



ELSEVIER

Seminars in

ROENTGENOLOGY

# The ABC's of Thoracic Trauma Imaging

Mary Costantino, MD, Marc V. Gosselin, MD, and Steven L. Primack, MD

The approach to thoracic trauma will be divided into two major categories in this article: parenchymal and extraparenchymal injuries. The radiographic and CT appearance of pulmonary edema, aspiration, atelectasis, and contusion will be reviewed. The extraparenchymal injuries will revolve around the ABC approach outlined by Dr Jud Gurney.<sup>1</sup> Review of common presentations as well as evolving diagnostic and pathological etiologies will be discussed in the article.

The morbidity and mortality of trauma in the United States is substantial. Trauma is the third leading cause of death overall and most frequent cause for those less than 35 years of age. Thoracic injuries account for 25 to 35% of trauma-related deaths.<sup>2</sup> Mortality from blunt chest trauma is usually due to aortic or great vessel injury. While great vessel injuries account for much of the mortality, they occur in only 1% of victims suffering blunt chest trauma. Pulmonary parenchymal injuries such as contusion and laceration are more common, occurring in 30 to 70% of blunt thoracic trauma victims.

Currently, the supine chest radiograph is obtained as a screening examination for the vast majority of trauma patients and remains an excellent tool for early detection of life-threatening thoracic injuries. In the emergency setting, a "quick read" assists the primary team in patient triage. Though chest CT scans along with CT scans of the abdomen and pelvis are obtained with increased frequency in blunt trauma patients, radiographs are not obsolete and maintain an important role in early diagnosis and patient triage.

The evaluation of the radiographs will be divided in this article by common parenchymal and extraparenchymal injuries. Parenchymal injuries include aspiration, atelectasis, contusion, and edema. In the evaluation of extraparenchymal injuries we describe an algorithm set forth by Dr Jud Gurney ([www.chestx-ray.com](http://www.chestx-ray.com)) modeled after the ABC's of clinical management, which addresses the mechanical disruption of thoracic anatomy and its physiologic consequences.

## Pulmonary Parenchymal Injuries

### Pulmonary Contusion, Laceration, and Traumatic Lung Cyst

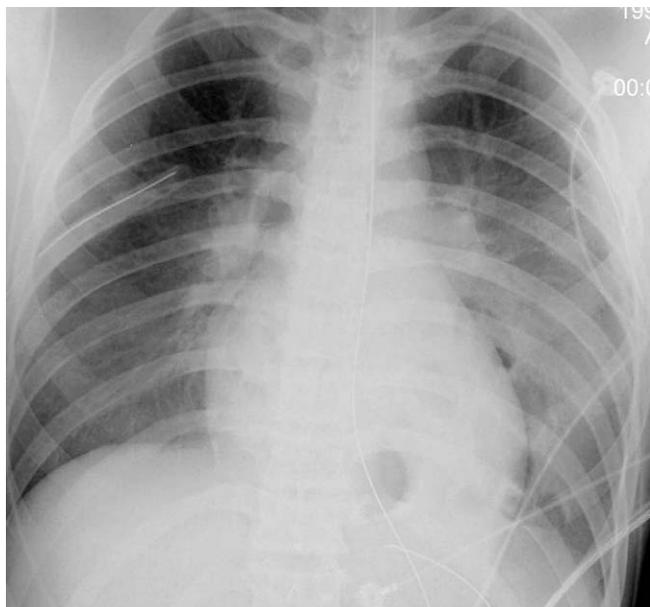
Pulmonary parenchymal contusion is the most common cause of pulmonary opacification in blunt chest trauma, occurring in 30 to 75% of patients. The mortality rate for lung contusion varies from 14 to 40%, varying by severity and extent of disease. The causative force of a pulmonary contusion is a direct blow immediately adjacent to normal lung or a contre-coup injury within the pulmonary parenchyma. Clinical symptoms include hemoptysis from transudation of blood within the alveoli and airways, mild fever, tachypnea, hypoxia, bronchorrhea, decreased cardiac output, and acute respiratory failure. Up to 50% of patients with lung contusion present without symptoms.

A contusion represents a combination of parenchymal edema and hemorrhage secondary to disruption of the alveolar capillary membrane. A variety of traumatic forces may increase the alveolar capillary membrane permeability. In the case of sudden deceleration found in motor vehicle accidents, low-density alveoli are sheared from the adjacent, higher density bronchovascular bundles, not dissimilar from diffuse axonal injury in the brain. Traumatic forces may directly disrupt pulmonary alveoli as in the case of a fractured rib lacerating pulmonary parenchyma. Spallation and implosive forces may cause contusion, laceration, and traumatic lung cysts. Spallation occurs when transmitted kinetic energy from a direct blow deposits energy locally at a liquid-gas interface such as the air-blood relationship in pulmonary alveoli.<sup>3</sup> Implosion force expands the gas component within alveoli with the passage of low-pressure shock waves.

Radiographically pulmonary contusion presents as focal or multifocal areas of confluent ground glass opacity or consolidation. Contusions are not limited by segmental boundaries and are usually in the lung periphery adjacent to site of direct trauma. There are frequently associated injuries such as rib or spine fractures. Pulmonary contusions generally affect the lung bases due to increased basilar mobility. Air bronchograms are usually absent secondary to blood filling the small airways. Contusions may not be radiographically apparent on the initial chest radiograph, but develop within 6 hours of the initial injury. They demonstrate maximum radiographic conspicuity within 24 to 72 hours and gradually

Oregon Health and Science University, Department of Radiology, Portland, OR.

Address reprint requests to Marc V. Gosselin, MD, Oregon Health and Science University, Department of Radiology, Mail Code L340, 3181 Sam Jackson Park Road, Portland, OR 97201. E-mail: [gosselin@OHSU.edu](mailto:gosselin@OHSU.edu)



**Figure 1** A 25-year-old male in a motor vehicle accident (MVA). Bilateral lower lobe consolidation, left much greater than right. Note the lucent cysts in the left lower lobe, characteristic for traumatic lung cysts (type 1).

clear over 3 to 10 days. Pulmonary opacities, which do not clear or increase radiographically over such a timeframe, raise the suspicion of developing secondary infection or Acute respiratory distress syndrome (ARDS).

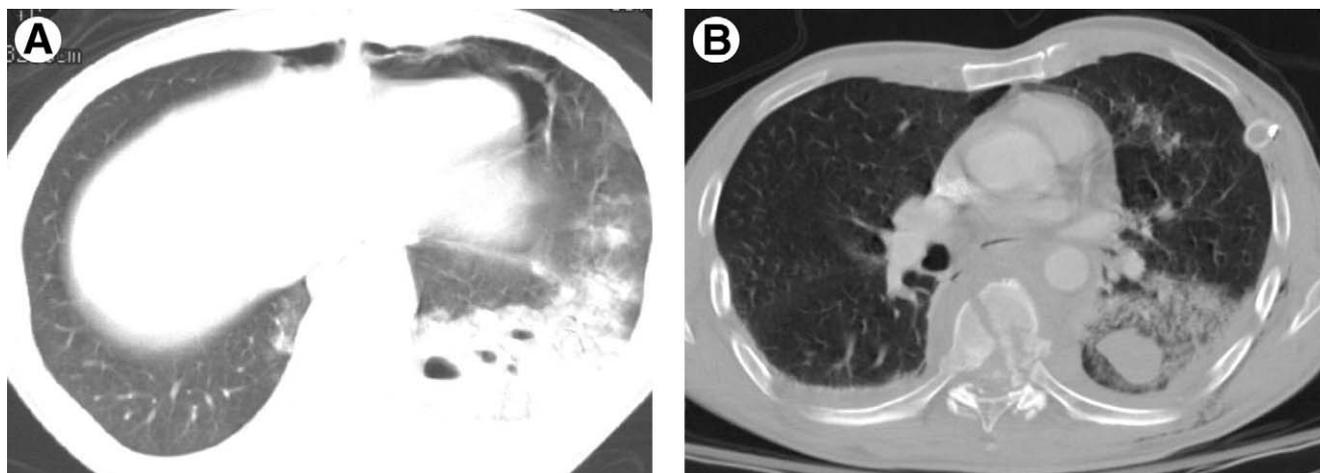
The detection of a contusion is significantly better on CT scans compared with conventional radiographs. In one experimental model, CT detected pulmonary contusion in 100% of cases compared with 37.5% by chest radiographs, and CT more accurately revealed the size and extent of injury when correlating with pathological specimens.<sup>4</sup> In trauma patients with unexplained respiratory symptoms and unremarkable radiographs, CT can depict underlying pathology that may be useful in altering clinical care such as more

aggressive pulmonary toilet and oxygen therapy. However, in one clinical series of 73 blunt chest trauma victims, only pulmonary contusions visible by plain film radiographs were clinically significant.<sup>3</sup>

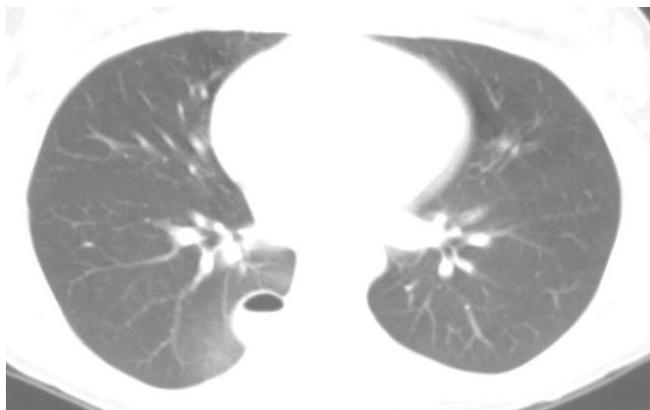
Pulmonary laceration represents a tear of lung parenchyma secondary to shearing stress. Clinically, pulmonary laceration may manifest as hemoptysis. Radiographically pulmonary lacerations are ovoid radiolucencies secondary to the elastic recoil properties of the surrounding lung parenchyma (Fig. 1). However, lacerations are frequently obscured on initial radiographs secondary to adjacent parenchymal contusion. While lacerations are often obscured on radiographs, CT scans clearly demonstrate lacerations as localized air collections within areas of contusion.

With the clearing of adjacent lung contusion pulmonary lacerations appear as thin-walled cysts that fill with air and/or blood secondary to involvement of the adjacent bronchial tree and pulmonary vasculature, respectively (Fig. 2). In the presence of both air and blood within the cyst air fluid levels can be seen. When it becomes completely opacified with blood, the traumatic lung cyst is termed a pulmonary hematoma. Lacerations usually spontaneously resolve over 3 weeks to a year. Although uncommon, they may persist indefinitely as a pulmonary nodular opacity due to retained blood products within the cystic space. Alternatively a traumatic laceration may manifest as a pulmonary cyst years later if all the blood products are expectorated. Traumatic lacerations are occasionally complicated by persistent bronchopleural fistula or infection within the cyst requiring surgical treatment.

Pulmonary lacerations and traumatic lung cysts are classified into four categories based on type of traumatic force and subsequent characteristic CT findings. Type I lacerations result from compression injury producing an air-filled or air-fluid-filled cyst within contused lung parenchyma. Type II lacerations demonstrate cysts within the paravertebral lung secondary to a lateral compression shear force when lung parenchyma presses against a vertebral body (Fig. 3). Frac-



**Figure 2** (A) Left lower lobe consolidation with multiple oval cysts and fluid levels. The presence of these type 1 traumatic lung cysts confirms the consolidation is a contusion. (B) Ground glass opacity and nodular opacity in left lower lobe, 1 day after a severe trauma. This was a contusion with a concurrent hematoma.



