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Concepts and Clinical Practice

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63

Pleural Disease

Alysa S. Davis and Nicholas M. Mohr

KEY CONCEPTS

- Point of care thoracic ultrasound can be used to rule out pneumothorax with high sensitivity.
- For healthy young patients with a small primary spontaneous pneumothorax, observation with supplemental oxygen is an appropriate treatment option. For larger symptomatic primary spontaneous pneumothorax, simple aspiration with a catheter is often successful.
- In most cases of secondary pneumothorax, tube thoracostomy should be considered because less invasive approaches are associated with treatment failure.
- The most common causes of pleural effusion in the United States are congestive heart failure, malignancy, and bacterial pneumonia. Pulmonary embolism is an uncommon cause of pleural effusion.
- Thoracentesis should be performed under ultrasound guidance to minimize the risk of complications.
- On a frontal (anteroposterior or posteroanterior) projection, a volume of at least 200 mL of pleural fluid is required to detect a pleural effusion. Ultrasound can detect as little as 50 mL of pleural fluid and can be easily preformed at bedside.

INTRODUCTION

Emergent pleural disease presentations range in severity from asymptomatic pleural effusion to life-threatening tension pneumothorax. This chapter reviews the two most common nontraumatic pleural conditions: spontaneous pneumothorax and pleural effusion. Traumatic pleural diseases are covered in Chapter 37.

SPONTANEOUS PNEUMOTHORAX

Foundations

Background and Importance

Pneumothorax is defined as an abnormal collection of air in the pleural space between the parietal and visceral pleura and can range from benign to life-threatening. A spontaneous pneumothorax occurs in the absence of any precipitating external factors, such as trauma or thoracic procedures. A spontaneous pneumothorax can be either a primary spontaneous pneumothorax with no clinically apparent underlying pulmonary disease or a secondary spontaneous pneumothorax in patients with underlying pulmonary disease. Tension pneumothorax is a pneumothorax that leads to a life-threatening increase in pleural pressure associated with displacement of mediastinal structures and hemodynamic compromise.

Primary spontaneous pneumothorax most commonly occurs in healthy young men of above average height and is three times more likely to occur in men than in women. Marfan syndrome and mitral valve prolapse are associated with increased risk for primary spontaneous pneumothorax. Environmental risk factors include smoking and changes in ambient atmospheric pressure. Genetic factors also predispose to primary spontaneous pneumothorax, although this is a rare cause.

As with primary spontaneous pneumothorax, the incidence of secondary spontaneous pneumothorax is three times higher in men. Secondary spontaneous pneumothorax occurs in the setting of chronic pulmonary disease, with chronic obstructive pulmonary disease (COPD) being the most common cause in the United States (Box 63.1). Pneumothorax is also a known complication of *Pneumocystis jirovecci* pneumonia in patients with HIV. In developing countries, tuberculosis and lung abscess are the leading causes of secondary spontaneous pneumothorax.

Both primary and secondary spontaneous pneumothorax are relatively rare in children. Causes of secondary spontaneous pneumothorax include asthma, cystic fibrosis, foreign body aspiration, and connective tissue disease, such as juvenile idiopathic arthritis. The principles associated with the diagnosis, treatment, and surgical management of spontaneous pneumothorax in children are similar to those in adults.

Anatomy and Physiology

Anatomically, the visceral and parietal pleura lie in close approximation with only potential space between them. Normally, the intrapleural pressure remains negative during inspiration, meaning that it is slightly less than atmospheric pressure. The alveolar walls and visceral pleura form a barrier that separates the intrapleural and intraalveolar space and maintains the transpulmonary pressure gradient. When this barrier is disrupted, air enters the pleural space until either the pleural defect is sealed or until the intraalveolar and the intrapleural pressures equalize. In primary spontaneous pneumothorax, the alveolar-pleural barrier is disrupted when a subpleural bleb or bulla ruptures into the pleura space. Blebs are small subpleural thin-walled air-containing pockets that can easily rupture. Increased intrabronchial pressures and intraalveolar pressures generated by bronchospasm and intrinsic positive end expiratory pressure (PEEP) can play a role in the rupture of these blebs. In secondary spontaneous pneumothorax, underlying lung disease and chronic inflammation can also weaken the alveolar-pleural barrier and lead to rupture of blebs.

When negative intrapleural pressure is lost, the ipsilateral lung collapses. A large pneumothorax can result in a restrictive ventilation impairment with reduced vital capacity, functional residual capacity,

BOX 63.1 Causes of Secondary Spontaneous Pneumothorax

Airway Disease
Chronic obstructive pulmonary disease
Asthma
Cystic fibrosis

Infections
Necrotizing bacterial pneumonia, lung abscess
Pneumocystis jiroveci pneumonia
Tuberculosis

Lung Abscess

Interstitial Lung Disease
Sarcoidosis
Idiopathic pulmonary fibrosis
Lymphangiomyomatosis
Tuberous sclerosis
Pneumoconioses

Neoplasms
Primary lung cancers
Pulmonary or pleural metastases

Connective Tissue Diseases
Marfan syndrome
Ehlers-Danlos syndrome
Scleroderma
Rheumatoid arthritis

Miscellaneous
Pulmonary infarction
Endometriosis, catamenial pneumothorax

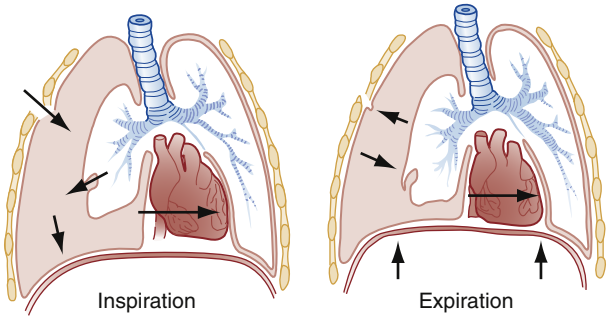

Fig. 63.1 Tension pneumothorax with total collapse of the right lung and shift of mediastinal structures to the left. Air is forced into the pleural space during inspiration and cannot escape during expiration.

and total lung capacity. Shunting of blood through poorly ventilated atelectatic lung tissue may lead to acute hypoxemia, but this effect is a late finding because it is mitigated by compensatory hypoxic vasoconstriction in the collapsed lung.

In tension pneumothorax, the alveolar-pleural defect acts as a one-way valve, allowing air to pass into the intrapleural space during inspiration and trapping the air in the pleural space during expiration (Fig. 63.1). This leads to progressive accumulation of intrapleural air and increasing intrapleural pressure, causing mediastinal shift and compression of the mediastinal venous structures and the contralateral lung. This leads to worsening hypoxemia and can impair venous return to the heart. Untreated, tension pneumothorax progresses to cardiovascular collapse and death.

