INTRODUCTION

Pleural disease in itself is an unusual cause for admission to the intensive care unit (ICU). Conditions potentially requiring ICU admission include a large pleural effusion causing acute respiratory failure, hemothorax producing respiratory or hemodynamic compromise, secondary spontaneous pneumothorax with respiratory failure, empyema with sepsis, and reexpansion pulmonary edema. Pleural complications of disease processes and procedures performed in the ICU are common, however, and the changes in respiratory physiology are additive to that of the underlying lung disease. The development of a pneumothorax in a critically ill patient, particularly in mechanically ventilated patients, may be a life-threatening event. Pleural effusions may be overshadowed by the illness requiring ICU admission in the critically ill patient. Pleural effusions and pneumothoraces may not be detected on chest radiographs because the radiologic appearance may differ in the supine patient.

PLEURAL EFFUSIONS IN THE INTENSIVE CARE UNIT

Radiologic Evaluation

In the normal pleural space, air and fluid tend to distribute following gravitational influences, with air initially accumulating between the superior portion of the lung and the apex of the thorax, while fluid accumulates between the inferior margin of the lung and the diaphragm. Pleural air and fluid collections shift location when radiographs are obtained in positions other than the erect position. Because radiographs in critically ill patients are taken in the supine or semierect position, the major radiographic finding of a pleural effusion may thus change.

In normal humans in the supine position, the radiolucency of the lung base is equal to or greater than that of the lung apex due to the anteroposterior diameter of the lung being greatest at the lung base. In addition, in the supine patient, breast and pectoral tissues will tend to move laterally away from the lung base. A pleural effusion should be suspected when increased homogeneous density is present over the lower lung fields as compared with the upper lung fields. Patient rotation, an off-center x-ray beam, prior lobectomy, or a pleural or chest wall mass may produce a unilateral homogeneous density that simulates the appearance of a pleural effusion (1). Cardiomegaly, a prominent epicardial fat pad, and lobular collapse or consolidation may obscure the detection of a pleural effusion on a supine radiograph.

Approximately 175 to 525 mL of pleural fluid will produce blunting of the costophrenic angle on an erect chest radiograph (2). This quantity of pleural fluid can usually be detected on a supine radiograph as an increased density over the lower lung zone. Blunting of the costophrenic angle (meniscus sign), silhouetting of the hemidiaphragm, and apical capping may be seen with larger effusions (3). An apparent elevation of the hemidiaphragm may be secondary to a subpulmonic collection of pleural fluid. A diffuse increase in the radiodensity of the hemithorax, or “veiling,” may be seen with very large effusions in the supine radiograph. Thus, the major radiographic finding of a pleural effusion in the supine patient is an increased homogeneous density over the lower lung field that does not obliterate normal bronchovascular markings, does not demonstrate air bronchograms, and does not produce hilar or mediastinal displacement until the effusion is massive. If a pleural effusion is suspected in the supine patient, obtaining an erect or lateral decubitus radiograph may be helpful.

Because the critically ill patient often has underlying parenchymal lung disease, the diagnosis of pleural effusion can be problematic. Ultrasound (US) helps to diagnose, quantify, and guide drainage of pleural fluid and can be performed at bedside. Small-sized pleural effusions are common in the ICU; occasionally, they might be large enough to necessitate drainage (4). Pleural effusions appear on ultrasound as hypoechoic areas between the parietal and visceral pleura. If the fluid collection is sufficiently large, the lung may be seen floating on it like a “jelly fish” (5). Effusions may be echogenic and septate if exudative in nature; hemothorax and pyothorax are typically hyperechoic on ultrasound (6,7). The approximate volume of pleural effusion may be estimated by measuring the distance between the parietal and visceral pleura at the lung base with the breath held in midexpiration. In one study of 74 ICU patients evaluated by both chest radiograph and US, the latter detected a pleural effusion that was not appreciated on chest radiograph in 10 additional patients (29% of patients determined to have a pleural effusion) (4). In another study, US was helpful in making a diagnosis in 27 of 41 (66%) patients and influenced treatment planning in 17 of 41 critically ill patients (41%) (8). US-guided thoracentesis at the bedside was successful in 24 of 25 patients in that same study. Other studies have noted the usefulness of US to safely guide bedside thoracentesis in mechanically ventilated patients (9–11). The presence of complex septated, complex nonseptated, and homogeneously echogenic patterns within pleural fluid collections are typically indicative of an exudative pleural effusion (12). Homogeneously echogenic effusions suggest hemorrhagic effusions or empyemas, whereas US evidence of fibrin septae suggests a parapneumonic effusion, empyema, hemothorax, or malignant effusion (12). Disadvantages of US include impedence of the ultrasound wave by air in the lung or pleural space, a restricted field of view, inferior evaluation of the lung parenchyma compared to computed tomography (CT), and operator dependence (1).

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CT may also be helpful in assessing pleural processes in the critically ill patient, and has the advantages of better lung parenchymal imaging, evaluation of the mediastinum, and ability to distinguish pleural from parenchymal abnormalities (1). On CT, free-flowing pleural fluid produces a sickle-shaped opacity in the most dependent part of the thorax (13). Loculated pleural fluid collections are seen as lenticular or rounded opacities in a fixed position with a relatively homogeneous water density (13). CT may be particularly helpful in the diagnosis and management of loculated pleural effusions (14). The most reliable sign of empyema, the split pleura sign, is usually identified during the organizing phase. Following administration of intravenous contrast, the parietal and visceral pleura will be thickened and enhanced and will be noted to be separated, and the extrapleural fat between the empyema and the chest wall may be increased in size (15,16). In one study, this sign was present in only 68% of patients, however (15). CT may be helpful in assessing inadequately drained fluid collections in patients with persistent fevers or sepsis due to malpositioned chest tubes (17). CT is much more sensitive at detecting pleural fluid, and in one study CT identified a pleural effusion in 13% of patients in the ICU which were missed by CXR (18).

Diagnostic Thoracentesis

Pleural effusions are common in the ICU. In one prospective study of 100 consecutive patients admitted to a medical ICU, pleural effusions were found on chest radiographs and/or by US in 62% of patients (4). Patients with a pleural effusion provide the opportunity to diagnose, at least presumptively, the underlying process responsible for the accumulation of pleural fluid. Although disease of any organ system can cause a pleural effusion in critically ill patients, the diagnoses listed in Table 115.1 represent the most common causes in the ICU.

When a pleural effusion is suspected on physical examination and confirmed radiologically, a diagnostic thoracentesis should be considered to establish the cause of the effusion.

Table 115.1 Causes of Pleural Effusions in ICU Patients

<table>
<thead>
<tr>
<th>Cause</th>
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<tr>
<td>Abdominal surgery</td>
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<tr>
<td>Acute respiratory distress syndrome (ARDS)</td>
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<tr>
<td>Atelectasis</td>
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<tr>
<td>Chylothorax</td>
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<tr>
<td>Congestive heart failure</td>
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<td>Coronary artery bypass surgery</td>
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<tr>
<td>Empyema</td>
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<td>Esophageal rupture</td>
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<td>Esophageal sclerotherapy</td>
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<td>Hemothorax</td>
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<td>Hepatic hydrothorax</td>
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<td>Hypoaalbuminemia</td>
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<td>Iatrogenic</td>
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<tr>
<td>Central venous catheter placement</td>
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<tr>
<td>Nasogastric tube placement</td>
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<tr>
<td>Vascular erosion by central venous catheter</td>
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<tr>
<td>Intra-abdominal abscess</td>
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<tr>
<td>Malignancy</td>
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<tr>
<td>Pancreatitis/pancreatic pseudocyst</td>
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<tr>
<td>Pneumonia</td>
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<tr>
<td>Postcardiac injury syndrome</td>
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<tr>
<td>Pulmonary embolism</td>
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<tr>
<td>Uremia</td>
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Observation alone may be reasonable in situations in which the clinical diagnosis is reasonably secure and a small amount of pleural fluid is present, such as in atelectasis or uncomplicated heart failure (19). Thoracentesis should be performed, however, if the patient’s clinical condition changes. When the distance from the pleural fluid line to the inside of the chest wall is less than 1 cm on lateral decubitus radiograph, the risk of thoracentesis probably outweighs the value of pleural fluid analysis. If the underlying disease causing the pleural effusion becomes clinically problematic, the effusion will often increase in size and allow for safe thoracentesis. When sampling of a small-volume pleural effusion is indicated, thoracentesis should be performed with US guidance. Several studies (20,21) suggest that US-guided diagnostic thoracentesis can be successfully performed in up to 88% of patients after unsuccessful clinically guided thoracentesis.

