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Pneumothorax

Marc Noppen[a] Tom De Keukeleire[b]

[a]Interventional Endoscopy Clinic, Respiratory Division, and Chief Executive Officer, [b]Interventional Endoscopic Clinic, Respiratory Division, University Hospital UZ Brussel, Brussels, Belgium

Key Words
Pneumothorax, pathogenesis · Pneumothorax, iatrogenic · Pneumothorax, spontaneous · Pneumothorax, traumatic

Abstract
Pneumothorax represents a common clinical problem. An overview of relevant and updated information on epidemiology, pathophysiology, and management of spontaneous (primary and secondary), catamenial, and traumatic (iatrogenic and noniatrogenic) pneumothorax is given.

Introduction

Pneumothorax is defined as the presence of air in the pleural space. Although intrapleural pressures are negative throughout most of the respiratory cycle [1], air does not enter into the pleural space because the sum of all the partial pressures of gases in the capillary blood averages only 93.9 kPa (706 mm Hg). Hence, net movement of gases from the capillary blood into the pleural space would require pleural pressures lower than –54 mm Hg (i.e., lower than –36 cm H_2O), which hardly ever occur in normal circumstances [2]. Hence, if air is present in the pleural space, one of three events must have occurred: (1) communication between alveolar spaces and pleura, (2) direct or indirect communication between the atmosphere and the pleural space, or (3) presence of gas-producing organisms in the pleural space. From a clinical standpoint, pneumothorax is classified as spontaneous (no obvious precipitating factor present) and nonspontaneous (table 1) [2, 3]. Primary spontaneous pneumothorax (PSP) is defined as the spontaneously occurring presence of air in the pleural space in patients without clinically apparent underlying lung disease.

Primary Spontaneous Pneumothorax

PSP has an incidence of 7.4–18 cases (age-adjusted incidence)/100,000 population per year in males, and 1.2–6 cases/100,000 population per year in females [4, 5]. PSP typically occurs in tall, thin subjects. Other risk factors are male gender and smoking. PSP typically occurs at rest [6]. Precipitating factors may be atmospheric pressure changes (which may account for the often observed clustering of PSP) [7] and exposure to loud music [8]. Almost all patients with PSP report a sudden ipsilateral chest pain, which usually resolves spontaneously within 24 h [2]. Dyspnea may be present but is usually mild. Physical

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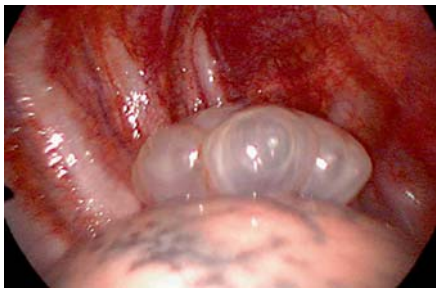
Marc Noppen, MD, PhD
Interventional Endoscopy Clinic, Respiratory Division
University Hospital UZ Brussel, 101, Laarbeeklaan
BE–1090 Brussels (Belgium)
Tel. +32 2 477 55 01, Fax +32 2 477 55 15, E-Mail marc.noppen@uzbrussel.be



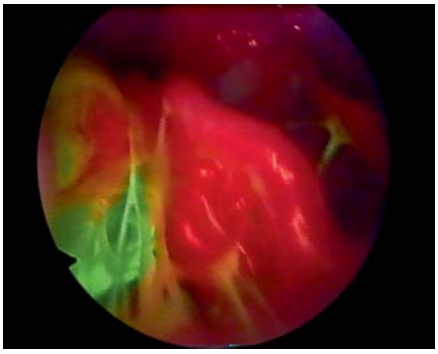


Fig. 1. Large bulla at the apex of the left lung in a 12-year-old boy with recurrent PSP.

Fig. 2. Air leak identified by fluorescein-enhanced autofluorescence thoracoscopy in a 27-year-old man with recurrent PSP. The air leak was situated at the base of a highly vascularized, severe malformation of the apex of the lung.

