chapter

89

SPONTANEOUS PNEUMOTHORAX AND PNEUMOMEDIASTINUM

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Key Points

- Primary spontaneous pneumothoraces occur in young patients without lung disease; secondary spontaneous pneumothoraces occur in patients with chronic obstructive pulmonary disease (COPD).
- The most common cause of a primary spontaneous pneumothorax is the rupture of small subpleural blebs.
- Pneumothoraces are considered small if they measure less than 3 cm and large if they measure more than 3 cm.
- Conventional tube thoracostomy with underwater seal drainage remains the procedure of choice for the initial management of moderate to large pneumothoraces.
- Surgery is indicated if there is recurrence after a first episode.
- Surgery is indicated at the time of the first episode if the pneumothorax is complicated by persistent air leak, hemothorax, or failure of the lung to re-expand.
- Resection of blebs and bullae and obliteration of the pleural space by pleurectomy or pleural abrasion, alone or in combination, are the two major goals in the surgical treatment of spontaneous pneumothoraces.
- With video-assisted thoracic surgery (VATS), analgesic requirements and length of hospital stay are reduced, but the recurrence rate is slightly higher.
- Rarely, surgeons use chemical pleurodesis as first-line treatment for recurrences of pneumothorax.
- Spontaneous pneumomediastinum (SPM) is defined as nontraumatic presence of free air in the mediastinum in a patient with no known underlying disease.
- After specific causes of mediastinal emphysema have been excluded, primary SPM can be treated expectantly.

Pneumothorax is defined by the presence of air in the intrapleural space, with secondary lung collapse. Although such air may originate from various sources, rupture of the visceral pleura with air leakage from the lung parenchyma is by far the most common cause.

Pneumothoraces can be classified as spontaneous, posttraumatic, and iatrogenic (Table 89-1). Whereas primary spontaneous pneumothoraces occur in young patients without lung disease, secondary spontaneous pneumothoraces occur in patients with clinical or radiographic evidence of underlying lung disease, most often COPD. Posttraumatic pneumothoraces are the result of blunt injuries to the bronchi, the lung, or the esophagus. An open pneumothorax happens when a penetrating trauma induces a disruption of the chest wall.

Iatrogenic pneumothoraces may occur during a diagnostic or therapeutic procedure in the hospital environment. Artificial therapeutic pneumothorax refers to the historical treatment for tuberculosis.

Most patients with a spontaneous pneumothorax seek medical attention because of sudden chest pain and dyspnea. If the spontaneous pneumothorax progresses to become under tension, the symptoms are more severe, and significant hemodynamic and respiratory instability may develop and require urgent treatment.

HISTORICAL NOTE

Boerhaave in 1724 was the first to identify the presence of abnormal air in the chest cavity, and Meckel (1759) was the first to describe a tension pneumothorax at the time of a postmortem examination.[3] The term *pneumothorax* was introduced by Etard in 1803, but it was Laennec (1819) who first described the clinical signs and symptoms accompanying a spontaneous pneumothorax (Killen and Gobbel, 1968).[1,2] For a long period, tuberculosis was considered as the principal cause of pneumothoraces. Kjaergaard (1932) was the first to propose rupture of isolated blebs located at the apex of the lung as the most common cause of spontaneous pneumothorax in young healthy adults.[3]

Currently, COPD and diffuse interstitial lung disease are among the leading causes of the secondary pneumothoraces in the aging population.[4,5] Secondary spontaneous pneumothoraces are also found in other lung diseases such as acquired immunodeficiency syndrome (AIDS) and severe acute respiratory syndrome (SARS) (Table 89-2).[6,7]

Before intercostal tube thoracostomy became the treatment of choice, several weeks of bed rest was the only treatment for these patients.[8]

Thoracotomy for the resection of apical blebs was suggested by Bigger in 1937 and adopted by Tyson and Crandall, who reported their results a few years later.[9,10]

Churchill (1941) was the first to suggest gauze abrasion of the parietal pleura to generate pleural adhesions in order to reduce recurrences.[2] Gaensler and later Thomas and Gebauer proposed parietal pleurectomy as the best method for an effective pleurodesis.[11,12] There has been some debate over the years about the best method to reduce recurrences. Clagett did not agree with pleurectomy, which he thought was too aggressive for the treatment of a benign disease.[13] He recommended pleural abrasion, and Youmans and colleagues, in 1970, provided additional clinical and experimental evidence of the efficacy of pleural abrasion.[14]

Deslauriers and associates were the first to report very low recurrences with a combination of blebectomy and limited apical pleurectomy performed through a transaxillary incision.[15] This proved to be an ingenious way to deal effectively

TABLE 89-1 Classification of Pneumothorax

Spontaneous
Primary
Secondary (underlying pulmonary disease)
Chronic obstructive pulmonary disease infection
Neoplasm
Catamenial
Posttraumatic
Blunt penetrating
Iatrogenic
Inadvertent
Diagnostic
Therapeutic

with the disease and reduce the morbidity related to a standard thoracotomy. Levi and coworkers introduced video-assisted thoracic surgery (VATS) for the surgical treatment of spontaneous pneumothorax.[16] With the development of minimally invasive surgery, the VATS approach has gradually replaced the transaxillary approach (Table 89-3).

BASIC SCIENCE

Anatomy of the Pleural Space

The visceral pleura covers the entire surface of the lung and has no plane of dissection with the parenchyma; the parietal pleura is a serous membrane that covers the inner surfaces of the mediastinum, the chest wall, the diaphragm, and the apex of the chest cavity. The presence of the endothoracic fascia between the pleura and the chest wall provides a plane of dissection that makes it easy for surgeons to perform a parietal pleurectomy. The parietal pleura is vascularized through branches originating from intercostal arteries and the apical pleura through branches of the subclavian artery. The parietal pleura, unlike the visceral pleura, has somatic innervation, and pain stimuli are transmitted through the intercostal and phrenic nerves.[17]

Physiology of the Pleural Space

When a patient is at rest (i.e., at functional residual capacity), the elastic forces of the chest wall and lung tend to separate the parietal pleura from the visceral pleura, creating a negative pressure with respect to atmospheric and alveolar pressure. This negative intrapleural pressure is not uniform throughout the pleural space; a gradient exists between the apex and the base of the lung. At the apex, the pressure is more negative than at the base; this difference tends to favor greater distention of the apical alveoli. In tall individuals, this gradient may be even greater and probably contributes to the development of pneumothoraces through the rupture of apical blebs.

Physiologic Changes Secondary to a Pneumothorax

Uncomplicated Pneumothorax

When a communication develops between the lung and the pleural space, the positive pressure of intra-alveolar air makes the air flow from the lung into the pleural space until there is no difference between the intrapleural pressure and the atmospheric pressure (Fig. 89-1). The same mechanism of pressure equilibration occurs when there is a communication between the opened chest wall and the pleural cavity.

Alveolar hypoventilation and hypoxemia are the consequences seen in patients with any significant (>25%) pneumothorax.[18] According to Moran and coauthors, hypoxemia is related to an alteration of the ventilation-perfusion ratio.[19] Anatomic shunting also contributes to the low arterial oxygen pressure.[20] Anthonisen suggested that the ventilation mismatch often seen in pneumothorax was secondary to the airway closure at low lung volumes.[21]

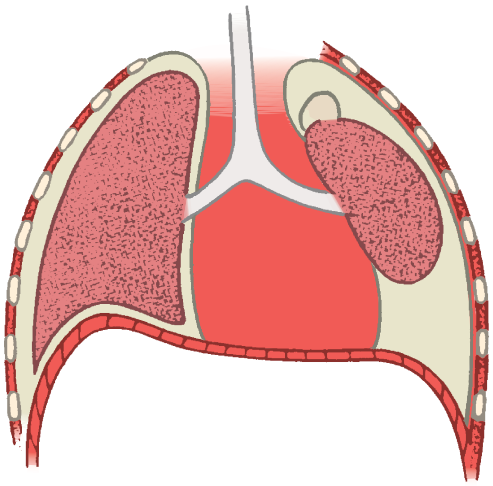
A pneumothorax also affects the mechanics of the lung and leads to reductions in lung compliance, vital capacity, total capacity, and functional residual capacity.[22] Finally, the normal

