-bottle” sign) on frontal radiographs and wide separation of the epicardial and retrosternal fat (“epicardial fat-pad,” “Oreo cookie,” sandwich, or stripe sign) on lateral radiographs (Fig. 22). CT can aid greatly in the characterization of pericardial lesions and effusion contents.

Pneumopericardium is air located within the pericardial cavity and external to the rest of the heart. Loculation within the pericardial sac can be shown by nondependent shift on decubitus radiographs. On erect chest radiographs, a radiolucent band of air surrounding the heart (halo sign) and air in the transverse pericardial sinus (“transverse band of air” sign) can be seen. Lateral radiographs may show retrosternal hypolucency anterior to the cardiac base and aortic root (“triangle of air” sign). In tension pneumopericardium, marked compression of the heart with a decreased cardiothoracic ratio may be visible (“small heart” sign) (Fig. 23).

Because of pericardial compliance, gradually developing effusions may not cause noticeable symptoms. However, rapid accumulation...
Chest Radiography in Thoracic Polytrauma

Fig. 18—Mediastinal bleeding and infection.
A, 43-year-old man with penetrating injury to chest. Frontal chest radiograph identifies mediastinal widening (double-headed arrow), suggestive of vascular injury. CT confirmed mediastinal hematoma.
B, 28-year-old man with history of mediastinal infection. Frontal chest radiograph identifies mediastinal widening (double-headed arrow) and pulmonary edema. CT confirmed presence of mediastinitis.

Fig. 19—Tracheobronchial injuries.
A, 39-year-old man injured in motor vehicle crash. Frontal chest radiograph shows irregularity of left main bronchus (arrow) and mediastinal widening (double-headed arrow), indicative of paratracheal hematoma.
B, 21-year-old woman 1 week after tracheobronchial injury. Frontal chest radiograph shows collapse of left lung with inferolateral displacement (fallen lung sign) (asterisk).
C, 40-year-old man 4 months after tracheobronchial injury. Frontal chest radiograph shows diffuse tracheal stenosis (arrows).

Fig. 20—Esophageal injuries.
A, 31-year-old man with Boerhaave’s syndrome. Frontal chest radiograph shows bilateral pneumomediastinum (arrows).
B, 34-year-old woman with hiatal hernia. Frontal chest radiograph shows large retrocardiac opacity (arrows).
of even small amounts of fluid or air can produce cardiac tamponade, in which increased pericardial pressure causes significant hemodynamic compromise. On CT, distention of the venae cavae and hepatic and renal veins is seen, indicative of severe cardiac congestion. Immediate pericardiocentesis is indicated for recovery of normal cardiovascular function [7, 8, 15].

Cardiac Trauma

Myocardial contusions are caused by rupture of intramyocardial vessels after severe cardiac trauma. On chest radiographs, chest wall hematomas and cardiomegaly due to hemopericardium may be seen. Myocardial stunning may lead to congestive heart failure, with pulmonary edema visualized on radiographs. Associated findings include skeletal fractures and pulmonary contusions.

Cardiac aneurysms, which are focal outpouchings in the septal or free walls of the cardiac chambers, can result from severe blunt trauma. They are most commonly seen in the left ventricular anterior wall or apex. True aneurysms can be managed conservatively, but should be monitored carefully because of the increased risk of rupture. Cardiac pseudoaneurysms, which form when wall ruptures are contained by epicardial hematoma and pericardial tissue, are typically sequelae of penetrating trauma. They are usually located in the posterolateral wall of the left ventricle. Immediate surgical repair is necessary to prevent complete rupture (Fig. 24A).

Complete cardiac rupture can be seen in severe blunt and penetrating trauma. The right ventricle is often involved because of its thinner walls and anterior location in the chest. The left ventricle, right atrium, and left atrium are less frequently affected. Ruptures of the free wall and fistulization to adjacent organs can produce hemopericardium and pericarditis. Ruptures of the interventricular septum, papillary muscles, and valves also may occur (Fig. 24B). Severe torsional stresses can produce cardiac avulsion, with separation of the heart from the great vessels. Chest radiography reveals an enlarged or irregular heart shadow, often in conjunction with pulmonary edema and pleural effusions. Immediate surgical correction is indicated.

