Pleural Disease

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Pleural disease is commonly encountered in the emergency department (ED). Presentations range in severity from asymptomatic pleural effusion to tension pneumothorax. This chapter reviews the two most common nontraumatic pleural problems: spontaneous pneumothorax and pleural inflammation with effusion. Pleural space problems associated with trauma are discussed in Chapter 17, and the approach to a patient presenting with pleuritic chest pain in Chapter 17.

SPONTANEOUS PNEUMOTHORAX

Perspective

Under normal conditions, the visceral and parietal pleurae lie in close apposition, with only a potential space between them. Pneumothorax is defined as the presence of free air in the intrapleural space. A spontaneous pneumothorax occurs in the absence of any external precipitating factor, either traumatic or iatrogenic. Primary spontaneous pneumothorax occurs in individuals without clinically apparent lung disease. Secondary spontaneous pneumothorax arises in the context of an underlying pulmonary disease process.

The incidence of primary spontaneous pneumothorax is approximately 15 cases per 100,000 population per year among men and 5 cases per 100,000 population per year among women. Primary spontaneous pneumothorax typically occurs in healthy young men of taller than average height. Factors associated with primary spontaneous pneumothorax include cigarette smoking and changes in ambient atmospheric pressure. Familial patterns suggest an inherited propensity in some cases of primary spontaneous pneumothorax. Mitral valve prolapse and Marfan’s syndrome are associated with spontaneous pneumothorax in the absence of clinically apparent lung disease.

Approximately one third of spontaneous pneumothoraces occur in the context of underlying pulmonary disease (Box 75-1). The incidence of secondary spontaneous pneumothorax is three times higher in men. The most common condition associated with secondary spontaneous pneumothorax is chronic obstructive pulmonary disease (COPD), which accounts for nearly 70% of cases. Patients with severe COPD (e.g., with forced expiratory volume in 1 second <1 L) are at highest risk. The incidence of spontaneous pneumothorax among patients hospitalized for emphysema is 0.8% and for asthma 0.3%.

Spontaneous pneumothorax occurs in approximately 2% of patients with acquired immunodeficiency syndrome, almost always in the setting of Pneumocystis jiroveci (previously known as Pneumocystis carinii) pneumonia.1 Bilateral pneumothoraces are common with P. jiroveci pneumonia, as are problems with delayed reexpansion and recurrences. Mortality in these patients is high.2

Malignancy is another common etiology of secondary spontaneous pneumothorax. The occurrence of spontaneous pneumothorax in a patient with known malignancy should suggest lung metastases. In developing countries, tuberculosis and lung abscess remain leading causes of secondary spontaneous pneumothorax.

Catamenial pneumothorax is a rare condition in which recurrent spontaneous pneumothorax occurs in association with menses (typically within 72 hours of onset).3 Although it is termed thoracic endometriosis syndrome and often responds to ovulation-suppressing medications, the exact etiology of catamenial pneumothorax is uncertain.

Spontaneous pneumothorax is rare in childhood. The principles of diagnosis, imaging, treatment, and surgical management for pediatric primary spontaneous pneumothorax are similar to those for adult pneumothorax.4

Pathophysiologic Principles

Normally, intrapleural pressure is negative (less than atmospheric), fluctuating from −10 mm Hg to −12 mm Hg during inspiration to approximately −4 mm Hg during expiration. Intrabronchial and intra-alveolar pressures are negative during inspiration (−1 to −3 mm Hg) and positive during expiration (+1 to +3 mm Hg). The alveolar walls and visceral pleura form a barrier that separates the intrapleural and intra-alveolar spaces and maintains the pressure gradient. If a defect occurs in this barrier, air enters the pleural space until either the pressures equalize or the communication seals.

With the loss of negative intrapleural pressure in one hemithorax, the ipsilateral lung collapses. A large pneumothorax results in restrictive ventilation impairment, with reduced vital capacity, functional residual capacity, and total lung capacity. Shunting of blood through nonventilated lung tissue may result in acute hypoxemia, although over time this effect is mitigated by compensatory vasoconstriction in the collapsed lung.

