

Pneumothorax

Won-Il Choi, M.D., Ph.D.

Department of Internal Medicine, Keimyung University School of Medicine, Daegu, Korea

Pneumothorax—either spontaneous or iatrogenic—is commonly encountered in pulmonary medicine. While secondary pneumothorax is caused by an underlying pulmonary disease, the spontaneous type occurs in healthy individuals without obvious cause. The British Thoracic Society (BTS, 2010) and the American College of Chest Physicians (ACCP, 2001) published the guidelines for pneumothorax management. This review compares the diagnostic and management recommendations between the two societies. Patients diagnosed with primary spontaneous pneumothorax (PSP) may be observed without intervention if the pneumothorax is small and there are no symptoms. Oxygen therapy is only discussed in the BTS guidelines. If intervention is needed, BTS recommends a simple aspiration in all spontaneous and some secondary pneumothorax cases, whereas ACCP suggests a chest tube insertion rather than a simple aspiration. BTS and ACCP both recommend surgery for patients with a recurrent pneumothorax and persistent air leak. For patients who decline surgery or are poor surgical candidates, pleurodesis is an alternative recommended by both BTS and ACCP guidelines. Treatment strategies of iatrogenic pneumothorax are very similar to PSP. However, recurrence is not a consideration in iatrogenic pneumothorax.

Keywords: Pneumothorax; Pneumothorax, Primary Spontaneous; Pulmonary Bullae Causing Pneumothorax

Introduction

A pneumothorax is characterized by dyspnea and chest pain originating from the lung and chest wall and may interfere with normal respiration owing to the presence of gas bubbles in the pleural cavity or gas retention in the pleural space that occur following bullae ruptures. Pneumothorax is categorized as either spontaneous—pulmonary collapse without any cause—or induced by trauma.

Spontaneous pneumothorax is further classified into pri-

mary pneumothorax and secondary pneumothorax. Primary pneumothorax develops following bullae ruptures in healthy people with no underlying pulmonary disease. Secondary pneumothorax is caused by rupture of damaged pulmonary tissue, and occurs primarily in patients diagnosed with pulmonary disease, such as pulmonary emphysema.

Pathophysiology of Pneumothorax

In a healthy person, the pleural pressure remains negative relative to atmospheric pressure throughout the entire respiratory cycle. This pressure difference between pulmonary alveoli and the pleural cavity is called the transpulmonary pressure, and this pressure causes elastic recoil of lung. In pneumothorax, the pulmonary alveoli or airway becomes connected to pleural cavity, and air migrates from the alveoli to the pleural cavity until the pressures of both areas are in equilibrium. Similarly, when the chest wall and the pleural cavity are connected, air moves into the pleural cavity from the environment until the pressure difference is no longer present or until the connection is closed. When the air present within the pleural cavity is sufficient to increase the pleural pressure from -5 cm H_2O to -2.5 cm H_2O , the transpulmonary pressure reduces from 5 cm H_2O , to 2.5 cm H_2O , and the pulmonary

Address for correspondence: Won-Il Choi, M.D., Ph.D.

Department of Internal Medicine, Keimyung University School of Medicine, 56 Dalseong-ro, Jung-gu, Daegu 700-712, Korea

Phone: 82-53-250-7572, **Fax:** 82-53-250-7434

E-mail: wichoic@dsmc.or.kr

Received: Jan. 16, 2014

Revised: Jan. 23, 2014

Accepted: Feb. 3, 2014

©It is identical to the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>).

Copyright © 2014

The Korean Academy of Tuberculosis and Respiratory Diseases.

All rights reserved.

vital capacity decreases by 33%. Space for the influx of pleural cavity air is created by compressing the lung, which decreases the vital capacity by 25%. In addition, the intra-pleural cavity pressure change increases the thoracic volume, resulting in an alteration of the thoracic wall recoil and an approximately 8% decline in vital capacity. When the pressure of pleural cavity increases, the mediastinum moves in the opposite direction, expanding the thorax of the same side, and depressing the diaphragm. These changes are observed in tension pneumothorax as well as other types of pneumothorax.