Myocardial infarction is seen with increased frequency in trauma patients because of coronary artery injury and occlusion. Chest radiography can reveal complications such as pulmonary edema secondary to heart
failure. Thrombolysis, percutaneous coronary intervention, or coronary artery bypass grafting may be required in severe cases. Chronic sequelae include myocardial thinning, fibrosis, and calcification (Fig. 24C). There is an increased risk of cardiac aneurysm and pseudoaneurysm formation with subsequent rupture [7, 8, 15].

**Aorta**

Traumatic aortic injury (TAI) refers to a spectrum of injuries caused by blunt aortic trauma, which produces differential deceleration of thoracic structures with associated solid and fluid mechanical effects. The aortic isthmus is most frequently involved, followed by the aortic root and diaphragmatic aorta. Forces affecting the aortic isthmus include shearing stress, in which the freely movable aortic arch separates from the fixed descending aorta; bending stress, with flexion of the aorta over the left pulmonary artery and mainstem bronchus; and osseous pinch, involving compression of the aorta between the spine and anterior bony structures. In the ascending aorta, torsion stress occurs at the level of the aortic valve because of cardiac displacement, and the water-hammer effect is produced by abrupt increases in intravascular pressure with possible pericardial rupture and cardiac tamponade. Possible injuries include aortic tearing or laceration, in which sections of the aorta are forcibly pulled apart; transection or transverse circumferential division of the aorta; and rupture, with massive disruption of tissue. Any or all layers of the arterial wall may be affected, with resultant hematoma formation in a variety of locations. Survival in complete ruptures requires pseudoaneurysm formation with containment of active bleeding by adventitia, thrombus, or mediastinal structures. Immediate open surgical repair or endovascular stent-grafting is advised. Indirect radiographic signs of TAI include mediastinal widening, irregularity or obscuration of the aortic contour, opacification of the aortopulmonary window, depression of the left mainstem bronchus, rightward tracheal and esophageal deviation, widened paratracheal and paraspinal stripes, and hemotorax or left apical capping (Figs. 25A and 25B).

Traumatic aortic dissection is characterized by an intimomedial tear, which allows bleeding into the medial wall layer and formation of a false lumen. Chest radiography is nonspecific and may show an irregular aortic silhouette, discontinuous calcification of the aortic knob (“broken halo” sign), or intraluminal displacement of a calcified aortic intima (ring sign) (Fig. 25C). Type B (descending aortic) dissections can be managed conservatively, whereas type A (ascending aortic) dissections require immediate surgery because of the risks of pericardial bleeding, coronary artery laceration, and aortic valve rupture.

Traumatic aortic aneurysms represent localized dilatation of the aorta involving all three arterial wall layers and are susceptible to rupture. An enlarged and irregular aortic silhouette is seen on chest radiographs (Fig. 25D). Open surgery is recommended for ascending aortic aneurysms that are symptomatic, rapidly expanding, or greater than 5.0–5.5 cm in diameter. Descending aortic

![](A.png)
aneurysms exceeding 6.0 cm can usually be repaired by endovascular stent-grafting.

Penetrating aortic injuries depend on the mechanism of trauma and thus vary widely in size and location. Vessel laceration, truncation, or arteriovenous fistulization may result. Most survivors show a small pseudoaneurysm at the site of vessel injury. Irregular aortic contours and luminal narrowing may also be seen [7, 8, 16–21].

Vascular Trauma

Great Vessels

More than 90% of injuries to the great vessels are caused by penetrating trauma. The aortic branch vessels, venae cavae, and pulmonary veins are also susceptible to blunt injury via mechanisms similar to those of TAI. Formation of local hematomas and hemopericardium are noted complications (Fig. 26). If bleeding cannot be controlled, surgical intervention is indicated to maintain the integrity of the cardiovascular circulation [7, 8, 16–20].