In tension pneumothorax, the alveolar-pleural defect acts as a one-way valve, allowing air to pass into the pleural space during inspiration and trapping it there during expiration (Fig. 75-1). This trapping leads to progressive accumulation of intrapleural air and increasingly positive intrapleural pressure,
BOX 75-1  CAUSES OF SECONDARY SPONTANEOUS PNEUMOTHORAX

Airway Disease
- Chronic obstructive pulmonary disease
- Asthma
- Cystic fibrosis

Infections
- Necrotizing bacterial pneumonia/lung abscess
- Pneumocystis jiroveci pneumonia
- Tuberculosis

Interstitial Lung Disease
- Sarcoidosis
- Idiopathic pulmonary fibrosis
- Lymphangioleiomyomatosis
- Tuberculosis
- Pneumoconioses

Neoplasms
- Primary lung cancers
- Pulmonary/pleural metastases

Miscellaneous
- Connective tissue diseases
- Pulmonary infarction
- Endometriosis/catamenial pneumothorax

Figure 75-1. 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 expiration and cannot escape during inspiration.

causes compression of the contralateral lung with asphyxia and worsening hypoxia. Intrapleural pressure exceeding 15 to 20 mm Hg impairs venous return to the heart. If allowed to progress, cardiovascular collapse and death ensue.

In primary spontaneous pneumothorax, disruption of the alveolar-pleural barrier occurs when a subpleural bulla (or bleb), typically located at the lung apex, ruptures into the pleural space. Subpleural bullae are found in almost all patients who undergo surgical treatment for primary spontaneous pneumothorax and are identified on computed tomography (CT) of the chest in 90% of cases. The etiology of these bullae may be related to degradation of elastic fibers within the lung and an imbalance in the protease-antiprotease and oxidant-antioxidant systems.

In the case of secondary spontaneous pneumothorax, the underlying lung disease weakens the alveolar-pleural barrier. In patients with P. jiroveci pneumonia, the cytotoxic effects of repeated episodes of inflammation lead to bullous and cystic changes. In patients with COPD, chronic exposure to cigarette smoke results in the development of large, thin-walled bullae that are at an increased risk for rupture. Other factors, including increased intrabronchial and intra-alveolar pressures generated by bronchospasm and coughing, also play a role.

Clinical Features

Symptoms of primary spontaneous pneumothorax typically begin suddenly while at rest. Ipsilateral chest pain and dyspnea are the most common symptoms. At the outset, the pain is typically “pleuritic” in nature (i.e., often described as sharp and made worse with deep inspiration), but it often evolves over time into a dull, steady ache. Although patients frequently describe shortness of breath, extreme dyspnea is uncommon in the absence of underlying lung disease or tension pneumothorax. Cough is present in a few individuals. Occasionally, patients are asymptomatic or have only nonspecific complaints. Patients may wait several days before they seek medical attention, and a significant number delay presentation for 1 week or more. Without treatment, symptoms often resolve spontaneously within 24 to 72 hours, although the pneumothorax is still present.

Physical findings tend to correlate with the degree of symptoms. A mild sinus tachycardia is the most common physical finding. With a large pneumothorax, decreased or absent breath sounds with hyperresonance to percussion may be present. Other classic signs include unilateral enlargement of the hemithorax, decreased excursion with respirations, absent tactile fremitus, and inferior displacement of the liver or spleen. Absence of any or all of these findings does not exclude pneumothorax, however, and a chest radiograph should be obtained when pneumothorax is suspected.

With tension pneumothorax, signs of asphyxia and decreased cardiac output develop. Tachycardia (often >120 beats/min) and hypoxia are common. Hypotension is a late and ominous finding. Distention of the jugular veins is common but may be difficult to detect. Displacement of the trachea to the contralateral side is classically described but is an uncommon finding, usually occurring only in the immediately preterminal phase of the pneumothorax, if at all. Its absence should not be considered evidence that a tension phenomenon is not present.

In patients with significant underlying lung disease, pneumothorax presents differently. Because of poor pulmonary reserve, dyspnea is nearly universal, even when the pneumothorax is small, and symptoms tend not to resolve on their own. Physical findings, such as hyperexpansion and distant breath sounds, often overlap considerably with the underlying lung disease, making the clinical diagnosis difficult. For this reason, the diagnosis of pneumothorax should be considered whenever a patient with COPD presents with an exacerbation of dyspnea.