**Figure 3** Type 2 traumatic lung cyst in the right lower lobe along the thoracic spine in a 16-year-old girl 2 weeks after a “mild” MVA. She presented with hemoptysis.

tured ribs that penetrate the lung parenchyma generate a type III laceration manifested by a peripheral parenchymal linear lucency or cavity adjacent to the site of osseous trauma (Fig. 4). Pleuropulmonary adhesions (near the inferior pulmonary ligament) impart a tearing force on the lung parenchyma with blunt trauma and produce type IV lacerations that can be diagnosed only pathologically.<sup>5</sup>

Multiple types of lacerations can be seen in the same patient simultaneously. Interestingly, in one series the vast majority of compression injury lacerations (Type I and II) were seen in patients 39 years or younger presumably owing to the greater degree of chest wall pliability and transmitted force to the pulmonary parenchyma due to the laxity of the costovertebral ligaments in younger individuals.<sup>6</sup>

### Aspiration Pneumonitis

Distinguishing pulmonary injury due to aspiration or contusion in the trauma patient influences patient care given the dissimilar management of the two entities. Aspiration demonstrates a morphology and distribution of disease often distinctly different compared with contusion, particularly on CT. Whereas contusion presents as ill-defined areas of confluent ground glass opacity or consolidation not respecting segmental or lobar boundaries with possible associated traumatic lung cysts, aspiration produces “tree-in-bud” poorly defined centrilobular opacities usually in the dependent portions of the lung (Fig. 5).

The CT “tree-in-bud” appearance of aspiration reflects aspirated material filling the distal small airways, whereas the confluent ground glass opacity of contusion represents injury to the localized parenchymal area. The superior and posterior basilar segments of the lower lobes and the posterior segments of the upper lobes are most frequently involved in aspiration. Within a few hours, these “tree-in-bud” opacities become more confluent, resulting in a consolidative appearance. However, along the periphery, some isolated filled terminal bronchioles often persist, indicating the underlying pulmonary complication.

### Atelectasis

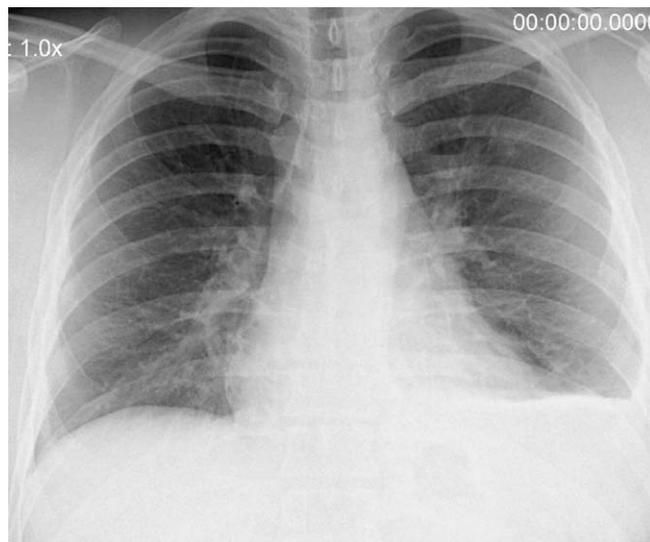
Lung collapse in the trauma patient can be separated broadly into the categories of obstructive, passive, compressive, and adhesive atelectasis. Obstructive atelectasis occurs due to mucus plug, foreign body (Fig. 6), endobronchial blood, traumatic bronchorrhea, or airway rupture. Presumably, separation of the parietal and visceral pleura by a hemothorax or pneumothorax disrupts the inherent elastic recoil properties of the lung producing passive atelectasis. Large traumatic lung cysts or contusions may cause compressive atelectasis from intrapulmonary mass effect. Pulmonary contusion causing decreased surfactant production creates adhesive atelectasis. Air bronchograms are often seen in all forms of atelectasis *except from a central bronchial obstructive*.

Atelectasis can usually be distinguished from contusion and aspiration given the denser, homogenous appearance and simultaneous fissure deviation. Atelectatic lung also demonstrates greater enhancement than consolidative lung, given the strong blush of contrast in the crowded pulmonary vasculature. Secondary imaging signs of volume loss such as shift of mediastinal and hilum structures, bronchovascular crowding, and diaphragmatic elevation may also be present.

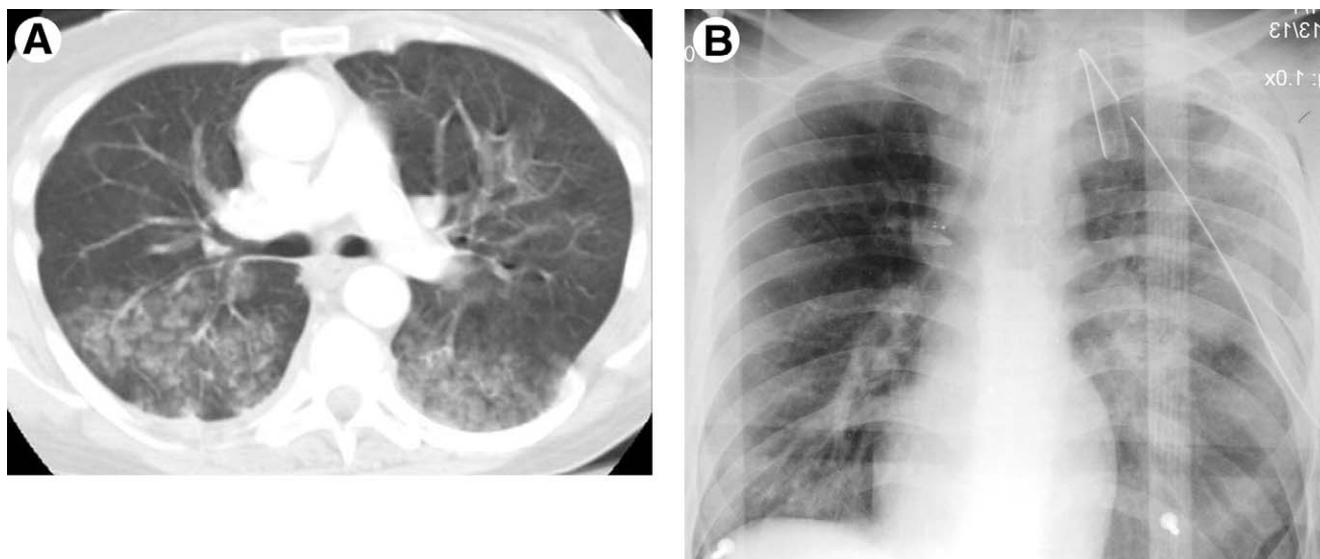
Atelectasis often causes hypoxia, which is at least partially corrected with increased O<sub>2</sub> delivery. Pulmonary blood flows through unventilated lung, thus mixing with the normally oxygenated blood. Although it has historically been associated with fever, there are no definite physiological mechanisms or studies to support this claim. On the contrary, studies have found no relationship of atelectasis and fever.<sup>7</sup> Therefore, the clinician should seek another potential source for the fever.

### Noncardiogenic Pulmonary Edema

Early, immediate pulmonary edema in the trauma patient often reflects neurological injury, airway obstruction, or toxic



**Figure 4** Type 3 traumatic lung cyst with a fluid level in left upper lobe anterior segment. It lays adjacent to a displaced anterior left 2nd rib fracture, which is not well seen on the radiograph.



**Figure 5** (A) Bilateral dependent traumatic aspiration pneumonitis. The distribution and “tree-in-bud” appearance are characteristic. (B) Radiograph demonstrating bilateral lower lobe opacification. The presence of an incisor tooth in the proximal right upper lobe bronchus supports at least some aspiration.

inhalation of gas. Pulmonary edema within the first 24 hours usually is due to overhydration or transfusion reaction, while pulmonary edema beyond 24 hours of presentation usually represents developing ARDS, fat embolism syndrome, or overhydration.

Traumatic causes of cardiogenic edema such as overhydration with underlying poor cardiac output or cardiac contusion versus traumatically induced noncardiogenic edema from fat embolism syndrome, neurogenic injury, or airway obstruction often appear radiographically distinct. Cardiogenic edema produces widening of the vascular pedicle, central and lower lobe pulmonary vascular haziness, and interlobular septal thickening. Noncardiogenic edema often demonstrates more diffuse vascular indistinctness/ground glass opacity without septal thickening or widened vascular pedicle. Although usually distinguishable on radiographs, the imaging differences of cardiogenic and noncardiogenic edema are more apparent on CT scans.

Though the presence of fat emboli to distant organs after significant skeletal fracture occurs in over 90% of cases, development of fat embolism syndrome is quite rare, at an incidence of 1 to 2% with isolated long bone shaft fractures and 5 to 10% with multiple fractures.

Clinical symptoms related to distal embolization and hypoxia include dyspnea; tachypnea; cyanosis; mental status changes; oliguria; fever; tachycardia; petechiae of the neck, axilla, and conjunctiva; and retinal petechial hemorrhage. Although symptoms can occur within a few hours, symptoms usually occur 1 to 4 days from the time of trauma. Treatment includes oxygen therapy, heparin, and intravenous steroids. Untreated, fat embolism syndrome is often fatal particularly in the patient with fulminant disease affecting the central nervous system.

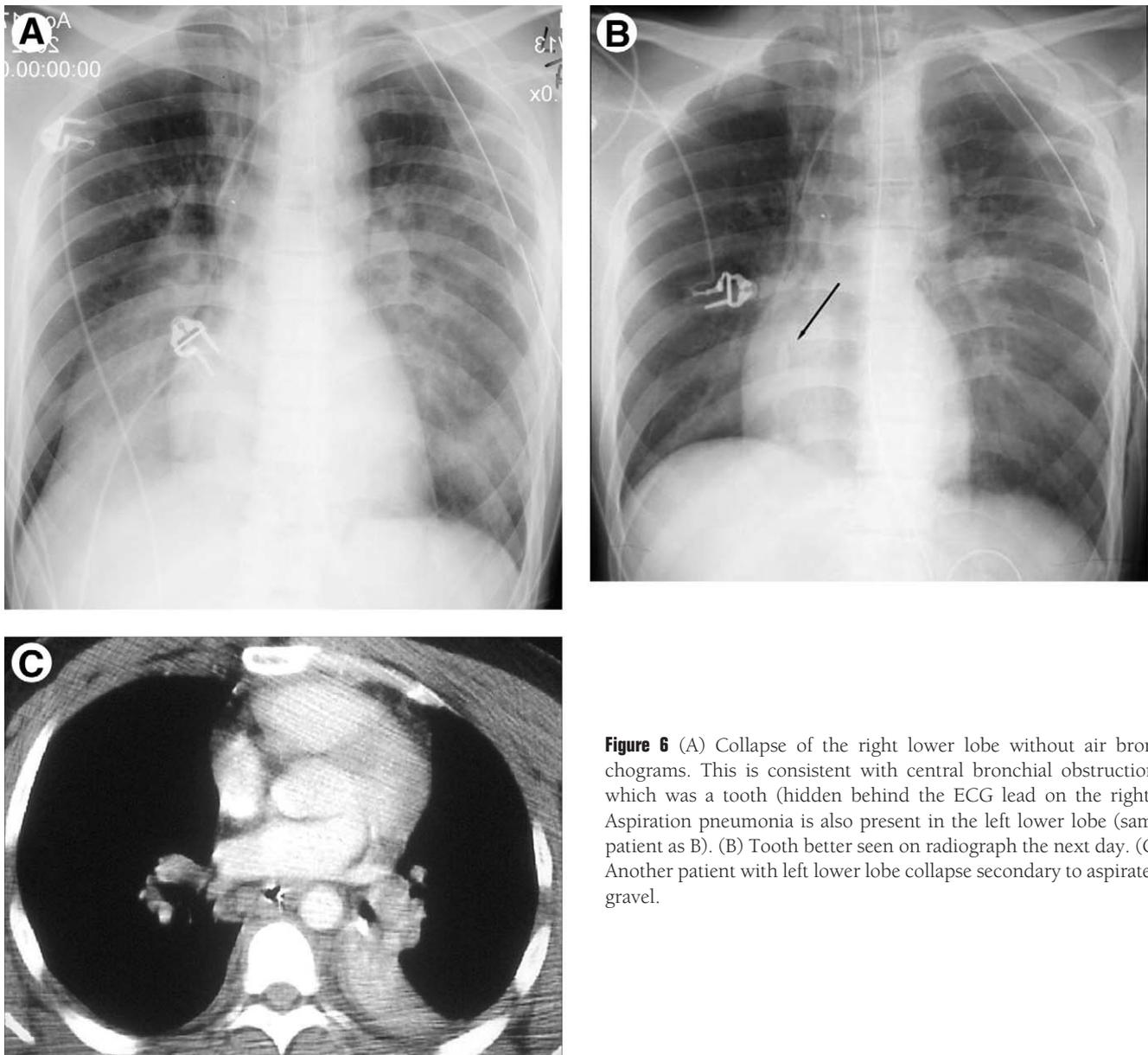
Fat emboli incite lung parenchymal damage and second-