Table 1. Clinical classification of pneumothorax

Spontaneous
Primary: no apparent underlying lung disease
Secondary: clinically apparent underlying disease (e.g., COPD, cystic fibrosis)
Catamenial: in conjunction with menstruation (Neonatal)
Traumatic
Iatrogenic: secondary to transthoracic and transbronchial biopsy, central venous catheterization, pleural biopsy, thoracentesis
Noniatrogenic: secondary to blunt or penetrating chest injury

examination can be normal in small pneumothoraces. In larger pneumothoraces, breath sounds and tactile fremitus are typically decreased or absent, and percussion is hyperresonant. Rapidly evolving hypotension, tachypnea and tachycardia, and cyanosis should raise the suspicion of tension pneumothorax, which is, however, extremely rare in PSP.

Diagnosis can be confirmed in the majority of cases with an upright posteroanterior chest radiograph, which also makes it possible to estimate the pneumothorax size with good accuracy [9]. In small pneumothoraces, computer tomography may be necessary to diagnose the presence of pleural air. Expiratory chest radiographs are useless [10]. It is important to realize that a contralateral shift of the trachea and mediastinum is a completely normal phenomenon in spontaneous pneumothorax and not at all suggestive of tension pneumothorax; this observation should therefore in no way influence treatment strategies [1]. In a minority of patients, some pleural fluid is present. Rarely, PSP may be associated with a spontaneous hemothorax.

Pathogenesis

The exact pathogenesis of PSP is unknown. The key issue is the spontaneous occurrence of a communication between the alveolar spaces and the pleura. Most authors believe that spontaneous rupture of a subpleural bleb, or of a bulla, is the cause of PSP [10] although alternative explanations are available [11–13]. Although the majority of PSP patients, including children [14], present blebs or bullae (usually at the apices of the lungs) (fig. 1) [15–18], it is unclear how often these lesions are actually the site of air leakage [19–21]. Only a small number of blebs are ruptured at the time of thoracoscopy or surgery, whereas in the remaining cases other lesions are present, often referred to as 'pleural porosity' [19–21]: areas of disrupted mesothelial cells at the visceral pleura, replaced by an inflammatory elastofibrotic layer with increased porosity, allowing air leakage into the pleural space. The latter phenomenon may explain the high recurrence rates of up to 20% of bullectomy alone (without associated pleurodesis) as therapy [22–25]. The development of blebs, bullae and areas of pleural porosity may be linked to a variety of factors, including distal airway inflammation [21–26], hereditary predisposition [27], anatomical abnormalities of the bronchial tree [28], ectomorphic physiognomy with more negative intrapleural pressures [29] and apical ischemia [30] at the apices [31], low body mass index and caloric restriction [15, 32], and abnormal connective tissue [33, 34]. The role of increased plasma aluminium concentrations in the pathogenesis of PSP remains unresolved [35, 36].

These lesions may therefore predispose to PSP when combined with (largely unknown) precipitating factors; blebs and bullae indeed also occur in up to 15% of normal subjects [15–17]. New techniques, such as fluorescein-enhanced autofluorescence thoracoscopy [37] (fig. 2) or infrared thoracoscopy [38], may shed more light on this issue, and may be helpful in the detection of the culprit

areas during thoracoscopy or surgery. It should be clear, however, that every therapeutic intervention with the purpose of preventing recurrences of PSP should include a pleurodesis technique with or without an intervention at the level of the lung parenchyma [39].

Management

A multitude of therapeutic options are available for treatment of PSP, varying from conservative (observation, oxygen treatment, simple manual aspiration, small catheter drainage) over intermediate (chest tube drainage, medical thoracoscopic talc poudrage or pleural abrasion) to invasive [video-assisted thoracoscopic surgery (VATS) with bleb- or bullectomy, pleural abrasion or partial pleurectomy, or axillary thoracotomy] measures [40]. This and the paucity of large, prospective, randomized clinical trials, as well as the different medical specialists taking care of PSP (pulmonologists, surgeons, radiologists, emergency physicians), probably explain why the present national and international expert opinion-based guidelines [41–43] are only poorly followed [44–47]. An algorithmic approach can be proposed (fig. 3).

A patient presenting with a first episode of a small (i.e., only partial, usually apical) dehiscence of the lung should not be treated, but can safely be discharged and followed on an outpatient basis.