Although suggested by the patient’s history and physical examination, the diagnosis of pneumothorax is generally made with the chest radiograph. The classic radiographic appearance is that of a thin, visceral pleural line lying parallel to the chest wall, separated by a radiolucent band devoid of lung markings. The average width of this band can be used to estimate the size of the pneumothorax with a fair degree of accuracy (Fig. 75-2), but in general, it is more reasonable simply to characterize the pneumothorax as small, moderate, large, or total. The estimated size of the pneumothorax and the patient’s clinical status can be useful in guiding management decisions.

Tension pneumothorax is a clinical diagnosis, and delaying treatment to obtain radiographic confirmation is inadvisable. When the diagnosis of tension pneumothorax is not apparent clinically and a chest radiograph 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 mediastinal
Determination of average interpleural distance to predict pneumothorax size. PA, posteroanterior.

**Figure 75-2.** Determining the size of a pneumothorax. Calculation of average interpleural distance to predict pneumothorax size. PA, posteroanterior.

**Figure 75-3.** Radiograph of tension pneumothorax with mediastinal shift to left.

structures across the midline (Fig. 75-3). In patients with underlying pulmonary disease, however, pleural adhesions and lack of lung elasticity may mask the fact that a pneumothorax is under significant positive pressure.

When pneumothorax is suspected but not seen on a standard chest radiograph, an expiratory film may be obtained. Theoretically, the volumes of the lungs and the chest cavity are reduced during expiration so that the relative size of the pneumothorax is enhanced. Although occasionally helpful in identifying a small apical pneumothorax, routine use of expiratory films does not improve diagnostic yield.7 In critically ill patients for whom only a supine chest radiograph can be obtained, the finding of a “deep sulcus” (i.e., a deep lateral costophrenic angle) can suggest the presence of pneumothorax on that side (see Fig. 75-3).

Special care should be taken when viewing the chest radiographs of patients with underlying lung disease. In patients with COPD, the relative paucity of lung markings makes pneumothorax more difficult to detect. At the same time, giant bullae may simulate the radiographic appearance of pneumothorax. A clue to differentiating a pneumothorax from a giant bulla is that the former tends to run parallel to the chest wall, whereas the latter tends to have a more concave appearance. When the diagnosis is unclear, computed tomography (CT) can differentiate between the two entities.8

While CT is considered the gold standard for the diagnosis of pneumothorax, it requires that patients be stable enough for transport. Bedside ultrasound is also a rapid and accurate diagnostic aid.9,10 Assessment for pneumothorax begins over the upper anterior chest wall in the midclavicular line and proceeds inferolaterally toward the anterior axillary line. Once the pleural line is identified, the presence of lung sliding during respiration effectively rules out a pneumothorax in the area being scanned. The differential diagnosis of pneumothorax includes numerous conditions associated with chest pain and dyspnea. Among the most important of these is pulmonary embolism, which may present in similar fashion with unilateral pleuritic chest pain. Most pleural-based processes (pneumonia, embolism, tumor) have characteristic radiographic findings. Rarely, pneumothorax may mimic an acute myocardial infarction with electrocardiogram changes simulating an acute injury pattern.11

Spontaneous pneumomediastinum is a closely related clinical entity, diagnosed by the presence of subcutaneous emphysema and the finding of mediastinal air on chest radiography. In contrast to spontaneous pneumothorax, spontaneous pneumomediastinum typically occurs during exertion, particularly after a strenuous Valsalva maneuver. Most cases of spontaneous pneumomediastinum occur in the absence of known underlying disease and have a benign course. Secondary causes of pneumomediastinum (e.g., Boerhaave’s syndrome) are more serious, and treatment is aimed at the underlying disorder.

Spontaneous hemopneumothorax is a rare but potentially serious condition that occurs when collapse of the lung is associated with rupture of a vessel in a parietopleural adhesion. The clinical presentation is similar to that of spontaneous pneumothorax but may be accompanied by symptoms and signs of hemorrhagic shock. Treatment entails large-caliber tube thoracostomy to evacuate the pleural space, reexpand the lung, and tamponade bleeding.