ary noncardiogenic edema via three mechanisms. Fat particles directly occlude the pulmonary capillaries leading to increased pulmonary vascular resistance, pulmonary arterial hypertension, and secondary local edema. Pulmonary endothelial lipase hydrolyzes the neutral fat globules into free fatty acids, which usually takes between 24 and 72 hours after the initial embolization. The free fatty acids elicit a direct chemical injury to the vascular endothelium, thus disrupting the capillary membrane. Injury-induced local release of serotonin, histamine, and other vasoactive peptides likely contributes to the localized edema and vascular disruption.

Radiographically the characteristic noncardiogenic pulmonary edema and parenchymal damage of fat emboli syndrome appears as ill-defined 5- to 10-mm nodular opacities predominantly affecting the lung periphery and bases, likely secondary to the small size of the fat emboli and their hematogenous spread (Fig. 7). Cardiac enlargement, widening of the vascular pedicle, and interlobular septal thickening indicative of cardiogenic pulmonary edema are lacking.

Neurogenic pulmonary edema can result from any CNS abnormality such as trauma, vascular infarction, or brain tumor that raises intracranial pressure. Radiographically, neurogenic pulmonary edema may initially involve the upper and peripheral lung zones before evolving into a diffuse distribution. The characteristic secondary signs of cardiogenic pulmonary edema are generally absent (Fig. 8).

Increased intracranial pressure leading to overactivation of the sympathetic nervous system has been theorized to induce an acute elevation of pulmonary venous pressure, thus inverting the normal distribution of pulmonary blood flow with more blood going to the upper lobes. This re-



**Figure 6** (A) Collapse of the right lower lobe without air bronchograms. This is consistent with central bronchial obstruction, which was a tooth (hidden behind the ECG lead on the right). Aspiration pneumonia is also present in the left lower lobe (same patient as B). (B) Tooth better seen on radiograph the next day. (C) Another patient with left lower lobe collapse secondary to aspirated gravel.

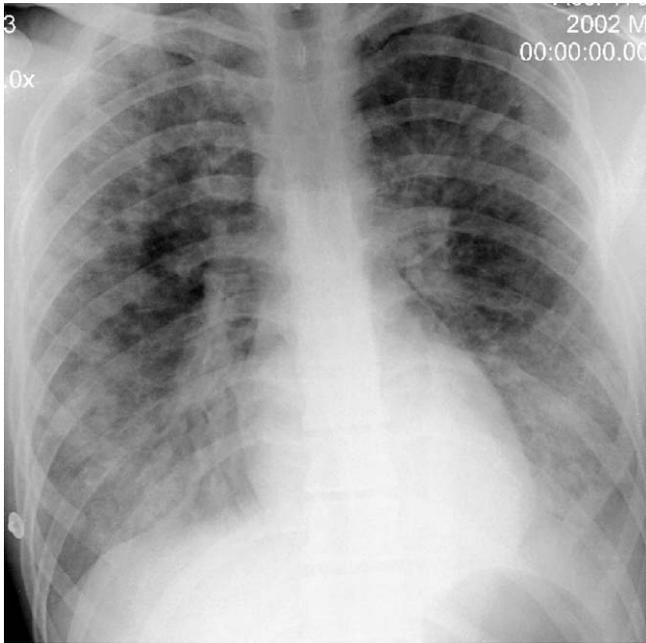
distribution of flow may transport chemical mediators, such as bradykinin and histamine, to the upper lobes, causing disruption of the capillary endothelium.

Acute airway obstruction from etiologies such as tracheal-laryngeal edema accounts for rare causes of noncardiogenic pulmonary edema. As in neurogenic pulmonary edema, the upper lung zones are often predominantly affected initially. Though poorly understood, hypotheses concerning the distribution of airway obstruction induced pulmonary edema exist. In most cases patients with airway obstruction assume an upright position to maximize ventilation, which predominantly decreases upper lung zone intrapleural pressures. Markedly decreased intrapleural pressures experimentally disrupt the pulmonary capillary membrane with subsequent development of protein-rich pulmonary edema.

## Extraparenchymal Thoracic Injuries

To address the extraparenchymal evaluation of the chest radiograph in the trauma patient, a systematic approach presented by Dr Gurney on [chestxray.com](http://chestxray.com) will be outlined, which is based on the ABC's taught with the clinical management. This approach may assist the radiologist to search for the other various thoracic injuries:

- A: Aortic injury
- B: Bronchial Tree
- C: Cord
- D: Diaphragm
- E: Esophagus
- F: Fractures



**Figure 7** Fat emboli syndrome in a 28-year-old male. The diffuse ground glass and consolidation bilaterally, more confluent in the lower lobes and slightly more ill-defined nodular opacities in the upper lobes. The patient is 48 hours posttrauma with multiple long bone and pelvic fractures.

G: Gas  
H: Heart  
I: Iatrogenic

### Aorta (Traumatic Aortic Injury)

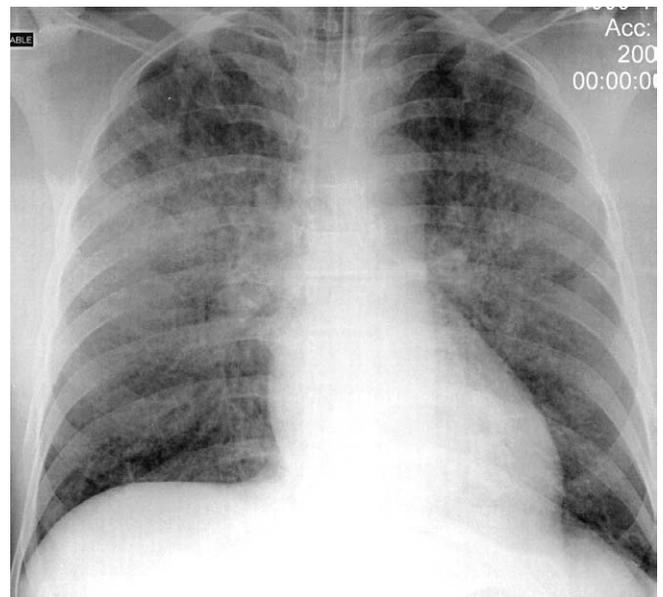
Traumatic aortic injury includes a spectrum of injury, from a small hematoma due to disruption of the vaso vasorum to a complete transection of the aorta with rapid exsanguination. Few patients with aortic transection are alive on arrival to the hospital, and on arrival, those in shock or with BP <90 have nearly a 100% early mortality. In a study of 104 patients with aortic transection, death occurred within 1 hour in 94% and within 24 hours in 99%<sup>8</sup>; the need for rapid diagnosis is clear. In diagnosing aortic transection, mechanism of injury remains the most important factor: falls from >10 feet, motor vehicle crashes at speeds >30 mph, unrestrained drivers, ejected passengers, and pedestrians struck by motor vehicles are all considered highly suspicious mechanisms.

The presence of a widened mediastinum (>8 cm) on supine portable chest radiograph may be very misleading in the trauma setting. Originally described in 1976, radiographic technique, including an increase in patient-to-tube distance (and therefore magnification), has changed substantially. Cassettes are not placed directly under the patient, but rather under the trauma board or in the trauma table. Mediastinal widening has been shown to be sensitive to hematoma and/or pseudoaneurysm formation, but it has a very poor specificity. This can be explained several ways: A portable radiograph obtained in the midst of resuscitation infrequently reflects a true AP projection. More commonly, the radiograph is ob-

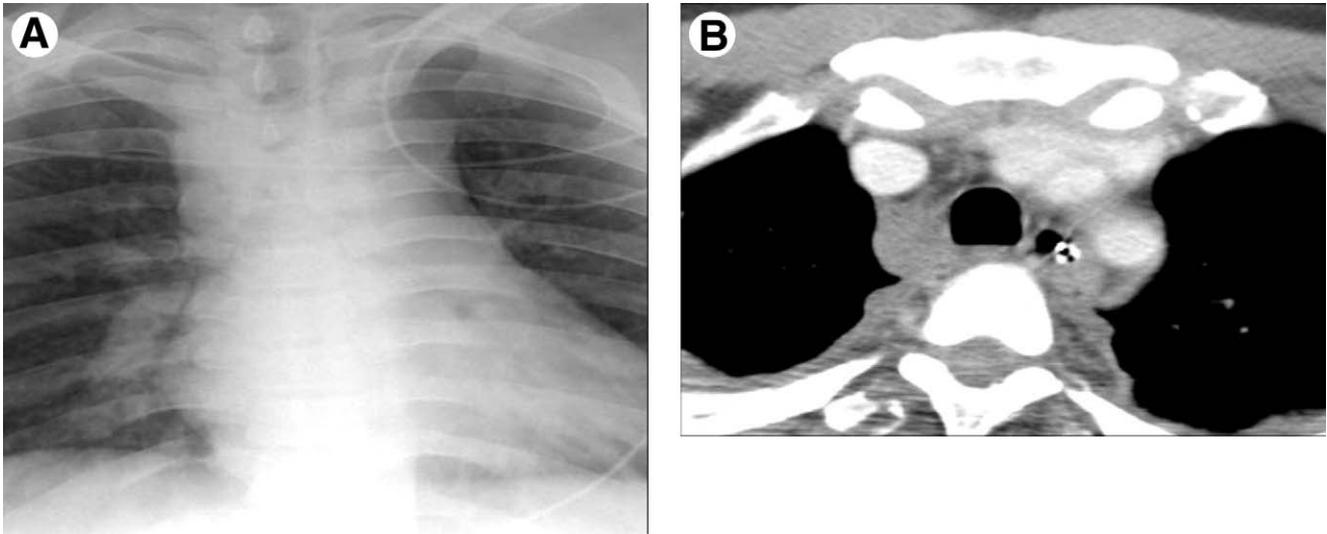
tained with a caudal-to-cephal angulation with the patient often rotated, thus splaying out the mediastinum. There is a variable tube-to-patient distance, magnifying the heart and mediastinum on the film. Often, there is no time and frequently inadequate patient compliance to breathing instructions. A radiograph not obtained in full inspiration results in a perceptual change in the mediastinum, making it appear shorter and wider. Therefore, a wide mediastinum is perhaps too sensitive of a sign.