TABLE 89-2 Historical Landmarks

Author (Year)	Landmark
Boerhaave (18th century)	Ruptured esophagus associated with presence of air in the pleural cavity
Meckel (18th century)	Postmortem description of a tension pneumothorax
Etard (19th century)	Autopsy description; introduction of the term pneumothorax
Laennec (19th century)	Description of clinical signs and symptoms
Kjaergaard (1932)	Rupture of lung blebs replacing tuberculosis as the most frequent cause of primary spontaneous pneumothorax
Getz and Beasely (1983)	Chronic obstructive lung disease as a frequent cause of pneumothoraces
Wait and Estrera (1992)	*Pneumocystis carinii* pneumonia, cytomegalovirus pneumonia, and atypical mycobacterial infections associated with AIDS are common causes of pneumothorax

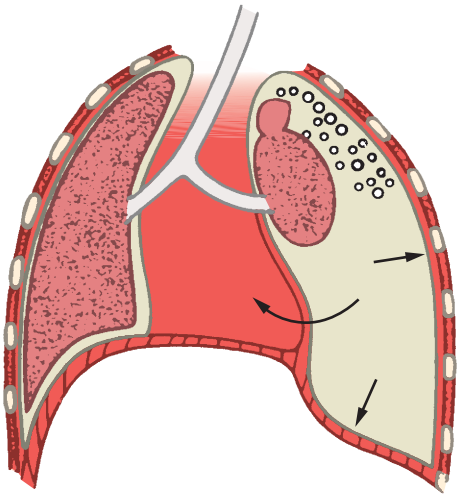
TABLE 89-3 Evolution of Therapy

Author (Year)	Therapy
Tyson and Crandall (1941)	Thoracotomy, resection of blebs
Gaensler (1956) Thomas and Gebauer (1958)	Subtotal parietal pleurectomy
Deslauriérs et al (1980)	Axillary thoracotomy, bleb resection, and apical pleurectomy
Levi et al (1990)	Video-assisted thoracoscopic surgery for bleb resection, pleural abrasion, or pleurectomy



↑ Air flow until no pressure difference
↓ Apex to base pressure gradient
↓ Lung compliance
↓ Functional residual capacity
↓ Ventilation
↓ Oxygenation
Slight shunt

FIGURE 89-1 Some of the physiologic characteristics of spontaneous pneumothorax.



↑ Continuous air flow (one-way valve)
↑ Intrapleural pressure
↑ Mediastinal shift (alteration of lung mechanics)
↓ Ventilation
↑ Shunt
↓ Oxygenation
↑ Cardiac stroke volume
↑ Heart rate

FIGURE 89-2 Physiologic characteristics of a tension pneumothorax.

gradient of pressure between apex and base in the pleural space tends to disappear (see Fig. 89-1).[23]

Tension Pneumothorax

During an episode of tension pneumothorax, a positive intrapleural pressure builds up during the expiration phase. This is because the air flow accumulates in the pleural space without any possibility of evacuation from a closed chest cavity.

It was long believed that a tension pneumothorax induces mediastinal compression and a decrease in venous return to the heart that ultimately decreases the cardiac output.[24] According to Gutsman and colleagues, however, only a fraction of the increase in intrapleural pressure is transmitted to the mediastinum, suggesting that mediastinal compression by accumulated air is not the sole explanation for the cardiovascular changes observed in a tension pneumothorax.[25]

In an experimental model, Hurewitz and associates showed that a fall in cardiac stroke volume as well as a progressive reduction in systemic oxygen transport and tissue oxygenation occurred during an episode of tension pneumothorax.[26] The insufficient tissue oxygenation of the heart resulted in the inability to increase the cardiac output.

Tension pneumothorax can also induce a shunt and thereafter reduce oxygenation and cardiac output (Fig. 89-2).

Resorption of Pleural Gas

The normal pleural space is free of gas. The pleural membrane is a semipermeable structure through which gases move by simple diffusion and equilibration. The rate of resorption of a pneumothorax is related to the quality of the pleural membrane. With a fibrotic pleura, for instance, the rate of resorption is slower than with a normal pleura. The rate of resorption is also proportionate to the total surface area of the pleura and to the amount of residual gas in the pleural cavity. The greater the amount of gas, the longer it takes for resorption. In pneumothoraces, the pressure gradient between the gas in the pleural space and the gas in the subpleural venous system is the driving force directing this diffusion process. Each gas is resorbed independently of the other.

Gas resorption from the pleural space takes place gradually and in successive phases. During the first phase, there is equilibration of oxygen and carbon dioxide partial pressures; during the second phase, there is a progressive resorption of the remaining intrapleural gases. Gradually, the intrapleural pressure recovers its negative pressure favoring lung re-expansion. If the lung does not re-expand, a transudate fills the pleural cavity.[27]

Finally, the composition of gases in the pleural space can also vary. For example, oxygen is more diffusible and soluble, and its transfer from pleura to circulation is faster than that of carbon dioxide or nitrogen.

PRIMARY SPONTANEOUS PNEUMOTHORAX

Etiology and Epidemiology

The most common cause of a primary spontaneous pneumothorax is the rupture of small subpleural blebs. This may occur when a patient is at rest or during exertion, and it is

seen stereotypically in young, healthy, thin and tall male smokers.[28,29] With the increased incidence of smoking, women are now also at risk.[30] In North America, the incidence varies from 6 to 7 per 100,000 in men and from 1 to 2 per 100,000 in women.[28] A familial incidence has been reported.[31]

According to Brooks, primary spontaneous pneumothorax occurs more commonly on the right side.[32] Bilateral pneumothoraces occur in fewer than 10% of patients.[33]

In addition to smoking, other explanations for the development of a spontaneous pneumothorax are still open to discussion. Exertion is probably not a major factor, but change in atmospheric pressure is considered an important cause.[34] In one study, the rate of hospital admission for spontaneous pneumothorax was significantly greater during the first 48 hours after a fall in the atmospheric pressure.[35] Studies on flight personnel with prior episodes of pneumothorax also showed that small apical bullae increase in size when subjected to decreasing atmospheric pressure in altitude chambers.[36]

Histopathology

Blebs are small (<2 cm) subpleural collections of air contained within the visceral pleura. They result from ruptured alveoli with air trapping between the elastica interna and externa of the visceral pleura. Blebs are usually found at the apex of the upper lobes or over the superior apices of the lower lobes.[37] Blebs are well demarcated from the remaining normal lung, although they are still attached to it by a narrow neck. They are considered to represent the paraseptal variety of emphysema, which can occur independently of widespread centriacinar or panacinar emphysema. Blebs are often accompanied by apical fibrosis of the lung.[38]

The formation of blebs is associated with the degradation of elastic fibbers. This process of elastolysis, in which neutrophils and macrophages play a role, is caused by imbalances between proteases and antiproteases and between oxidants and antioxidants.[39-42]

Bullae are found in secondary pneumothoraces. Bullae usually result from the alveolar wall destruction typically seen in diffuse emphysema.[43]

In 1966, Reid proposed a classification of the types of bullae found in the lung.[44] Type I bullae have thin walls made of pleura and connective tissue with few blood vessels. These bullae are located at the apices of upper lobes or over the edges of other lobes. They correspond to overinflation of a small volume of parenchyma and communicate with the lung by a narrow neck. Blebs are type I bullae. In type II bullae, the mesothelial cells are relatively well preserved but alveolar structures are destroyed at their base. The diseased alveoli are in continuity with the bullae through a broad neck. In type III bullae, the base of the bulla is large and extends deep into the lung.

Diagnosis and Staging

The severity of symptoms such as chest pain and dyspnea usually, but not always, correlates with the degree of lung collapse. Chest pain is very common and can be quite severe. It is often described as an early sharp pain followed by a steady pain. The symptoms of dyspnea and chest pain usually decrease gradually and resolve during the 24 hours following the episode.[45] Occasionally, patients have a nonproductive cough. Most episodes occur while the patient is at rest. Physical findings may be totally absent if lung collapse is minimal, but when a significant pneumothorax is present, there is often a decrease in chest wall movement on the affected side. On percussion, the chest cavity may be hyperresonant, and at auscultation, breath sounds are diminished or absent. A pleural friction rub can sometimes be heard, and tachycardia is almost invariably present.