Pneumothorax has a readily available, highly reliable confirmatory diagnostic test (i.e., chest radiography). Absence of a pneumothorax on chest radiography should prompt a search for an alternate diagnosis.
Management

Whether in the field or in the ED, if the clinical circumstances suggest tension pneumothorax, treatment should not be delayed by awaiting further cardiovascular compromise or definitive diagnosis by chest radiography. As soon as tension pneumothorax is believed to be present, the pleural space should be decompressed. This decompression may be accomplished by insertion of an intravenous catheter or by immediate tube thoracostomy, depending on the availability of equipment and the expertise of the providers. The diagnosis is confirmed by the hiss of air escaping under positive pressure as the needle or chest tube enters the pleural space. Needle decompression is only a temporizing procedure, and definitive management requires prompt tube thoracostomy. In morbidly obese patients, the needle and catheter may be of insufficient length to reach the pleural space, and a longer needle may be required.

The management of spontaneous pneumothorax has two goals: (1) to evacuate air from the pleural space, and (2) to prevent recurrence. Pursuit of the latter goal extends well beyond the realm of the ED but may influence the initial approach to management. Therapeutic options for treatment of pneumothorax range from simple observation or aspiration with a catheter to video-assisted thoroscopic surgery or thoracotomy. Decisions must be individualized and consider several factors, including size of the pneumothorax, severity of signs, presence of underlying pulmonary disease, other comorbidities, history of previous pneumothoraces, patient reliability, degree and persistence of the air leak, and available follow-up monitoring.

For otherwise healthy, young patients with a small primary spontaneous pneumothorax (i.e., <20% of the hemithorax), observation alone may be appropriate. The intrinsic reabsorption rate ranges from 1 to 2% per day, a rate that is accelerated by a factor of 4 with the administration of 100% oxygen. By lowering the alveolar partial pressure of nitrogen, supplemental oxygen increases the rate at which air diffuses across the pleural-alveolar barrier. The disposition of patients managed noninterventionally for a small pneumothorax varies by institution. Most physicians admit these patients for at least 6 hours of observation, often in an ED-based observation unit. A repeat chest radiograph can be obtained before discharge to document that there is no increase in the size of the pneumothorax. Discharged patients should be able to obtain emergency medical services quickly and should have definitive follow-up evaluation in 24 hours. Air travel and underwater diving must be avoided until the pneumothorax has completely resolved. Unreliable patients are not candidates for this approach.

For primary spontaneous pneumothoraces that are larger in size (i.e., ≥20% of the hemithorax), aspiration with an intravenous catheter may be attempted. If 6 hours after aspiration the chest radiograph shows no reaccumulation of the pneumothorax, the catheter is removed, and the patient can be discharged home, with the same caveats that apply to patients managed with observation alone.

Although there is no universal agreement on the optimal treatment of patients presenting with a first episode of primary spontaneous pneumothorax, data suggest that aspiration may be equally effective as chest tube drainage. Advantages of simple aspiration include low morbidity, lack of invasiveness, and overall cost savings, with reported rates of successful outcome ranging from 45% to 71%. Success is less likely when the patient is older than 50 years or the volume of air aspirated exceeds 2.5 L, suggesting a continuing air leak. If aspiration fails to reexpand the lung fully, the catheter can be attached to a water-seal device or to a one-way Heimlich valve and managed like a small-caliber chest tube.

Most secondary spontaneous pneumothoraces should be managed with tube thoracostomy because less invasive approaches (i.e., observation or simple aspiration) are associated with significantly lower success rates. Similarly, patients who present with respiratory distress, have tension pneumothorax, or are likely to require mechanical ventilation should undergo tube thoracostomy to reexpand the lung definitively. Also, if there is detectable pleural fluid (hemothorax or hydrothorax), tube thoracostomy is required. Finally, tube thoracostomy may be considered in uncomplicated cases of primary spontaneous pneumothorax either as a first-line intervention or after a less invasive approach (i.e., observation or simple aspiration) fails.

For most primary spontaneous pneumothoraces, placement of a small-caliber (7–14F) tube is generally sufficient because air leakage tends to be minimal. Small-caliber tubes are easy to insert, are well tolerated by patients, and leave only a small scar after removal. Complications associated with small-caliber tubes include kinking, malposition, inadvertent removal, occlusion by pleural fluid or clotted blood, and large persistent air leaks. For secondary spontaneous pneumothorax, a standard size (20–28F) thoracostomy tube is recommended. When there is detectable pleural fluid or an anticipated need for mechanical ventilation, a larger tube size (≥28F) is required.