In case of complete dehiscence of the lung and/or in case of pneumothorax symptoms air evacuation treatment is warranted. There is now sufficient evidence coming from eight papers [48–55] and three meta-analyses and reviews [56–58] that simple manual aspiration should be the first-line treatment approach in these PSP patients (table 2). Success rates vary between 50 and 80% of cases, averaging two thirds of cases. Complications are absent, pain and discomfort are minimized, recurrence rates are similar to those seen after typical chest tube drainage, outpatient treatment with immediate discharge is possible in over half the cases, and length of stay, when necessary, is significantly shortened. Alternatively, because repeat aspiration or insertion of a catheter is necessary in one third of patients, some authors propose immediate placement of a small catheter attached to a Heimlich valve followed by immediate discharge [59, 60].

There is also good consensus and clinical evidence that PSP recurrence prevention should only be proposed after a first recurrence [39, 40] based on the observation that there is a recurrence in about one third of patients [18], but this may increase to 62% after a first recurrence, and to 83% after a third [61]. Exceptions may be patients at professional risk (aviation personnel, divers), when

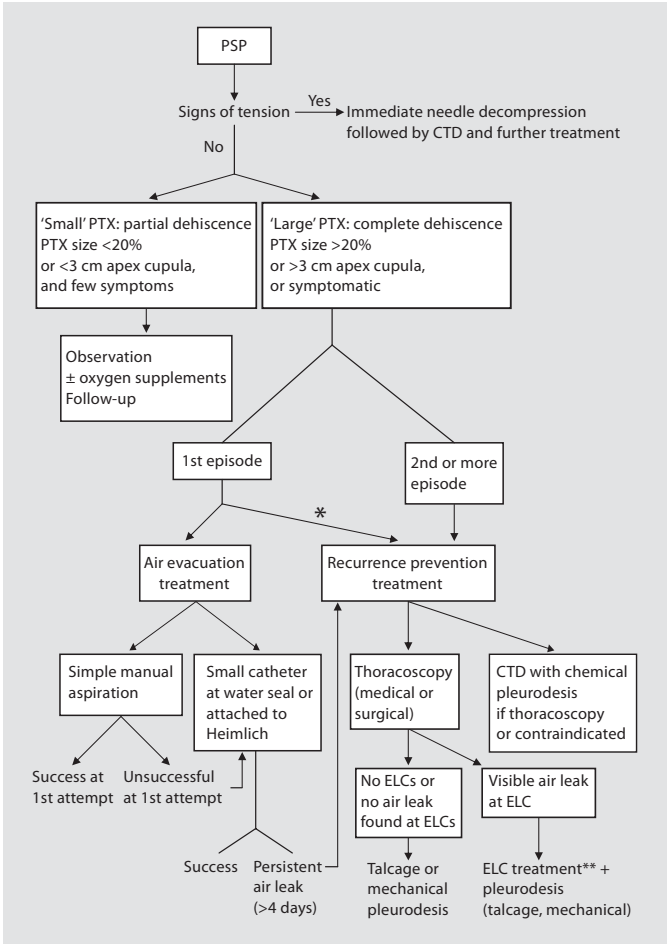


Fig. 3. An algorithmic approach to the treatment of PSP. * After informed consent or in certain patient groups (aircraft personnel, divers). ** Staple bleb/bullectomy, electrocoagulation, ligation. CTD = Chest tube drainage; PTX = pneumothorax; ELCs = emphysema-like changes.

preferred by anxious patients [39], or when a prolonged air leak (>4 days) [42] is present. Of note, intrapleural insertion of a catheter or tube has only a minimal (if any) effect on recurrence prevention (34–36% observed recurrence rates after chest tube drainage only) [40, 62]. The optimal procedure for recurrence prevention remains controversial because of the paucity of prospective, randomized, large, head-to-head comparative studies.

Intermediate recurrence prevention success rates can be achieved by administration of a sclerosing agent through a chest tube (e.g., talc slurry, tetracycline, minocycline, or doxycycline) [40]. This approach is therefore acceptable only in those patients who are unfit for or refuse thoracoscopy or more invasive surgery.