The diagnosis of pneumothorax is best confirmed by erect posteroanterior (PA) and lateral chest radiographs through identification of the visceral pleura, which normally is not recognizable, and the presence of abnormal air in the pleural cavity (Fig. 89-3). Expiration films are useful in demonstrating a small pneumothorax that may have been missed on a standard film (Fig. 89-4). Lateral decubitus views may be advantageous in confirming the presence of a pneumothorax.[46]

Pseudopneumothoraces such as skin folds or chest wall alterations must always be ruled out. Occasionally, a pulmonary cyst or emphysematous bulla may also be mistaken for a pneumothorax. The diagnosis of a tension pneumothorax is suggested by complete collapse of the lung with contralateral shift of the heart and mediastinum and inversion of the hemidiaphragm (Fig. 89-5).

Size of the Pneumothorax

The American College of Chest Physicians defines *small pneumothoraces* as those in which the visceral pleura is less

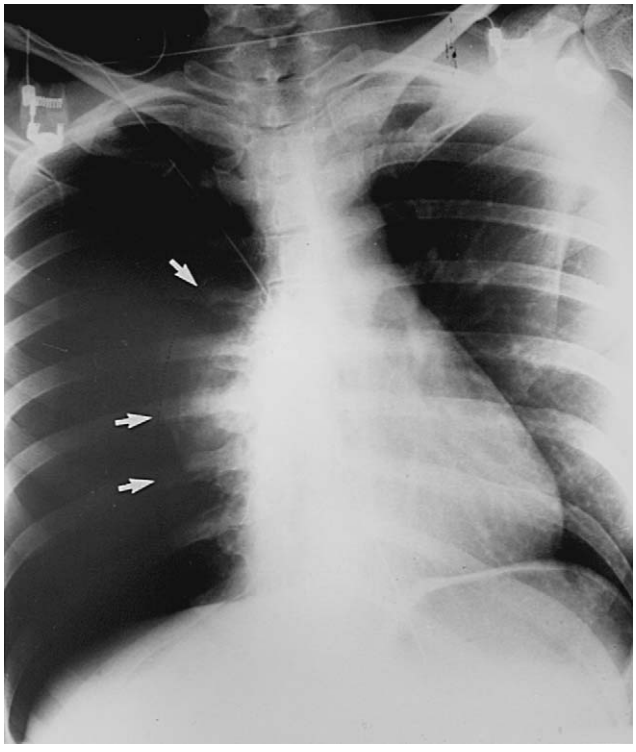
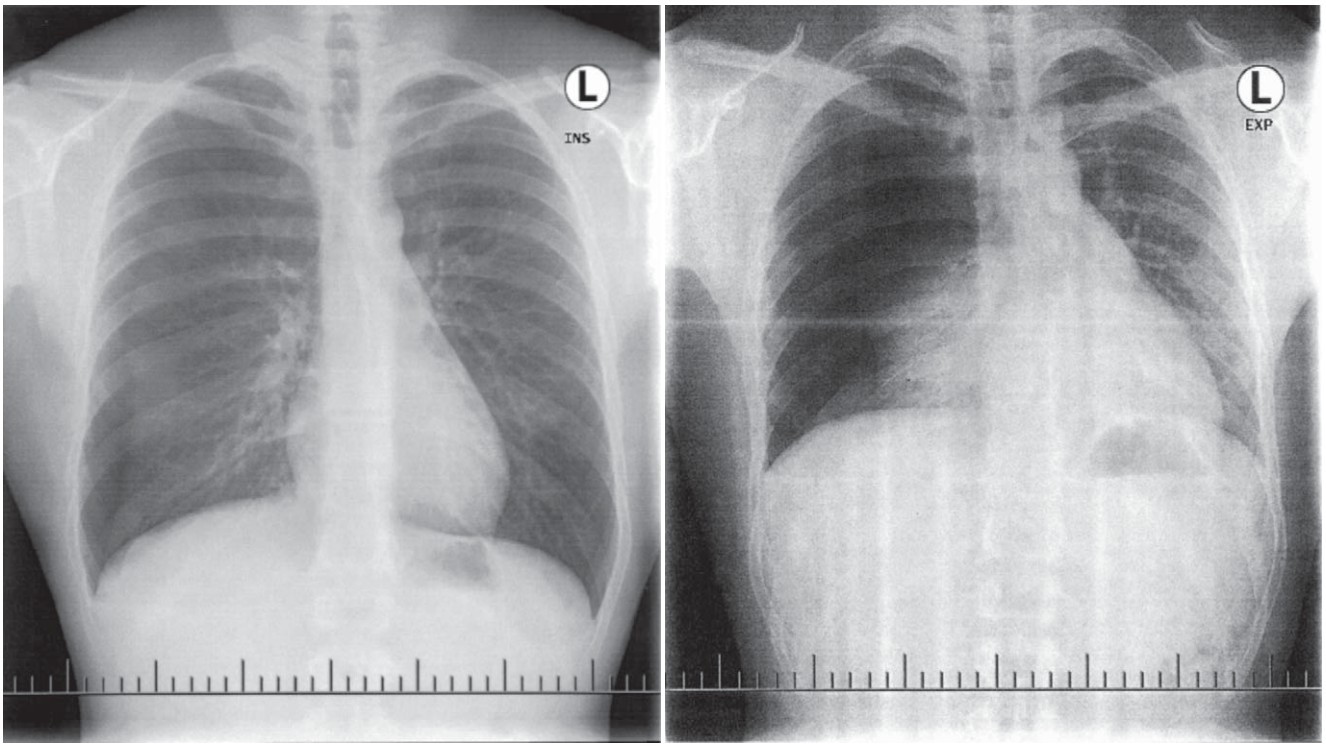


FIGURE 89-3 A pneumothorax is identified by finding the location of the visceral pleura (*arrows*).



A B

FIGURE 89-4 A, Spontaneous pneumothorax (regular film). **B,** Increased evidence with expiration film.

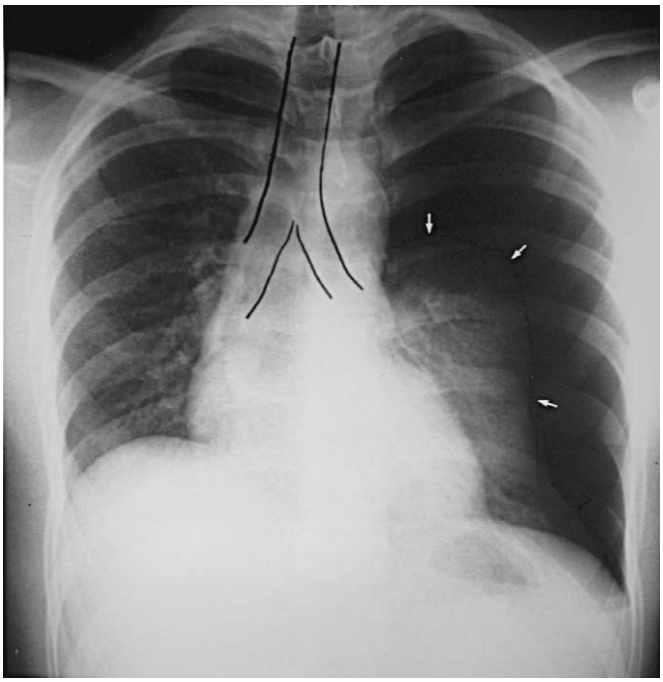


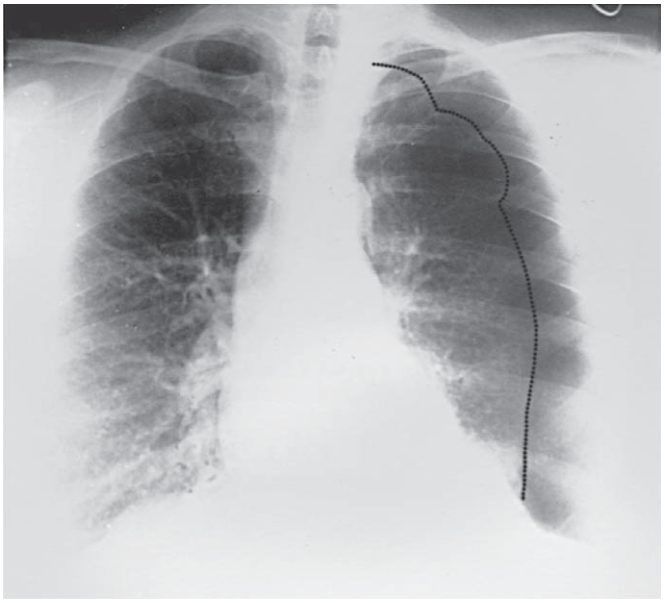
FIGURE 89-5 Chest radiograph showing a tension pneumothorax with mediastinal shift and deviated trachea. The trachea is outlined. *Arrows* indicate location of visceral pleura.