After insertion, the tube is attached to a water-seal device and left in place until the lung has reexpanded fully and the air leak has ceased. A Heimlich valve, which consists of a one-way flutter valve covered in transparent plastic, can be used in place of a water-seal device and allows unhindered ambulation. Specific complications associated with the use of a Heimlich valve include accidental disconnection and occlusion by fluid.

Routine application of suction neither increases the rate at which the lung reexpands nor improves patient outcome and is no longer recommended after standard tube thoracostomy. Rather, the use of suction (with a pressure of 20 cm H₂O) is reserved for situations in which the lung fails to reexpand after drainage through a water-seal device or Heimlich valve for 24 to 48 hours.

In most cases, chest tube management requires hospital admission, although outpatient management of spontaneous pneumothorax with a small-caliber tube and Heimlich device is described. Common complications of chest tube placement include incorrect placement, pleural infection, and prolonged pain. Reexpansion pulmonary edema and reexpansion hypotension are rare occurrences after rapid evacuation of large pneumothoraces.

Outcome

Most spontaneous pneumothoraces resolve within 7 days of tube thoracostomy. Air leaks that persist for longer than 2 days are less likely to resolve on their own. If an air leak persists beyond 4 to 7 days, tube thoracostomy is considered to have failed, and surgical intervention generally is recommended.

Failure of tube thoracostomy is more common with secondary spontaneous pneumothoraces because these tend to be associated with larger and more persistent air leaks. In the setting of COPD, healing of the alveolar-pleural barrier may be impaired by chronic inflammatory changes and loss of vascularity in pulmonary tissue. The success rate also decreases substantially with recurrent episodes of pneumothorax, declining from 91% for treatment of a first pneumothorax to

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outcome ranging from 45% to 71%. Success is less likely when the patient is older than 50 years or the volume of air aspirated exceeds 2.5 L, suggesting a continuing air leak. If aspiration fails to reexpand the lung fully, the catheter can be attached to a water-seal device or to a one-way Heimlich valve and managed like a small-caliber chest tube.

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52% for treatment of a first recurrence and to 15% for treatment of a second recurrence. 25

Recurrences of spontaneous pneumothorax are common. The risk of recurrence after a primary spontaneous pneumothorax is approximately one in three, with studies reporting rates between 16 and 50%.26 Younger age, lower weight-to-height ratio, and history of smoking are associated with an increased rate of recurrence. Recurrence rates after a secondary spontaneous pneumothorax are slightly higher (39–47%).6

Recurrences may be life-threatening for patients with serious underlying lung disease, and intervention is advocated 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. Preventive treatment also is recommended for patients who plan to continue activities such as flying or diving that increase the risk of serious complications if a pneumothorax recurs. CT can be used in primary spontaneous pneumothorax to detect emphysematous changes, predict the likelihood of recurrence, and guide intervention decisions.27

A variety of operative and nonoperative interventions prevent recurrences. One strategy promotes adherence of parietal and visceral pleura, which obliterates the pleural space. Pleurodesis can be accomplished by mechanical pleural abrasion or by instillation of sclerosing agents. 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, allow for resection of bullae and pleurodesis.26 Patients with extensive bullae may require thoracotomy for wider visualization of lesions. Success rates are generally good, ranging from 86 to 100%.

**PLEURAL INFLAMMATION AND EFFUSION**

**Perspective**

Under normal circumstances, a thin layer of fluid lies between the visceral and the parietal pleura, which obliterates the pleural space. Pleural effusion implies the presence of an abnormally large amount of fluid in the pleural space. Pleural effusions are relatively common.29 The most common cause of pleural effusions in Western countries is congestive heart failure, followed by malignancy, bacterial pneumonia, and pulmonary embolism.29 In other countries, tuberculosis is the leading cause of pleural effusions. Other conditions commonly associated with pleural effusions include viral infections of the lung parenchyma or pleura, uremia, myxedema, cirrhosis, nephrotic syndrome, ovarian hyperstimulation syndrome, collagen vascular diseases (e.g., systemic lupus erythematosus, rheumatoid arthritis), and intra-abdominal processes (e.g., acute pancreatitis, subphrenic abscess, ascites). Esophageal perforation is a rare but uniquely morbid cause of a pleural effusion.

Pleuritis (also referred to as pleurisy) is a nonspecific term denoting inflammation of the pleura. Pleuritis can occur with or without significant exudation of fluid into the pleural space. Pleuritis is a common presentation for a range of disease processes, from self-limited viral syndromes to more serious acute conditions, such as pneumonia and pulmonary embolism, to chronic illnesses, such as systemic lupus erythematosus and other connective tissue diseases.