The indications for diagnostic thoracentesis are not different in the ICU patient, and receiving mechanical ventilation is not a contraindication. Establishing the diagnosis quickly in critically ill patients may be more important than in the noncritically ill. The reported incidence of pneumothorax in nonventilated patients ranges from 4% to 30% (22–25). Various risk factors for developing a pneumothorax after thoracentesis have been reported, although operator inexperience, baseline lung disease, and use of positive pressure mechanical ventilation appear to be the most established risk factors. Several earlier studies have demonstrated that the incidence of pneumothorax after blind thoracentesis in mechanically ventilated patients, 5% to 10%, is similar to that of nonventilated patients, and it is thus safe to perform blind thoracentesis in mechanically ventilated patients (26,27). If the patient on mechanical ventilation does develop a pneumothorax, however, a significant risk of progression to a life-threatening tension pneumothorax exists. As such, some authors have advocated the routine use of ultrasound guidance for all thoracentesis procedures in mechanically ventilated patients, given the observed pneumothorax rates of 0% to 3% with ultrasound guidance in nonventilated patients (25,28), as well as in patients receiving mechanical ventilation (9–11). Strong consideration should be given to using US guidance in patients with small or moderate effusions. US or CT guidance should be used to sample loculated pleural fluid collections. There are no absolute contraindications to diagnostic thoracentesis; the major relative contraindications are a bleeding diathesis or anticoagulation. In one study of 207 patients requiring thoracentesis with mild to moderate coagulopathy, defined as a prothrombin time (PT) or partial thromboplastin time (PTT) up to twice normal, or a platelet count from 50,000 to 100,000 cells/μL, no increase in bleeding complications was noted (29). Thoracentesis should not be performed through an area of active skin infection. Analysis of pleural fluid in the ICU patient is similar to that in other settings and is beyond the scope of this chapter.

Therapeutic Thoracentesis and Physiologic Effects

The primary indication for therapeutic thoracentesis or chest tube drainage of a pleural effusion is relief of dyspnea, although pulmonary mechanics and oxygenation may be improved in some patients (30). Contraindications to and complications of therapeutic thoracentesis are similar to those
of diagnostic thoracentesis, with the additional complications of hypoxemia, reexpansion pulmonary edema, and hypovolemia. An increased risk of pneumothorax has been noted with therapeutic thoracentesis in some studies (24,25,31) although not others (32,33). We would recommend the use of a catheter-over-needle system in performing therapeutic thoracentesis to reduce the risk of developing a pneumothorax. In patients with pleural effusion and ipsilateral shift suggesting endobronchial obstruction, chronic atelectasis, or a trapped lung, the risk of reexpansion pulmonary edema may be increased, and the patient may be less likely to experience a beneficial effect. In addition, patients with initial negative pleural pressures and those with more precipitous falls in pleural pressures with fluid removal also likely have trapped lung or endobronchial obstruction and are less likely to benefit from therapeutic thoracentesis (34).

Pleural effusions compress the lung, causing atelectasis, ventilation/perfusion mismatch, and shunt physiology with resultant hypoxemia (35). Pleural fluid tends to enlarge the volume of the hemithorax more than it compresses lung volume. Studies in humans have shown that total lung capacity following thoracentesis increases by only approximately one-third of the thoracentesis fluid volume, and forced vital capacity increases by approximately half of the increase in total lung capacity. Studies evaluating gas exchange in non-ventilated patients have been mixed. One found a decrease in PaO₂ (36), while another found no change in PaO₂ (37), and a third showed a mild increase in PaO₂ (38). More recent studies have also shown variable results, with one study reporting a small increase in PaO₂, and decrease in alveolar-arterial O₂ gradient (39), although another noted no change in PaO₂, alveolar-arterial O₂ gradient or shunt, while the amount of blood flow to low ventilation/perfusion units increased slightly (35).

Despite these mixed results, some patients requiring mechanical ventilation may benefit from pleural fluid drainage. Talmar et al. (30) reported that 19 patients with acute respiratory failure and pleural effusions who had a poor response to positive end-expiratory pressure (PEEP), defined as the inability to wean FiO₂ to 0.5 with PEEP up to 20 cm H₂O, benefited from chest tube drainage of the pleural effusions. The PaO₂ increased from 125 to 199 mmHg, and the PaO₂/FiO₂ ratio increased from 151 to 254. Fourteen patients had a unilateral effusion, and five patients had bilateral effusions necessitating bilateral chest tube placement. More recently, Doelken et al. (40) studied the effects of thoracentesis on respiratory mechanics and gas exchange in eight mechanically ventilated patients. Following removal of 800 to 1,950 mL (mean 1,495 mL), no significant change in PaO₂ or dead space ventilation was observed. No significant changes were noted for peak and plateau pressures, dynamic and effective static compliance, respiratory system resistance, and intrinsic PEEP. Mean work performed by the ventilator did significantly decrease, however. A recent study of 20 patients showed that drainage of more than 500 mL of pleural fluid in mechanically ventilated patients improved respiratory mechanics with a decrease in plateau pressure and a large increase in end-expiratory transpulmonary pressure. Improvement in the PaO₂/FiO₂ ratio from baseline to 24 hours post drainage was positively correlated with the increase in end-expiratory lung volume but not with the amount of fluid drained. There was no significant effect of large volume thoracentesis on patient hemodynamics (41).

Further studies are required to confirm these results. In patients who are difficult to wean from mechanical ventilation, we would consider a trial of therapeutic thoracentesis.

### COMMON CAUSES OF PLEURAL EFFUSIONS IN THE ICU

#### Abdominal Surgery

Approximately one-half of patients undergoing abdominal surgery will develop small unilateral or bilateral pleural effusions 24 to 48 hours following surgery (42,43). The incidence of pleural effusions is higher in procedures involving the upper abdomen, in patients having ascitic fluid at time of surgery, and in patients who have postoperative atelectasis (34). Larger left-sided effusions are common following splenectomy. The effusion after abdominal surgery is usually exudative with a normal glucose level, pH more than 7.40, and less than 10,000 nucleated cells/μL (42). Small effusions generally do not require diagnostic thoracentesis and resolve spontaneously without becoming clinically significant. Thoracentesis is indicated to exclude empyema if the effusion is relatively large or loculated or if the possibility of a subdiaphragmatic abscess related to the surgery exists.

#### Acute Respiratory Distress Syndrome

The presence of pleural effusions in acute respiratory distress syndrome (ARDS) has not been well appreciated or studied. In a retrospective study of 25 patients with ARDS, 36% were found to have pleural effusions (44). All patients had extensive alveolar infiltrates in addition to pleural effusions. Pleural effusions have been observed in animal models of ARDS using α-naphthylthiourea, oleic acid, and ethchlorvynol (45,46). In the oleic acid model, 35% of the excess lung water collected in the pleural spaces (45). Effusions are likely undiagnosed in ARDS because the patient has bilateral alveolar infiltrates and the radiograph is taken in the supine position. In experimental models of ARDS, the effusions are serous to serosanguineous with a predominance of polymorphonuclear leukocytes (PMNs) (46). In a post hoc analysis of a study looking at the effect of large volume thoracentesis in mechanically ventilated patients, no significant improvement in PaO₂/FiO₂ ratio was observed in patients with ARDS (41). These effusions resolve as the ARDS resolves and require no specific therapy.

#### Atelectasis

Atelectasis is a common cause of small pleural effusions in the ICU due to patients being immobile (4). Atelectasis and small effusions are commonly observed following thoracostomy or abdominal surgery; other potential causes include endobronchial obstruction from tumor, foreign body, or mucus plugging as well as extrinsic airway compression from malignancy. With lung collapse, local areas of increased negative pressure are created by

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*Effusions are termed exudative—or not—by lactate dehydrogenase (LDH) and protein criteria. Only LDH and protein values are used in the Light criteria although cholesterol level is used by some. pH, glucose, and cell counts are not part of the classification of transudate versus exudate.*
the separation of the lung and chest wall. The decrease in pleural pressure favors the movement of fluid into the pleural space, presumably from the surface of the parietal pleura (19).

Pleural effusions from atelectasis are serous transudates with a few mononuclear cells, a glucose concentration equal to serum, and a pH of 7.45 to 7.55. The pleural effusions dissipate over several days when the atelectasis resolves.

Chylothorax

A chylothorax is defined as the accumulation of chyle in the pleural space. The predominant mechanisms of chylothorax formation include disruption of the thoracic duct, extravasation from pleural lymphatics, and transdiaphragmatic efflux from chylous ascites (47). The most common cause of chylothorax is lymphoma, accounting for 37% of chylothoraces in a series of 191 patients (48); the second most frequent cause is surgical trauma, which represented 25% of cases in the same series of 191 patients (48). The incidence of chylothorax following thoracic surgery has been reported to be 0.36% to 0.42% (49,50) and 1.9% following lower neck surgery (46).

A higher proportion of chylothoraces are noted following esophagectomy. Virtually all intrathoracic surgical procedures, including lobectomy, pneumonectomy, and coronary artery bypass grafting, have been reported to cause chylothorax (48). Nonsurgical trauma, including blunt and penetrating injuries to the neck, thorax, and upper abdomen as well as obstruction of the superior vena cava or thrombosis of the left subclavian vein from indwelling central venous catheters (CVCs), may produce chylothoraces in ICU patients (51).

The patient may be asymptomatic if the effusion is small and unilateral, or may be dyspneic with a large unilateral effusion or bilateral effusions. The pleural fluid is usually milky but can be serous, serosanguineous, or bloody. The fluid may not have a milky appearance if the patient is malnourished or not eating (52). The pleural fluid typically has less than 7,000 nucleated cells/μL, which are over 80% lymphocytes. The pH is alkaline (7.40–7.80), and the triglyceride levels exceed plasma levels (19). A pleural fluid triglyceride concentration greater than 110 mg/dL makes the diagnosis of chylothorax highly likely, whereas a concentration below 50 mg/dL makes the diagnosis highly unlikely. With triglyceride concentrations of 50 to 110 mg/dL, lipoprotein electrophoresis is indicated to demonstrate the presence of chylomicrons, which confirms the diagnosis of chylothorax (52).