than 3 cm from the chest wall; in large pneumothoraces, the distance is greater than 3 cm.[47] However, according to Light, measuring the average diameter of the collapsed lung and of the involved hemithorax provides a better approximation of the size of the pneumothorax.[48] One must understand that the lung is in a three-dimensional space, and the volume of a pneumothorax approximates the ratio of the cube of the lung diameter to the hemithorax diameter. If a pneumothorax is measured to be 1 cm from the apex to the bottom on the chest radiograph, it occupies approximately 25% of the hemithorax volume. Similarly, in a 2-cm radiographic pneumothorax, approximately 49% of the volume of the hemithorax is occupied by abnormal air. According to the British Thoracic Society guidelines, a pneumothorax of more than 2 cm represents a condition that needs immediate attention.[49]

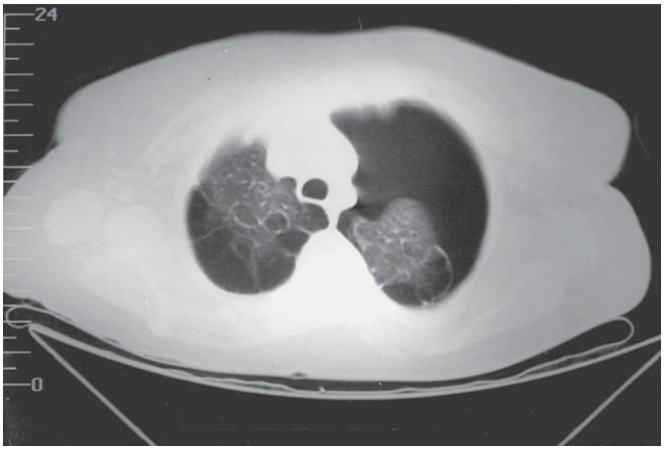
Considering the three dimensions of the pleural cavity, we define a small pneumothorax as one with a diameter of less than 3 cm if located solely at the apex or one in which there is less than a 2-cm rim of air between the lung and the parietal pleura. For a large pneumothorax, the space between the lung and the parietal pleura is greater than 2 cm from the apex to the bottom of the pleural cavity.

Computed tomographic (CT) scanning is the most precise method to estimate the size of a pneumothorax, especially in patients with emphysema or other underlying diseases.[50-52]

A detailed description of the number, size, and location of the blebs by CT scanning is also useful for predicting contralateral occurrences (Fig. 89-6).[53]



A



B

FIGURE 89-6 A, Chest radiograph showing left-sided pneumothorax and normal right lung. **B,** CT scan of the same patient showing ipsilateral and contralateral bullae with left-sided pneumothorax.

Patients with pneumothoraces can also have abnormal electrocardiographic readings. For example, in patients with a left-sided pneumothorax, the electrocardiogram may show changes such as a right-sided shift of the QRS axis with a decrease in precordial R-wave voltage and QRS amplitude and a precordial T-wave inversion; these changes usually resolve with re-expansion of the lung.[54] If a large pneumothorax is present, the interposition of air between the heart and the electrodes may also lead to a decrease in QRS amplitude as well as that of the R and T waves, simulating an anterior myocardial infarction.[55]

COMPLICATIONS OF SPONTANEOUS PNEUMOTHORAX

A tension pneumothorax is a serious complication that occurs when alveolar air continuously accumulates in the pleural space.[56] The patient usually shows signs of respiratory distress, with tachycardia, anxiety, dyspnea, and chest pain. Occasionally, the patient rapidly becomes hypotensive, with peripheral cyanosis and tracheal deviation. It is a life-threatening situation. Immediate decompression of the pleural space with a needle, chest drain, or other instrument is imperative.

Pneumomediastinum is secondary to the dissection of air along the bronchi and vascular sheets of pulmonary vessels. Usually, it has no clinical significance; however, in certain circumstances, an injury to major airways or perforation of the esophagus needs to be ruled out.

Pneumoperitoneum secondary to a pneumothorax is uncommon. In this situation, a perforated abdominal hollow viscus must be ruled out.[57]

Interstitial emphysema and subcutaneous emphysema, which often accompany secondary pneumothorax, are usually of no clinical consequence. Rarely, massive subcutaneous emphysema causes respiratory embarrassment that may require a tracheal intubation and cutaneous incision on the chest wall for decompression.

Hemopneumothoraces occur rarely and are more frequent in men than in women. The hemorrhage is caused by a torn vascular adhesion between the parietal and visceral pleura or, less frequently, by a rupture of vascularized blebs or bullae. Although lung re-expansion may help tamponade the bleeding site, early surgery is often warranted.[58,59]

Bilateral pneumothoraces occur in 10% to 15% of cases. Although these may be synchronous, more often they are sequential.[60]

Recurrence after initial treatment is probably the most frequent complication of primary spontaneous pneumothorax; it occurs in approximately 10% to 15% of the cases. Most recurrences are seen within 2 years of the first episode, and the majority are ipsilateral; after a second pneumothorax, the risk of having a third one increases to 40% to 50%.[61,62]

The most frequent complications after the initial treatment of a pneumothorax are listed:

1. An air leak that does not resolve after 48 to 72 hours in an apparently well-drained chest cavity
2. An incomplete re-expansion of the lung after suction has been applied to the chest tube
3. An important air leak that cannot be controlled successfully with a chest tube

In the aforementioned conditions, surgery is often required to solve the problem.

MANAGEMENT

Management of the First Uncomplicated Episode

At the time of a first episode, various therapeutic strategies such as observation, aspiration, chest tube drainage, and surgery may be used, depending on the size of the pneumothorax and the presence of symptoms.

Observation

Asymptomatic patients in good health who have a small pneumothorax may be treated expectantly if there is no clinical or radiographic evidence of progression.[63]

Air resorption from the pleural space is estimated to be 1.25% of the volume of the pneumothorax per 24 hours (50 to 70 mL/day).[64] Administration of oxygen, which is reabsorbed faster than room air, is an effective method to enhance the rate of resolution.[65] It may avoid the need for a chest tube drainage in a small pneumothorax.[66]

Patients who are treated expectantly are observed very closely, especially if they are sent home. Before hospital discharge, they are instructed about the potential hazards of developing a tension pneumothorax, and they must be monitored with weekly chest radiographs until the pneumothorax has completely resolved. This method of treatment exceeds tube thoracostomy in duration. In addition, one must remember that mortality secondary to unrecognized tension pneumothorax can happen in patients treated by observation alone.[67] Nevertheless, careful observation remains a valid therapeutic option for patients with small pneumothorax.

Aspiration

Aspiration of the pneumothorax with a 16-gauge intravenous canula connected to a three-way stopcock and 60-mL syringe is a treatment option that is successful in approximately 50% of patients.[68] According to the British Thoracic Society, it is the method of choice for treatment of the first episode of a spontaneous pneumothorax.[49] It is successful in 83% of patients without lung disease but in only 33% of patients with preexisting lung disease. Those who appear to benefit from this approach are patients younger than 50 years of age whose pneumothorax is smaller than 50%.[69,70]

Because of a 20% to 50% recurrence rate with this method, small-caliber catheters are often used to decompress iatrogenic pneumothoraces.[71]

Since the introduction of the dart technique for the emergency treatment of pneumothoraces, several devices have become commercially available. The McSwain dart is a 16 Fr, 15-cm polyethylene catheter with a winged flange attached to a flutter valve, and the thoracic vent is a small-bore 13 Fr urethane catheter trocar with a one-way valve connected to a suction apparatus or underwater seal system.[72] Chest tubes smaller than 9 Fr are often associated with malfunction and occlusion.