A pleural effusion associated with bacterial pneumonia, bronchiectasis, or lung abscess is called a parapneumonic effusion. The term complicated parapneumonic effusion refers to parapneumonic effusions that require tube thoracostomy for their resolution. Empyema (or pus in the pleural space) requires the presence of bacteria on Gram’s staining of the pleural fluid.

Fluid anatomically confined and not freely flowing in the pleural space is termed a localized effusion. Loculated effusions occur when there are adhesions between the visceral and the parietal pleurae. Hemothorax and chylothorax (i.e., from rupture of the thoracic duct) are special instances of pleural effusion that are approached separately.

**Pathophysiologic Principles**

Pleural fluid is produced from systemic capillaries at the parietal pleural surface and absorbed into pulmonary capillaries at the visceral pleural surface. Lymphatics also play an important role in removing pleural fluid. Movement of fluid across the pleural surfaces is governed by Starling’s law. Under normal circumstances, the direction of pleural fluid flow is largely governed by the difference in hydrostatic pressure between the systemic and the pulmonary circulations (Fig. 75-4). Pleural fluid exists in a dynamic equilibrium in which influx equals efflux, with approximately 1 L of fluid traversing the pleural space in 24 hours. Under normal conditions, the amount of fluid that remains in the pleural space is small (~0.1–0.2 mL/kg body weight) and clinically or radiographically undetectable. Pleural effusion develops whenever influx of fluid into the pleural space exceeds efflux. Numerous disorders can lead to formation of a pleural effusion. Pleural effusions classically are

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**Figure 75-4.** Diagram representing pressures involved in formation and absorption of pleural fluid. (Modified from Fraser RG, et al: Diagnosis of Diseases of the Chest, 3rd ed. Philadelphia, WB Saunders, 1988.)

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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/pleural metastases
  - Lymphoma
- **Connective Tissue Disease**
  - Rheumatoid arthritis
  - Systemic lupus erythematosus
- **Abdominal/Gastrointestinal Disorders**
  - Pancreatitis
  - Subphrenic abscess
  - Esophageal rupture
  - Abdominal surgery
- **Miscellaneous**
  - Pulmonary infarction
  - Uremia
  - Drug reactions
  - Postpartum
  - Chylothorax

Transudates are essentially ultrafiltrates of plasma, containing very little protein. A transudative effusion develops when there is an increase in the hydrostatic pressure or decrease in the oncotic pressure within pleural microvessels. The primary cause of increased hydrostatic pressure is congestive heart failure, which is responsible for about 90% of transudative effusions. In hepatic cirrhosis and nephrotic syndrome, increased hydrostatic pressure is combined with loss of plasma oncotic pressure because of significant decreases in serum albumin. Patients with severe malnutrition may develop transudative effusions resulting from severe isolated hypoalbuminemia.

Exudates contain relatively high amounts of protein, reflecting an abnormality of the pleura itself. An exudative effusion is the result of increased membrane permeability or defective lymphatic drainage. Any pulmonary or pleural process associated with inflammation can result in an exudative effusion. In the absence of clinically apparent effusion, pleuritic symptoms may still be present. The most common form of exudative effusion is a parapneumonic effusion, in which infection of the adjacent lung elicits an intense inflammatory response in the pleura, disrupting normal membrane permeability. Malignant effusions are the second most common form of exudative effusion and often reflect alterations in pleural permeability and problems with lymphatic drainage. Exudative effusions also may arise in response to inflammatory abdominal processes, such as pancreatitis or subphrenic abscess, presumably owing to altered permeability of the diaphragm itself. Exudative effusions may be reabsorbed or organize into fibrous tissue, resulting in pleural adhesions.

Some pleural effusions can present as either transudates or exudates or may have characteristics of both. In the case of pulmonary embolism, the pathogenesis of pleural effusion is often multifactorial, reflecting increased pulmonary vascular pressure (a transudative process) and ischemia and breakdown of the pleural membrane (an exudative process).

Massive effusions (>1.5–2 L) are most commonly associated with malignancy but also can arise in the setting of congestive heart failure, cirrhosis, and other conditions. Massive effusions may restrict respiratory movement, compress the lungs, and result in intrapulmonary shunting. In extremely rare cases, tension hydrothorax can develop, with mediastinal shift and circulatory embarrassment.