Up to 2 to 3 L of chyle may drain daily, causing loss of fluid, electrolytes, protein, fat, fat-soluble vitamins, and lymphocytes. Severe nutritional depletion and immunodeficiency may result if these losses are not addressed. In addition to chest tube drainage, initial conservative management consists of intravenous hydration and a nonfat, high-protein, high-calorie diet with medium-chain triglycerides, which are absorbed directly into the portal system, or discontinuing all oral feeding and initiating total parenteral nutrition. If the chylothorax fails to resolve with conservative measures after 7 to 14 days, then surgery with thoracic duct ligation may be considered (53), although pleuropertitoneal shunting has also been used.

Congestive Heart Failure

Congestive heart failure (CHF) is the most common cause of all transudative pleural effusions and in one study was the most common cause of pleural effusions in a medical ICU (4). Pleural effusions due to CHF are associated with increases in pulmonary venous pressure. In a study of 37 patients admitted for CHF, the mean pulmonary capillary wedge pressure (PCWP) was higher in patients with pleural effusions than in those without—24.1 versus 17.2 mmHg, respectively (54). Isolated increases in right heart pressures were not associated with pleural effusions. Patients with chronic obstructive pulmonary disease (COPD) and cor pulmonale, in the absence of left ventricular dysfunction, thus, rarely have pleural effusions, and other causes for pleural effusions should be sought in these patients.

Most patients with pleural effusion secondary to CHF have the usual signs and symptoms. The chest radiograph classically demonstrates cardiomegaly and bilateral small to moderate pleural effusions of similar size, with right-sided effusions often being slightly greater than the left. Radiographic evidence of pulmonary edema is usually present, with the severity of pulmonary edema correlating with the presence of effusions. In patients who have been hospitalized, records will usually show intake greater than output for several days, weight gain, an increasing alveolar-arterial O₂ gradient, and decreasing compliance in those patients requiring mechanical ventilation. Some patients without a history of CHF may not be suspected of having CHF until intravenous hydration produces pleural effusions and subsequent echocardiograms demonstrate left ventricular dysfunction (4).

Pleural effusions from CHF are transudates and have less than 1,000 nucleated cells/μL, which are mainly mesothelial cells and lymphocytes. Acute diuresis may increase the protein concentration of the pleural fluid and thus change the classification of the fluid from transudative to exudative in 8% to 38% of patients (55). In the afebrile patient with clinical CHF and cardiomegaly with bilateral effusions of relatively equal size on chest radiograph, the diagnosis is reasonably secure and observation is appropriate. Thoracentesis should be considered in patients who are febrile, have pleuritic chest pain, or are noted on chest radiograph to have effusions of disparate size, unilateral effusions, a larger effusion on the left than the right, or absence of cardiomegaly.

Treatment consists of decreasing preload and improving cardiac output with diuretics, inotropes, and afterload-reducing agents. With appropriate management, the pleural effusions will resolve over days to weeks.

Coronary Artery Bypass Surgery

A small left pleural effusion is virtually always present following coronary artery bypass surgery (CABG). The effusion is associated with left lower lobe atelectasis and elevation of the left hemidiaphragm on chest radiograph. Approximately 10% of patients will have a larger effusion occupying more than 25% of the hemithorax. These large effusions can be separated into early effusions occurring within the first 30 days of surgery that are bloody exudates with a high percentage of eosinophils, and late effusions occurring more than 30 days after surgery that are clear yellow lymphocytic exudates (56). Factors associated with higher incidence of post-CABG effusions and large effusions are internal mammary artery grafting, on pump surgery, and topical cardiac hypothermia with cold saline.
Esophageal Rupture

Spontaneous esophageal rupture—Boerhaave syndrome—is a potentially life-threatening event and requires immediate diagnosis and therapy. Esophageal rupture or perforation may rarely occur with blunt thoracic trauma or as a complication of endoscopy and nasogastric/orogastric tube placement. The history in spontaneous esophageal rupture is usually severe retching or vomiting; however, activities that generate a Val-salva maneuver can cause esophageal rupture, and in some patients the perforation may be silent (38). The findings on chest radiograph may vary depending on the time between perforation and obtaining of the chest radiograph, the site of perforation, and integrity of the mediastinal pleura. Mediastinal emphysema is present in less than half of patients, and may take 1 to 2 hours to be observed, whereas mediastinal widening may take several hours. Pneumothorax, indicating rupture of the mediastinal pleura, is present in 75% of patients; 70% of pneumothoraces are on the left, 20% are on the right, and 10% are bilateral (59). Pleural effusion, with or without associated pneumothorax, occurs in 75% of patients. A presumptive diagnosis should be confirmed radiographically with an esophagram as soon as possible. Because rapid passage of the contrast in the upright patient that may not demonstrate a small perforation, the study should be done with the patient in the appropriate lateral decubitus position.

Pleural fluid findings depend on the degree of perforation and the timing of thoracentesis. Early thoracentesis without mediastinal perforation shows a sterile serous exudate with a predominance of PMNs and a pH greater than 7.30. Amylase of salivary origin appears in the fluid in high concentration following disruption of the mediastinal pleura. With the seeding of the pleural space by anaerobic bacteria, the pH falls rapidly and progressively to approach 6.00. The presence of food particles and squamous epithelial cells in the pleural fluid also suggests esophageal rupture (60). Management is usually operative intervention in conjunction with pleural space drainage and antibiotics. Nonoperative therapy with antibiotics and chest tube drainage alone may be considered in a nontoxic patient with small perforations due to instrumentation.

Esophageal Sclerotherapy

Pleural effusions are found in approximately 50% of patients 48 to 72 hours following esophageal sclerotherapy (61). Effusions may be unilateral or bilateral, with no predilection for side. The effusions tend to be small serous exudates with variable nucleated (38,000–90,000 cells/mL) and red cell counts (126,000–160,000 cells/mL) and glucose concentrations similar to serum. The mechanism for development of these effusions is likely extravasation of the sclerosant beyond the esophageal mucosa, resulting in mediastinal and mediastinal pleural inflammation. An effusion that is not associated with fever, chest pain, or signs of perforation is not important clinically, and will usually resolve over several days to weeks without specific therapy. A diagnostic thoracentesis should be performed and an esophagram considered in patients with symptomatic effusions for 24 to 48 hours to exclude empyema and esophageal perforation.

Hemothorax

Hemothorax needs to be differentiated from a hemorrhagic pleural effusion, as the latter can be the result of only a few drops of blood in serous pleural fluid. The arbitrary definition of a hemothorax is a pleural fluid to blood hematocrit ratio greater than 50%. Hemothorax can be divided into three categories based on etiology: spontaneous, iatrogenic, and traumatic hemothorax; most hemothoraces result from blunt or penetrating thoracic trauma (62). Other etiologies include invasive procedures, pulmonary infarction, malignancy, and ruptured aortic aneurysms. Anticoagulation therapy or coagulopathy may rarely cause a spontaneous hemothorax. Hemothorax should be suspected in any patient with blunt or penetrating chest trauma with a pleural effusion on chest radiograph. Chest tube thoracostomy with a 28-Fr chest tube, or larger, should be performed in these patients and pleural fluid hematocrit measured. In patients with suspected iatrogenic or spontaneous hemothorax, thoracentesis should be performed first, and if positive, a chest tube should be inserted. Chest tube drainage allows the monitoring of the rate of bleeding, may potentially tamponade the bleeding, and will evacuate the pleural space, thus decreasing the risk of developing empyema or a subsequent fibrothorax (62,63). Prophylactic use of antibiotics (usually a first-generation cephalosporin) for at least 24 hours after the start of chest tube drainage for traumatic hemothorax, reduces the incidence of pneumonia and empyema (64). Whether antibiotic prophylaxis is useful for spontaneous hemothorax has not been investigated accurately.

Intrapleural fibrinolytic therapy can be applied in an attempt to evacuate residual blood clots and breakdown adhesions when initial chest tube drainage is inadequate. The dose, time to initiate, frequency, and duration of fibrinolytic use is unclear from existing literature. Generally, it is advised to evacuate the clotted hemothorax within 7 to 10 days. We recommend 5 to 10 mg of tissue plasminogen activator (tPA) in 50 mL of normal saline given intrapleurally once daily for 3 days starting on day 4 or later of the development of the hemothorax and evaluating the response at the end of 3 days with a chest CT. Indications for surgical exploration vary between clinicians, but general guidelines are hemodynamic instability despite adequate resuscitation, initial drainage greater than 1,500 mL, continued bleeding of more than 200 mL/hr for 3 consecutive hours, continued bleeding of more than 1,500 mL/day, and radiographic evidence of significant retained clot despite adequate noninvasive management as mentioned above (greater than one-third of the pleural space).

Hepatic Hydrothorax

Pleural effusions are present in approximately 6% of patients with cirrhosis and clinically apparent ascites (65,66). The effusions result from movement of ascitic fluid through congenital
or acquired diaphragmatic defects. Rarely, a hepatic hydrothorax may be found in a patient without clinical ascites but with ascites demonstrated only by US, implying the presence of a large diaphragmatic defect. With a small pleural effusion, the patient may be asymptomatic, whereas with large to massive effusions, the patient may have varying degrees of dyspnea. The chest radiograph usually demonstrates a normal cardiac silhouette and a right-sided pleural effusion in 70% of patients, which can vary from small to massive. Effusions are less commonly isolated to the left pleural space (15%) or are bilateral (15%). The pleural fluid is a serous transudate with a low nucleated cell count and a predominance of mononuclear cells, pH greater than 7.40, a glucose level similar to serum, and an amylase less than serum amylase (19). The diagnosis is substantiated by demonstrating that the pleural fluid and ascitic fluid have similar chemistries. If the diagnosis is still in question, injection of a radionuclide into the ascitic fluid with subsequent detection on chest imaging supports the diagnosis (67).