Clinical Features

Symptoms of primary spontaneous pneumothorax are similar in adults and children. Symptoms often begin suddenly with ipsilateral pleuritic chest pain and dyspnea. Over time, the pain may evolve to a dull steady ache. Although dyspnea is common, it may not be severe in the absence of underlying lung disease or tension pneumothorax. Symptoms are often mild, and patients may wait several days before seeking medical attention. Without treatment, symptoms may spontaneously resolve within 24 to 72 hours, even though the pneumothorax may still be present.

Physical exam findings often correlate with the degree of symptoms. Sinus tachycardia is the most common early physical exam finding. With a large pneumothorax, hypoxia and decreased breath sounds with hyperresonance to percussion may be present. In children, breath sounds are distributed throughout the thorax, which makes unilateral breath sounds more challenging to identify. Other classic physical exam findings include unilateral hemithorax enlargement, unequal chest wall movement with exhalation, absent tactile fremitus, and inferior displacement of the liver or spleen (on the affected side). Absence of these findings does not exclude pneumothorax, and imaging should be obtained when pneumothorax is suspected. In children, routine chest radiography is not recommended in all cases of chest pain, but it should be performed if history or physical examination findings suggest that pneumothorax may be present.

Symptoms of tension pneumothorax include hypoxia, increased work of breathing, and tachycardia. Hypotension is a late finding. Distention of the jugular veins is common but may be difficult to detect. Displacement of the trachea is also a classically described sign but is usually a late finding. Absence of tracheal deviation does not rule out tension. Complete cardiovascular collapse, including cardiac arrest, may occur in tension pneumothorax if intervention is delayed.

In secondary spontaneous pneumothorax, the severity of signs and symptoms are related to both the size of the pneumothorax and the degree of underlying lung disease. Because of poor pulmonary reserve, dyspnea is nearly universal, even in the setting of a small pneumothorax. Symptoms are unlikely to resolve on their own. Physical exam findings such as hyperexpansion and distant breath sounds often overlap considerably with the findings of underlying lung disease, which makes clinical diagnosis difficult. The diagnosis of pneumothorax should be considered whenever a patient with COPD or other significant underlying lung disease has increasing dyspnea, and for this reason chest radiography is recommended in all patients with exacerbations of chronic lung disease.

Differential Diagnosis

The differential diagnosis of spontaneous pneumothorax includes many conditions that also manifest with chest pain and dyspnea. Among the most important is pulmonary embolism (PE), which has similar symptoms of pleuritic chest pain and dyspnea. Other pleural-based diseases such as pneumonia, tumor, and pleural effusion have characteristic radiographic findings that can help to make the diagnosis. Rarely, pneumothorax may mimic acute myocardial infarction or pericarditis with corresponding electrocardiographic changes. Pericardial effusion with or without tamponade can also present with chest pain, dyspnea, and tachycardia, but can be easily diagnosed with bedside ultrasound.

Spontaneous pneumomediastinum is a closely related clinical entity with similar symptoms, and it is diagnosed by the presence of subcutaneous or mediastinal emphysema on chest radiograph. Most cases of spontaneous pneumomediastinum occur in the absence of underlying disease and have a benign course. Secondary causes of pneumomediastinum (e.g., Boerhaave syndrome) are more serious, and treatment focuses on the underlying disorder.

Spontaneous hemopneumothorax is a rare but potentially serious condition that occurs when collapse of the lung is associated with the rupture of a vessel in a parieto-pleural adhesion. The clinical presentation is similar to spontaneous pneumothorax alone but may be accompanied by signs of hemorrhagic shock and effusion on chest imaging. Treatment entails tube thoracostomy to evacuate the pleural space, reexpand the lung, and limit ongoing bleeding.

Diagnostic Testing

Although the history and physical exam may suggest the diagnosis of spontaneous pneumothorax, the diagnosis is made with chest imaging, which may include chest radiography, ultrasound, or computed tomography (CT).[1] The classic radiographic finding is a thin visceral pleural line lying parallel to the chest wall, separated by a radiolucent band devoid of lung markings (Fig. 63.2). The average width of this band can be used to estimate the size of the pneumothorax, such as with the Rhea method which estimates the size of the pneumothorax by plotting the average of three different distances on a nomogram (Fig. 63.3). However, precise quantification is often inaccurate and, in general, it is more practical to characterize the pneumothorax using a semiquantitative approach as small, moderate, or large. The British Thoracic Society guidelines define size based on the measurement of the intrapleural distance at the level of the hilum: small, less than 1 cm; moderate, 1 to 2 cm; and large, more than 2 cm. The American College of Chest Physicians recommends measuring from the apex to the cupola with small being defined as less than 3 cm and large as greater than 3 cm. The estimated size, the patient's clinical status, and associated comorbidities are all useful in guiding management decisions.

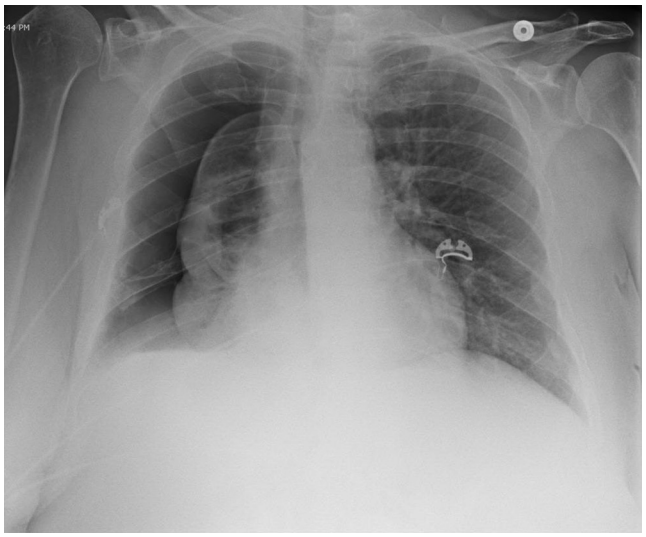

Fig. 63.2 Classic radiographic finding of a pneumothorax showing a thin visceral pleural line lying parallel to the chest wall, separated by a radiolucent band devoid of lung markings.

Tension pneumothorax is a clinical diagnosis, and there should be no delay in treatment for radiographic confirmation. When the diagnosis of tension pneumothorax is not clinically apparent and a chest x-ray is obtained, the classic appearance is one of complete lung collapse with gross distention of the thoracic cavity on the affected side and shift of the mediastinal structures across the midline. In critically ill patients for whom only a supine chest x-ray can be obtained, the finding of a deep sulcus (i.e., a deep lateral costophrenic angle) can suggest the presence of a pneumothorax on that side. Small to moderate pneumothoraces may be difficult to detect in the supine position.