### Clinical Features

Symptoms associated with pleural effusion are most often due to the underlying disease process and not the effusion itself. Small pleural effusions can be entirely asymptomatic. A new pleural effusion may be heralded by localized pain or pain referred to the shoulder. Viral pleuritis and pulmonary infarction commonly are associated with pleuritic chest pain. When the volume of pleural fluid reaches 500 mL, dyspnea on exertion or at rest may occur as a result of compromised pulmonary function.

The patient’s history often helps to establish the diagnosis for pleural effusion or pleural inflammation. A history of congestive heart failure, liver disease, uremia, or malignancy can direct subsequent evaluation. The pain of viral pleuritis usually is preceded by several days of a typical viral prodrome, with low-grade fever, sore throat, and other upper respiratory or constitutional symptoms. In the absence of such prodromal symptoms, an alternate etiology for pleuritis such as pulmonary embolism must be sought.

Physical findings depend on the size of the effusion but are often either dominated or obscured by the underlying disease process. Classic physical signs of pleural effusion include diminished breath sounds, dullness to percussion, decreased tactile fremitus, and occasionally a localized pleural friction rub. The simple technique of auscultatory percussion (i.e., percussing the chest while listening for a dullness with the stethoscope) may be even more sensitive and specific for the physical diagnosis of pleural effusion. Egophony and enhanced breath sounds can often be appreciated at the superior border of the effusion because of underlying atelectatic lung tissue. In the setting of pleuritis, a pleural friction rub may be appreciated. With massive effusions, signs of mediastinal shift may be present.

Chest radiography confirms the clinical suspicion of pleural effusion and occasionally reveals a pleural effusion as an incidental finding. The classic radiographic appearance of a pleural effusion is blunting of the costophrenic angle on the upright chest radiograph. On a frontal (anteroposterior or posteroanterior) projection, a volume of 250 to 500 mL of pleural fluid is required before radiographic demonstration is possible. A lesser amount of fluid may be visible in the posterior costophrenic gutter on a lateral projection. With larger effusions, the hemidiaphragm is obscured, and an upwardly concave

### BOX 75-3

**Light's Criteria for Differentiating Transudates from Exudates**

1. Pleural fluid protein level: serum protein level > 0.5
2. Pleural fluid lactate dehydrogenase (LDH) level: serum LDH level > 0.6
3. Pleural fluid LDH level > \( \frac{1}{3} \times \) (upper limit of normal for serum LDH level)

In the presence of an exudative effusion, additional pleural fluid analyses further classify the effusion. A pleural fluid pH of less than 7.3 is associated with parapneumonic effusions, malignancies, rheumatoid effusions, tuberculosis, and systemic acidosis. A pH of less than 7.0 strongly suggests empyema (or esophageal rupture). A pleural fluid pH of less than 7.0 and glucose less than 50 mg/dL are reasonable indications for tube thoracostomy.

Normal pleural fluid contains less than 1000 white blood cells/mm³; exudative pleural fluid may contain over 10,000 white blood cells/mm³. Although the absolute cell count has limited diagnostic value, a predominance of neutrophils suggests an acute process, such as pneumonia, pulmonary embolus, or acute tuberculous pleuritis. A predominance of monocytes or lymphocytes suggests a more chronic process, such as malignancy or established tuberculosis. Pleural fluid from any patient with an undiagnosed exudative pleural effusion should undergo Gram’s staining and culture for bacteria (aerobic and anaerobic), mycobacteria, and fungi.
In the absence of a traumatic tap, bloody fluid suggests trauma, neoplasm, or pulmonary infarction. If the hematocrit of the pleural fluid is more than 50% that 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 (e.g., ruptured aortic aneurysm).

If the diagnosis of a malignant pleural effusion is being considered, pleural fluid should be submitted for cytologic examination. Contrary to popular perception, the sensitivity for diagnosis of pleural malignancy does not depend on the volume of pleural fluid extracted during thoracentesis. Cytologic analysis provides the diagnosis of cancer in 40 to 87% of malignant effusions.