A mediastinal hematoma may be secondary to venous hemorrhage, thoracic vertebral body fracture, or spinal ligamentous injuries. Although the presence of a hematoma necessitates further investigation, in a review of 149 cases of mediastinal hematoma, the aortic adventitia was shown to be intact in 60% of cases.<sup>9</sup> It should be remembered that about 7% of patients with a great vessel injury who survive the transportation to the hospital have a normal mediastinal appearance on the radiograph. Thus, if there is a high clinical index of suspicion for an aortic injury based on mechanism, then it may be prudent to further evaluate with CTA, regardless of the radiograph. A repeat radiograph after primary clinical survey with care given to tube positioning and respiratory cycle may be helpful, but intimal injuries or a small contained rupture are not generally seen.

Perhaps a more useful and specific radiographic sign is to closely evaluate for the presence of a hematoma, either free or contained. One may use the right paratracheal density (Fig. 9). The left paratracheal space is dense due to the left-sided thoracic aortic arch. On a normal radiograph the right paratracheal space is relatively lucent compared with the arch. This is easily explained by the vertical orientation of the superior vena cava (SVC) versus the more horizontal



**Figure 8** A 32-year-old with neurogenic pulmonary edema. There is ground glass and some early bilateral consolidation with a slightly mid to upper lung distribution. The patient had extensive cerebral edema from diffuse axonal injury.

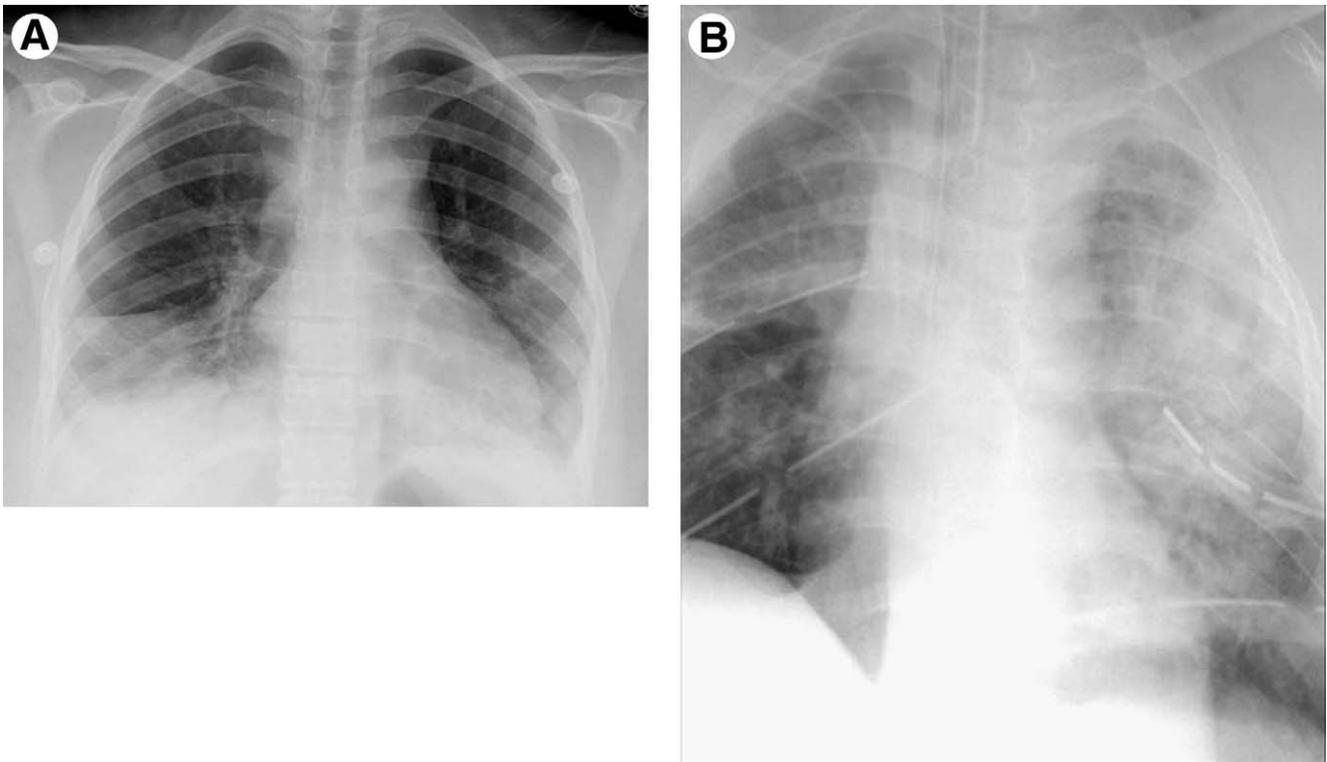


**Figure 9** (A) A 25-year-old male in an MVA with a dense right paratracheal mediastinum (equal to more dense than aortic arch). (B) CT scan demonstrates blood in the mediastinum extending to the right side. This is an example of flowing blood from a partially contained aortic isthmus transection (not shown).

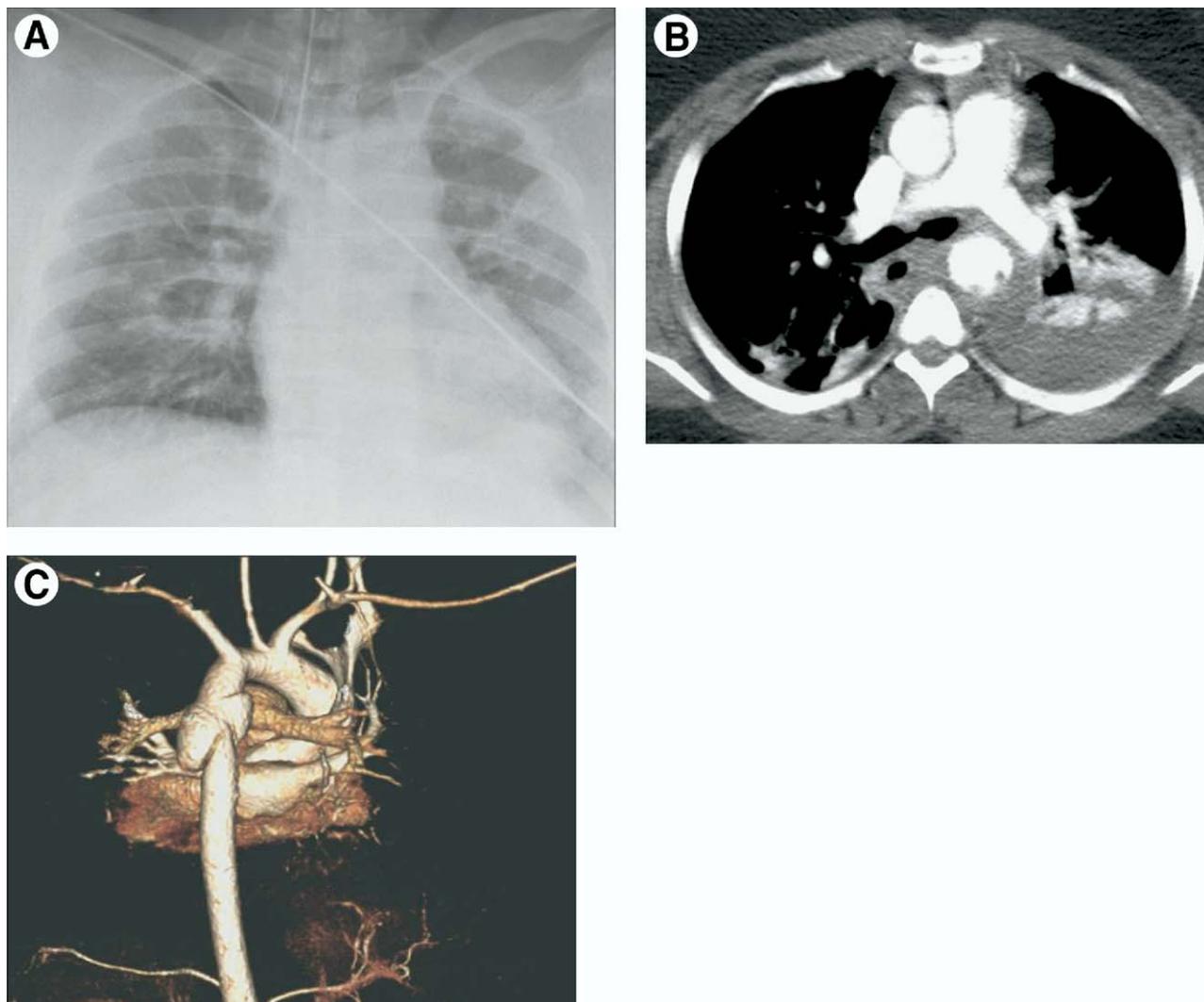
aortic arch. When the density of the right paratracheal space equals or is greater in density than the left paratracheal aortic arch, a partial flowing mediastinal hematoma should be a primary consideration (Fig. 10). A contained aortic rupture will manifest as mass effect and focal in-

creased density around an ill-defined arch. Apparent widening of the descending thoracic aorta from the spine is also strongly suggestive of mural blood extending caudally (Fig. 11).

A fundamental shift in our radiographic approach to the



**Figure 10** (A) “Widened” mediastinum in an 18-year-old patient hit by a car. However, the right paratracheal mediastinum is less dense than the aortic arch; azygos vein and aortic arch are well seen. The CT scan was negative for blood or great vessel injury. (B) A 23-year-old male in a MVA. Large left contusion and multiple left anterior lateral rib fractures. The mediastinum is uniformly dense with a left apical cap. The patient was found to have a large mediastinal hematoma and aortic transection.



**Figure 11** (A) A 36-year-old female in an MVA with mass effect on the trachea and NG tube, which are deviated to the right (the patient is mildly rotated to the left). The arch is not seen and the descending aorta is widened and dense. (B) CT scan demonstrates the aortic transection, mural blood, and mass effect on the trachea. (C) Reformatted 3D images show the complexity of the pseudoaneurysm. (Color version of figure is available online.)

mediastinum should reflect a search for a hematoma, rather than the conventional teaching of a “wide” mediastinum. Indirect signs of aortic injury on a chest radiograph may be grouped as follows:

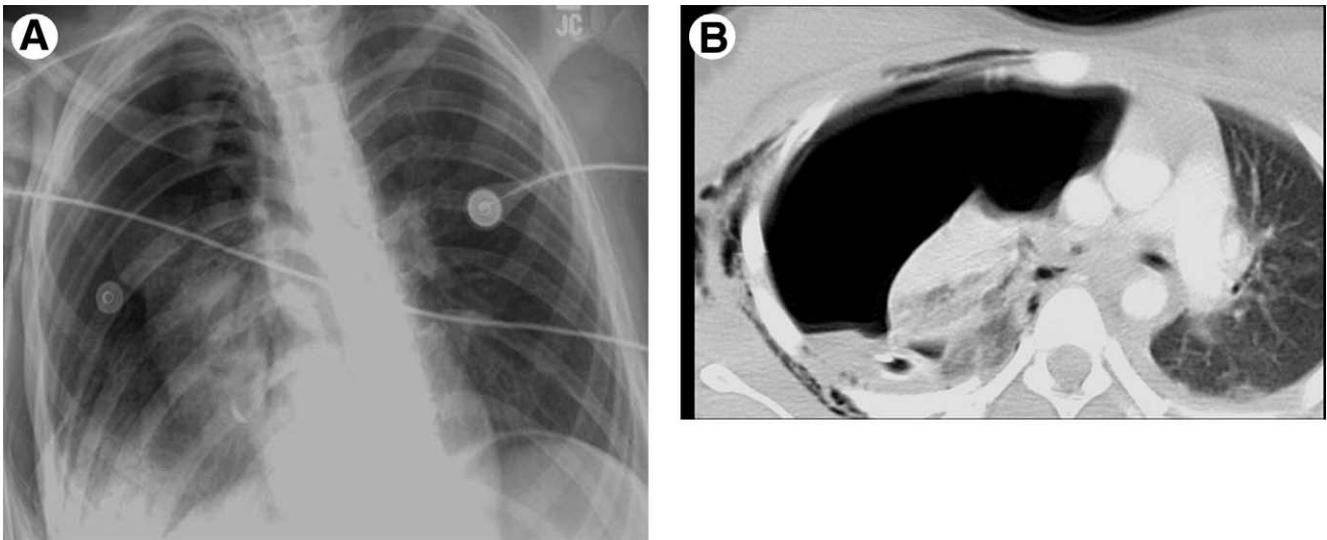
1. None—normal mediastinum
2. Free or partial flowing blood—increased right paratracheal density relative to the arch, loss of aortic arch definition, apical cap, increased width and density of descending aorta  $\pm$  a wide mediastinum.
3. Contained rupture/hematoma—rightward deviation of the trachea/NG tube, downward displacement of the left bronchus, loss of aortic arch definition, increased arch density, increased width and density of descending aorta.