Special care should be taken when viewing chest radiographs of patients with underlying lung disease, especially COPD. In patients with COPD, the relative paucity of lung markings throughout the lung makes pneumothorax more difficult to detect. In addition, large bullae may mimic the radiographic appearance of pneumothorax. A clue to differentiating pneumothorax from a giant bulla is that pneumothorax often shows a pleural line that runs parallel to the chest wall, whereas bullae usually have a more concave appearance. When the diagnosis is unclear, a CT scan can differentiate between the two and evaluate for underlying pathology. Chest CT should be obtained in patients with significant underlying lung disease who present with new-onset dyspnea or hypoxia with a nondiagnostic chest x-ray. Chest CT pulmonary angiography with intravenous contrast is necessary to rule out PE, but a noncontrast chest CT can identify occult pneumonia, pneumothorax, or progression of underlying chronic lung disease. CT can also be used in primary spontaneous pneumothorax to identify bullae, predict the likelihood of recurrence, and guide intervention decisions. Given the increasing use of CT, occult pneumothorax is an increasingly

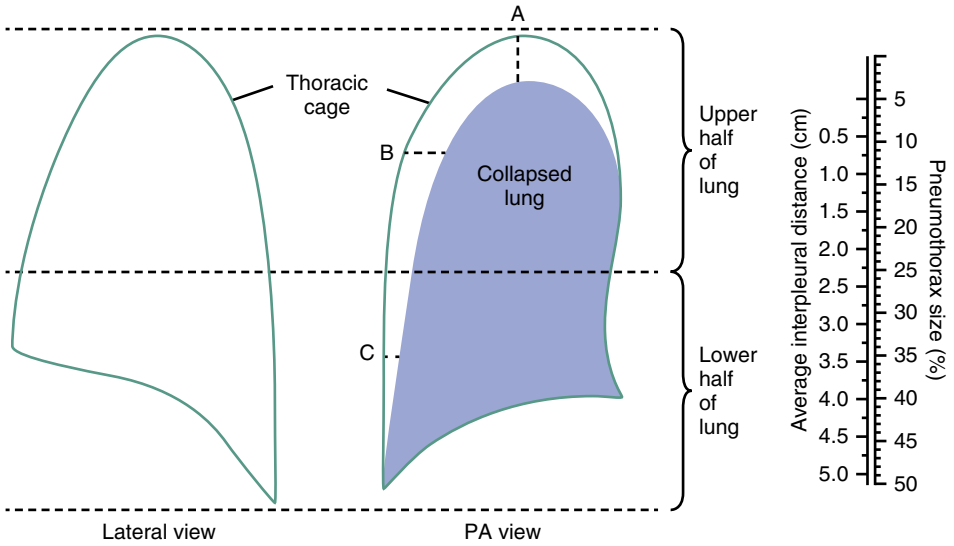

Fig. 63.3 Rhea method of calculating pneumothorax size involves taking the average of the maximal apical intrapleural distance (A), the intrapleural distance of the midpoint of the upper half of the lung (B), and the intrapleural distance of the midpoint of the lower half of the lung (C) and plotting that average distance on the nomogram to get the pneumothorax size (%).

common diagnosis. Typically, an occult pneumothorax not apparent on chest -ray can be managed conservatively in stable patients. Ventilated patients with an occult pneumothorax require close monitoring or early intervention due to the potential for expansion of the pneumothorax with positive-pressure ventilation.

Point of Care Ultrasound

Point of care ultrasound (POCUS) is increasingly being used to diagnose pneumothorax.[1] Bedside thoracic ultrasound is a very sensitive test for the diagnosis of pneumothorax. Sensitivity of chest radiographs in the diagnosis of pneumothorax is 28% to 52% whereas the sensitivity of ultrasound is 79% to 98%. The specificity of chest radiograph (99%) in the diagnosis of pneumothorax is similar to that of ultrasound (98%). In addition to using ultrasound for the diagnosis of spontaneous pneumothorax, it can also be used for screening of postprocedural pneumothorax (e.g., after central line placement). Ultrasound assessment for pneumothorax is best done with a high-frequency linear transducer. A phased array transducer or curvilinear transducer may also be used when set to a shallow depth. There are three features on two-dimensional ultrasound that have been described to be consistent with pneumothorax: lung sliding, B-lines, and a lung point. M-mode may also be used to clarify the ultrasound interpretation in the assessment of pneumothorax.[2]

In the absence of pneumothorax, the visceral and parietal pleura are closely approximated, and they create a characteristic shimmery or sliding appearance with respiratory variation (Fig. 63.4A). Visualization of lung sliding rules out pneumothorax in the area of examination. Visualization of lung sliding throughout the lung rules out pneumothorax, while absence of pleural sliding is suggestive of a pneumothorax. Because lung sliding only rules out pneumothorax in a local region, it is critical to obtain multiple views to rule out pneumothorax. Air generally goes to the most anterior portion in a supine patient and ultrasound should include the most nondependent portion of the patient when assessing for pneumothorax. Identification of a lung point, or the boundary between normal lung sliding and the absence of lung sliding, is highly specific for the detection of pneumothorax and can give clues about its size.

M-mode may be used to better capture the appearance of pneumothorax in a still image: normal lung sliding in M-mode creates a "seashore" sign while the absence of lung sliding creates a "barcode" appearance (Fig. 63.4B). Recent studies have shown that the addition of M-mode helps add to the diagnostic accuracy of lung sliding and is especially useful in less experienced sonographers.[2]

B-lines are long, wide bands of hyperechoic artifact of the visceral pleura that extend vertically through the entire image. If B-lines are present, the visceral pleura is visible by ultrasound, so no pneumothorax separates the two pleural layers. The presence of B-lines rules out pneumothorax in that area. The presence of B-lines can help rule out pneumothorax in patients with pleural disease that would otherwise cause poor lung sliding, such as blebs, fibrosis, or history of pleurodesis or pneumonectomy. In addition, mainstem intubation may lead to the absence of lung sliding in the contralateral hemithorax strictly because ventilation is not applied to the affected side, but B-lines will still be present.

Management

The management of spontaneous pneumothorax has two goals: (1) to evacuate air from the pleural space; and (2) to prevent recurrence. Therapeutic options for treatment of spontaneous pneumothorax include simple observation, aspiration with a catheter, tube thoracostomy, video-assisted thoracoscopic surgery, and thoracotomy. Treatment decisions should be individualized, with considerations for the size of the pneumothorax, severity of the signs/symptoms, presence of underlying pulmonary disease, other comorbidities, history of previous pneumothorax, patient reliability, availability of follow-up, and degree of persistent air leak.

Primary Spontaneous Pneumothorax

For young, healthy patients with a small primary spontaneous pneumothorax and minimal symptoms, supplement oxygen and observation are appropriate. The intrinsic resorption rate is 1-2% per day and the rate of absorption can be accelerated with the administration of 100% oxygen via nonrebreather mask. If observation is selected as the treatment of choice, the patient should be observed for at least 4 hours with supplemental oxygen in the emergency department (ED). Repeat chest radiography prior to discharge should confirm that there is no interval worsening. The patient should follow up with a primary care provider or return to the ED in 24 to 48 hours for reevaluation.