The main physiological change in pneumothorax is a reduction of arterial oxygen tension in addition to the reduced vital capacity. Patients experiencing primary pneumothorax endure the vital capacity reduction relatively well, but in patients suffering from secondary pneumothorax and underlying pulmonary disease, the vital capacity reduction can lead to alveolar hypoventilation and respiratory failure. In a study evaluating 12 patients diagnosed with spontaneous pneumothorax, 9 patients (75%) had a $PO_2 \leq 80$ mm Hg, and 2 patients, who were both diagnosed with secondary pneumothorax, had a $PO_2 \leq 55$ mm Hg¹.

Reduced oxygen tension may be caused by an anatomic shunt and, in some cases, alveolar hypoventilation in the pneumothorax area created from the reduced ventilation-perfusion ratio in the pulmonary alveoli. In the aforementioned 12 patient study, right-to-left vascular shunting occurred at a ratio higher than the 10% mean found in normal patients¹. When the pneumothorax encompasses <25% of a lateral thorax, vascular shunts do not increase. However, when the pneumothorax increased beyond this size, the shunts also increased in size.

Generally, once the air retained in the pleural cavity is removed, oxygen saturation increases within a few hours. For example, in a patient suffering from pneumothorax and a shunt $\geq 20\%$, the shunt decreased to $\leq 10\%$ within 90 minutes of evacuating the intra-pleural air, though about 5% of the shunt still remained¹. The slow recovery rate of the shunt was likely associated with the duration of the pneumothorax.

Causes and Risk Factors

Primary spontaneous pneumothorax (PSP) commonly occurs in tall, thin males aged 10–30 years; a substantial proportion of affected patients have a history of smoking. Smoking is closely related to the PSP. For example, 91% of patients diagnosed with PSP are current or past smokers, and as smoking increases, the risk of pneumothorax also increases². Smoking-associated bronchiolitis likely accounts for the occurrence of pneumothorax in smokers.

Most PSP cases result from spontaneous rupture of a subpleural bleb or bulla, which leaks air into the pleural cavity. A pulmonary bleb or bulla is a small air sac formed between the

lung tissues and pleura, originating from a pulmonary alveoli enlargement (diameter 1–2 cm) and usually developing at an apical area³.

There are two purposed mechanisms for bleb or bulla formation. One mechanism is congenital; the upper pulmonary lobe grows more quickly than the vasculature, causing a lack of blood supply and development of a bullae. The second mechanism is related to the pleural cavity pressure, which becomes more negative at the apical region of the lungs. In a tall individual, the negative pleural cavity pressure is increased at the upper pulmonary lobe, and the alveolar pressure similarly increases. This increase can cause the formation of numerous bullae and pneumothoraxes.

Rarely, PSP occurs in persons with specific inherited genes. Responsible genes have an autosomal dominant inheritance with a variety of penetrations. Genetic risk factors include the HLA haplotype A2B40, alpha-1 antitrypsin phenotypes M1M2, and FBN1 genetic mutation.

Even in patients that have underlying pulmonary diseases such as asthma or pneumonia, pulmonary abscess, and pertussis, PSP may occur; it can even occur in patients diagnosed with Marfan syndrome or lung cancer.

Secondary spontaneous pneumothorax (SSP), unlike PSP, develops in patients diagnosed with a pulmonary disorder. The most common associated etiology is chronic obstructive pulmonary disease (COPD), also known as chronic bronchitis or pulmonary emphysema. Multiple bullae can develop in affected patients accompanied by pneumothorax.

1. Clinical manifestations of spontaneous pneumothorax

Ninety-five percent of affected patients complain of acute and sudden thoracic pain (chest pain) accompanied by shortness of breath. This pain may be more severe at inhalation and localized to the site that the pneumothorax developed. The severity of symptoms such as dyspnea is proportional to the size of pneumothorax, but 5% of patients may be asymptomatic; such patients usually have an overall poor systemic condition. Spontaneous pneumothorax usually occurs at rest, and only 10% of cases occur during exercise⁴. In patients suffering PSP, the pain and dyspnea usually resolve within 24 hours, but patients experiencing SSP usually have more severe symptoms. When the pleural cavity gas caused by the PSP occupies less than 25% of the entire pleural cavity, hypoxemia does not develop easily. However, in patients diagnosed with pulmonary emphysema, even a small amount of intra-pleural cavity gas can cause serious hypoxemia and hypercapnia.