Treatment of hepatic hydrothorax is directed at resolution of the ascites with sodium restriction, diuretics, and paracentesis. It is not uncommon for the effusion to persist until all of the ascitic fluid is mobilized. If the patient is acutely dyspneic or hypoxemic, therapeutic thoracentesis may be done as a temporizing measure. Chest tube drainage should be avoided, as it can cause infection of the fluid, and the prolonged drainage can lead to volume depletion, protein and lymphocyte depletion, and may precipitate renal failure. Chemical pleurodTreeNode cannot be used to treat symptomatic hepatic hydrothorax refractory to medical management (65,66), as has video-assisted thoracoscopic surgery to patch the diaphragmatic defect followed by pleural abrasion or talc poudrage (68).

Hepatic hydrothorax may occasionally be complicated by spontaneous bacterial empyema (SBE), also known as spontaneous bacterial pleuritis, in 13% to 16% of the cases (69). The formation of SBE is a result of either bacterial translocation from infected ascitic fluid or bacteremia and seeding of a hepatic hydrothorax. However, in approximately 40% of cases, SBE can occur in the absence of spontaneous bacterial peritonitis (SBP) and even in the absence of ascites (70). The diagnostic criteria for SBE are similar to those for SBP, requiring a serum/pleural fluid albumin gradient greater than 1.1, a PMN count more than 250 cells/μL with a positive culture, or a PMN count greater than 500 cells/μL with negative cultures and exclusion of contiguous infections. The treatment for SBE is a third-generation cephalosporin given IV for 7 to 10 days. Given the significant mortality rate and its proven benefit in SBP, IV albumin 1.5 mg/kg on day 1 and 1 mg/kg on day 3 post diagnosis is recommended, although albumin use has not been specifically studied in SBE. Chest tube is generally not recommended in SBE unless frank pus is present, because it can lead to life-threatening fluid depletion, protein loss, and electrolyte imbalance (71).

**Hypoalbuminemia**

Many patients admitted to the medical ICU have chronic illnesses and associated hypoalbuminemia. Pleural effusions may be observed when the serum albumin is less than 1.8 g/dL. In one study evaluating the association of pleural effusions with hypoalbuminemia, 3 of 21 (14%) patients with serum albumin less than 2.0 g/dL had pleural effusions (72). Since the normal pleural space has an effective lymphatic drainage system, pleural fluid tends to be the last site of collection of extravascular fluid in patients with low oncotic pressure. It is, therefore, unusual to find a pleural effusion solely due to hypoalbuminemia in the absence of anasarca. The chest radiograph usually shows small to moderate bilateral effusions with a normal heart size. The pleural fluid is a serous transudate with pH ranging from 7.45 to 7.55, and the glucose level is similar to serum. Since hypoalbuminemia is an extremely rare cause of pleural effusion, recognition in these patients should prompt careful clinical evaluations to identify other potential causes for the effusion. The effusions resolve when the hypoalbuminemia is corrected.

**Iatrogenic**

Insertion of a CVC or extravascular migration of a CVC into the pleural space can cause a pneumothorax, hemothorax, chylothorax, or transudative pleural effusion (73,74). The incidence of this complication appears to be approximately 0.4% to 1.0% of catheter placements, but it may be higher considering that some cases remain unrecognized. It is more common with insertion into the left subclavian and internal jugular veins due to the horizontal orientation of the left brachiocephalic vein in relation to the superior vena cava (58). Catheterization via the internal jugular vein may result in fewer malpositions than catheterization via the subclavian vein (75). The postprocedure chest radiograph should always be assessed for proper catheter placement, with catheter positioning parallel to the long axis of the superior vena cava and tip positioning at the right tracheobronchial angle indicating proper placement (76).

Symptoms include chest pain and dyspnea in the conscious patient. Depending on the volume and rate of infusion of fluid into the mediastinum, tachypnea, respiratory distress, and cardiac tamponade may occur. The chest radiograph demonstrates the catheter tip in an abnormal position, a widened mediastinum, and unilateral or bilateral effusions. The effusion can have characteristics similar to the infusate (milky if lipid is being given), and may be hemorrhagic and neutrophil predominant due to trauma and inflammation. If a glucose-containing solution is being infused, the pleural fluid to serum glucose ratio is greater than 1.0 (74). The CVC should be removed immediately. Observation is sufficient if the effusion is small; if the effusion is large or causes respiratory distress, thoracentesis or tube thoracostomy should be performed. If a hemothorax is discovered, a chest tube should be placed.

**Pancreatitis**

Pleural effusions are commonly associated with pancreatitis due to the close proximity of the pancreas to the diaphragm. Pleural effusions have been noted in 3% to 20% of patients with pancreatitis (77). They are usually small to moderate left-sided effusions (60%), although effusions may be isolated to the right side (30%) or occur bilaterally (10%) (78). Pleural effusions related to acute pancreatitis have been shown to be an independent negative prognostic factor as well as a predictor of subsequent pseudocyst development (77–79). The diagnosis is confirmed by an elevated pleural fluid amylase...
Parapneumonic Effusions and Empyema

Pleural effusions are a common finding in patients with pneumonia. More than 40% of patients with bacterial pneumonia, and 60% of patients with pneumococcal pneumonia, develop parapneumonic effusions. While treatment with antibiotics leads to resolution in most patients, some patients develop a more fibrinous reaction, with the presence of frank pus in the pleural space, although many clinicians extend the definition to include pleural fluid that has a positive Gram stain for bacteria or a positive bacterial culture. Complicated parapneumonic effusions are defined as pleural effusions in the setting of pneumonia that have either a pH below 7.2, glucose less than 60 mg/dL, lactate dehydrogenase (LDH) more than 1,000 IU/L, or seaptations or loculations, whereas uncompl icated parapneumonic effusions are the ones which do not meet the criteria for complicated parapneumonic effusion or empyema (84–87).

The usual presentation is similar to the non-ICU patient with fever, dyspnea, chest pain, purulent sputum, leukocytosis, and a new alveolar infiltrate on chest radiograph. In the elderly, debilitated, or immunosuppressed patient, however, many of these findings may be absent. Although pleural space infections most commonly occur in association with pneumonia, it should also be recognized that pleural space infections may result from thoracic surgery, chest tube placement, penetrating chest trauma, esophageal perforation, mediastinitis, subdiaphragmatic abscesses, SBP, and bacteremic seeding of a pre-existing effusion (84).

Once the diagnosis of parapneumonic effusion or empyema has been made, treatment options should be considered, which center around antibiotic therapy and pleural drainage. Pleural drainage can include therapeutic thoracentesis, image-guided catheter placement, tube thoracostomy, and surgical drainage procedures. There are multiple consensus guidelines to aid clinician with decisions regarding pleural drainage (88). In essence, free-flowing effusions less than 10 cm on lateral decubitus can be managed with antibiotics and close observation. For small to moderate effusions the fluid should be sampled, but further drainage procedures are not necessary in absence of empyema or evidence of complicated parapneumonic effusion, as diagnostic thoracentesis can completely drain the pleural space, and thus also serve a therapeutic role. In patients with complicated parapneumonic effusion or empyema, formal pleural drainage with a chest tube is indicated. Considerable controversy exists about what size chest tube is appropriate in which clinical settings. Typically, chest tubes are divided into small-bore (≤14 Fr) versus large-bore (≥14 Fr) tubes with smaller tubes generally placed by percutaneous Seldinger approaches and larger-bore tubes by open incision. Prospective evaluation of the outcomes relative to quartiles of chest tube size (<10 Fr, 10–14 Fr, 15–20 Fr, or >20 Fr) was performed in one study and demonstrated no difference in mortality or need for surgery based on chest tube size (89). In general, a larger-bore catheter has a theoretical advantage of facilitating effective drainage. However, this must be balanced against the increased pain associated with larger-bore tube insertion, which could compromise a patient with a tenuous respiratory status. When using smaller-bore tubes, the authors urge all practitioners to develop protocols to assure catheter patency. This approach would include routine checks to identify catheter kinking including careful anchoring at the entry site, routine catheter flushes, and connection to a closed pleural drainage system with continuous, regulated suction.

Evidence of pus, loculations, pleural thickening, and progressive organization in the chest would suggest that less invasive drainage procedures may be unsuccessful. These are the patients in whom intrapleural fibrinolytic therapy should be considered based on the results of the MIST2 trial, a four-arm double-blinded, double-dummy study of tPA plus placebo, deoxyribonuclease (DNase) plus placebo, combined tPA and DNase, or double placebo. Unlike the outcome in the MIST1 trial, the MIST2 study demonstrated improvement in pleural opacity in the tPA–DNase group compared with either agent alone or placebo. Combined therapy also demonstrated a reduction in surgical referral and length of hospital stay (90). Consequently, we recommend that, in a stable patient, chest tube drainage including fibrinolytic and DNase therapy (twice daily intrapleurally for 3 days) be considered first, reserving surgical intervention for clinical or radiographic fibrinolytic failure (72–75).