Please refer to the article by Dr Rivas in this journal for a more extensive clinical and imaging discussion of great vessel injury, especially with multidetector CT technology.

### Bronchial (Bronchial Fracture)

The majority of bronchial fractures are caused by rapid deceleration with the more mobile right and left main bronchi shearing from the fixed carina. Cartilaginous support of the trachea and proximal bronchi progressively decreases to a predominately membranous support of the distal bronchi. Accordingly, 80% of bronchial fractures occur within 2.5 cm of the carina, where the rigid cartilaginous bronchi are fixed against the adjacent more membranous distal bronchi.

Early chest radiographic findings include pneumomediastinum, pneumothorax with atelectasis of the affected lung, soft-tissue emphysema, and malposition of the endotracheal tube. The two classic radiographic signs of tracheobronchial injury are exceedingly rare: The “double wall sign” represents rupture of the trachea or the main bronchi with intramural gas in the proximal airways. The “fallen lung sign” represents inferior lung collapse due to the disruption of the normal central anchoring attachments of the lungs. Normally, the



**Figure 12** (A) A 14-year-old hit by a car. A large right pneumothorax with right-sided soft-tissue air and pneumomediastinum. The lung falls dependently rather than toward the hilum. (B) CT scan demonstrates the “fallen” lung dependently with rupture of the bronchus intermedius.

lung will retract toward the hilum. The diagnosis of most bronchial fractures is made on delayed imaging, with persistent or increasing soft-tissue emphysema and/or a persistent pneumothorax with a continuous chest tube air leak (Fig. 12).

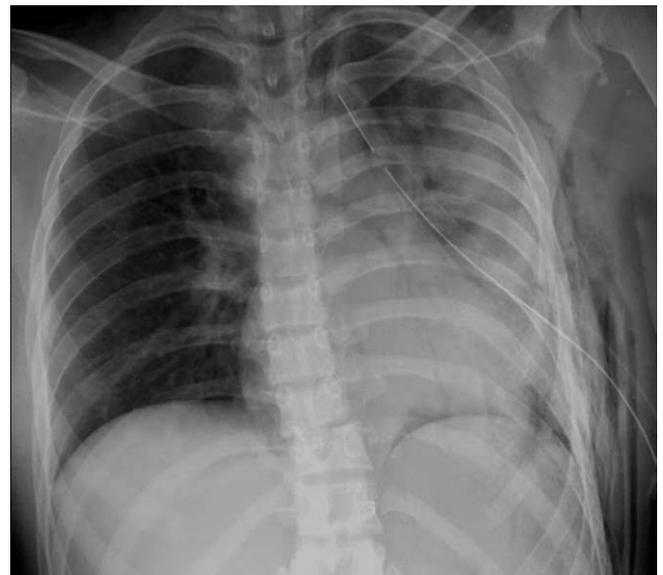
On CT, a small tract of air may be seen coming from a bronchus or the trachea. A discontinuity of the central airway may be seen, especially with the thinner collimation available on the multidetector scanners. High tracheal or hypopharyngeal injuries may be quite subtle with imaging, but should be considered with persistent pneumomediastinum/air leak.

### Cord Injury

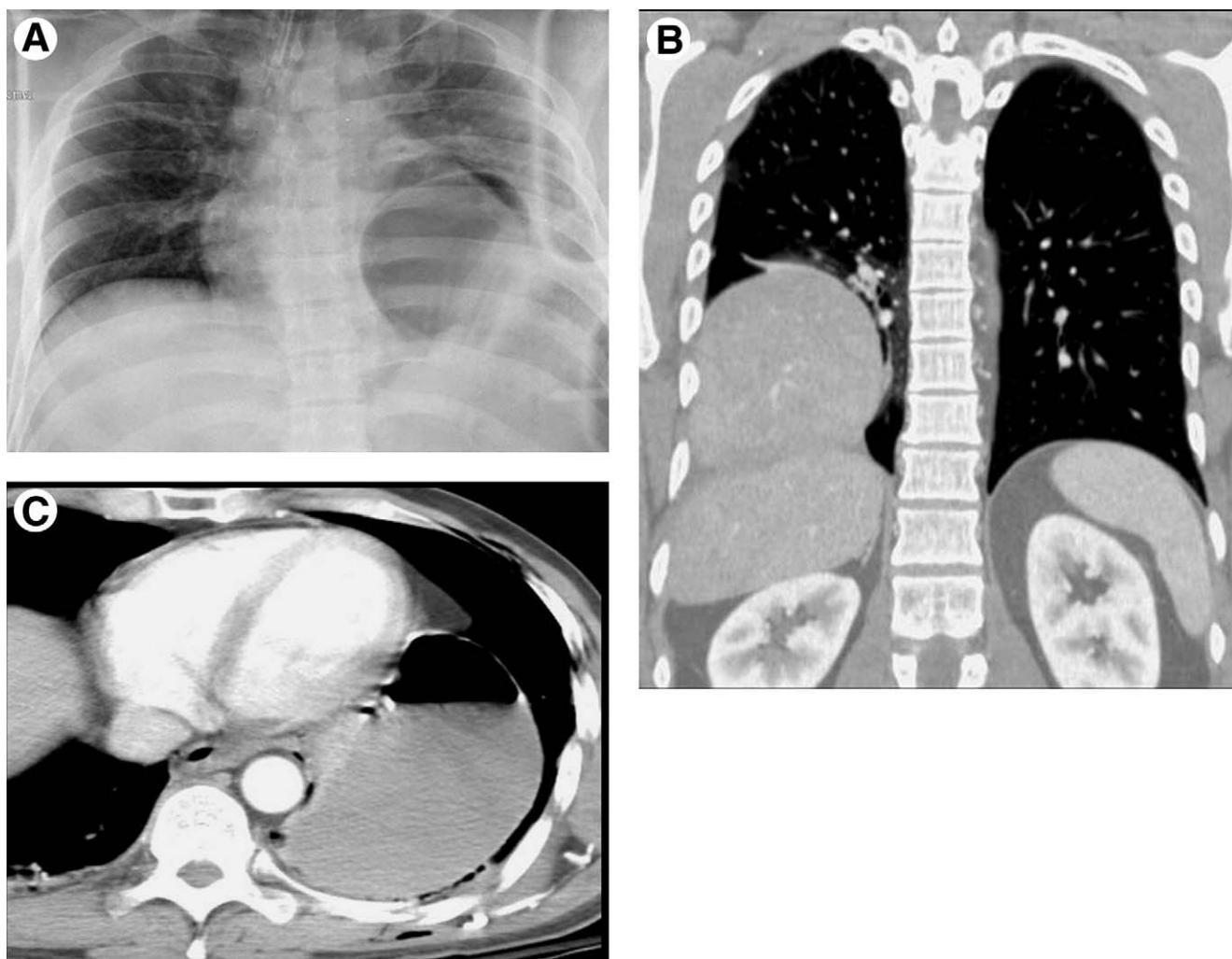
Complete evaluation of the thoracic spinal column and central canal is best performed on the lateral view, which is not obtained in most acute trauma settings. On AP view the thoracic vertebrae should be aligned with visualization of the pedicles, endplates, and spinous processes. Initial evaluation includes identifying fracture lucencies; increased paraspinous or mediastinal hematoma; uniform caliber of the spinal canal; radiopaque bony fragments within the canal; misalignment/dislocation; as well as noting congenital or acquired conditions, which may predispose a patient to cord trauma (Fig. 13). Undetected unstable fractures can progress to significant cord compression and paralysis. This is particularly relevant in the thoracic spine where fractures result in the highest percentage of spinal cord injury. In the thoracic spine the spinal cord occupies a greater percentage of the thecal space and has a more tenuous arterial supply.

Motor vehicle accidents are the most common mechanism for spinal fracture. The most common location for fracture is the lumbar spine (48%), followed by the cervical spine (43%), and the thoracic spine (28%), with 19% sustaining a spinal fracture in more than one location.<sup>10</sup> Therefore, if a fracture is identified, the entire spine should be imaged. No

good comparative studies of the sensitivity of plain radiograph versus CT in thoracic or cervicothoracic junction vertebral body fractures have been done, though CT has proven to be far superior in the evaluation of cervical spine fractures alone (3 to 65% plain radiograph versus 99% CT).<sup>11</sup> With multidetector CT scanning, sagittal and coronal reformations are readily obtained and at our institution are proving to add to the sensitivity in fracture detection, particularly with fracture in the coronal plane. However, one must have a proactive search pattern to visualize one of these injuries on the AP chest radiograph. In addition to vertebral body fractures, isolated fractures of the neural arch and transverse processes can also occur.



**Figure 13** A 19-year-old in an MVA with left-sided contusions, rib fractures, and T8 to 9 thoracic spinal fracture/dislocation.



**Figure 14** (A) A left-sided diaphragmatic rupture with the gastric fundus located in the inferior medial hemithorax. Note the narrowed portion at the site of herniation (“collar” sign). (B) CT reformatted coronal image demonstrates a large right diaphragmatic rupture with the liver herniated into the right hemithorax. This is 4 weeks after a MVA. (C) CT demonstrates a left diaphragmatic rupture with the stomach lying dependently on the posterior hemithorax rather than on the diaphragm (“dependent viscera” sign).

## Diaphragmatic Rupture

Diaphragmatic rupture is most commonly caused by blunt trauma (74%) and penetrating injuries. The incidence of diaphragmatic rupture in blunt trauma is 0.8%. The left hemidiaphragm is affected in 90% of cases, which is thought to be due to absorption of right-sided forces by the liver. However, it is possible that left hemidiaphragmatic rupture is more frequently diagnosed due to the ease of herniation of left-sided bowel compared with right-sided liver. Bilateral diaphragmatic tears have been reported, though are rare. Most diaphragmatic tears are longer than 10 cm and occur in a posterolateral location. Up to 30% of patients are symptomatic, with symptoms including abdominal pain, shortness of breath, chest pain, abdominal pain, and decreased breath sounds. Ninety-four percent have associated additional injuries, especially multiple lower thoracic rib fractures, and intraabdominal injuries are usually present.