2. Evaluation of spontaneous pneumothorax

The guidelines published by the American College of Chest Physicians (ACCP) define a small pneumothorax as a decrease in apical length of less than 3 cm⁵. However, the British

Thoracic Society (BTS) designates a marginal depth measured from the chest wall to the outer pulmonary edge of less than 2 cm as a small pneumothorax, while a marginal depth of ≥ 2 cm is considered a large pneumothorax^{6,7}. The ACCP guidelines only consider a pneumothorax at the pulmonary apex for evaluation, and therefore the method can be inadequate in assessing the pneumothorax volume. On the contrary, the BTS guidelines are a bit more objective in assessing the pneumothorax volume. Under ACCP guidelines, a patient is considered clinically stable when able to maintain a respiratory rate of less than 24 breaths/min, a pulse rate between 60–120 beats/min, a normal blood pressure, $\text{SPO}_2 \geq 90\%$ on room air, and is able to speak an entire sentence⁵. BTS guidelines consider a patient clinically stable state when there is no respiratory disturbance^{6,7}.

Thoracic computed tomography (CT) is effective at identifying the cause of spontaneous pneumothorax, but the ACCP does not recommend CT as the basic imaging modality, whereas BTS does emphasize the technique's importance in measuring the pneumothorax volume and differentiating diagnoses^{8,9}.

3. Treatment of spontaneous pneumothorax

There are three different treatment strategies in addition to supplying the patient with oxygen, observation, aspiration, or chest tube placement. Although both ACCP and BTS guidelines discuss detailed treatment guidelines for pneumothorax, they differ in determination of pneumothorax severity and the importance of aspiration.

1) Oxygen therapy: Gas within the pleural cavity is absorbed by diffusion and can be facilitated by changing the composition of the intra-pleural cavity gas. Oxygen is absorbed 62 times faster than nitrogen, and carbon dioxide (CO_2) is absorbed 23 times faster than oxygen. When the patient inhales 100% oxygen, nitrogen will disappear from the pleural cavity, leaving only oxygen, which is absorbed faster from the pleural cavity into veins.

The absorption rate of intra-pleural cavity gas is approximately 1.25% per day in ambient air¹⁰; a pneumothorax occupying 25% of the cavity will require 20 days to be completely absorbed. By contrast, when the patient receives oxygen supplementation, the absorption rate accelerates 3–4 times; this effect is particularly prominent when a large volume of gas occupies the pleural cavity^{11,12}. BTS guidelines recommend the use of high-flow oxygen (10 L/min) in symptomatic patients. However, caution must be exercised to avoid hypercarbia in patients with COPD⁷.

2) Observation: Observation can be performed in both PSP and SSP cases. Because PSP has a low mortality rate, stable patients can be carefully observed while the gas is absorbed passively from the pleural cavity. Both the ACCP and

BTS recommend observation only of clinically stable patients diagnosed with a small volume pneumothorax. According to ACCP guidelines, clinically stable patients should be observed for 3–6 hours and can be discharged home if a repeat chest radiograph excludes progression of the pneumothorax, which indicates that the causal lesion has closed. However, for patients residing far from a hospital or health care center, admission for observation is best. Even if the patient is not admitted, the patient should be provided with careful instructions for follow-up examination within 2 days, depending on the circumstances. For patients diagnosed with a SSP, both the ACCP and BTS recommend hospitalizing patients for treatment, even if the pneumothorax is small.

3) Simple aspiration: Aspiration of a pneumothorax is performed using a small catheter. The catheter is inserted into the pleural cavity and either removed immediately after evacuating the air from the pleural cavity or left inserted while the patient is observed. When left inserted in the thoracic wall, the catheter is still considered a chest tube despite its small size. In patients diagnosed with spontaneous pneumothorax, the mean success rate of aspiration is between 53% and 58%. Specifically, the mean success rate for PSP is 75%, which is comparatively higher than the SSP mean success rate of approximately 37%¹³.