Tube Thoracostomy

Conventional tube thoracostomy with underwater seal drainage remains the procedure of choice for the initial management of moderate to large pneumothoraces. Significant symptoms, radiographic progression, complete collapse of the lung, tension pneumothorax, contralateral pulmonary disease, and failure of re-expansion after aspiration are all indications for tube thoracostomy. With proper tube drainage, the lung re-expands rapidly, and in most patients the air leak stops within 48 hours. Although underwater seal drainage is sufficient for most cases, we personally prefer to use negative pressure (–10 to –20 cm of H_2O) on the chest tube to maintain lung re-expansion during the first 24 hours.

The chest tube is removed after the air leak has stopped for at least 24 hours and preferably 48 hours. A radiologic control is always recommended before the patient is discharged. In spite of satisfactory lung re-expansion, immediate relapse after removal of the chest tube is not uncommon.

Although most of our patients are kept in the hospital for 24 hours with underwater seal drainage and suction, in selected cases we use the Heimlich valve. The Heimlich flutter valve is a passive, one-way, ambulatory chest tube drainage system.[73] It has been shown to be safe and efficient for the outpatient treatment of primary spontaneous pneumothorax.[74] In our experience, it is installed only after underwater seal drainage has been in place for several hours to allow for a full re-expansion of the lung. After the lung is re-expanded and if air leak is minimal, a Heimlich valve is installed, and the patient is discharged from the hospital. If, during the early follow-up period, the lung does not re-expand, the patient is admitted to the hospital for underwater seal drainage.

Indications for Surgery at the Time of the First Episode

Complicated Pneumothorax

Surgery may be indicated at time of the first episode if the pneumothorax is complicated by persistent air leak, hemothorax, or failure of the lung to re-expand. Bilaterality, tension pneumothorax, and complete pneumothorax may also be indications for surgery (Table 89-4). In contrast, surgery is rarely indicated for psychological reasons such as fear of pain related to the chest tube insertion.[75]

A prolonged air leak lasting longer than 4 days is probably the most frequent indication for surgery at the time of the first episode. Most air leaks in patients with primary spontaneous pneumothorax seal within 24 to 48 hours after the insertion of a chest tube. Only 3% to 5% of patients have a persistent air leak. Today, there are very few good reasons for prolonged chest tube drainage.[76] Considering the efficacy and the low morbidity of surgery, as well as the low recurrence rate, early surgical intervention is advocated.[77]

The occurrence of simultaneous bilateral pneumothoraces is uncommon, but when it occurs it is followed by definitive surgery, at least on one side but preferably on both sides.

TABLE 89-4 Indications for Surgery in Primary Spontaneous Pneumothorax

First Episode
Early complications
Prolonged air leak
Non–re-expansion of the lung
Bilaterality
Hemothorax
Tension
Complete pneumothorax
Potential hazards
Occupational hazard
Absence of medical facilities in isolated areas
Associated single large bulla
Psychological
Second Episode
Ipsilateral recurrence
Contralateral recurrence after a first pneumothorax

Patients with a significant hemothorax may need urgent surgery for definitive control of the bleeding site. Large clotted hemothoraces (>1000 mL), if inadequately drained, may lead to complications such as empyema and fibrothorax and therefore needs to be evacuated.

Patients at risk of developing pneumothoraces in relation to their occupation (e.g., airline personnel, scuba divers) may be treated with surgery at the time of the first episode.[78] Patients living in isolated areas and patients who travel frequently, especially those with evidence of bullae on chest radiographs, may also be candidates for early surgery. The management of a pneumothorax that occurs in a pregnant woman during the first trimester or near parturition needs to be conservative.

A first episode of a tension pneumothorax is an indication to proceed with surgery before the patient leaves the hospital.

If a single large bulla or multiple blebs are identified on chest radiography or CT at the time of a spontaneous pneumothorax, surgery is also recommended.

Uncomplicated Pneumothorax

The option for surgery at the time of a first uncomplicated episode is now a matter of debate.[79-82] The choice for surgery is based on patient preference in certain cases but more often on economic concerns such as potentially decreased duration of work disability and decreased overall medical costs.[83-85] The argument against surgery is that most patients (80%) will not have any recurrence after the first episode and therefore would undergo unnecessary surgical risk.[86-88] We maintain that easy access to VATS does not modify the indications for surgery at the time of the first episode.

Management of Recurrences

The most frequent reason for considering surgery for a spontaneous pneumothorax is recurrence after conservative treatment. Approximately 20% of the patients have a recurrence within 2 years after a first episode. After three or more episodes of spontaneous pneumothorax, the incidence of recurrence in the following 2 years increases up to 50%, and in our opinion surgery is the only good option for these patients. If a recurrence occurs during pregnancy, surgical treatment may be carried out safely during the second trimester.[89]

Surgical Strategies

Resection of blebs and bullae and obliteration of the pleural space by pleurectomy or pleural abrasion, alone or in combination, are the two major goals in the surgical treatment of spontaneous pneumothorax.[90]

If apical blebs are present, wedge resection of the blebs is necessary.[91,92] Multiple wedge resections may be required if there are blebs at several sites. Anatomic segmentectomies or lobectomies are rarely indicated. Blebs are not always found at the time of surgery. A single bleb may have ruptured and therefore is no longer visualized.[93] In addition, there are several patterns of the disease. In the first pattern, which occurs in about 30% to 40% of the patients, the lung is normal. In the second pattern, in 12% to 15% of patients, the lung appears normal but there are pleuropulmonary adhesions, suggesting previous pneumothoraces. It is in the third group, comprising 30% to 40% of patients, where one finds single or multiple blebs. Finally, in 5% to 10% of patients, one finds multiple bullae larger than 2 cm. Retrospective studies showed that apical stapling reduced the recurrence rate from 23% when no resection was made to 1.8% when resection was accomplished.[94,95] Resection of the apex decreased the rate of recurrence even if no abnormalities were found.[96] Although resection of the apex of the lung where the blebs are located is very effective to prevent recurrences, obliteration of the pleural space by pleurectomy, pleural abrasion, or pleurodesis is a second maneuver to ensure the lowest possible recurrence rate.

Obliteration of the pleural space can be accomplished by parietal pleurectomy or mechanical abrasion of the parietal pleura because each of these methods creates an inflamed surface with secondary fixation of the lung to the endothoracic fascia. Parietal pleurectomy limited to the apex creates sufficient adhesions to prevent recurrences. Gaensler found no evidence of any respiratory functional restriction after pleurectomy.[11] The morbidity associated with apical pleurectomy is low, although significant complications such as hemorrhage can occasionally happen.[97] When used as the sole procedure, pleurectomy produces excellent results, with a recurrence rate varying from 1% to 5%.[15,98-100]

Pleural abrasion also produces effective obliteration of the pleural space. It has the advantage of preserving the entire pleura and resulting in fewer hemorrhagic complications than pleurectomy.[101,102] In nine reported series reviewed by Weeden and Smith, the recurrence rate of pneumothorax after pleural abrasion alone was 2.3%.[97] It appears that a combination of resection of the blebs and pleurodesis is a safer treatment.[103]

We routinely proceed with a wedge resection of the apex, followed by an apical pleurectomy and pleural abrasion of the rest of the parietal pleura. Occasionally, the inferior pulmonary ligament is taken down. A single 28 Fr chest tube is left in the pleural place and can often be removed within 48 hours after surgery. Most patients are discharged from the hospital by the third or fourth postoperative day.

Surgical Approaches

A full posterolateral thoracotomy is seldom necessary for the surgical treatment of spontaneous pneumothorax. The apex of the lung can be resected, and a pleurectomy or pleural abrasion can easily be performed through a less invasive and more cosmetically acceptable axillary incision[15,98] or a limited thoracotomy.

The standard VATS with three ports is very popular and is currently the technique of choice in the surgical community. Uniportal VATS has recently been proposed as an alternative to reduce the neurologic complications.[104]

VATS is performed with the patient under general anesthesia with a double-lumen endotracheal tube. Three ports of entry are usually necessary to introduce a rigid telescope with video attachment and instruments to perform the blebectomy, pleurectomy, or pleural abrasion. Proper port place-

ment is essential to produce the triangulation that is required to fully access all lung zones and to eliminate criss-crossing of instruments. Selection of the appropriate intercostal spaces for placement of trocars is adjusted according to the patient's morphologic form. Blebs are best resected with an endoscopic linear stapler, and this is followed by pleural abrasion or subtotal parietal pleurectomy.