A chest radiograph is diagnostic of diaphragmatic rupture in 28 to 70% of patients with thoracoabdominal trauma. Serial chest radiographs increase the diagnosis by 12%.<sup>12</sup> Radiograph signs include asymmetric elevation and herniation of abdominal contents into the thoracic cavity with or without mass effect on the mediastinum. Lower lobe opacification suggests diaphragmatic injury in the presence of an asymmetric diaphragmatic elevation (Fig. 14). Eventration or central “peaking” of the hemidiaphragm should not be mistaken for asymmetric elevation relating to trauma. In the latter case, the entire hemidiaphragm will be elevated rather than just the central portion. Definitive diagnosis can be made by surgical exploration. CT imaging may demonstrate the diaphragmatic “collar sign” or discontinuity of the diaphragm.

The mortality of diaphragmatic rupture approaches 30% and may be affected by delay in diagnosis. A high index of suspicion is required for this injury and dedicated search for

any diaphragmatic irregularities should at least make the radiologist consider the diagnosis.

## Esophageal Rupture

Esophageal perforation secondary to blunt trauma is rare, with perforation being more commonly iatrogenic or secondary to foreign body ingestion. The etiology of blunt traumatic esophageal rupture is thought to relate to increased intraluminal pressures by decompressive forces. Perforation most commonly occurs at the level of the cervical esophagus due to a direct blow to the neck or laceration by adjacent bony fracture fragments. Rupture of the distal esophagus is limited to several case reports. Esophageal rupture has a high morbidity and mortality, increasing from 10 to 25% if diagnosed within 24 hours to 40 to 60% when treatment is delayed beyond 48 hours. Chest pain is the most common symptom, though chest pain is highly nonspecific in the trauma patient. With delayed diagnosis and treatment, leak of gastric materials and esophageal flora can result in periesophageal abscess, mediastinitis, and formation of an esophageal–tracheal fistula. Management of esophageal rupture is controversial, with some advocating conservative management and others advocating surgical repair or resection.

Pneumomediastinum is the most common finding of esophageal rupture on conventional radiograph. However, it is important to remember that the etiology of pneumomediastinum is overwhelmingly due to pulmonary alveolar rupture and not esophageal rupture. The “V sign of Naclerio” is a V-shaped collection of air tracking along the mediastinum and diaphragm, indicating pneumomediastinum and pneumothorax. This is described in cases of esophageal rupture secondary to Boerhaave's Syndrome. Additional CT findings of esophageal rupture include deep cervical emphysema, periesophageal gas, pleural effusion, and thickening of the esophagus. Diagnosis of esophageal rupture should be confirmed by an esophagram, either with conventional fluoroscopy or with CT scan after ingesting or placing water-soluble contrast into the esophagus.

## Fracture

Assessing the osseous structures is important for two reasons: one, a subtle fracture may explain the patient's pain, which will decrease the need for further investigation; and two, fractures indicate the location of greatest traumatic force and can predict patterns of injury. The screening radiograph is also important to detect complications of fractures, such as pneumothorax or pulmonary contusion from a punctured lung. In the setting of a rib fracture hemorrhage most frequently occurs due to disruption of the adjacent intercostal arteries, resulting in an extrapleural hematoma. The extrapleural hematoma is a useful red flag of an underlying fracture(s) (Fig. 15).

Most fractures occur at the level of the mid thorax posteriorly. Expiratory, oblique, coned-down, or rib views are usually not necessary unless nonaccidental trauma is suspected. In the presence of a flail chest, CT with 3D reconstruction is preformed for surgical planning. Fractures may

be easily detected with osseous windowing on chest CT, although obviously an occult fracture is not an indication for a dedicated CT examination.

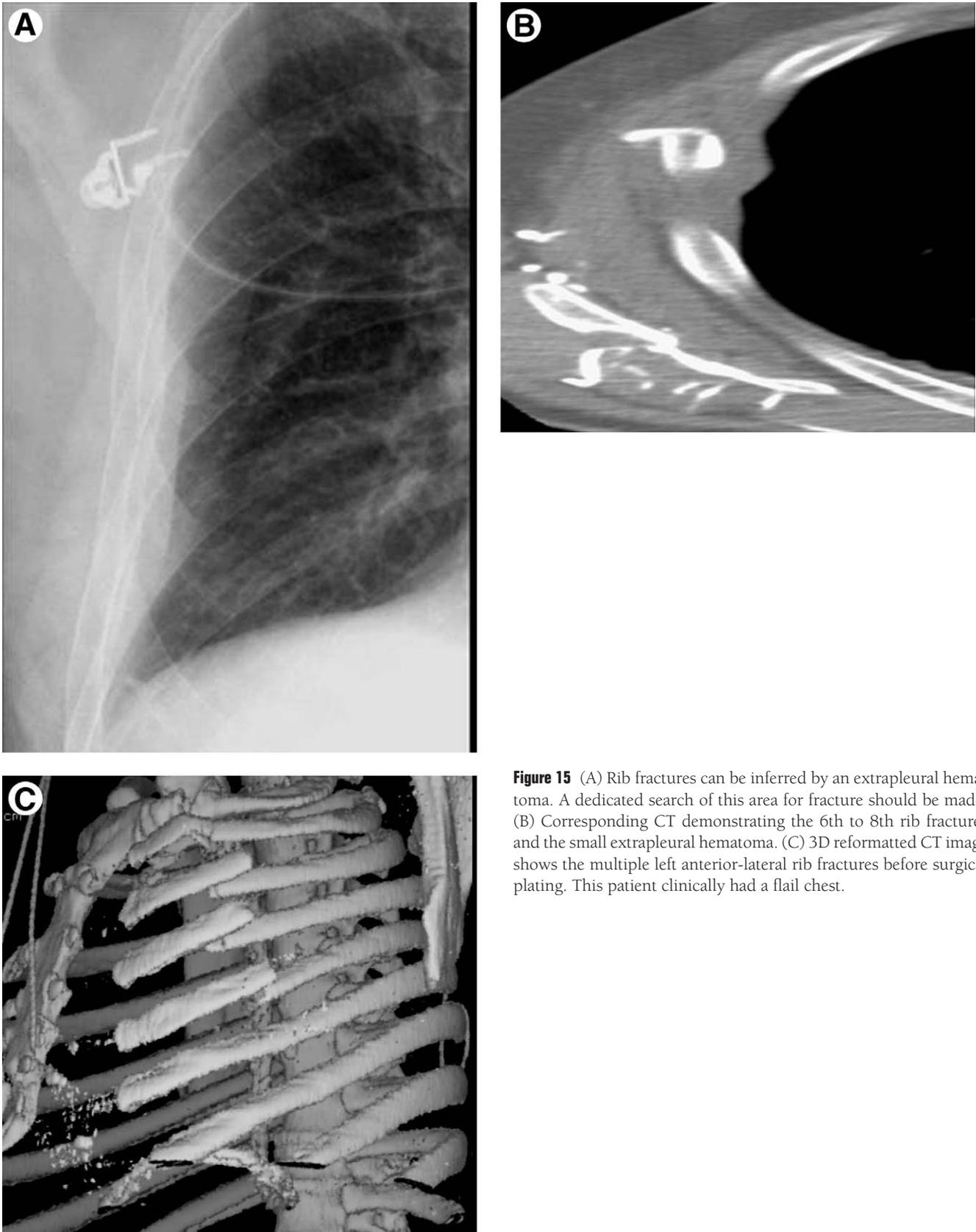
Rib fractures occur in approximately 10% of all trauma cases and are the most common blunt chest injury in adults. Ribs 1 to 3 are very stable, being protected by the shoulder girdle and surrounding musculature. Therefore, a fracture at this location usually requires an extreme force. However, in the absence of additional clinical and radiographic signs, CT angiography of the aorta is not usually indicated. There is a high correlation of first rib fractures with brachial plexus, cranial, or maxillofacial injuries.

Fractures of ribs 4 to 9 are common and most significant when associated with flail chest. Flail chest is defined as chest wall instability with focal paradoxical motion of the chest wall during respiration. There are usually segmental (anterior and posterior) fractures of >3 congruent ribs or single fractures of >4 ribs. In this setting nonvisualized injury to the intercostals musculature may be much more important in patient morbidity and long-term disability than the visualized osseous fractures. The number and location of rib fractures may be primarily useful in predicting intercostal muscle disruption. Surgical plating up to 6 days postinjury is an early alternative treatment for flail chest. Stabilization of the fractured ribs may allow for some underlying intercostal muscle healing and in a more anatomic fashion. This may have the benefit of reducing or even obliterating the discoordinated motion of the chest wall and therefore reducing some of the patient's long-term disability. Fractures of ribs 10 to 12 are less common and are associated with visceral damage to the liver, spleen, or kidneys.

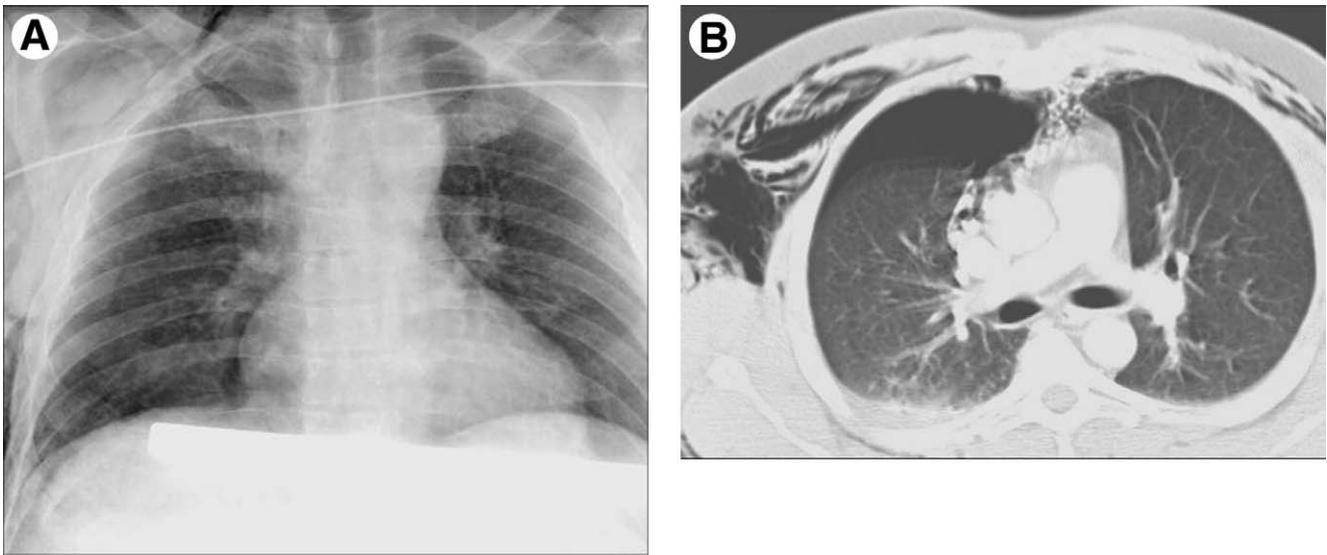
The clavicle, sternum, and scapula should also be evaluated for fracture. Fractures of the clavicle comprise 4% of all fractures in adults and are classified according to their location. Group I fractures are fractures of the midclavicular body (80%); group II fractures involve the distal 1/3 of the clavicle lateral (15%), and group III fractures involve the medial 1/3 of the clavicle (5%). A Serendipity View is a dedicated 40-degree cephalic tilt view used to visualize the medial 1/3 of the clavicle for fracture or dislocation. Most fractures demonstrate upwards displacement of the distal fracture fragment, are of the closed type, and are managed conservatively. Distal clavicular fractures have a high incidence of nonunion, though surgical intervention is only indicated for the small percentage of patients who are symptomatic.