(1) Primary spontaneous pneumothorax: ACCP recommends the use of simple aspiration in clinically stable patients diagnosed with PSP whose conditions worsen under observation. However, BTS recommends the use of simple aspiration in all patients diagnosed with PSP who need intervention. The BTS guidelines were established based on a study conducted by Noppen et al.¹⁴. The study compared clinical outcomes in patients diagnosed with PSP who received aspiration with a 16G IV catheter and chest tube insertion (16F or 20F); there was no difference in therapeutic effectiveness between the two techniques, but the hospitalization duration was reduced in patients receiving simple aspiration. It is worth noting that the sample size was very small and the treatment protocols of the two groups were quite different, and therefore, it is difficult to conclude that there is no difference between two treatment methods. The BTS guidelines do consider these limitations and recommends performing simple aspiration only at facilities possessing practical experience in the procedure and relevant equipment⁶. Further study is required on the therapeutic effectiveness of the simple aspiration and small diameter chest tube placement.

(2) Secondary spontaneous pneumothorax: While the ACCP recommends simple aspiration in patients diagnosed with SSP in a very limited manner, BTS recommends the procedure for treating small pneumothorax with mild respiratory symptoms in patients less than 50 years old. The simple aspiration is quite low in the SSP¹³, in particular, the failure rate is high in patients ≥ 50 years old⁶. After performing simple aspiration, the patient should be hospitalized for observation during

recovery.

4) Chest tube placement

(1) Primary spontaneous pneumothorax: ACCP recommends the placement of a chest tube in a case of large pneumothorax, regardless of whether the patient is clinically stable or unstable, and that in most instances, patients with a large pneumothorax should be hospitalized. In comparison, BTS recommends placement of a chest tube when the simple aspiration procedure fails to resolve the pneumothorax.

(2) Secondary spontaneous pneumothorax: ACCP recommends either observation or chest tube placement in clinically stable patients diagnosed with a small SSP. In a clinically stable patient diagnosed with a large pneumothorax or a clinically unstable patient, ACCP recommends chest tube placement. BTS also recommends chest tube placement, except in patients diagnosed with a very small pneumothorax (1–2 cm) and no respiratory symptoms.

5) Chest tube management

(1) Thickness of chest tube: Both ACCP and BTS recommend physicians avoid placing a thick chest tube in both primary and SSP. The ACCP does recommend inserting a thick chest tube (24 to 28F) in a patient experiencing a large-scale air leak, such as a bronchopleural fistula, or receiving mechanical ventilation. In the stable patient diagnosed with PSP, ACCP recommends a chest tube thickness of 14 to 22F or less. BTS always recommends using a 14F chest tube, as there is no evidence that a thick chest tube (20–24F) is more clinically effective than a thin chest tube (10–14F)⁷.

(2) Suction of the chest tube: The efficacy of suction after the placement of chest tube is not well verified; one study reported that suction was not an effective treatment in patients diagnosed with the primary or the SSP¹⁵. In another study investigating pneumothorax treatment in 71 patients, the lungs re-expanded and the air leaks dissipated without the clinicians performing any suction through the chest tube in 77% of the patients treated¹⁶. Similarly, in another report, chest tube suction did not affect the severity of lung collapse¹⁷. ACCP recommends suction only when the lung fails to re-expand following chest tube placement and observation. BTS does not recommend suction because of the risk pulmonary edema induced by re-expansion. Alternatively, Suction may be applied if the air leak lasts ≥ 48 hours or there is no pulmonary re-expansion after chest tube placement. The BTS also recommends performing suction at a higher velocity and lower pressure (–10 to –20 cm H₂O)⁷.

(3) Chest tube removal: Chest tubes should be removed only when a chest radiograph demonstrates re-expansion of lung, complete resolution of the pneumothorax, and no clinical evidence of an ongoing air leak¹⁸. ACCP and BTS provide similar guidelines for the timing of chest tube removal. ACCP recommends that any suction in progress be suspended prior to chest tube removal. However, the two organizations have different recommendations on clamping the chest tube

closed before its removal. ACCP has a 47% consensus for using clamping in PSP and a 59% of consensus in SSP. Those who support clamping express concern for a potential small air leak and feel that clamping may be useful in locating a leak. BTS does not recommend clamping when there is no air leak visible. However, it recommends additional precautions in observing the patient if clamping is performed⁶.