For a primary spontaneous pneumothorax that is larger in size (i.e., >2–3 cm) or a smaller pneumothorax that causes significant symptoms or expands during observation, therapeutic options include needle aspiration, placement of a small-bore (8–14 Fr) pleural catheter, or observation alone. Optimal treatment for those with a first episode of primary spontaneous pneumothorax has been controversial and no available treatment options have been demonstrated to be clearly superior. Advantages of a simple needle aspiration include low morbidity, less invasiveness than chest tube placement, fewer follow-up visits, and overall cost savings. Recent data suggest that conservative management (observation alone) in patients with uncomplicated moderate to large spontaneous pneumothorax is noninferior to drainage with a lower rate of serious adverse events.[3] Currently, either observation alone or simple aspiration is a reasonable approach, and symptoms and patient shared decision-making may be used to select an appropriate treatment option.

Needle aspiration is often performed with a thoracentesis kit, which typically contains an 8 French catheter over an 18-gauge needle and a three-way stopcock. In the needle aspiration technique, the small catheter is placed into either the second anterior intercostal space at the midclavicular line or laterally at the fourth or fifth intercostal space at the anterior axillary line after prepping and sterile draping of the patient and local anesthesia infiltration. A three-way stopcock is then attached, and a large syringe is used to aspirate aliquots of air until resistance is met. The catheter is then removed. If a repeat chest radiograph after the observation period shows no reaccumulation of the pneumothorax, the patient may be discharged home with return precautions and close follow-up. Success of a catheter aspiration is more likely when a patient is younger than 50 years or when the volume of air aspirated is less than 2.5 L. Aspiration of larger volumes of air suggests a continuing air leak. If aspiration fails to reexpand the lung fully, a small-bore chest tube (8–14 Fr) should be placed.

For most patients with primary spontaneous pneumothorax requiring ongoing pleural drainage, placement of a small-bore tube or pigtail catheter is sufficient, because air leak is usually minimal. Small-caliber tubes are easy to insert, are well tolerated by patients, and leave only a small scar after removal. Complications with small-caliber tubes include kinking, malposition, inadvertent removal, occlusion by pleural fluid or clotted blood, and failure due to large persistent air leak. Pigtail catheters have similar outcomes to small-bore chest tubes and are easily placed using the Seldinger technique. Traditionally, patients with indwelling chest tubes or catheters were managed in the hospital, but those with access to close outpatient follow-up can also be managed at home with a one-way Heimlich valve, which consists of a one-way flutter valve covered in transparent plastic.

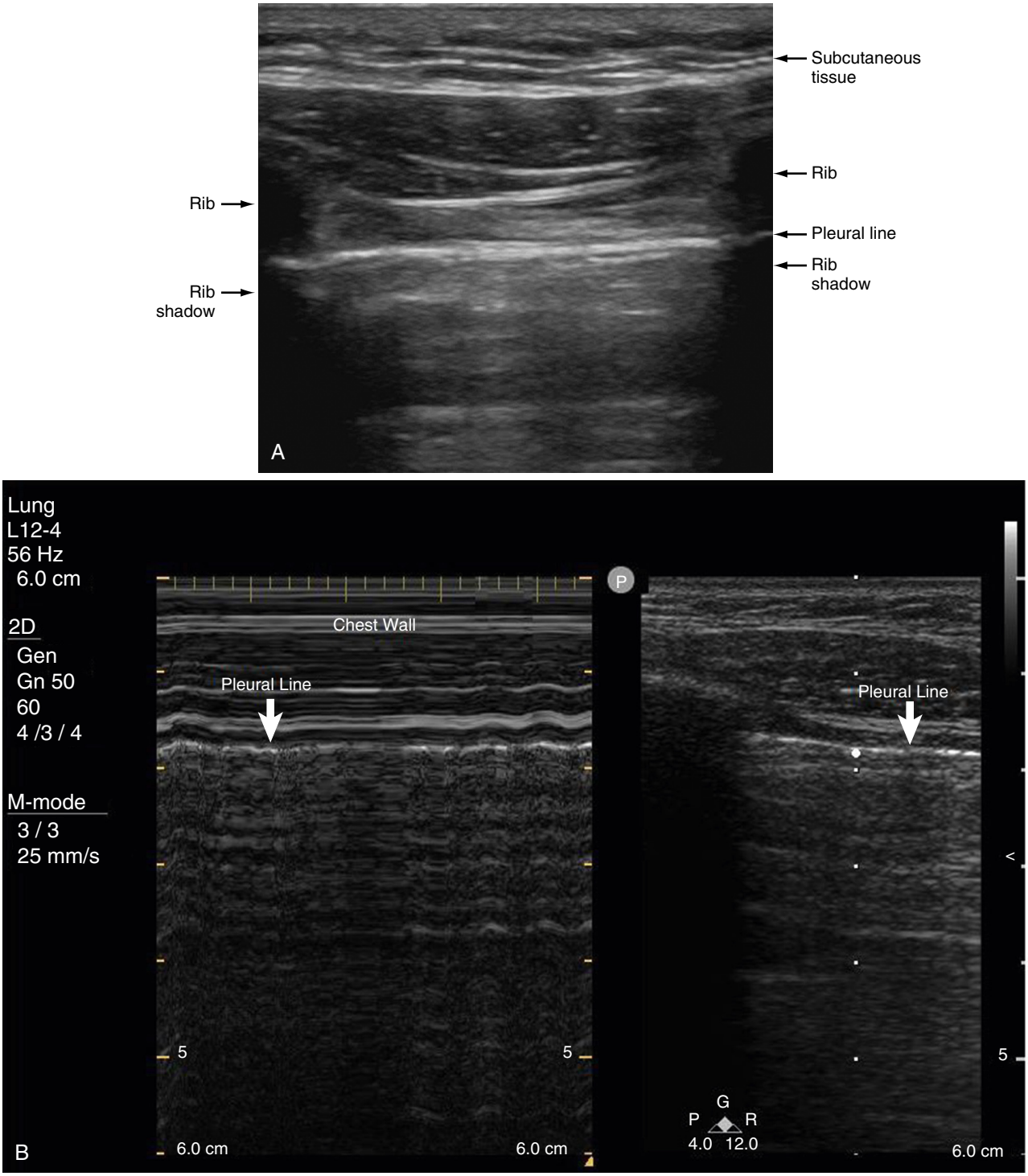

Fig. 63.4 (A) Ultrasound demonstrating location of pleural line. Lung sliding is dynamic and visualized in real time as shimmering or sliding of the pleural line. (B) The use of M-mode creates a "seashore" sign with normal lung sliding.

Secondary Spontaneous Pneumothorax

For patients with secondary spontaneous pneumothorax that is small (<1 cm), admission or placement in an ED observation unit for oxygen therapy and at least 24 hours of observation is appropriate. Secondary spontaneous pneumothorax has a higher likelihood of failing observation, which is why admission is recommended. In patients with secondary spontaneous pneumothoraces of moderate size (1–2 cm), a trial of simple needle aspiration is appropriate. If aspiration is successful and the pneumothorax is resolved or less than 1 cm in size, the patient should be admitted or placed in an ED observation unit for 24 hours of observation. If the pneumothorax is larger (>2 cm) or fails simple aspiration, then a tube thoracostomy is recommended with a small size chest tube or pigtail catheter (8–14 F).