Fractures of the sternum are most often the result of a motor vehicle accident (84%). Sternal fractures are important to note because of an association with fracture of the thoracic spine. Interestingly, there is no association with visceral chest injury, including cardiac contusion. One theory is that when the sternum is fractured, the force of the impaction is absorbed by the sternum and not transmitted through the anterior chest wall to the underlying heart. Patients with sternal fractures have decreased overall injury when compared with trauma patients without sternal fracture.

Scapular fractures occur in 1% of blunt trauma and have a 99% association with additional injuries. Fractures may occur in the body or spine (40 to 75%), the acromion (8 to



**Figure 15** (A) Rib fractures can be inferred by an extrapleural hematoma. A dedicated search of this area for fracture should be made. (B) Corresponding CT demonstrating the 6th to 8th rib fractures and the small extrapleural hematoma. (C) 3D reformatted CT image shows the multiple left anterior-lateral rib fractures before surgical plating. This patient clinically had a flail chest.



**Figure 16** (A) A 48-year-old hit by a car. Multiple right-sided rib fractures and right chest wall and neck soft-tissue air. This combination is diagnostic for a right-sided pneumothorax, even though one is not seen on the radiograph. (B) CT scan confirms a moderate right anterior pneumothorax.

16%), the neck (5 to 32%), the glenoid (10 to 25%), and the coracoid (3 to 13%). With a suspected scapular fracture, a transcapular view may be obtained in addition to the routine AP view. Scapular fractures have traditionally been associated with increased morbidity and mortality, and the association of scapular fracture to blunt thoracic aortic injury is controversial. A large review of 11,500 patients demonstrates an association with thoracic injury (49% versus 6% in the control group) but no increased mortality or neurovascular morbidity.<sup>13</sup> A second large review of >35,000 blunt trauma patients demonstrated 1.0% of patients with scapular fractures and 0.6% with thoracic aortic injury. Of those with scapular fractures only 4/408, or 1%, also sustained aortic injury.<sup>14</sup> The more common associated injuries were rib fractures (43%), lower extremity (36%), and upper extremity (33%) fractures.

## Gas

Pneumothorax and pneumomediastinum are radiographic signs of disrupted pleura and/or viscera and range from barely detectable to life-threatening. The barely detectable pneumothoraces are important to note as they could increase in size, especially in intubated patients, leading to acute decompensation.

The etiology of a pneumothorax is thought to relate to pulmonary alveolar pressures surpassing pulmonary interstitial pressures. With this increased gradient the alveoli burst and release air into the surrounding interstitium. The same forces that disrupt the alveoli also disrupt the underlying pleura, allowing air to track to the pleural space. When air is present between the parietal and visceral pleura, normally a potential space, the suspensory forces on the lung is disrupted and atelectasis/collapse ensues. The diagnosis of a large pneumothorax is straightforward, with visualization of the pleural surface and atelectasis/collapse of the ipsilateral

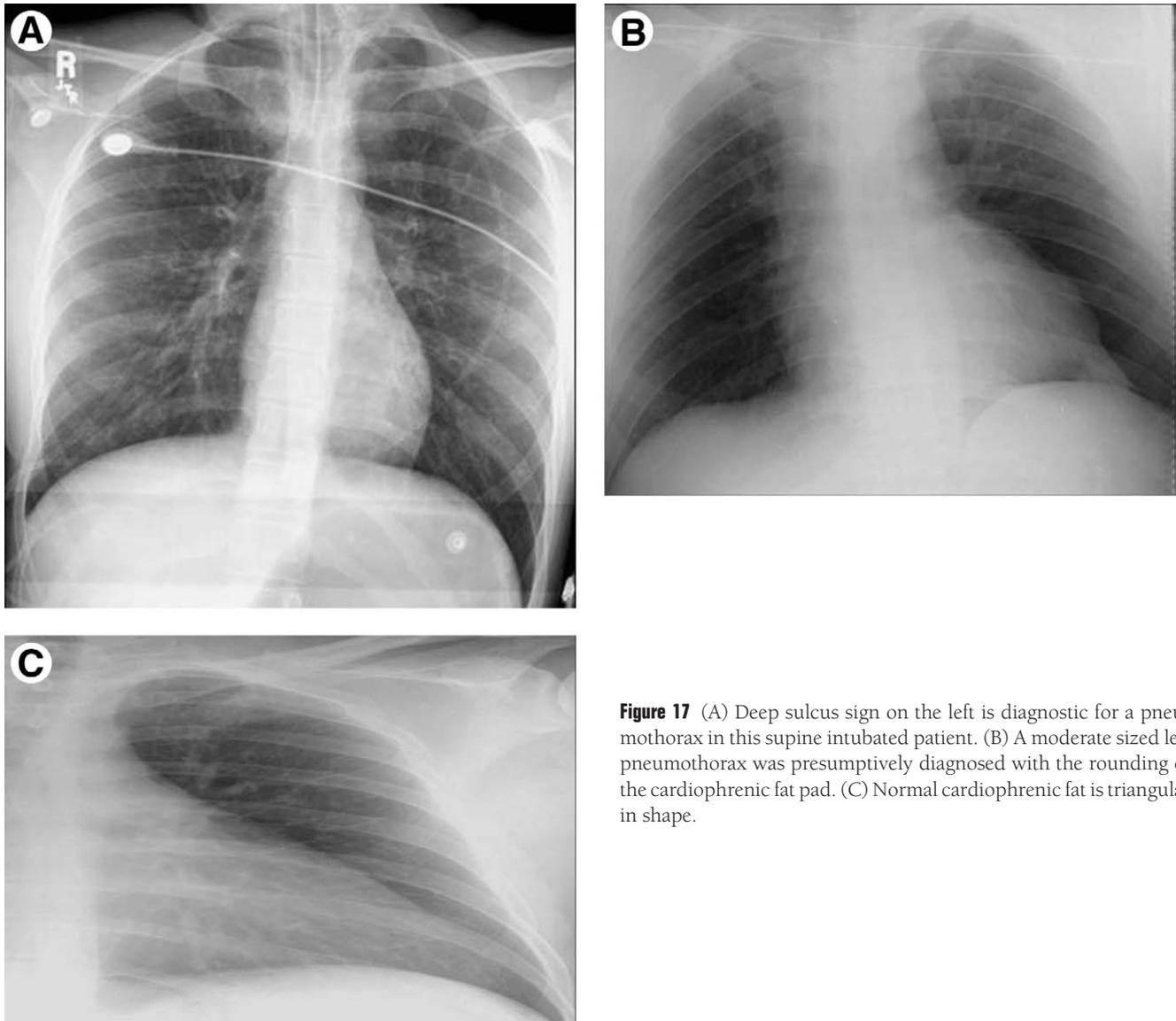
lung toward the hilum. Smaller pneumothoraces may be more difficult to see and diagnosis often relies on secondary signs.

In the trauma patient chest radiographs are obtained in a supine or semirecumbent position. In these positions, 30% of pneumothoraces are not visualized.<sup>15</sup> The left lateral decubitus view is the most sensitive to detect a pneumothorax (88%) as compared with the upright (59%) and supine (38%) views,<sup>16</sup> but obviously not useful in the trauma setting. If a pneumothorax is not detected on a standard AP view, the clinical significance of a small pneumothorax is likely to be very low, and additional views are not routinely recommended. However, if the patient is intubated, a small nondetectable pneumothorax can quickly become life-threatening with positive pressure ventilation.

In the setting of rib fractures and ipsilateral chest wall soft-tissue emphysema, a pneumothorax should be presumed present, *even* if it is not detected on the radiograph. In the intubated patient, the presence of soft-tissue gas with underlying rib fractures should make the radiologist forewarn the clinician that an underlying pneumothorax is present and close a follow-up examination would be prudent if no chest tube is initially placed (Fig. 16).

In the supine and semirecumbent patient, the antero-medial (38%) and subpulmonic (26%) recesses are the most commonly involved. Subpulmonary pneumothoraces represent air between the lung base and the diaphragm and can occur anteriorly or posteriorly. An AP radiograph demonstrates a hyperlucent upper abdomen, a sharply outlined hemidiaphragm, a deep sulcus sign, a rounded cardiophrenic fat pad, and demarcation of the inferior surface of the lung.

Radiographic signs of pneumothorax include direct visualization of the thin pleural surface of the lung, usually seen at the lung apices. The “deep sulcus sign” represents



**Figure 17** (A) Deep sulcus sign on the left is diagnostic for a pneumothorax in this supine intubated patient. (B) A moderate sized left pneumothorax was presumptively diagnosed with the rounding of the cardiophrenic fat pad. (C) Normal cardiophrenic fat is triangular in shape.

air coursing into the pleural space lateral to the diaphragm with resultant increased lucency of the lateral costophrenic angle (Fig. 17).

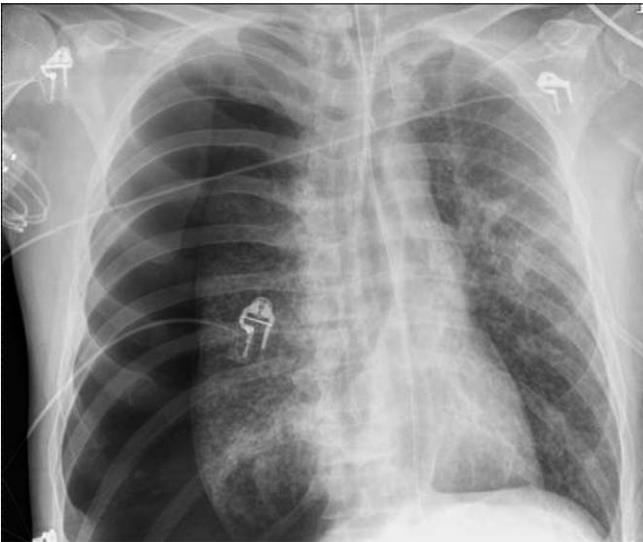
The radiograph should be evaluated for complications of pneumothorax. These include collapse of the ipsilateral lung and a contralateral shift of the mediastinal structures. Poor respiratory outcome is secondary to compromised gas exchange in both the collapsed lung and the compressed contralateral lung.

A tension pneumothorax is a more serious form, resulting from a pulmonary parenchymal or bronchial injury that acts as a one-way valve; therefore, with each respiration air moves into the pleural space but cannot exit (Fig. 18). Untreated this will rapidly evolve into life-threatening pulmonary and cardiac compromise. In the setting of a tension pneumothorax the radiograph should be evaluated for four important findings, including widening of the lateral diameter of the thorax, a deep sulcus sign, mediastinal shift, and a depressed diaphragm. The diaphragmatic depression is helpful since ab-

dominal pressure is 0 atm. Therefore, the pleural space has to be  $>0$  atm, also known as a “tension” pneumothorax. Treatment of a tension pneumothorax is rapid decompression with a large bore hollow needle, which converts a tension pneumothorax into an open one. An intercostal chest tube is then placed for further management. Detection of tension pneumothorax requires rapid direct communication to the trauma team.

A false-positive diagnosis of pneumothorax is often due to a skin fold, bulla, or misinterpreting a rib margin for a pleural line. Visualization of lung vascularity lateral to the mistaken pleural line provides strong (although not absolute) evidence that there is no pneumothorax. Management of pneumothorax is limited to observation if the pneumothorax is estimated to be small or if the patient has no respiratory distress. In cases of a larger pneumothorax, a mechanically ventilated patient, or if the patient is short of breath, then management is often with the insertion of a chest tube.