(6) Definition of persistent air leaks: Intervention to eliminate the leak is generally recommended if the air leak persists for 2 days up to 14 days^{19–21}. ACCP recommends intervention for air leaks persisting beyond 4 days in PSP cases and over 5 days in SSP cases. BTS recommends thoracic surgery if the air leaks persist beyond 2 days or if the lung does not re-expand. If air leaks caused by spontaneous pneumothorax are allowed to persist, the cost of treatment increases, and the therapeutic success rate of thoracoscopy decreases²².

7) Interventional procedure for prevention of recurrence and air leaks

(1) Surgery: ACCP and BTC recommend surgical intervention to prevent recurrence or to stop persistent air leaks. Although the advantage of surgical treatment is not clearly identified yet, ACCP recommends either parietal pleurectomy and bullectomy, or parietal pleural abrasion of one pleural upper half and bullectomy. BTS recommends several possible interventions including parietal pleurectomy in addition to parietal pleural abrasion and talc-utilized pleurodesis.

(2) Pleurodesis and Heimlich valve: ACCP recommends performing pleurodesis, using medications such as talc and doxycycline administered through the chest tube, in cases of primary and SSP if the patient declines surgical intervention or is not a suitable surgical candidate. Similarly, BTS recommends pleurodesis for patients that are not suitable surgical candidates. Appropriately sized talc may actually reduce the risk of respiratory failure²³. In patients diagnosed with SSP who cannot undergo surgery, outpatient treatment with a Heimlich valve may be considered.

(3) Timing of intervention for recurrence prevention of spontaneous pneumothorax: Excluding persistent air leaks, 85% of ACCP panel members recommend surgical intervention for the second recurrence of PSP, whereas 81% supported surgical intervention at the first recurrence in cases of SSP. BTS recommends surgical treatment when the pneumothorax at the second occurrence on the same side, the first recurrence on the opposite site, and in cases of bilateral pneumothorax.

Iatrogenic Pneumothorax

Iatrogenic pneumothorax is increasing owing to the increase in invasive diagnostic and treatment methods. The most common causes of iatrogenic pneumothorax are trans-thoracic needle aspiration, subclavian vein catheterization, thoracentesis, transbronchial lung biopsy, pleural biopsy,

and mechanical ventilation. Pneumothorax should always be suspected if the cardiopulmonary function becomes unstable after these procedures. In some cases, the pneumothorax can occur immediately after the procedure, but clinicians should remember that it also could occur several days later. The clinical profile of iatrogenic pneumothorax is affected by any underlying diseases and health conditions of patient, as well as the pneumothorax etiology. A patient in poor general condition may experience severe symptoms even from a small volume pneumothorax. In addition, when performing positive pressure ventilation, the pneumothorax volume can increase more quickly and a tension pneumothorax may result. Trans-thoracic needle aspiration increases the risk of pneumothorax when performed on a small lesion located deep in the lung or when emphysema is present at the biopsy location.

Unlike spontaneous pneumothorax, there is no concern about recurrence of iatrogenic pneumothorax. Non-invasive treatment is recommended, depending on the clinical profile or underlying disease of the patient. Treatment many include observation, as well as simple aspiration or the chest tube placement using a small catheter. If the condition occurs in a patient diagnosed with large volume pneumothorax or COPD, or during mechanical ventilation, chest tube placement is the preferred treatment.

Pneumothorax in Specific Form: Tension Pneumothorax

In tension pneumothorax, air flows into the pleural cavity during inhalation but is retained in the pleural cavity during exhalation and thus cannot exit, leading to a gradual increase in intra-pleural cavity pressure. Tension pneumothorax can develop from either a spontaneous pneumothorax or traumatic pneumothorax. During tension pneumothorax, the affected lung ipsilateral to the pneumothorax completely collapses, and the contralateral lung and heart are pressurized. The result is severe dyspnea, cyanosis, and hypotension, leading to death. Thus, tension pneumothorax should be treated with immediate needle decompression.