Postcardiac Injury Syndrome

Postcardiac injury syndrome (PCIS) is characterized by the onset of fever, pleuropericarditis, and parenchymal infiltrates typically 3 weeks (2–86 days) following injury to the myocardium or pericardium (91,92). PCIS includes different pleuropericardial syndromes that are elicited by an initial traumatic
Pleuritic chest pain is reported by virtually all patients, whereas one-half of patients are noted to have dyspnea, fever, pericardial rub, and rales. Half of the patients have a leukocytosis, and almost all have an elevated erythrocyte sedimentation rate. The chest radiograph is abnormal in most patients, with the most common abnormality being left-sided or bilateral pleural effusions (92). Pulmonary infiltrates are present in 75% of patients and are most commonly seen in the left lower lobe (91). The pleural fluid is a serosanguineous or bloody exudate with pH greater than 7.30 and glucose level greater than 60 mg/dL. Nucleated cells range from 500 to 39,000 cells/μL, with a predominance of PMNs (95). Corticosteroid therapy at low doses (i.e., prednisone 0.2–0.5 mg/kg/day) is useful when aspirin/NSAID are either contraindicated or not well tolerated, and to reduce the possible interference of aspirin/NSAID therapy with oral anticoagulant therapy (96); adjunctive colchicine is also advised in such cases. Following treatment, the pleural effusion resolves within 1 to 3 weeks. It is important to not misdiagnose PCIS as a pulmonary embolism, as anticoagulation therapy may lead to pericardial hemorrhage and tamponade.

Pneumothorax In the Intensive Care Unit

Pneumothorax is defined as air identified within the pleural space and is a commonly encountered problem in the critical care setting. The prevalence of pneumothorax in ICU patients trigger affecting the pericardium/myocardium and/or pleura after cardiac surgery, an invasive percutaneous intervention, myocardial infarction, or chest trauma. The incidence following myocardial infarction has been estimated at up to 4%, and up to 30% following cardiac surgery. Based on available data, it appears that PCIS results from an autoimmune reaction following myocardial or pericardial injury (93).

PCIS is usually self-limited and may not require treatment if symptoms are minor. Spontaneous recovery occurs in 66% of patients. Hornfeiter et al. conducted a trial for treatment of PCIS, comparing ibuprofen 600 mg orally four times per day, indomethacin 25 mg orally four times per day, or placebo for 10 days. The rate of resolution of the postpericardiotomy syndrome was 90% in the ibuprofen group, 89% in the indomethacin group, and 63% in the placebo group (p = 0.003), while recurrences at 30 days were 25% in the ibuprofen and indomethacin groups, and 50% in the placebo group (95). Corticosteroid therapy at low doses (i.e., prednisone 0.2–0.5 mg/kg/day) is useful when aspirin/NSAID are either contraindicated or not well tolerated, and to reduce the possible interference of aspirin/NSAID therapy with oral anticoagulant therapy (96); adjunctive colchicine is also advised in such cases. Following treatment, the pleural effusion resolves within 1 to 3 weeks. It is important to not misdiagnose PCIS as a pulmonary embolism, as anticoagulation therapy may lead to pericardial hemorrhage and tamponade.

Pulmonary Embolism

Pleural effusions occur in up to 50% of patients with pulmonary embolism (86). Pulmonary embolism has been established to be the fourth main cause of pleural effusion in the United States after congestive heart failure, parapneumonic effusion, and malignant effusion (97). The pathogenesis of pleural effusions in pulmonary embolism includes ischemia and inflammatory mediator-induced increased pleural capillary permeability, imbalance in microvascular and pleural space hydrostatic pressures, pleuropulmonary hemorrhage, and atelectasis. With pulmonary infarction, necrosis and hemorrhage into the lung and pleural space may result. More than 80% of patients with pulmonary infarction will have bloody pleural effusions, while up to 40% of patients without radiographic evidence of infarction will also have hemorrhagic fluid (98). Ipsilateral pleuritic chest pain occurs in most patients with pleural effusions complicating pulmonary embolism. A coexistent pulmonary infiltrate is noted on chest radiograph in approximately half of patients with pulmonary embolism and pleural effusion.

Pleural fluid analysis is variable and may demonstrate either an exudate or a transudate (19,99). A bloody pleural effusion in the absence of chest trauma, recent cardiac injury, asbestos exposure, or malignancy should increase the suspicion of pulmonary embolism. The pleural fluid is hemorrhagic in two-thirds of patients, although the number of red blood cells exceeds 100,000 cells/μL in less than 20% (19). The nucleated cell count ranges from less than 100 (presumably atelectatic transudates) to 50,000 cells/μL (pulmonary infarction). When thoracentesis is performed near the time of acute symptoms, PMNs are predominant; with later thoracentesis, lymphocytes represent the majority of cells, and eosinophils may be present as well. The effusion from pulmonary embolism is usually apparent (92%) on the initial chest radiograph and reaches a maximum volume during the first 72 hours. In patients who demonstrate progression of effusions after 72 hours of therapy, recurrent embolism, hemothorax secondary to anticoagulation, an infected infarction, or an alternative diagnosis should be considered. The effusions usually resolve in 1 week in the absence of an infiltrate on chest radiograph. When an infiltrate is present, presumably representing a pulmonary infarction, the resolution time is longer, typically 2 to 3 weeks (98).

The association of a pleural effusion with pulmonary embolism does not alter therapy. The presence of a bloody effusion is not a contraindication to full-dose anticoagulation, since hemothorax is a rare complication of heparin therapy for pulmonary embolism (100,101). An enlarging pleural effusion on therapy necessitates thoracentesis to exclude hemothorax, empyema, or another cause. The development of a hemothorax during therapy requires discontinuation of anticoagulation, chest tube thoracostomy, and placement of a vena cava filter.

Uremia

Uremic pleural effusions have been reported in 3% to 5% of patients undergoing chronic dialysis (102). In one study evaluating the cause of pleural effusions in 100 patients requiring long-term hemodialysis, uremic pleurisy accounted for 16% of cases (103). Patients may manifest fever, cough, chest pain, and pleural friction rubs. The chest radiograph usually shows a moderate unilateral effusion, although massive and bilateral pleural effusions have been reported (104–106). The pleural effusion is a serosanguineous or bloody exudate, with less than 1,500 nucleated cells/μL, predominantly lymphocytes. The creatinine concentration is high, although the pleural fluid exceeds 100,000 cells/μL (pulmonary infarction). When thoracentesis is performed near the time of acute symptoms, PMNs are predominant; with later thoracentesis, lymphocytes represent the majority of cells, and eosinophils may be present as well. The effusion from pulmonary embolism is usually apparent (92%) on the initial chest radiograph and reaches a maximum volume during the first 72 hours. In patients who demonstrate progression of effusions after 72 hours of therapy, recurrent embolism, hemothorax secondary to anticoagulation, an infected infarction, or an alternative diagnosis should be considered. The effusions usually resolve in 1 week in the absence of an infiltrate on chest radiograph. When an infiltrate is present, presumably representing a pulmonary infarction, the resolution time is longer, typically 2 to 3 weeks (98).

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PNEUMOTHORAX IN THE INTENSIVE CARE UNIT

Pneumothorax is defined as air identified within the pleural space and is a commonly encountered problem in the critical care setting. The prevalence of pneumothorax in ICU patients...
Pneumothoraces in ICU patients.

The mechanisms of spontaneous generation of extra-alveolar air were first delineated by Macklin and Macklin (112). In situations in which intra-alveolar pressure is increased, a gradient is produced between the alveolus and the adjacent vascular sheath, causing the alveoli to rupture at their bases. Following rupture, air is introduced in the perivascular adventitia, resulting in interstitial emphysema. The air then dissects proximally to the lung hilum and mediastinum due to a lower mean pressure in the mediastinum compared to that of the lung parenchyma. Once in the mediastinum, the accumulated air may decompress along paths of least resistance into the subcutaneous tissues or, less commonly, into the pericardium, peritoneum, and retroperitoneum. If mediastinal pressure increases abruptly or if decompression via these routes is not sufficient, the mediastinal parietal pleura may rupture, resulting in pneumothorax. Alternatively, air from ruptured alveoli may dissect to the periphery of the lung and rupture via subpleural blebs through the visceral pleura into the pleural space (113).

Pneumothoraces are classified as spontaneous, which occur without preceding trauma or other obvious causes, and traumatic, which occur as a result of direct or indirect trauma to the chest. Spontaneous pneumothoraces can be subdivided into primary spontaneous, which occur in otherwise healthy patients without clinical lung disease, and secondary spontaneous, which occur in patients with underlying lung disease. Traumatic pneumothoraces can be subdivided into the categories of iatrogenic and related to blunt or penetrating chest trauma. In addition, pneumothoraces can be classified as simple or complicated, with complicated pneumothoraces consisting of tension pneumothorax, hemopneumothorax, pyopneumothorax, and open pneumothorax in which the integrity of the chest wall is disrupted. The potential causes of pneumothoraces in critically ill patients are listed in Table 115.2. We will focus mainly on iatrogenic pneumothoraces and pneumothoraces resulting from barotrauma, as these are the most common causes of pneumothoraces in ICU patients.