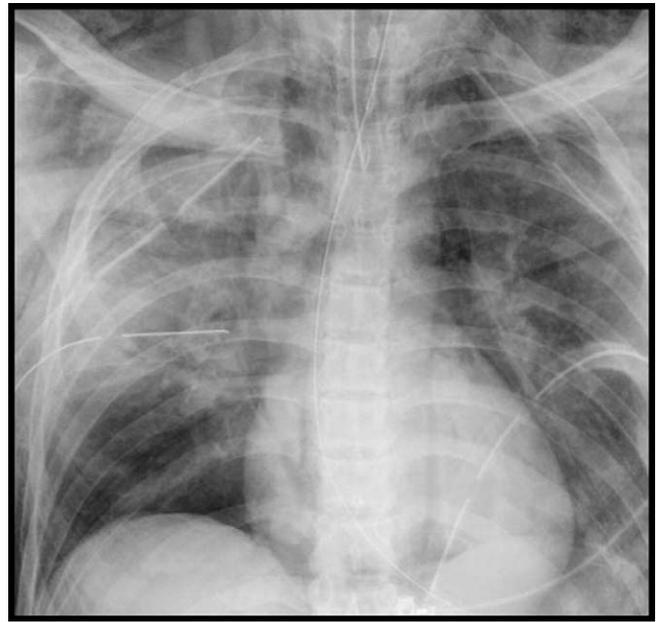
In addition to pneumothorax, pneumomediastinum is the



**Figure 18** A large right pneumothorax with severe mediastinal shift to the left and caudal displacement of the right hemidiaphragm. This is characteristic for a tension pneumothorax.

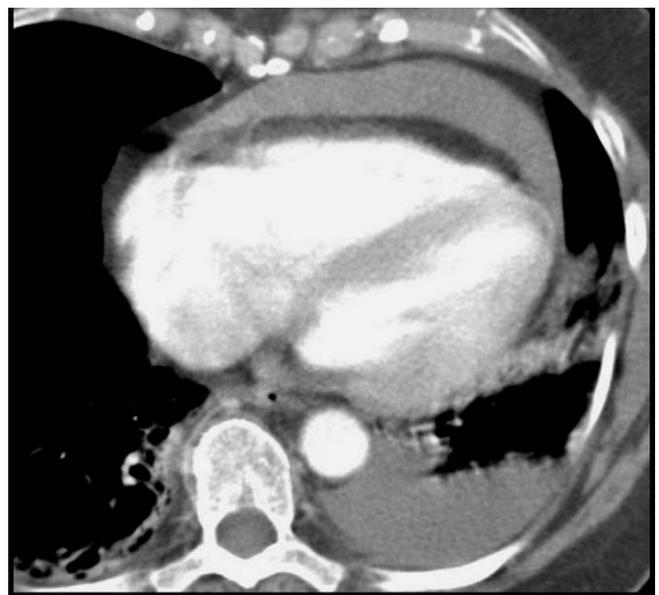
second form of abnormal gas that can be seen on the radiograph or CT. Pneumomediastinum has many potential sources. Intrathoracic sources include the lung, tracheobronchial tree, and rarely, the esophagus. Extrathoracic sources include the head and neck and the anterior retroperitoneum. (The mediastinum and retroperitoneum communicate at the level of the sternocostal attachment of the diaphragm as well as the periaortic and periesophageal tissue planes.) The most common source of pneumomediastinum is the terminal air spaces of the lung. It may result from an increased intra-alveolar pressure surpassing the interstitial pressure, inducing an alveolus rupture from the increased gradient. The air tracks via a peribronchial sheath to the mediastinum. This is termed the “Macklin Effect,” which specifically implies alveolar damage. In this scenario with positive pressure ventilation, pneumomediastinum will increase as air is forced into the ruptured alveoli.

Pneumomediastinum is best visualized on an AP film as a lucency bordered by a thin white pleural line coursing along the pericardium and lateral mediastinum. Differentiating pneumomediastinum from medial pneumothorax and pneumopericardium can be difficult. With medial pneumothorax, other evidence of pneumothorax should be sought, including the presence of rib fractures and soft-tissue air. With pneumopericardium there may be direct visualization of the pericardium. Also, the air collection will respect the boundaries of the pericardium, and as the air collection grows, it may appear as more ovoid in shape rather than linear given the constrictions of the pericardium. As a pneumomediastinum increases in size, there will be dissection into the neck/subcutaneous tissues. The “Hammond sign” is crackles or crunch heard in the left lateral decubitus position and is a clinical indicator of pneumomediastinum. A large pneumomediastinum should also make airway or esophageal laceration a strong consideration (Fig. 19).



**Figure 19** A large pneumomediastinum with moderate soft-tissue air is present. The patient was found to have a 3-cm posterior wall tracheal laceration on bronchoscopy, just distal to the Endotracheal tube (ETT). Note the lucent air along the descending aorta, brachiocephalic vessels, and the medial visceral pleural line lifted along the mid left hemithorax.

Radiographic signs of pneumomediastinum on an AP film include the “double bronchial wall sign,” in which air accumulates adjacent to a bronchial wall and allows visualization of both sides of the wall. The “continuous diaphragm sign” is air trapped posterior to the pericardium with the entire diaphragm visualized from medial to lateral. The “tubular artery



**Figure 20** The patient was in a head-on MVA and had arrhythmias on ECG, consistent with a cardiac contusion. CT scan demonstrates a concurrent hemopericardium, confirmed at surgery. A left pleural effusion was also present.



**Figure 21** A left chest tube was placed in the trauma bay, but it extended into the left upper quadrant of the abdomen. This patient had a concurrent left diaphragmatic rupture and hernia.

sign” results from mediastinal air outlining the medial side of the aorta with normally aerated lung outlining the lateral side. The “ring around the artery” sign results from air surrounding the pulmonary artery. In children, elevation of the thymus is diagnostic with a large amount of mediastinal air.

A false-positive pneumomediastinum may be caused by the Mach effect, wherein a thin linear lucency outlines the dense opaque cardiac silhouette. This effect occurs at the level of the retina and is due to a visual perception of edge enhancement. The opaque cardiac silhouette tricks the eye into seeing an adjacent lucency without a white visceral pleural line.

## Heart

Evaluation of the heart on radiograph or CT in a trauma patient does have limitations. A pericardial effusion/hemopericardium is the main diagnostic possibility with radiographs and, more likely, CT scans (Fig. 20). A large contusion is conceivable on enhanced CT imaging by seeing a relative area of decreased myocardial enhancement, although much more research is needed. Contusion is often made clinically with alterations in ECG findings and elevation of cardiac enzymes.

## Iatrogenic

To round out a complete evaluation of the chest radiograph, the position of all tubes and lines placed by EMTs, emergency, and trauma personnel should be evaluated. Common instrumentation includes endotracheal tubes, nasogastric tubes, chest tubes, and central venous catheters.

Rapid sequence intubation is preformed in the trauma patient by emergency technicians as well as emergency and anesthesiology physicians. Rapid sequence intubation remains the definitive method of securing an airway in trauma management. Endotracheal tubes should terminate about 2 to 3 cm above the carina, with the most common erroneous

position being a right main bronchus intubation. Right main bronchus intubation occurs in 1 to 6% and esophageal intubation occurs in 4 to 8% of cases. In the case of right-sided intubation, incorrect positioning was first indicated on the chest radiograph in 3% of cases.<sup>17</sup>

Nasogastric tube placement is often clinically straightforward, as correct placement results in return of gastric contents, allowing for a bedside determination of correct positioning. Most malpositioned tubes terminate within the distal esophagus, in which the only deleterious effect is ineffective gastric suctioning. Scenarios in which this could conceivably alter care are in a small upper gastrointestinal bleed or gastric emptying to prevent aspiration. Airway or pleural placement should be communicated immediately to the emergency/trauma personal, even if it is obvious to the radiologist.

Complications of central catheter placement include pneumothorax (<2%), venous air embolism (<2%), arterial puncture (1%), and catheter malfunction (<1%). Unusual complications of nontunneled central venous catheter placement include intrapleural placement, cardiac tamponade, contralateral hemothorax, arteriovenous fistula, central venous stenosis or thrombosis, aortic injury with tamponade, mediastinal catheterization, inadvertent puncture of the subclavian artery, and pseudoaneurysm formation.

Chest tube placement is a common iatrogenic potential. They may be placed into the fissure, often with little clinical significance. Placement against mediastinal structures may require them to be pulled back and repositioned. Chest tubes occasionally end up in the lung parenchyma, especially if the lung is contused. The chest tube should be removed carefully and replaced into the pleural space. Placement outside the pleural space is less common, but well-described, especially in the chest wall soft tissues or upper abdomen (Fig. 21). The latter is a potential with diaphragmatic hernia or severe diaphragmatic eventration.

## Summary

Traumatic pulmonary parenchymal injuries demonstrate a characteristic appearance in distribution, morphology, and temporal evolution on the chest radiograph and CT that often allows the radiologist to provide a narrow differential or definitive diagnosis. Accurate, focused diagnosis on the part of the radiologist can significantly aid the trauma surgeon/clinician in tailoring treatment and appropriate follow-up to optimize patient care with reduction in morbidity and mortality.

## References

1. Gurney JW: Chest X-ray: The Art & Science of Thoracic Imaging. Available at: <http://www.chestx-ray.com>
2. Tocino I, Miller MH: Computed tomography in blunt chest trauma. *J Thorac Imaging* 2(3):45-59, 1987
3. Smejkal R, O'Malley KF, David E, et al: Routine initial computed tomography of the chest in blunt torso trauma. *Chest* 100(3):667-669, 1991
4. Schild HH, et al: Pulmonary contusion: CT versus plain film radiographs. *J Comp Assist Tomogr* 13(3):417-420, 1989

5. Wagner RB, Crawford WO Jr, Schimpf PP: Classification of parenchymal injuries of the lung. *Radiology* 167:77-82, 1988
6. Wagner RB, Crawford WO Jr, Schimpf PP: Classification of parenchymal injuries of the lung. *Radiology* 167:77-82, 1988
7. Engoren M: Lack of association between atelectasis and fever. *Chest* (107):81-84, 1995
8. Williams JS: Aortic injury in vehicular trauma. *Ann Thorac Surg* 57(3): 726-730, 1994
9. Ayella RJ, Hankins JR, Turney SZ, et al: Ruptured thoracic aorta due to blunt trauma. *J Trauma* 17:199-205, 1977
10. Brown CVR, Antevil JL, Sise MJ, et al: Spiral computed tomography for the diagnosis of cervical, thoracic, and lumbar spine fractures: its time has come. *J Trauma* 58(5):890-896, 2005
11. Woodring JH, Lee C: The role and limitations of computed tomographic scanning in the evaluation of cervical trauma. *J Trauma* 33(5): 698-708, 1992
12. Rodriguez-Morales G, Rodriguez A, Shatney CH: Acute rupture of the diaphragm in blunt trauma: analysis of 60 patients. *J Trauma* 26:438-444, 1986
13. Stephens NG, Morgan AS, Corvo P, et al: Significance of scapular fracture in the blunt-trauma patient. *Ann Emerg Med* 26(4):439-442, 1995
14. Brown CV, Velmahos G, Wang D, et al: Association of scapular fractures and blunt thoracic aortic injury: fact or fiction? *Am Surg* 71(1): 54-57, 2005
15. Tocino IM, Miller MH, Fairfax WR: Distribution of pneumothorax in the supine and semirecumbent critically ill adult. *AJR Am J Roentgenol* 144(5):901-905, 1985
16. Carr JJ, Reed JC, Choplin RH, et al: Plain and computed radiography for detecting experimentally induced pneumothorax in cadavers: implications for detection in patients. *Radiology* 183(1):193-199, Apr 1992
17. Omert L, Yeane W, Mizikowski S, et al: Role of the emergency medicine physician in airway management of the trauma patient. *J Trauma* 51(6):1065-1068, 2001