Patients with respiratory distress, those with tension pneumothorax, those likely to require mechanical ventilation, and those with associated hemothorax or pleural effusion may require tube thoracostomy to definitively reexpand the lung with a moderate-sized chest tube (14–28 Fr). Larger-bore tubes (>28 Fr) may be required for patients with hemothorax or a large air leak, but typically are unnecessary in spontaneous pneumothorax.

After insertion, a chest tube is attached to a water seal device and left in place until the lung has fully reexpanded and the air leak has ceased. The routine use of suction neither increases the rate at which the lung reexpands nor improves patient outcomes and is no longer recommended. The use of suction (with a pressure of 20 cm H_20) is reserved for situations where the lung fails to reexpand after drainage through a water seal device or Heimlich valve for 24 to 48 hours.

Complications from chest tube placement include tube malposition, pleural space infection, and pain at the chest tube site. Reexpansion pulmonary edema and reexpansion hypotension are rare occurrences after evacuation of a large pneumothorax. Chest x-ray is routinely obtained after chest tube placement to assess for adequacy of placement and complications.

Tension Pneumothorax

If the clinical circumstances suggest a tension pneumothorax, treatment should be initiated prior to definitive diagnostic testing. Emergency management of tension pneumothorax includes rapid pleural decompression. This decompression may be accomplished by inserting a large bore intravenous catheter followed by tube thoracostomy or by immediate finger or tube thoracostomy. The determination of which procedure to perform should be guided by the setting, training of the care team, and available equipment.

Needle decompression is a temporizing procedure that releases high pressure air from the pleural space and restores hemodynamic stability. Definitive management is still required with a prompt tube thoracostomy. Needle decompression is completed by inserting a large bore IV catheter (14–16 gauge in adults, 18 gauge in children) of adequate length (at least 3–5 cm in adults) into the pleural space via the second intercostal space anteriorly at the midclavicular line or via the 4th/5th intercostal space at the midaxillary line. The diagnosis is confirmed by air escaping under positive pressure as the needle or chest tube enters the pleural space or by rapid hemodynamic improvement. The needle can then be removed, and the IV catheter remains in the chest wall open to air until definitive tube thoracostomy is completed. After tube thoracostomy, the catheter can be removed. In obese patients, a standard IV catheter may be of insufficient length to reach the pleural space and a longer needle or immediate finger or tube thoracostomy is required.

Disposition

Disposition decisions should consider the type and size of pneumothorax, underlying comorbidities, hemodynamic stability, and availability of follow-up. Admission to the hospital is generally not required for young, otherwise healthy patients with a small pneumothorax and no hypoxemia or hemodynamic abnormalities. After a period of observation (4–6 hours), these patients can be discharged home. Discharged patients should have the capability to return to an ED if their condition worsens and should undergo follow-up with a primary care provider or in the ED in 24 to 48 hours. Air travel and underwater diving must be strictly avoided until the pneumothorax has completely resolved, to prevent pressure-related pneumothorax expansion.

In most cases, chest tube management requires hospital admission although outpatient management of primary spontaneous pneumothorax with a small-caliber tube and Heimlich device is possible with good outpatient follow-up. Those with secondary spontaneous pneumothorax require either 24 hours of observation or admission for ongoing chest tube management.

Spontaneous pneumothorax usually resolves within 7 days of tube thoracostomy. Air leaks that persist beyond 2 days are less likely to resolve on their own. If the air leak persists beyond 4 to 7 days, tube thoracostomy is considered to have failed and surgical intervention is generally recommended. Failure of tube thoracostomy is more common in secondary spontaneous and recurrent pneumothorax because they tend to be associated with larger and more persistent air leaks.

Recurrence of spontaneous pneumothorax is common (approximately 30%). Younger age, lower weight/height ratio, and ongoing smoking are associated with increased risk of recurrence. There are a variety of operative and nonoperative interventions aimed at decreasing the rate of recurrence. Pleurodesis involves pleural instillation of a sclerosing agent or mechanical pleural abrasion to promote scarring of the parietal and visceral pleura, which obliterates the pleural potential space. Another strategy involves resection of apical bullae or other lesions at risk for causing recurrences. Often the two strategies are combined. Minimally invasive procedures, such as video-assisted thoracoscopic surgery (VATS), allow for resection of bullae and pleurodesis. Patients with extensive bullae may require thoracotomy for wider visualization of lesions.

Recurrence may be life-threatening for patients with serious underlying lung disease, and intervention may be used to prevent recurrence as part of the initial approach to secondary spontaneous pneumothorax. In contrast, for patients with primary spontaneous pneumothorax, interventions typically are not considered until after a second ipsilateral pneumothorax. However, those with a first episode of primary spontaneous pneumothorax with bullae greater than 2 cm have less recurrence when they undergo surgical resection after the first episode.[4] Preventive treatment is also recommended for patients who plan to continue activities that increase the risk of severe complications if a pneumothorax recurs, such as flying or diving.

PLEURAL EFFUSION

Foundations

Background and Importance

Pleural effusion is an abnormal collection of fluid in the pleural space. The most common causes of pleural effusion in the United States are congestive heart failure, malignancy, bacterial pneumonia, and pulmonary embolism. Tuberculosis remains a leading cause of pleural effusion in endemic areas. Other conditions commonly associated with pleural effusion include viral infections, cirrhosis, nephrotic syndrome, uremia, ovarian hyperstimulation syndrome, collagen vascular disease, myxedema, and intraabdominal inflammation. Esophageal perforation is a rare and uniquely morbid cause of pleural effusion (Box 63.2).

A pleural effusion that is associated with bacterial pneumonia or lung abscess is termed a *parapneumonic effusion*. The term *pleural empyema* (or pyothorax) implies the presence of pus within the pleural space. Fluid in the pleural space that is anatomically confined and is not free flowing within the pleural space is termed a *loculated effusion*. Loculated effusions occur when there are adhesions between the visceral and parietal pleurae. Traumatic hemothorax is a distinct type of pleural effusion that is approached in Chapter 37.

Anatomy and Physiology

Under normal circumstances, a thin layer of fluid lies between the visceral and parietal pleura. Pleural fluid is produced from systemic capillaries at the parietal pleural surface and absorbed into the pulmonary capillaries at the visceral pleural surface. Although lymphatics play an

BOX 63.2 Causes of Pleural Effusion

Transudates
Congestive heart failure
Cirrhosis with ascites
Nephrotic syndrome
Hypoalbuminemia
Myxedema
Peritoneal dialysis
Glomerulonephritis
Superior vena cava obstruction
Pulmonary embolism

Exudates
Infections
Bacterial pneumonia
Bronchiectasis
Lung abscess
Tuberculosis
Viral illness

Neoplasms
Primary lung cancer
Mesothelioma
Pulmonary or pleural metastases
Lymphoma

Connective Tissue Disease
Rheumatoid arthritis
Systemic lupus erythematosus

Abdominal or Gastrointestinal Disorders
Pancreatitis
Subphrenic abscess
Esophageal rupture
Abdominal surgery

Miscellaneous Conditions
Pulmonary infarction
Uremia
Drug reactions
Postpartum
Chylothorax

essential role in removing pleural fluid, the direction of pleural fluid flow is generally governed by the difference in hydrostatic pressures between the systemic and pulmonary circulations (Fig. 63.5). Under normal circumstances, pleural fluid exists in a dynamic equilibrium with approximately 1 L of fluid traversing the pleural space every 24 hours. The net accumulation of fluid in the pleural space is small (approximately 0.1–0.2 mL/kg body weight) and clinically insignificant. Pleural effusions develop when the influx of fluid into the pleural space exceeds the efflux.