Treatment for Bilateral Disease

Some surgeons select simultaneous bilateral thoracotomy or single-stage bilateral transaxillary thoracotomies even if the pneumothorax is unilateral.[105] VATS is a good option for performing bilateral bleb excisions during the same procedure, but the surgical risk needs to be weighed against the benefits.[106] To avoid incisions on both sides, an ipsilateral VATS with surgery on the contralateral lesion has been described.[107] A median sternotomy has been advocated when bilateral disease is identified before surgery.[108] We prefer bilateral staged operations if bilateral disease is present.[109]

Morbidity of VATS

Most patients have minimal postoperative morbidity and discomfort; however, chronic postoperative pain requiring analgesic medication is always possible after any VATS procedure. Postoperative chest wall pain can be very distressing. The pain is secondary to trocar insertion and manipulation of the instruments in the intercostal space, which contributes to injury of the intercostal nerves. Placing the incisions anteriorly in wider intercostal spaces for the bigger trocars and using the posterior trocar for smaller instruments (5-7 mm) may help prevent postoperative pain. The postoperative chest wall pain that occurs after VATS is not negligible and may represent a significant problem in young patients.[95]

Results of VATS

In a retrospective study, Cole and coworkers failed to demonstrate any advantage of VATS over axillary thoracotomy in terms of hospital stay or morbidity.[85] Kim and colleagues found no advantage of VATS over axillary thoracotomy with regard to operating time, amount of analgesics used during the first postoperative day, duration of chest tube drainage, and number of postoperative recurrences.[110] Horio and colleagues showed that the recurrence rate after VATS was double that seen after limited axillary thoracotomy.[111] In 1997, Dumont and associates compared the results of axillary thoracotomy with thoracoscopy and found no major differences between the two groups with regard to chest tube duration or hospital stay.[112] The overall morbidity rate was 16% in the axillary thoracotomy group and 11% in the thoracoscopy group. In a prospective randomized study comparing VATS with axillary thoracotomy, Freixenet and coauthors found no significant difference for postoperative blood loss, respiratory function (maximum inspiratory and expiratory pressures, forced expiratory volume in the first second, and forced vital capacity), postoperative pain (analogue visual scale), supplementary doses of analgesics, postoperative complications, hospital stay, or resumption of normal activity.[113]

In a prospective, controlled randomized study, 60 patients with spontaneous pneumothorax were treated by VATS or posterolateral thoracotomy.[114] Patients undergoing VATS had significantly longer operative time, but patients who had a thoracotomy had more severe alterations of their postoperative pulmonary functions. No differences were found in duration of chest tube drainage, treatment failure, recurrence, or operative mortality. VATS appears to be superior to thoracotomy because of lower postoperative analgesic requirements and decreased duration of hospitalization.[114]

In 1997, Jiménez-Merchan and coworkers reported the results of a retrospective analysis comparing 110 patients with pneumothorax treated by VATS with 627 patients treated by thoracotomy. The VATS group has decreased postoperative pain, faster recovery, and shorter length of hospital stay.[115]

In a recent systematic review of randomized clinical trials comparing VATS with conventional strategy, it was found that use of pain medication and length of hospital stay were reduced, but the recurrence rate was slightly higher, averaging 5%.[116]

Most of the series reported in the literature represented the early period of experimentation with VATS. We are convinced that the future will show the superiority of the VATS approach. The operation is easy for a single operator, fast, and cosmetically acceptable with a minimum of residual pain, and the results are as good as with the other approaches. The learning curve is probably completed for most thoracic surgeons in practice today, thus the posterolateral thoracotomy must be abandoned as a routine approach in favor of VATS.

Management of Recurrence After VATS

There is very little experience with the surgical management of recurrence after VATS. Many surgeons would consider that VATS is not a good option for reoperation because of the presence of postoperative pleural adhesions. However, Cardillo and colleagues, in a small series of 19 patients undergoing repeat VATS who were monitored for 32 months, reported no recurrences. The conversion rate was 5.2%.[117] VATS was also used with success in patients who had talc pleurodesis for the treatment of recurrent spontaneous pneumothorax.[118] VATS is an acceptable approach for recurrence after a previous VATS. However, one should not hesitate to convert to full posterolateral thoracotomy if VATS becomes difficult.

Other Treatment Options

Medical Thoracoscopy and VATS for Pleurodesis Alone

In Europe, medical thoracoscopy has long been used for the diagnosis and treatment of spontaneous pneumothoraces.[93,119] According to its proponents, the technique allows for proper staging of the disease followed by treatment. According to Verschoof and associates, thoracoscopy needs to be performed routinely at the time of the first episode of a spontaneous pneumothorax to classify the patients in one of three categories:

1. No obvious abnormalities
2. Small apical blebs
3. More generalized bullous disease

Treatment is then selected according to the findings.[120] A chemical pleurodesis is used in the first two groups, and surgery is offered to patients with generalized bullae.[120]

Talc poudrage is an acceptable technique for patients who present with a secondary spontaneous pneumothorax, but it is controversial in the management of primary spontaneous pneumothorax.

Chemical Pleurodesis

Rarely, surgeons use chemical pleurodesis as first-line treatment for recurrences of pneumothorax. Many agents, such as quinacrine, autologous blood, and bleomycin as well as tetracycline and talc, have been used for this purpose.[127-130]

Talc is a powder of hydrous magnesium silicate containing various contaminants that has been shown to be effective in treating malignant pleural effusions and spontaneous pneumothoraces. Commercially available purified talc is free of asbestos and is considered safe for therapeutic use. It is effective in inducing pleural fibrosis and adhesions, but the morbidity associated with its use includes fever and pain. The most compelling concern about using talc is the risk of inducing respiratory failure in a young patient. Because of this possible complication, the maximum dose recommended for intrapleural use is 5 g. Talc may be injected through a chest tube in suspension form (2-5 g diluted in 50-250 mL of normal saline) or sprinkled with a syringe over the entire pleural surface during the VATS procedure. It may also be insufflated in its powder form through a thoracoscope or with a talc insufflator.

A recent series involving a large number of patients treated by videothoracoscopic talc poudrage showed a high success rate and a low morbidity.[119]

Some surgeons are concerned by the use of chemopleurodesis in patients who may eventually require a thoracotomy because it may be associated with higher morbidity rates. At present, chemical pleurodesis is used only in selected cases of surgery.

In our opinion, chemical pleurodesis in primary spontaneous pneumothorax is not recommended except in exceptional situations.

Aerosol of fibrin glue nebulized over the lung surface under thoracoscopic control has been used with a success rate of 80%.[121] We think surgery can offer better results.

Electrocautery, using laser or the argon beam electrocoagulator for ablation of the blebs with the VATS approach, has become an option in the management of pneumothoraces.[122-126]

Outcome After Treatment

Although most of the studies evaluating the results are retrospective, they show a low incidence of recurrences whether pleurectomy or pleural abrasion was used. In surgical series, including 977 patients from 1957 to 1993, the recurrence rate after a follow-up of between 5 and 10 years was 1.5%.[131] Patients treated with bed rest, needle aspiration, and chest tube drainage had a recurrence rate of 30%.

TABLE 89-5 Common Causes of Secondary Spontaneous Pneumothorax

Airway and Pulmonary Disease
Chronic obstructive pulmonary disease (bullous or diffuse emphysema)
Asthma
Cystic fibrosis
Interstitial Lung Disease
Pulmonary fibrosis
Sarcoidosis
Infectious Disease
Tuberculosis and other mycobacterial infections
Bacterial infections
Pneumocystis jiroveci (P. carinii) infection
Parasitic infections
Mycotic infections
Acquired immunodeficiency syndrome (AIDS)
Neoplastic Disease
Bronchogenic carcinoma
Metastatic disease (lymphoma or sarcoma)
Catamenial (endometriosis)
Miscellaneous
Marfan's syndrome
Ehlers-Danlos syndrome
Histiocytosis X
Scleroderma
Lymphangiomyomatosis
Collagen diseases

SECONDARY SPONTANEOUS PNEUMOTHORAX

Spontaneous pneumothorax can occur secondary to a variety of pulmonary and nonpulmonary disorders (Table 89-5).