### Diagnostic Evaluation

The diagnosis of pneumothorax in the critically ill patient can sometimes be made with information from the history and physical examination, noting acute onset of dyspnea or chest pain, tachycardia, hypotension, decreased breath sounds, pulsus paradoxus, and contralateral tracheal deviation. Although clinical features can be used to diagnose the presence of a pneumothorax, it should be noted that many of these findings are nonspecific and have not been a reliable indicator of size. As a result, radiologic data remains the gold standard for the diagnosis of pneumothorax (114). Chest radiographs have traditionally been the first test ordered for suspected pneumothorax. In the critically ill, the traditional erect posterior–anterior expiratory film is not practical and thus, the supine or semirecumbent anterior–posterior film is frequently obtained. The radiographic signs of pneumothorax in the supine patient frequently differ from the classic visceral pleural line seen on erect views. In a review of 88 critically ill patients with 112 pneumothoraces, only 22% of pneumothoraces were in the classic apicolateral location (115). In this same study, 30% of pneumothoraces were not detected initially, and of these, half progressed to a tension pneumothorax. The anteromedial position is the most common location for pneumothoraces in the supine patient since this area is the least dependent pleural space (116).

### Table 115.2 Causes of Pneumothoraces in ICU Patients

<table>
<thead>
<tr>
<th>Secondary Spontaneous</th>
<th>Barotrauma/Volutrauma</th>
<th>Trauma</th>
<th>Iatrogenic</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Airway diseases</td>
<td>• Mechanical ventilation</td>
<td>• Blunt chest trauma</td>
<td>• Endotracheal intubation</td>
</tr>
<tr>
<td>• Chronic obstructive pulmonary disease (COPD)</td>
<td>• Acute respiratory disease syndrome (ARDS)</td>
<td>• Penetrating chest trauma</td>
<td>• Tracheostomy</td>
</tr>
<tr>
<td>• Status asthmaticus</td>
<td>• Status asthmaticus</td>
<td>• Tracheobronchial injuries</td>
<td>• Central venous catheter placement</td>
</tr>
<tr>
<td>• Cystic fibrosis</td>
<td>• COPD</td>
<td>• Rib fractures</td>
<td>• Thoracentesis</td>
</tr>
<tr>
<td>• Parenchymal lung diseases</td>
<td>• Inhalational drug usage</td>
<td>• Esophageal rupture</td>
<td>• Naloxone tube placement</td>
</tr>
<tr>
<td>• Idiopathic pulmonary fibrosis</td>
<td>• Decompression injury</td>
<td>• Bronchoscopy with bronchoalveolar lavage (BAL) or biopsies</td>
<td>• Postoperative</td>
</tr>
<tr>
<td>• Sarcoidosis (stage IV)</td>
<td></td>
<td>• Bag/valve/mask ventilation</td>
<td>• Bronchoalveolar lavage (BAL) or biopsies</td>
</tr>
<tr>
<td>• Langerhans cell histiocytosis (histiocytosis-X)</td>
<td></td>
<td>• Cardiopulmonary resuscitation</td>
<td>• Status asthmaticus</td>
</tr>
<tr>
<td>• Malignancy</td>
<td></td>
<td>• Cardiopulmonary resuscitation</td>
<td>• Chronic obstructive pulmonary disease (COPD)</td>
</tr>
<tr>
<td>• Pulmonary infections</td>
<td></td>
<td>• Cardiopulmonary resuscitation</td>
<td>• Status asthmaticus</td>
</tr>
<tr>
<td>• Pneumocystis jiroveci</td>
<td></td>
<td>• Cardiopulmonary resuscitation</td>
<td>• Chronic obstructive pulmonary disease (COPD)</td>
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<tr>
<td>• Necrotizing bacterial pneumonia</td>
<td></td>
<td>• Cardiopulmonary resuscitation</td>
<td>• Status asthmaticus</td>
</tr>
<tr>
<td>• Tuberculosis</td>
<td></td>
<td>• Cardiopulmonary resuscitation</td>
<td>• Chronic obstructive pulmonary disease (COPD)</td>
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<td>• Fungal pneumonia</td>
<td></td>
<td>• Cardiopulmonary resuscitation</td>
<td>• Status asthmaticus</td>
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<td></td>
<td></td>
<td>• Cardiopulmonary resuscitation</td>
<td>• Chronic obstructive pulmonary disease (COPD)</td>
</tr>
</tbody>
</table>
pneumothoraces on CT that were not apparent or not appreciated on conventional radiographs (17,117). Occasionally, a pneumothorax may be confused with a large bulla in patients with COPD and other pulmonary diseases that generate cystic changes. In these instances, a CT may be helpful in making the correct diagnosis (113). Ultrasonographic assessment of pneumothorax has emerged recently as an alternative modality in facilities with physicians trained in bedside ultrasonography (US). US is the preferred first-line diagnostic test to exclude pneumothorax in the ICU. Because pleural air would block the visualization of the underlying lung, the presence of B lines and lung sliding rules out a pneumothorax with a negative predictive value of 100% in the location of the chest probe. The lung point (area where normal lung sliding meets an area where no lung sliding is seen) can be visualized with both B-mode and M-mode ultrasonography, and when seen, has 100% specificity for pneumothorax (118). A study comparing ultrasonography to CT scan and chest radiographs for the diagnosis of occult pneumothoraces showed that the use of US detected 92% of occult pneumothoraces diagnosed with CT scan (119).

Primary and Secondary Spontaneous Pneumothorax

Patients with pneumothorax have a decrease in vital capacity and an increase in the alveolar-arterial O₂ gradient, with hypoxemia being present in some patients. The hypoxemia is thought to be secondary to development of both anatomic shunts and areas of low ventilation/perfusion in the atelectatic lung. Patients with primary spontaneous pneumothorax rarely require admission to the ICU, as the contralateral lung can maintain the necessary alveolar ventilation and hypoxemia can be managed with supplemental oxygen. Patients with secondary spontaneous pneumothoraces may need ICU admission because the gas exchange abnormality caused by the pneumothorax is superimposed on pre-existing gas exchange abnormalities and, thus, severe hypoxemia can occur. Patients with secondary spontaneous pneumothoraces are more likely to develop hypercapnic respiratory failure than are patients with primary spontaneous pneumothorax (120,121).

Iatrogenic Pneumothorax

As the number of operative procedures performed for diagnostic and therapeutic purposes increase in training and research hospitals, iatrogenic pneumothorax will become the most encountered type of pneumothorax after traumatic pneumothorax. This complication prolongs the hospitalization period, worsens the patients' physical condition and increases morbidity and mortality, especially in patients who develop barotrauma due to mechanical ventilation. Insertion of CVCs is the most common cause of iatrogenic pneumothoraces in the ICU. In two studies of mechanical complications of CVCs, 1.1% of 534 patients and 1.0% of 713 patients suffered a pneumothorax (122,123). Cannulation of the subclavian vein is associated with a higher risk of pneumothorax than cannulation of the internal jugular vein (124,125). Most pneumothoraces occur at the time of the procedure from direct lung puncture, but delayed pneumothoraces have been noted. Bilateral pneumothoraces have been reported to occur from unilateral cannulation attempts (126).

The second most frequent cause of iatrogenic pneumothorax is thoracentesis; the reported incidence is between 3% and 19% (32,22). Risk factors in thoracentesis are the experience of the personnel, coughing of the patient during the procedure, the underlying lung disease, and the number of passes performed. When the procedure is performed with the assistance of ultrasound, the rate of complications decreases to 2% to 3%. Ultrasound is especially beneficial when there is a very low amount of fluid present and for loculated fluids (22).

Cardiopulmonary resuscitation has been reported as a cause of iatrogenic pneumothorax. Pneumothorax in this setting may arise either from barotrauma as a consequence of bag-ventilation or from rib fractures sustained during the resuscitation. Hillman and Albin (127) described three patients who developed subcutaneous emphysema and pneumothoraces, one of whom had bilateral pneumothoraces following cardiopulmonary resuscitation with bag-ventilation. Shulman et al. (128) reported two patients in whom barotrauma was observed following resuscitation. One of the patients was ventilated with a self-inflating bag, whereas the other was ventilated with a positive pressure demand valve. Other cases of pneumothorax related to cardiopulmonary resuscitation or malfunctioning valves in self-inflating bags have been reported (129,130).

Pneumothoraces may rarely occur following endotracheal intubation, usually due to rupture of the posterior membranous portion of the trachea (131). In a prospective study of translaryngeal intubation in 297 critically ill patients in a teaching hospital, pneumothorax occurred in 1% of patients (132). Pneumothoraces may also result from tracheostomy, either from open procedures or bedside percutaneous dilational tracheostomy (133). The incidence of pneumothorax after tracheostomy in adults has been reported to be between 0% and 4% (134).

Bronchoscopy in critically ill patients may also cause pneumothoraces. The risk is higher when transbronchial biopsies are obtained, although the degree of increased risk compared to nonventilated patients and the influence of high airway pressures and PEEP are unknown. It should be recognized that performing bronchoalveolar lavage (BAL) alone may produce a pneumothorax (135–137).