Pathophysiology

Pleural effusions are classically divided into two groups according to the composition of the pleural fluid: transudates and exudates. Transudative effusions are ultrafiltrates of the plasma and contain very little protein. A transudative effusion develops due to an increase in hydrostatic pressure or decrease in oncotic pressure within the pleural microvessels. The most common cause of transudative effusion is congestive heart failure with an associated increase in hydrostatic pressure. Patients with severe malnutrition also develop transudative effusions because of profound hypoalbuminemia and loss of plasma oncotic pressure. Other conditions such as cirrhosis and nephrotic syndrome are also associated with an increase in hydrostatic pressure and loss of plasma oncotic pressure.

In contrast, exudative effusions contain a relatively high amount of protein, reflecting an intrinsic abnormality of the pleura. Any pleural or pulmonary disease associated with inflammation can result in an exudative effusion. The most common cause of exudative effusion is a parapneumonic effusion. Malignant effusions are another common form of exudative effusion and often reflect alteration in pleural permeability and altered lymphatic drainage. Exudative effusions may also arise from inflammatory abdominal conditions, such as pancreatitis. As an exudative effusion is resorbed, the fibrinous tissue left behind can give rise to ongoing inflammation and pleural adhesions.

Some pleural effusions have characteristics of both transudative and exudative pleural effusions. For example, in the case of PE, the pathogenesis of pleural effusion is multifactorial and involves increased pulmonary vascular pressure (leading to a transudative effusion) and ischemia and inflammation of the pleural membrane (leading to an exudative effusion).

Massive pleural effusions (>1.5–2 L) are usually associated with malignancy but can also arise in the setting of heart failure and in other conditions of volume overload. Massive effusions can restrict respiratory movement, compress the lung parenchyma, and result in intrapulmonary shunting. In extremely rare cases, tension hydrothorax can develop with mediastinal shift and circulatory compromise.

Clinical Features

Pleural inflammation, with or without effusion, is associated with pleuritic pain (i.e., sharp and worse with deep breathing) or with pain referred to the ipsilateral shoulder. Symptoms generally depend on the size of the effusion and underlying cause. Small pleural effusions are typically asymptomatic, and generally dyspnea does not develop until the volume of pleural fluid in adults reaches at least 500 mL.

Physical findings also depend on the size of the effusion. A pleural friction rub may be the only finding in a patient with isolated pleurisy, whereas patients with massive pleural effusions can have hemodynamic compromise. Classical physical signs of pleural effusion include diminished or distant breath sounds, dullness to percussion, and decreased tactile fremitus. The technique of auscultatory percussion (i.e., percussing the chest while listening for dullness with the stethoscope) may be more sensitive and specific for the physical diagnosis of pleural effusion. Egophony and enhanced breath sounds are often appreciated at the superior border of the effusion because of the underlying atelectatic lung disease.

Differential Diagnosis

The differential diagnosis of pleural effusion includes a wide variety of diseases characterized by dyspnea or chest pain and ranges from congestive heart failure and volume overload, pneumonia, pulmonary embolism, and pericardial effusion. Of note, many of these conditions may coexist with pleural effusions. The presence of a pleural effusion requires thoughtful consideration of the underlying cause. Specifically, an unexplained pleural effusion should raise concern for malignancy and requires follow-up.

Diagnostic Testing

The diagnosis of pleural effusion should be confirmed by chest X-ray, CT, or bedside ultrasound. A volume of approximately 200 mL is required before a pleural effusion can be demonstrated on an upright,

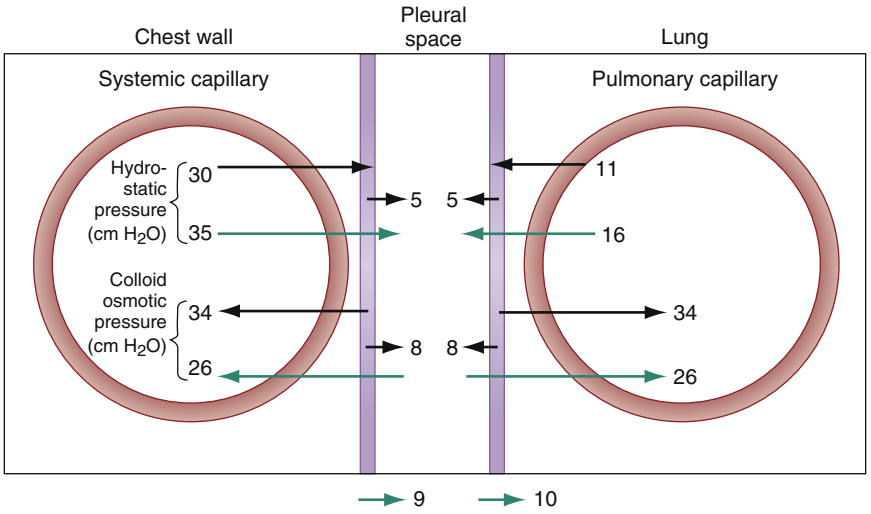

Fig. 63.5 Diagrammatic representation of the pressures involved in the formation and absorption of pleural fluid. (Adapted from Robert G, Fraser GA, Paré PD, et al. *Diagnosis of Diseases of the Chest*, ed 3, Philadelphia: WB Saunders; 1989.)

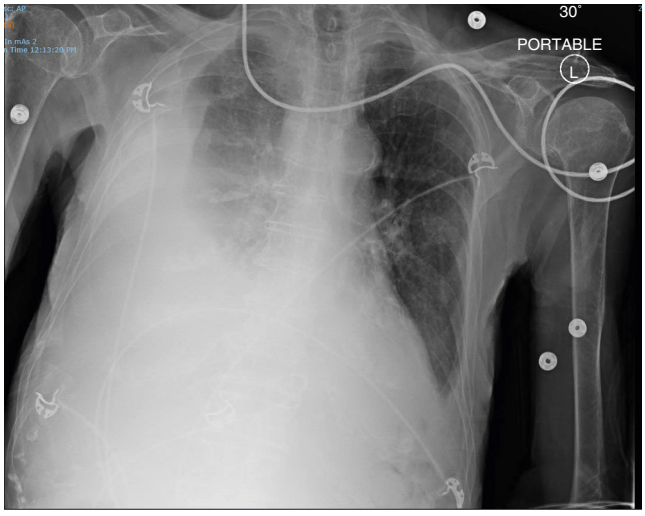

Fig. 63.6 Radiograph of a right-sided pleural effusion.

frontal chest x-ray. A smaller amount of fluid may be demonstrated in the posterior costophrenic gutter on the lateral projection. The classic radiographic appearance of a pleural effusion is blunting of the costophrenic angle. With larger pleural effusions, the hemidiaphragm may be obscured entirely, typically with an upward concave appearance because pleural fluid tends to layer higher laterally than centrally (Fig. 63.6). Pleural fluid can also extend up a major fissure and appear as a homogenous density in the lower portion of the lung field. Massive pleural effusions can completely opacify the hemithorax.