Chronic Obstructive Pulmonary Disease

COPD is the most common cause of secondary pneumothorax. It almost always occurs in patients older than 50 years of age and represents a troubling episode in the evolution of a patient with COPD. A spontaneous pneumothorax is a marker of the severity of the disease and a predictor of survival. Each pneumothorax occurring in these patients increases the chance of dying by almost fourfold.[132]

The explanation for the development of blebs and bullae was suggested by several studies to be a combination of inflammation provoked by smoking and resulting in an endobronchial obstruction. It is believed that endobronchial obstruction by inflammatory cells and peribronchial fibrosis are among the causes for the generation of bullae by increasing the pressure in the alveolar tissue. The result is rupture of pulmonary parenchyma and the development of bullae.

Clinical diagnosis of a pneumothorax can be difficult in these patients, who already have dyspnea. They often show increased dyspnea and acute respiratory distress with hypoxia, hypercarbia, and acidosis. Because of their limited pulmonary function, patients with COPD often show little tolerance to even a small pneumothorax. Diagnosis can be made by chest radiography, but again it can be difficult to interpret the radiographs because of the increased radiolucency of the

diseased lung. CT scanning is often helpful to confirm and localize the pneumothorax.[133,134]

More than 40% to 50% of patients will develop a second pneumothorax if pleurodesis is not performed at time of the first event.

Because of decreased lung vascularization, patients with COPD and pneumothorax are more likely to have prolonged air leak; they also have a higher incidence of in-hospital infection and of empyema.[135] A persistent bronchopleural fistula may necessitate early aggressive treatment.

The choice of therapy is based on the severity and duration of symptoms, severity of the underlying parenchymal disease, number of previous episodes, medical comorbidities of the patient, and experience of the surgical team. Patients with secondary spontaneous pneumothorax have, as a first line of therapy, a chest tube with underwater seal drainage. Negative pressure on the chest tube must always be used with caution.

Major operative risks tend to justify this prolonged conservative treatment before a surgical decision is made. However, surgery needs to be considered early, before the patient may contract a nosocomial infection or an empyema as a result of prolonged chest tube drainage. Chemical pleurodesis is not always possible or effective in these patients, in whom it is impossible to clamp the chest tube if the air leak is important.[136,137] It may even be dangerous because it could convert it into a tension pneumothorax. Medical thoracoscopy and talc poudrage are options that have been proposed, but we believe that, whenever possible, VATS performed under general anesthesia with a double-lumen intubation is preferable.[138,139] For many surgeons, a thoracotomy remains the safest approach because it allows a better vision in patients with COPD and large emphysematous bullae. We limit the resection with staplers to the site of the leak unless we plan to proceed with a lung volume reduction at the same time.

We normally do not resect all the bullae because we fear it may result in an unfilled pleural space and too many leaks from suture lines. We always add an extensive parietal pleurectomy and a chemical pleurodesis with talc poudrage.

The operative mortality rate may reach 10%, and the morbidity can be significant. In 1994, Waller and associates reported an operative morbidity rate of 23%, a mortality rate of 9%, and a mean postoperative stay of 9 days in a series of 22 patients who had undergone VATS for secondary pneumothorax.[139]

Other surgical procedures, such as electrocautery, carbon dioxide laser, or neodymium:yttrium-aluminum-garnet (Nd:YAG) laser, have been used to deal with the bullae, but we think resection with the endostaplers is the safest and most effective approach.

In individuals with poor overall physical condition in whom anesthesia may be too risky, alternative options such as tube chemical pleurodesis or permanent drainage with a Heimlich valve is considered for a prolonged air leak.

More than 40% to 50% of patients with a pneumothorax in the context of COPD eventually develop a second pneumothorax. It is then time to perform a chemical pleurodesis before the chest tube is removed.

Cystic Fibrosis

Pneumothorax occurs in about 10% of patients with cystic fibrosis. It may lead to a life-threatening situation in patients with poor lung function. Although conservative therapy is associated with a high rate of recurrence, the possibility of subsequent lung transplantation must be considered before a decision is made about surgery and pleurodesis.[140] The best surgical option is probably a thoracoscopic approach with lung resection and limited pleurodesis.[141] Localized apical thoracoscopic talc poudrage is also an option.[142]

Infection

Pneumothorax can be secondary to pulmonary infection (bacterial, viral, mycotic, or parasitic), pleural infection (empyema), or intra-abdominal infection (subphrenic abscess). Cavitary pulmonary infections are particularly prone to rupture with secondary pneumothoraces. Pulmonary tuberculosis, for instance, is known to be associated with pneumothoraces that often require prolonged tube drainage. Surgery is not undertaken until these patients have received adequate systemic antituberculosis therapy.

Acquired Immunodeficiency Syndrome

Since the early 1980s, several reports have described the association of spontaneous pneumothorax, pneumomediastinum, and AIDS.[143] DeLorenzo and associates reported a 6% incidence of pneumothorax in the AIDS population, with pneumothorax occasionally being the initial manifestation of the disease.[144,145] In AIDS patients, the pneumothorax may remain small and asymptomatic, but it may also increase rapidly and become under tension, causing severe respiratory failure. In the AIDS population, there is an increased incidence of synchronous bilateral pneumothoraces and bronchopleural fistulas, as well as higher rates of ipsilateral and contralateral recurrences.

The high incidence of pneumothorax in AIDS patients is probably the result of cystic lesions that are most common at the apices and consist of subpleural air spaces filled with eosinophilic exudates, *Pneumocystis jiroveci (P. carinii)* organisms, fibrous material, and macrophages. Histologic studies suggested that such lesions are the result of infection associated with tissue destruction and fibrosis.

Whenever possible, the initial management needs to be conservative; sometimes, small pneumothoraces resolve with observation alone. However, most patients have large and persistent air leaks that eventually require tube thoracostomy.[146] On occasion, these patients may be treated on an outpatient basis with a one-way flutter valve.[147]

To prevent recurrences, chemical pleurodesis has been used, but not always with good results.[148] Surgical resection of the diseased area with pleurectomy remains the most effective mode of therapy. Although significant operative mortality rates have been reported, AIDS patients can tolerate surgery reasonably well, and most do not require mechanical ventilation during the postoperative period.[149] Treatment with VATS and talc poudrage may also be used to control the pneumothorax and associated air leak.[150] The introduction of

new medical therapies for AIDS has led to a decrease in the incidence of related pneumothoraces.

Severe Acute Respiratory Syndrome

Spontaneous pneumothorax occurs as a complication in 1.7% of patients with SARS.[7] Clinically, all patients are usually very dyspneic with high oxygen requirements and show extensive radiologic manifestations of the disease. Such an extensive pulmonary injury is a predisposing factor for the development of pneumothorax in this category of patient. Regarding the management of pneumothorax, conservative measures appear to offer adequate treatment in most symptomatic patients. Chest tube drainage is used first, before one recommends video-assisted thoracoscopy for prolonged or persistent air leak. One must be careful not to rush into surgery with these very fragile patients.[151]

Neoplasia

Occasionally, bronchial obstruction by a lung cancer leads to a pneumothorax. A pneumothorax may also develop as the result of pleural space rupture of an ischemic primary tumor or metastasis. Pneumothoraces may also occur during chemotherapy or radiotherapy.[152]

Pneumothoraces are more commonly associated with metastatic sarcomas but have also been described with teratomas, Wilms' tumors, melanomas, carcinomas of the kidney and pancreas, gynecologic malignancies, lymphomas, choriocarcinomas, and lymphangiomatosis.[153-158]

In these patients, chest tube drainage is the therapy of choice; surgery is seldom indicated. Chemical pleurodesis may be used to prevent recurrences.[159]

Catamenial Pneumothorax

Pneumothoraces occurring within 48 to 72 hours after the onset of menstruation were first described by Maurer and Schall.[160] According to Nakamura, catamenial pneumothorax may afflict 3% to 6% of women between 20 and 30 years of age.[30] Most catamenial pneumothoraces are on the right side, and they may be recurrent over several years before being diagnosed. They are usually small, and patients present with chest pain and dyspnea.[160]

The pathogenesis of catamenial pneumothorax is still unclear.[161] Air may reach the pleural space from the cervix and abdomen through congenital diaphragmatic defects, or there may be focal thoracic endometrial implants on the visceral pleura or in the lung, with air leakage occurring during menstruation. Endometrial implants may also obstruct bronchioles, causing distal hyperinflation and alveolar rupture.[162] Increased levels of prostaglandin F_2 tromethamine at the time of menses may also cause bronchial and vascular constriction, leading to alveolar rupture and subsequent pneumothorax. It is likely that several of these mechanisms are involved simultaneously in the development of catamenial pneumothoraces.