Pneumothorax is a frequent, potentially lethal complication of mechanical ventilation–induced barotrauma. The patient requiring mechanical ventilation usually becomes symptomatic after developing a pneumothorax because of the underlying lung parenchymal disease, and this complication should be suspected whenever a sudden clinical deterioration occurs. If conscious, the patient becomes dyspneic and tachypneic, and may become dysynchronous with the ventilator; worsening oxygenation is often seen. Peak inspiratory pressures may increase with a coexisting decrease in lung compliance; a significant percentage of patients will develop a tension pneumothorax. There are multiple risk factors for acquiring a pneumothorax in mechanically ventilated patients in the ICU. Studies in mechanically ventilated patients with acute lung injury or ARDS have reported pneumothorax occurrence rates between 7% and 42% (128–133). A recent study by Papazian et al. (138) showed a significant reduction in pneumothoraces in patients with severe ARDS who were randomized to receive 48 hours of paralysis. An study in the pediatric population showed that the prevalence of pneumothorax in ventilated patients was significantly higher in the era before protective
Tension Pneumothorax

A tension pneumothorax occurs when intrapleural pressure exceeds atmospheric pressure throughout expiration, and often inspiration as well. This develops when a break in the visceral or parietal pleura produces a one-way valve that is open during inspiration, allowing air to enter the pleural space, but is closed during expiration, preventing the egress of air. Tension pneumothorax often inspiration as well. This develops when a break in the pleural space (103). Tension pneumothoraces most commonly develop as a complication of mechanical ventilation—barotrauma or volutrauma—or as a result of blunt and penetrating thoracic trauma, although tension pneumothoraces can occur in 1% to 4% of patients with spontaneous pneumothoraces (140,141).

Tension pneumothorax usually presents as an acute cardiopulmonary emergency beginning with respiratory distress and, if unrecognized and untreated, progresses to cardiovascular collapse and death. Patients with tension pneumothorax often exhibit decreased ipsilateral breath sounds, hyperresonance to percussion, distended neck veins, tracheal deviation to the contralateral side, and hypotension. The absence of physical examination findings, however, does not completely exclude the diagnosis of a tension pneumothorax and when suspected should be treated as such without any confirmatory tests.

On the chest radiograph in a patient with tension pneumothorax, in addition to the pneumothorax, there is often shift of the trachea and mediastinum to the contralateral side, ipsilateral diaphragmatic depression, and increased distance between contiguous ribs compared to the unaffected side. It should be emphasized, however, that tension pneumothorax is a clinical diagnosis, and these radiographic findings may be observed in patients without physiologic evidence of a tension pneumothorax. It should also be noted that patients may have cardiopulmonary compromise due to a tension pneumothorax without observing tracheal or mediastinal shift on chest radiograph (142,143).

In one study of 16 ARDS patients with tension pneumothorax, only 5 patients had subtle mediastinal shift (142). Of these 16 patients, 11 had flattening of the diaphragm and 8 had depression of the diaphragm. Diaphragmatic abnormalities may therefore be a more sensitive indicator of tension pneumothorax in patients with ARDS. In 15 of the 16 patients, the location of a loculated tension pneumothorax was subpulmonic or paracardiac. Potential explanations for these observations include the presence of adhesions between the parietal and visceral pleura and the noncompliance of lungs in patients with ARDS which may prevent collapse of the ipsilateral lung and compression of the contralateral lung, allowing a small volume of air to significantly increase intrapleural pressure (142,143).

It is important to note that patients with ARDS can develop tension pneumothoraces despite the presence of a chest tube on the ipsilateral side having been placed for a previous pneumothorax (142–145). In the 16 patients reported by Gobien et al. (142) and the 3 patients reported by Ross et al. (143), all patients had a functional ipsilateral chest tube and had localized pneumothoraces. In a study by Heffner et al. (144), 14 patients had recurrent pneumothoraces despite ipsilateral chest tubes, with 9 of the 14 having tension pneumothoraces, and all nine of these chest tubes being horizontally placed. In the latter study, 12 of the 14 chest tubes had horizontal as opposed to vertical placement on chest radiograph. Seven of the 14 patients had subsequent CT scans, with the finding that all seven chest tubes were placed within interlobar fissures. Thus, chest tubes placed into interlobar or posterior locations may not drain anterior gas loculations, the most common location of pneumothoraces in ARDS patients (115,146), allowing for development of localized tension pneumothoraces. In the patient reported by McConaghy and Kennedy (145), the chest tube was intraparenchymal.

Management of Pneumothoraces and Tension Pneumothoraces

Most critically ill patients in the ICU will have poor cardiopulmonary reserve and may be unable to tolerate a pneumothorax, even in the absence of tension physiology. In nonventilated patients who are hemodynamically stable and have adequate oxygenation and ventilation, simple pneumothoraces that affect oxygenation and ventilation, simple pneumothoraces that occur as a result of a procedure and are small may reasonably be managed with close observation and monitoring with serial radiographs. Patients with secondary pneumothoraces who require ICU care will usually require chest tube placement because of their poor pulmonary reserve. Patients who are not receiving positive pressure ventilation, but are hemodynamically unstable, should be treated with chest tube thoracostomy, since the additive effects of development of hypoxia or early tension physiology could quickly precipitate cardiopulmonary arrest. The role of manual aspiration in ventilated patients has not been studied, and there are currently no expert guidelines to suggest that manual aspiration has a role in the management of these patients.

In general, chest tube thoracostomy should be performed in mechanically ventilated patients with a pneumothorax of any size given the significant risk of progression to a tension pneumothorax. A wide variety of chest tube sizes exist, ranging from 6 Fr to 40 Fr. Traditionally, large-bore chest tubes were used for pneumothorax, but more recently, smaller-bore tubes inserted via a modified Seldinger technique have become widely available and have become commonly used in nonintubated patients. The risk of serious complications associated with small-bore catheters is minimal, with a frequency of injury of 0.2% and a malposition rate of 0.6%. The biggest risk is drain blockage, with a rate of 8.1%, and this is easily prevented with scheduled sterile flushing to maintain patency (147). A retrospective review by Lin et al. of 62 ventilated patients who underwent small-bore chest tube drainage as the primary management of pneumothorax found a 68.6% overall success rate, defined as no residual air seen in the follow-up...
BRONCHOPLEURAL FISTULA IN THE INTENSIVE CARE UNIT

A bronchopleural fistula (BPF) represents a communication between the bronchial tree and the pleural space. BPFs most commonly result from surgical procedures including pneumonectomy, segmentectomy, and wedge resections of the lung, with an incidence of 1.6% to 6.8% (154). The mortality in patients with BPFs following surgical resection is reported to be between 23% and 71%, usually due to infectious complications (154–156). BPFs may also result from blunt or penetrating chest trauma, pulmonary infarction, and as a complication of pulmonary and pleural infections such as tuberculosis, necrotizing pneumonia, lung abscess, or empyema (157,158). Last, BPFs may result as a complication of mechanical ventilation for acute respiratory failure, particularly in patients with ARDS, and, as such, represent a form of barotrauma/volutrauma (22,158,159). For this discussion, we will focus primarily on BPFs in the setting of patients requiring mechanical ventilation.

BPF in the ventilated patient is defined as an air leak that persists for more than 24 hours following placement of a chest tube. BPFs in patients receiving mechanical ventilation may present acutely with the development of a pneumothorax, with or without tension, or with sudden expectoration of potentially infected material from the pleural space, with flooding of the ipsilateral and contralateral airways leading to respiratory compromise.

Several potential adverse effects of a BPF in the mechanically ventilated patient have been noted. Depending on the size of the fistula, flow resistance through the fistula versus the airways and lung parenchyma, and pressure gradient between the airways and pleural space, air may be redirected from normal intrapulmonary routes to the BPF (160). This can cause loss of effective tidal volume, which may lead to difficulty in oxygenating and ventilating the patient and subsequent development of life-threatening hypoxemia and respiratory acidosis (159). If incomplete lung expansion due to the BPF is present, ventilation/perfusion mismatching and shunt may occur. There may be difficulty in maintaining PEEP with further decrements in oxygenation (161,162). If a high level of chest tube suction is used, the negative pressure may be transmitted to the proximal airways, causing inappropriate ventilator cycling (161–163). Finally, BPFs may cause pleural space infection or contamination of the airways.

The amount of air flow through a BPF is typically estimated by subtracting the expired tidal volume from the inspired tidal volume as measured by the ventilator. This method, however, becomes increasingly inaccurate as the size of the leak decreases, particularly when the size of the leak is less than 200 mL/breath. More accurate, albeit cumbersome, methods have been developed to quantify the amount of flow through a BPF (164–167). Air flows through BPFs have been reported up to 22 L/min (164). It has been recognized that the air escaping from a BPF does not flow passively from the airways into the pleural space but instead participates to some degree in physiologic gas exchange. In two studies evaluating CO₂ excretion occurring via the BPF (168).

The development of a BPF has been regarded as a serious and life-threatening complication of mechanical ventilation. In one of the largest series reported—1,700 consecutive patients receiving mechanical ventilation—Pierson et al. (159) observed that 39 (2.3%) patients developed a BPF. In that study, overall mortality in patients with BPF was 67%; mortality was higher in patients who developed a BPF late in their illness (94%) than when it occurred within 24 hours of admission (45%). Patients with air leaks greater than 500 mL/breath had a mortality of 100% compared with a mortality of 57% in patients with air leaks less than 500 mL/breath. Mortality...
was also higher in patients with ARDS than in patients without—81% versus 50%—and in patients with pleural space infections compared to those without said infection—87% versus 54%. A study of patients with ARDS by Weg et al. (169), however, suggested that mortality was not different between patients with or without air leaks, 46% versus 39%, respectively. In that study, however, the duration of mechanical ventilation was 4.3 ± 1.3 days, which may not be typical for many patients with BPF, and the subset of patients with BPF was not analyzed separately. It may be that the presence of a BPF is a marker for severity of lung injury and by itself does not directly contribute to mortality.