Pleural effusions are more difficult to diagnose on a supine radiograph. In the recumbent position, free pleural fluid gravitates superiorly, laterally, and posteriorly and thus, pleural effusions may not be clearly discernable. If the effusion is large enough, diffuse haziness or partial opacification of a hemithorax may be seen. Other findings on the supine radiograph may include apical capping, obscuring of the hemidiaphragm, or a widened minor fissure. Loculated fluid in a pleural fissure may assume a fusiform appearance and can simulate a mass. The lateral recumbent view historically has been useful for demonstrating small loculated effusions. It has been largely replaced by ultrasound or CT. Some pleural effusions can be challenging to diagnose on plain chest radiograph and further imaging may be required.

CT scan can detect as little as 3 to 5 mL of pleural fluid and is the gold standard for the diagnosis of small pleural effusions. CT is particularly useful in distinguishing between pleural based disease and parenchymal disease to identify an underlying cause (e.g., PE, malignancy, pneumonia). It can also help quantify the amount of fluid and may help guide thoracentesis.

Thoracic ultrasound is more sensitive than chest radiography in diagnosing and estimating the size of pleural effusion and is readily available at the bedside. Ultrasound can identify pleural effusions as small as 50 mL of fluid. On ultrasound, pleural effusions can be classified as simple or complex. Simple transudative pleural effusions often appear as hypoechoic fluid above the diaphragm and are best visualized with a curvilinear or phased array probe in the midaxillary line (Fig. 63.7). Often, compressed lung can be visualized within the effusion. Complex pleural effusions are subtyped as heterogenous or homogenous echogenicity. Hemothorax and pyothorax may appear heterogeneous, with echogenic material or septations extending through the effusion. Effusions with heterogenic echogenicity with swirling echoes suggest a high cellularity content often seen in malignant effusion. Fibrinous stranding, septations, and loculations also suggest an exudative effusion.

Identification of fluid above the diaphragm, compressed lung tissue, as well as localization of the diaphragm, liver, and spleen can help aid in the correct localization for thoracentesis or tube thoracostomy. If available, ultrasound should be used to identify pleural effusions and to guide interventions, such as thoracentesis. Iatrogenic pneumothorax is the most common complication after a thoracentesis, and it can be significantly reduced by using ultrasound guidance.[5]

Most patients with a pleural effusion should undergo diagnostic thoracentesis to determine the nature of the effusion (i.e., transudate, exudate) and identify an underlying cause. Occasionally, the clinical scenario provides an obvious apparent cause (e.g., congestive heart failure) and thoracentesis is unlikely to contribute to changes in therapy. Not all pleural effusions need to undergo thoracentesis in the ED. In the ED, thoracentesis may be performed to evaluate the etiology of life-threatening pathology (e.g., empyema, esophageal rupture) or to

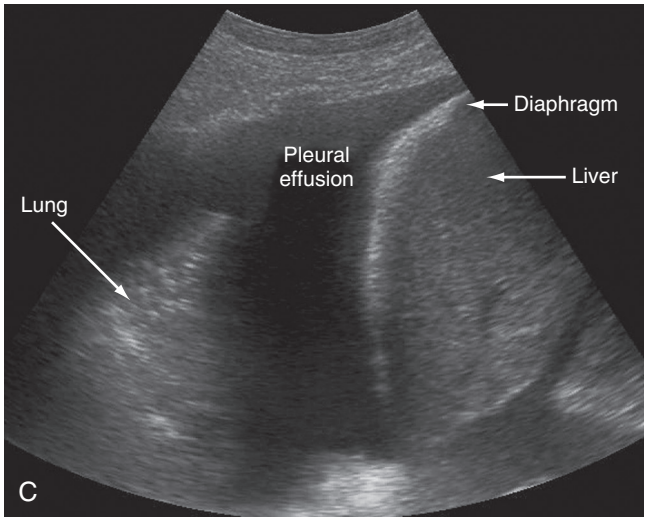

Fig. 63.7 Ultrasound image of the right upper quadrant demonstrating the typical hypoechoic appearance of a pleural effusion.

BOX 63.3 Light's Criteria for Differentiating Transudates from Exudates

Pleural fluid is considered an exudate if one or more of the following conditions are met:

1. Pleural fluid protein level/serum protein level exceeds 0.5.
2. Pleural fluid lactate dehydrogenase (LDH) level/serum LDH level exceeds 0.6.
3. Pleural fluid LDH level exceeds two-thirds of the upper limit of normal for the serum LDH level.

provide urgent symptomatic relief. In many other cases, diagnostic thoracentesis may be deferred.

Although numerous classifications have been proposed, Light's criteria remain the most widely accepted means of differentiating transudates and exudates (Box 63.3). Briefly, these criteria identify exudative effusions as being protein-rich and high in lactate dehydrogenase (LDH). A pleural fluid pH of less than 7.3 is associated with parapneumonic effusion, malignancies, rheumatoid effusions, tuberculosis, and systemic acidosis. A pH of less than 7.0 strongly suggests empyema or esophageal rupture and is an indication for tube thoracostomy. Pleural fluid should also be sent for Gram stain and culture to rule out empyema or parapneumonic effusion. If the diagnosis of malignant pleural effusion is being considered, and tumor is not already diagnosed, pleural fluid should be submitted for cytologic examination. The sensitivity for diagnosis of pleural malignancy does not depend on the volume of fluid collected during thoracentesis.

In the absence of a traumatic thoracentesis, bloody fluid suggests trauma, neoplasm, or pulmonary infarction. If the hematocrit of the pleural fluid is greater than 50% of the peripheral blood, the effusion is, by definition, a hemothorax. Atraumatic hemothorax is relatively rare but can occur with spontaneous rupture of a tumor or blood vessel.

Management

Most pleural effusions do not require emergent drainage, and there are few indications for urgent therapeutic thoracentesis in the emergency department. For the patients with massive effusions (>1.5–2 L), urgent thoracentesis may stabilize respiratory or circulatory status. Patients with empyema require timely chest tube drainage in the ED, operating room, or with interventional radiology in order to obtain source control and prevent complications. Traditionally, large-bore (28–40 Fr) chest tubes were placed in patients with empyema; however, small-bore tubes such as pigtail catheters (14 Fr) are now generally accepted as first-line therapy.[6] In most other cases, the timing of therapeutic thoracentesis can be individualized. For example, therapeutic thoracentesis would be reasonable to perform in the ED for a recurrent malignant pleural effusion if symptomatic relief would allow for discharge or would alter the diagnostic dyspnea workup.[7]

Relative contraindications to thoracentesis include coagulopathy and other bleeding disorders, history of pleurodesis, and chest wall infections. Pleural adhesions are also a relative contraindication to thoracentesis due to the potential for pneumothorax, but this risk can be minimized with ultrasound guidance.