Management is similar to that for other types of pneumothoraces. Small and asymptomatic episodes may be treated conservatively, whereas large and symptomatic episodes require chest tube drainage. The management of recurrence is more controversial, and several options are possible:

1. Treatment of each episode with tube thoracostomy
2. Use of oral contraceptives or weak androgens to suppress ovulation
3. Chemical pleurodesis
4. Hysterectomy and bilateral oophorectomy
5. Thoracotomy with pleural abrasion or pleurectomy[163]

In 1990, Fleisher proposed a treatment algorithm taking into consideration whether hormonal therapy is contraindicated and whether or not pregnancy is desired. Resection of blebs and pleurectomy or pleural abrasion are indicated if pregnancy is desired or if laparoscopic tubal ligation is contraindicated.[164]

The postoperative outcome may be influenced by the presence of diaphragmatic defects with or without endometriosis. Surgery is performed during menstruation for optimal visualization of pleurodiaphragmatic endometriosis. Coverage of the diaphragmatic surface with polyglactin mesh to prevent recurrence of catamenial pneumothorax, even when the diaphragm appears normal, is an option.[165] The use of talc poudrage is debatable in these young patients.

Miscellaneous

Other diseases that have sometimes been associated with pneumothorax include Marfan's syndrome, Ehlers-Danlos syndrome, histiocytosis X, pulmonary infarction, interstitial fibrosis, eosinophilic granuloma, sarcoidosis, and tuberous sclerosis.[166]

PRIMARY SPONTANEOUS PNEUMOMEDIASTINUM

Spontaneous pneumomediastinum (SPM) is defined as the nontraumatic presence of free air in the mediastinum in a patient with no known underlying disease. SPM is a rare condition with an incidence of 1 per 7000 to 12,000 hospital admissions. It has long been recognized as a self-limited, benign condition in young men. According to Munsell, pneumomediastinum was first recognized in the 17th century by a midwife to the Queen of France.[167] The first reported case in the modern era was by Louis Hamman in 1939.[168]

This entity develops as a result of the increased pressure in the intrabronchial and intra-alveolar space associated with a Valsalva maneuver, coughing, straining, or vomiting or during artificial ventilation. It is also linked to other causes, such as bronchial asthma, diabetic ketoacidosis, inhalation of drugs, and childbirth. According to Macklin and Macklin, SPM results from the rupture of terminal alveoli into the lung interstitium.[169] The air dissects along the pulmonary vasculature toward the hilum, centrally along the bronchoalveolar trunks, through the peribronchial space, or within the lymphatics, toward the mediastinum. The air can also dissect along the perivascular connective tissue into the pulmonary parenchyma, resulting in interstitial emphysema.

The clinical presentation of SPM can be subtle, and the diagnosis can be missed or delayed. A high index of suspicion is necessary for prompt diagnosis. The most frequent signs

and symptoms are pain in the chest and neck, exertional dyspnea, subcutaneous emphysema (40%-100% of the cases), hypotension, dysphagia, and cough. Hamman described the mediastinal crunch sign, consisting of crunch-like sound over the left hemithorax anteriorly. It is reported in about 50% of patients.[170] The initial differential diagnosis of SPM is broad and includes cardiac, pulmonary, musculoskeletal, and esophageal causes. Most of these can be excluded with a thorough history, detailed physical examination, the use of electrocardiographic examination, and radiographic or endoscopic assessment as indicated. CT scanning of the thorax may be required for more precision.

The diagnosis is confirmed by chest radiography, which shows streaky gas densities along the fascial planes of the mediastinum. The lateral display helps to obtain 100% accuracy. The most important differential diagnosis to be excluded is an esophageal perforation. That is why an esophagogram is often performed during the initial investigation of these patients. However, these patients are often overinvestigated.[171] After specific causes of mediastinal emphysema have been excluded, primary SPM can be treated expectantly.

Tension pneumomediastinum arises when air in the lung and mediastinum causes compression of pulmonary and mediastinal vessels and interferes with respiration by the splinting action of air in the interstitial tissues of the lung. Its clinical characteristics are dyspnea, cyanosis, prominent veins in the neck, tachycardia, and hypotension. It may mimic cardiac tamponade. Fatalities have been reported.

The treatment of malignant pneumomediastinum aims to reduce the high pressure in the alveoli by evacuating the air in the mediastinum with the use of multiple subcutaneous aspirations or incisions. Cervical mediastinotomy rarely may be necessary.

In conclusion, SPM is an uncommon and self-limited clinical entity. It is usually a benign condition that can be treated expectantly.[172] Recurrence is rare. Hospitalization and an aggressive approach are limited and individualized.

COMMENTS AND CONTROVERSIES

Primary spontaneous pneumothorax occurs in young, apparently healthy individuals. In such patients, radiographic signs of lung disease are absent, but the apical blebs that are the cause of the pneumothorax can usually be identified on CT scanning. Why these young and usually thin and tall people have apical blebs is a matter of controversy, but one of the most prevalent theories is that the lung becomes stretched during its growth, leading to areas of relatively diminished vascular supply (arterial and pulmonary) at the apices (i.e., farthest from the hilum). This, in turn, leads to bleb formation.

It is generally agreed that the first episode, if uncomplicated, can be managed by observation alone if the pneumothorax is small (≤20%) or by intercostal tube drainage if the pneumothorax is larger (>20%). If the first episode is complicated by tension, persistent air leakage (3%-5% of patients), or hemothorax (5% of patients) or if the pneumothorax is recurring (10%-12% incidence after a first episode), further measures are necessary. These involve resection of the offending blebs (blebectomy) and obliteration of the pleural space. It is worth noting that, in spontaneous pneumohemothoraces, the bleeding, which can be quite severe, usually results from a torn adhesion between the parietal and visceral pleura. Because the vessels contained in such adhesions have thin walls, they do not retract after disruption and therefore bleed actively in the empty, negative-pressure pleural space.

Most controversies concerning the surgical treatment of primary spontaneous pneumothoraces revolve around which surgical approach is best and which method should be recommended to achieve pleurodesis. Both of these issues are well discussed in this chapter. At present, the relative merits of the thoracoscopic approach (VATS) versus those of the limited axillary incision are still debated, although in most series the incidence of further recurrences is higher after VATS than what has been reported after transaxillary operations. Pleurodesis, which is the most important part of the operation, can be accomplished by either parietal pleurectomy or mechanical abrasion, the latter technique having the advantage of preserving the extrapleural plane should further surgery be required. Both procedures result in effective pleurodesis, although many surgeons believe that pleurectomy stimulates the formation of denser and more complete and permanent adhesions. If a pneumothorax still recurs after blebectomy and pleurodesis, it is more often caused by the inadequacy of the pleurodesis rather than the formation of new blebs.

The problems associated with pneumothoraces secondary to COPD are different because almost all patients are older and have associated comorbidities. Because of their limited pulmonary reserve, these patients may also show little tolerance to even a small pneumothorax, and they can present with acute respiratory distress, hypoxia, hypercapnia, and respiratory acidosis. Chances of recurrence are much higher in this group (30%-50%) than in patients with primary spontaneous pneumothorax (10%-12%). Because of the significant incidence of prolonged air leaks, the high risk of recurrence, and the potential fatality of this condition, surgical intervention should be considered at the time of first occurrence. The emergence of VATS techniques has considerably changed the magnitude of the operation, which can now be done with low operative morbidity and mortality even in high-risk cases. If the operative risks are considered prohibitive, management should consist of prolonged tube drainage with or without administration of sclerosing agents through the tube.

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