Pulmonary Arteries

In trauma patients, hypercoagulability and immobilization predispose to deep venous thromboses, which can circulate to the pulmonary arteries and produce pulmonary embolism (PE). This results in inflammation, hypoxemia, hemodynamic compromise with right heart strain (cor pulmonale), and pulmonary infarction with regional loss of surfactant. Chest radiography findings are largely nonspecific and include cardiomegaly, atelectasis, pulmonary edema, pleural effusion, and hemidiaphragmatic elevation. Classic imaging signs include regional oligemia (Westermark sign), central pulmonary artery enlargement (Fleischner sign), right descending pulmonary artery enlargement (“Palla” sign), and abrupt pulmonary artery tapering (“knuckle” sign). In the presence of acute infarction, focal subpleural opacities (Hampton
hump) may be seen, whereas linear fibrosis (Fleischner lines) and centripetal infarct resolution (“melting ice cube” sign) occur in later stages (Figs. 27A and 27B). More definitive tests for PE include nuclear ventilation–perfusion (V/Q) scintigraphy, CT angiography (CTA), and pulmonary angiography. Nevertheless, radiographs are still routinely used to screen for other sources of chest pain and to aid in the proper interpretation of V/Q scans. Immediate anticoagulation therapy is recommended for suspected PE.

Septic embolism occurs when infected material from organ rupture or foreign body injury travels to the lungs. Chest radiographs show diffuse bilateral nodules of various sizes and stages of cavitation, reflecting multiple embolic showers. Over time, lesions can progress to wedge-shaped peripheral opacities (Fig. 27C). Treatment requires antibiotic therapy and possible thoracentesis.

Air embolism is caused by organ rupture or penetrating injury affecting the systemic venous circulation. It also can be caused by barotrauma. Mortality depends on the amount and rate of gas entry. Chest radiographs may show hyperlucent areas in the right heart, pulmonary arteries, and systemic veins. Signs of pulmonary oligemia, edema, or right heart congestion may also be seen.

Fat embolism results from trauma to the long bones and pelvis, which can release fat particles and occlude capillaries. Production of free fatty acids causes a chemical pneumonitis within 12–72 hours of injury. Radiologic manifestations are similar to those of ARDS—that is, diffuse parenchymal opacities (Fig. 27D). Management is supportive, and the condition takes 7–10 days to resolve.

Pregnancy is a known risk factor for thromboembolic disease. The risk of radiation exposure to the fetus should be weighed against the clinical suspicion for PE. Affected patients should be treated with heparin because of the teratogenic effects of warfarin. In addition,
there is a risk of amniotic fluid embolism (AFE), in which amniotic fluid enters the uterine veins during labor or placental manipulation. Radiographically, this condition presents with diffuse bilateral opacities indistinguishable from PE, hemorrhage, and pneumonia (Fig. 27E). The prognosis is poor, and management is supportive. Immediate cesarean delivery should be performed in patients with cardiac arrest who are unresponsive to resuscitation.

Foreign body embolism can occur with fragmentation of foreign bodies. Material may travel through the arterial or venous circulations and become lodged in distal sites (Fig. 27F). Mortality depends on the location, duration, and severity of emboli. Cardiopulmonary injuries are common, and other risks include perforation, thrombosis, and infection [7, 8, 19, 21–23].

Conclusion

Chest radiography plays an important role in the initial evaluation of blunt and penetrating chest trauma, providing rapid imaging information to supplement the history and physical examination. In the emergency department, familiarity with the spectrum of injuries that can occur in the chest and upper abdomen is important for accurate interpretation of chest radiographs as well as establishment of appropriate recommendations for management and follow-up. An understanding of trauma pathophysiology and related imaging findings for injuries to the chest wall, diaphragm, pleura, lungs, mediastinum, heart, aorta, and great vessels will enable radiologists to interact rapidly and effectively with the other members of the health care team.

Acknowledgments

We thank D. Claire Anderson, Sanjeev Bhalla, Andrew Bierhals, David Gierada, Harvey Glazer, Guillermo Geisse, Cylen Javidan-Nejad, Gilbert Jost, Anosh Montaser, Stuart Sagel, Janice Semenkovich, Marilyn Siegel, and Pamela Woodard for contributing many of the cases featured in this article.

References