Following a diagnostic or therapeutic thoracentesis, a chest radiograph or bedside ultrasound assessment should be obtained to evaluate for complications such as iatrogenic pneumothorax. Other potential complications include hemothorax, lung laceration, shearing of the catheter tip, infection, and transient hypoxia due to ventilation-perfusion mismatch. Hypotension can also occur after the removal of large volumes of fluid, particularly in patients that are already volume depleted.

Reexpansion pulmonary edema is a rare complication of thoracentesis and is traditionally correlated with large volume (>1500 mL) drainage. The symptoms of reexpansion pulmonary edema are generally mild and can be managed with supplemental oxygen and gentle diuresis. Safe aspiration of larger volumes of fluid has been described while using pleural manometry, however complications are rare when aspirating greater than 1500 mL during a single thoracentesis attempt. Therefore, the British Thoracic Society recommends removing no more than 1500 mL of pleural fluid in a single procedure to reduce the risk of reexpansion pulmonary edema (Grade C).

Disposition

The natural progression of pleural effusions is determined largely by the underlying diagnosis. The decision to admit a patient with a pleural effusion to the hospital must be individualized, considering the patient's underlying diagnosis, respiratory and hemodynamic status, and predicted clinical course. For example, small pleural effusions are common after abdominal surgery and in the postpartum state, but they almost always resolve spontaneously within a few days. Viral pleuritis, with or without effusion, is generally self-limited and resolves with only symptomatic support. In patients with congestive heart failure, pleural effusions generally respond well to diuresis, but patients may require admission for intravenous diuretics. Effusions may persist in patients with poorly compensated disease. In nearly 20% of pleural effusions, no definitive diagnosis can be established, even after investigation, and most of these effusions resolve spontaneously without sequelae.

Parapneumonic effusions contribute significantly to the morbidity and mortality associated with pleural disease. For this reason, the presence of a parapneumonic effusion is a reason to hospitalize a patient with community-acquired pneumonia. Empyema will develop in 5% to 10% of parapneumonic effusions and early surgical drainage results in better outcomes than conservative management.

Pleural effusions associated with malignancy are a marker of significant morbidity and mortality. Therapeutic thoracentesis can provide symptomatic relief in the short term, but malignant effusions tend to recur and often do so rapidly. Close management of recurrent malignant pleural effusions improves the patient's quality of life. Strategies for managing recurrent pleural effusions include chemical or mechanical

pleurodesis to obliterate the pleural space or placement of a permanent indwelling catheter or pleural peritoneal shunt to provide continued drainage.[7] Consistent use of definitive procedures in recurrent pleural effusions leads to fewer ED performed procedures and fewer complications when compared with repeat thoracentesis.[8] Patients with benign but recurrent pleural effusions refractory to other treatments may also benefit from chemical or mechanical pleurodesis or placement of an indwelling pleural catheter.[9]

The references for this chapter can be found online at ExpertConsult.com.

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CHAPTER 63: QUESTIONS AND ANSWERS

1. What is the most common condition associated with secondary spontaneous pneumothorax in adults in the United States?

a. Chronic obstructive pulmonary disease
b. Collagen vascular disease
c. *Pneumocystis* pneumonia
d. Pulmonary malignancy

Answer: A. Chronic obstructive pulmonary disease is the most common condition associated with secondary spontaneous pneumothorax, although all the conditions listed may also be causes.

2. Which of the following are indications of a pneumothorax on bedside ultrasound?

a. Identification of a lung point
b. Lack of lung sliding identified
c. When in M-mode, you see a "barcode" appearance
d. All of the above

Answer: D. Pneumothorax may be difficult to identify on bedside ultrasound and there are several ways to help identify a pneumothorax. Identifying an area of lung that lacks lung sliding and has a "barcode" appearance on M-mode indicates that the parietal and visceral pleura are not sliding over each other and is highly suggestive of a pneumothorax. Identifying a lung point, the point where a pneumothorax transitions to a normal pleural interface, can also help identify a pneumothorax.

3. A 32-year-old male with no significant past medical history presents with acute onset of right pleuritic chest pain, cough, and shortness of breath. The symptoms occurred at rest. Physical examination is remarkable for a tachycardia of 110 beats/min and respiratory rate of 32 and use of accessory muscles to breathe. Chest radiography reveals an estimated 5-cm right pneumothorax. Which of the following would be suitable management?

a. Admission for 100% face mask oxygen and repeat radiography in 1 day
b. One-time air aspiration and repeat radiography in 6 hours
c. Reassurance and observation
d. Tube thoracostomy

Answer: D. Large (2 cm) primary spontaneous cases and patients who are showing signs of respiratory distress should be treated with tube thoracostomy. If the primary spontaneous pneumothorax is small (<2 cm), then observation alone is appropriate. For a larger primary spontaneous pneumothorax (> 2cm) without significant symptoms, either observation alone or intervention with either simple aspiration or tube thoracostomy may be appropriate and a shared decision-making discussion with your patient is appropriate to determine best course of action.

4. Which of the following statements is true regarding the routine application of suction after tube thoracostomy?

a. It improves the rate of lung expansion.
b. It increases the risk of reexpansion pulmonary edema.
c. It is associated with increased rates of empyema.
d. It is not routinely indicated.

Answer: D. Suction neither accelerates lung reexpansion nor improves outcomes. It is indicated when reexpansion with a Heimlich or water seal device does not occur after 24 to 48 hours. A pressure of at least −20 cm H_2O should be used.

5. A 68-year-old man with a history of esophageal cancer presents with progressive fever, chest pain, and shortness of breath over 24 hours. Chest radiography demonstrates a possible left lower lobe pneumonia and large left pleural effusion. Pleural fluid analysis reveals pH of 6.95, glucose level of 47 mg/dL, 11,500 white blood cells (WBCs)/mm^3 (82% neutrophils), and protein level 75% of plasma levels. What are the indicated next steps in management?

a. Antibiotics and fluid resuscitation
b. Antibiotics and tube thoracostomy
c. Antibiotics, tube thoracostomy, and esophageal oral contrast study
d. One-time pleural aspiration for fluid analysis

Answer: C. This is an exudative pleural effusion as defined by Light's criteria (see Box 63.3). A pH less than 7.0 suggests emphysema or esophageal rupture. This patient is at risk for both; hence, the need to assess esophageal integrity. A pH less than 7.0 with glucose level less than 50 mg/dL are indications for tube thoracostomy. Normal pleural fluid has a WBC count of less than 1,000/mm^3.