References

- Norris RM, Jones JG, Bishop JM. Respiratory gas exchange in patients with spontaneous pneumothorax. *Thorax* 1968;23:427-33.
- Bense L, Eklund G, Wiman LG. Smoking and the increased risk of contracting spontaneous pneumothorax. *Chest* 1987;92:1009-12.
- Schramel FM, Postmus PE, Vanderschueren RG. Current aspects of spontaneous pneumothorax. *Eur Respir J* 1997;10:1372-9.
- Bense L, Wiman LG, Hedenstierna G. Onset of symptoms in spontaneous pneumothorax: correlations to physical activity. *Eur J Respir Dis* 1987;71:181-6.
- Baumann MH, Strange C, Heffner JE, Light R, Kirby TJ, Klein J, et al. Management of spontaneous pneumothorax: an American College of Chest Physicians Delphi consensus statement. *Chest* 2001;119:590-602.
- Henry M, Arnold T, Harvey J; Pleural Diseases Group, Standards of Care Committee, British Thoracic Society. BTS guidelines for the management of spontaneous pneumothorax. *Thorax* 2003;58 Suppl 2:ii39-52.
- MacDuff A, Arnold A, Harvey J; BTS Pleural Disease Guideline Group. Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease Guideline 2010. *Thorax* 2010;65 Suppl 2:ii18-31.
- Engdahl O, Toft T, Boe J. Chest radiograph: a poor method for determining the size of a pneumothorax. *Chest* 1993;103:26-9.
- Phillips GD, Trotman-Dickenson B, Hodson ME, Geddes DM. Role of CT in the management of pneumothorax in patients with complex cystic lung disease. *Chest* 1997;112:275-8.
- Kircher LT Jr, Swartzel RL. Spontaneous pneumothorax and its treatment. *J Am Med Assoc* 1954;155:24-9.
- Chadha TS, Cohn MA. Noninvasive treatment of pneumothorax with oxygen inhalation. *Respiration* 1983;44:147-52.
- Northfield TC. Oxygen therapy for spontaneous pneumothorax. *Br Med J* 1971;4:86-8.
- Baumann MH, Strange C. Treatment of spontaneous pneumothorax: a more aggressive approach? *Chest* 1997;112:789-804.
- Noppen M, Alexander P, Driesen P, Slabbynck H, Verstraeten A. Manual aspiration versus chest tube drainage in first episodes of primary spontaneous pneumothorax: a multicenter, prospective, randomized pilot study. *Am J Respir Crit Care Med* 2002;165:1240-4.
- So SY, Yu DY. Catheter drainage of spontaneous pneumothorax: suction or no suction, early or late removal? *Thorax* 1982;37:46-8.
- Minami H, Saka H, Senda K, Horio Y, Iwahara T, Nomura F, et al. Small caliber catheter drainage for spontaneous pneumothorax. *Am J Med Sci* 1992;304:345-7.
- Sharma TN, Agnihotri SP, Jain NK, Madan A, Deopura G. Intercostal tube thoracostomy in pneumothorax: factors influencing re-expansion of lung. *Indian J Chest Dis Allied Sci* 1988;30:32-5.
- Baumann MH. Pneumothorax. *Semin Respir Crit Care Med* 2001;22:647-56.
- Chee CB, Abisheganaden J, Yeo JK, Lee P, Huan PY, Poh SC, et al. Persistent air-leak in spontaneous pneumothorax: clinical course and outcome. *Respir Med* 1998;92:757-61.
- Mathur R, Cullen J, Kinnear WJ, Johnston ID. Time course of resolution of persistent air leak in spontaneous pneumothorax. *Respir Med* 1995;89:129-32.

21. Jain SK, Al-Kattan KM, Hamdy MG. Spontaneous pneumothorax: determinants of surgical intervention. *J Cardiovasc Surg (Torino)* 1998;39:107-11.
22. Waller DA, McConnell SA, Rajesh PB. Delayed referral reduces the success of video-assisted thoracoscopic surgery for spontaneous pneumothorax. *Respir Med* 1998;92:246-9.
23. Maskell NA, Lee YC, Gleeson FV, Hedley EL, Pengelly G, Davies RJ. Randomized trials describing lung inflammation after pleurodesis with talc of varying particle size. *Am J Respir Crit Care Med* 2004;170:377-82.