### Management of Bronchopleural Fistulae

Numerous interventions, listed in Table 115.3, have been proposed in the management of BPFs. Many of these are based on the concept of decreasing the pressure gradient between the airways and the pleural space, with decreased air flow through the fistula allowing for earlier closure. Although the various manipulations theoretically make sense, they have not been evaluated in controlled trials. The suggested changes in ventilator settings may actually worsen oxygenation and ventilation in some patients with ARDS. We will discuss those interventions for which some data are available in the following section.

In the absence of difficulty oxygenating or ventilating the patient, it is unknown if active measures to close the BPF affect outcome. Definitive therapy for BPFs includes surgical procedures such as bronchial stump closure with thoracoplasty, myoplasty, or omentoplasty, or completion pneumonectomy (154,156). Unfortunately, most critically ill patients will not be sufficiently stable to undergo these procedures and must be managed medically. Adequate pleural space drainage, antibiotic therapy for pleural space infections, and support of nutritional status is vital in these patients.

Adequate chest tube drainage and full expansion of the lung should be assessed in patients with BPF. An appropriately sized chest tube should be placed, recognizing that air flow through a chest tube is inversely proportional to the length and radius to the fourth power of the tube. It has been suggested that a tube with an internal diameter of 6 mm (18 Fr) is the smallest acceptable size because it will allow a maximum possible flow rate of 15 L/min at –10 cm H₂O (170). Our preference is to use at least a 28-Fr chest tube in these patients. Placement of additional chest tubes or CT-guided percutaneous catheters—if the pleural space is complicated—should be considered if the lung is not fully expanded. As with the chest tube, resistance to flow of air through a chest tube drainage system may need to be considered. In an animal model of BPF, when the size of air leak reached 4 to 5 L/min, the Thora-Klex and Sentinel Seal systems become clinically impractical. The Pleur-Evac system can handle flow rates up to 34 L/min, although its use with rates greater than 28 L/min is impractical due to intense bubbling in the control chamber. The Emerson pump, which can be set to deliver chest tube suction greater than –20 cm H₂O, is capable of handling air flows up to 35 L/min and is the system of choice for BPFs with extremely high flow rates (170).

Manipulation of the level of chest tube suction may affect BPF air flow, and some authors have suggested using the least amount of suction that maintains lung inflation (159,160). An animal model demonstrated that increasing negative intrapleural pressures increased air flow in large BPFs but had no effect on small BPFs (171). Roth et al. (167) reported that increasing chest tube suction from 0 to 22.5 cm H₂O increased BPF flow in a patient from 24.6 to 26.7 L/min. In a study of six patients by Powner et al. (165), increasing chest tube suction from 0 to 25 cm H₂O increased BPF flow in two patients, had no effect in two patients, and decreased flow in two patients. To decrease air loss through the BPF when there is applied PEEP, some investigators have applied PEEP to the chest tube (162,172), while others have devised systems to synchronously occlude the chest tube during inspiration (173,174); a lack of success using these methods has been noted by other investigators, however (159). These techniques may pose a risk of increasing the size of the pneumothorax or causing a tension pneumothorax; thus, the patient should be closely monitored.

The goals of mechanical ventilation in patients with a BPF are to maintain adequate oxygenation and ventilation while reducing fistula flow. In general, strategies for conventional mechanical ventilation that limit airway pressure and tidal volumes may reduce the amount of air flow escaping through the BPF and allow the fistulous site to heal. As such, it has been recommended to use the lowest possible tidal volume, fewest mechanical breaths/min, lowest level of PEEP, and shortest inspiratory time.

Alternative methods of mechanical ventilation have been used in a few patients. High-frequency jet ventilation (HFJV) and high-frequency oscillatory ventilation (HFOV) have been used based on the principle that lower airway pressures may be

<table>
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<tr>
<th>TABLE 115.3</th>
<th>Potential Options for Management of Bronchopleural Fistula in Mechanically Ventilated Patients</th>
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<tbody>
<tr>
<td><strong>Chest Tube Drainage</strong></td>
<td>Adequate size chest tube, Drainage system with adequate ability to handle air leak, Additional chest tube placement if lung not fully expanded</td>
</tr>
<tr>
<td><strong>Reduce Airway Pressures</strong></td>
<td>Reduce delivered tidal volume, Use synchronized intermittent mandatory ventilation (SIMV) instead of assist-control mode, Decrease level of positive end-expiratory pressure (PEEP), Decrease inspiratory time (I:E ratio), Avoid Inspiratory pause, Minimize auto-PEEP</td>
</tr>
<tr>
<td><strong>Alternative Modes of Mechanical Ventilation</strong></td>
<td>High-frequency jet ventilation, High-frequency oscillatory ventilation, Independent lung ventilation</td>
</tr>
<tr>
<td><strong>Chest Tube Manipulation</strong></td>
<td>Decrease chest tube suction, Apply PEEP to chest tube, Inspiratory chest tube occlusion</td>
</tr>
<tr>
<td><strong>Direct Closure/Occlusion of Bronchopleural Fistula (BPF)</strong></td>
<td>Surgical closure or resection, Endobronchial occlusion of BPF, Cyanoacrylate-based tissue adhesives, Fibrin sealants, Synthetic hydrogel, One-way endobronchial valves, Stent placement, Pleurodesis, Blood patch, Talc, Doxycycline</td>
</tr>
</tbody>
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generated in these modes of ventilation and should, therefore, decrease BPF air flow. In one animal model of BPF, an increase in fistula flow was seen with increasing mean airway pressures, and effects on flow were similar whether mean airway pressure was changed by manipulating peak inspiratory pressure, PEEP, or inspiratory:expiratory (I:E) ratios (171). In another animal model, a nonsignificant trend toward increasing BPF flow with increasing peak inspiratory pressures, and a significant increase in BPF flow with increasing PEEP was observed (175).

Several studies comparing HFJV and HFOV with conventional ventilation using animal models have shown less BPF air flow during HFJV and HFOV (176–179), although one study using HFJV demonstrated no difference (180). In studies reporting blood gases, improved oxygenation was seen during HFJV and HFOV compared with conventional ventilation (177–179). Increasing levels of PEEP were also noted to increase BPF flow in two studies (176,180). It is problematic to extrapolate these studies to patients in the ICU because the animal models were cannulated in more proximal bronchi and the lung parenchyma was relatively normal.

Other modes of mechanical ventilation have also been used in patients with BPF. Case reports have reported independent lung ventilation to be of benefit (181–183). Case reports of combining independent lung ventilation with high-frequency, low tidal volume ventilation of the affected lung (184) and HFJV of the affected lung have been published (185,186). Differential lung ventilation using a single ventilator and a variable resistance valve attached to one lumen of a bifurcated endotracheal tube has also been described (187,188). Discussion of the techniques of independent lung ventilation and its attendant difficulties is beyond the scope of this chapter, and the reader is referred to other reviews (189,190).

Because many critically ill patients are unable to tolerate a major thoracic procedure, bronchoscopic techniques may provide viable alternatives for closure of BPFs. Endobronchial occlusion of BPFs has been reported with cyanoacrylate-based tissue adhesives (Histoacryl, Bucrylate), fibrin sealants (Tissel, Hemaseal), thrombin plus fibrinogen or cryoprecipitate, synthetic hydrogel (CoSeal), absorbable gelatin sponge (Gelfoam), vascular occlusion coils, doxycycline and blood, Nd:YAG laser, silver nitrate, and lead shot (191–193). The agent initially seals the leak by acting as a plug and subsequently induces an inflammatory process with fibrosis and mucosal proliferation, permanently sealing the area. Of these techniques, the uses of cyanoacrylate tissue adhesives and fibrin sealants have been most widely reported. Airway stents may be used to cover and seal the fistula in selected patients depending on the location of the fistula. BPFs due to breakdown of a stump after lobectomy or pneumonectomy, or bronchial dehiscence after lung transplantation or bronchoplasty procedures are the most amenable to successful closure with airway stenting. Successful closure of BPFs using bronchoscopic placement of endobronchial valves designed for emphysema has been described (194–196). More recently, synthetic hydrogel (CoSeal) was used to successfully seal the air leak in patients with BPF in a single center study (191).

Pleuradisome with various agents has also been tried to affect closure of BPFs. Autologous “blood patch” pleurodism has been described to be effective in some patients (197–199). Pleurodism with fibrin glue has also been reported (200,201). However, none of these patients was undergoing mechanical ventilation at the time of pleurodism.

Key Points

- The major radiographic finding of a pleural effusion in the supine patient is an increased homogeneous density over the lower lung field that does not obliterate normal bronchovascular markings, does not demonstrate air bronchograms, and does not produce hiliar or mediastinal displacement until the effusion is massive.
- The radiographic signs of pneumothorax in the supine patient frequently differ from the classic visceral pleural line seen on erect views. The anteromedial position is the most common location for pneumothoraces in the supine patient since this area is the least dependent pleural recess. The lucency sharply outlines adjacent vascular structures such as the ascending aorta, superior vena cava, and ayzygos vein.
- Ultrasonographic assessment of pneumothorax has emerged as a first-line diagnostic modality for physicians trained in bedside ultrasonography.
- Patients with ARDS can develop tension pneumothoraces despite the presence of a chest tube on the ipsilateral side being placed for a previous pneumothorax.
- In patients with complicated parapneumonic effusion or empyema, formal pleural drainage with a chest tube is indicated.
- Evidence of pus, loculations, pleural thickening, and progressive organization in the chest would suggest that patients would benefit from intrapleural fibrinolytic therapy.

References