Management

In patients with large effusions, urgent therapeutic thoracentesis may stabilize respiratory or circulatory status. The presence of empyema mandates insertion of a chest tube to drain the pleural space adequately and prevent the development of loculations. If an effusion is already loculated, streptokinase or urokinase can be injected by a thoracic surgeon, pulmonologist, or interventional radiologist into the pleural space in an attempt to dissolve adhesions and allow fluid to drain freely. Hemothorax requires tube thoracostomy to evacuate the pleural space, quantify bleeding, and allow apposition of the two pleural surfaces to tamponade hemorrhage. If bleeding exceeds 200 mL/hr, thoracotomy should be considered.

In most other cases, the decision to proceed with therapeutic thoracentesis in the ED can be individualized. For example, therapeutic thoracentesis may be considered in patients with known, recurrent malignant effusion, in whom symptomatic relief may permit discharge.

Pain relief is an important consideration in the management of patients with pleuritis, which may have a significant inflammatory component. Nonsteroidal anti-inflammatory drugs are relatively successful in treating pleural pain. Opioid analgesia is safe and effective, but care should be exercised in debilitated patients or patients with severe lung disease because of potential respiratory depression.

Relative contraindications to thoracentesis include coagulopathy and other bleeding disorders. Thoracentesis may be safe with a prolonged prothrombin time in the absence of active bleeding. Pleural adhesions, suggested by a prior history of empyema, represent a relative contraindication to thoracentesis because of the high risk of pneumothorax associated with blind needle insertion.

After thoracentesis is completed, a chest radiograph should be obtained to rule out iatrogenic pneumothorax. Other potential complications of thoracentesis include hemothorax, lung laceration, shearing of the catheter tip, and infection. Transient hypoxia caused by ventilation-perfusion mismatch often occurs, whereas unilateral, postexpansion pulmonary edema is rare except when large volumes (>1500 mL) are drained in one session. Hypotension also can occur after removal of a large volume of fluid, particularly in patients who are already intravascularly volume depleted.

For patients with congestive heart failure, pleural effusions generally respond well to diuretic therapy. If an effusion persists despite several days of aggressive diuresis, a diagnostic thoracentesis should be considered.

Pleural effusions associated with malignancy are a significant cause of morbidity in patients with advanced cancer. The presence of a malignant effusion indicates disseminated disease, and most of the malignancies that cause pleural effusions—mainly lung or breast carcinoma and lymphoma—are not curable by this stage. Therapeutic thoracentesis can relieve dyspnea in the short term, but malignant effusions tend to be recurrent, often rapidly so. Management strategies include chemical or mechanical pleurodesis to obliterate the pleural space or placement of a pleuroperitoneal shunt to provide continual drainage. Control of pleural effusions can improve quality of life in these patients.

Parapneumonic effusions contribute significantly to the morbidity and mortality of pneumonia. Therefore, the presence of a parapneumonic effusion may influence the decision to hospitalize a patient with community-acquired pneumonia. Empyema can develop in 5 to 10% of patients with a parapneumonic effusion, but in most cases it responds well to parenteral antibiotics and pleural drainage. Early surgical drainage results in shorter hospital stays and may be more cost-effective than conservative management. In nearly 20% of pleural effusions, no definitive diagnosis can be established even after extensive investigation. A sizable percentage of these effusions may be due to viral infections, and most resolve spontaneously without sequelae.

KEY CONCEPTS

- For healthy, young patients with a small (<20%) primary spontaneous pneumothorax, observation alone (with administration of 100% oxygen) is an appropriate treatment option; for larger symptomatic pneumothoraces, simple aspiration with an intravenous catheter is often successful.
- In most cases of secondary spontaneous pneumothorax, tube thoracostomy should be considered because less invasive approaches are associated with lower rates of success.
- Routine application of suction after tube thoracostomy is no longer recommended and does not accelerate lung reexpansion.
- The most common cause of pleural effusions in Western countries is congestive heart failure, followed by malignancy and bacterial pneumonia; however, the diagnosis of pulmonary embolism should not be overlooked in a patient with pleural effusion of unknown etiology.
- Therapeutic thoracentesis is indicated for the relief of acute respiratory or cardiovascular compromise.
- The clearest indication for diagnostic thoracentesis in the ED is to diagnose immediately life-threatening conditions, such as empyema or esophageal rupture in a toxic patient; in most other cases, diagnostic thoracentesis to distinguish between transudative and exudative processes can be deferred.

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