Table 2. Simple manual aspiration as first-line treatment of PSP: immediate success rates

	Immediate success rate, %
BTS, 1994 [48]	80
Andrivet et al., 1995 [49]	68.5
Noppen et al., 2002 [50]	59
Faruqi et al., 2004 [51]	83
Chan and Rainer, 2006 [52]	50.5
Camuset et al., 2006 [53]	69
Ayed et al., 2006 [54]	62
Masood et al., 2007 [55]	76

Table 3. Frequent and/or typical causes of SPP

Airway disease
Emphysema
Cystic fibrosis
Severe asthma
Infectious lung disease
Pneumocystis carinii pneumonia
Tuberculosis
Necrotizing pneumonia
Interstitial lung disease
Idiopathic pulmonary fibrosis
Sarcoidosis
Histiocytosis X
Lymphangioleiomyomatosis
Connective tissue disease
Rheumatoid arthritis, scleroderma, ankylosing spondylitis
Marfan's syndrome
Ehlers-Danlos syndrome
Malignant disease
Lung cancer
Sarcoma

The choice between 'medical' thoracoscopy, 'surgical' thoracoscopy (VATS) or open surgery [usually via anterolateral thoracotomy as access method to the pleural cavity depends upon the professional background of the operator (pulmonologist or surgeon), and local availabilities, preferences, beliefs and habits]. Open surgical approaches are slightly superior [63] or equally effective as 'closed' thoracoscopic methods [64], but carry a higher morbidity [65]. Therefore, unless there are specific clinical indications for more invasive surgery, it would seem reasonable for thoracoscopy to become the recommended approach [66]. Also, within the surgical community, there is a trend towards less invasive VATS approaches, such as uniportal VATS [67], needle thoracoscopy [68] or even awake VATS procedures [69], which narrows the spectrum between surgical and medical thoracoscopy to almost nil. More important than the technique of access to the pleural space is the procedure which is performed within this space. Bleb and bulla treatment by means of stapled resection, clipping, ligation, looping, laser or electrocautery ablation is still the surgical dogma. When performed without associated pleurodesis, recurrence rates are unacceptably high (up to 20%) [22–25]. It is therefore questionable, unless a bleb or bulla is clearly leaking (thus 'flat') during thoracoscopy, whether a parenchymal procedure is absolutely necessary [39, 40]. Adequate pleurodesis should be the cornerstone of thoracoscopic recurrence prevention. All pleurodesis techniques are based on the successful induction of some form of pleural inflammation [40, 70]. This can be achieved my mechanical abrasion, partial resection, or thoracoscopic instillation of an abrasive agent, usually talc. There is undisputable evidence that the use of size-calibrated talc is absolutely safe, in short- as well as long-term follow-up studies [71–74]: it does not cause cancer, pulmonary fibrosis, impaired pulmonary function or impaired subsequent thoracic surgery, and it is by far the cheapest agent. Thoracoscopic recurrence prevention techniques, be they 'medical' or 'surgical', usually show recurrence rates between 0 and 10%. As mentioned earlier, open surgical interventions might even be more successful in experienced hands.

Finally, these therapeutic recommendations are equally valid in children [75, 76] and in pregnancy [77, 78].

Secondary Spontaneous Pneumothorax

A multitude of respiratory disorders have been described as a cause of spontaneous pneumothorax. The most frequent underlying disorders are COPD with emphysema, cystic fibrosis, tuberculosis, lung cancer, HIV-associated *Pneumocystis carinii* pneumonia, followed by more rare but 'typical' disorders such as lymphangioleiomyomatosis and histiocytosis X (table 3). Because lung function in these patients is already compromised, secondary spontaneous pneumothorax (SSP) often presents as a potentially life-threatening disease, requiring immediate action, in contrast with PSP which is more of a nuisance than a dangerous condition. The general incidence is almost similar to that of PSP. Depending upon the underlying disease, the peak incidence of SSP can occur later in life, e.g. at 60–65 years of age in the emphysema population [2].

In SSP, dyspnea is the most prominent clinical feature; chest pain, cyanosis, hypoxemia, and hypercapnia, sometimes resulting in acute respiratory failure, can also be present. Diagnosis is confirmed on a posteroanterior chest radiograph; in bullous emphysema, the differential diagnosis with a giant bulla can be difficult, necessitating CT confirmation [79]. As in PSP, air may enter the pleural space through various mechanisms: direct alveolar rupture (as in emphysema or necrotic pneumonia) via the lung interstitium, or backward via the bronchovascular bundle and mediastinal pleura (pneumomediastinum). Recurrence rates usually are higher as compared to those for PSP, ranging up to 80% of cases as is observed in cystic fibrosis [80].

Management

SSP requires immediate air evacuation followed by recurrence prevention at the first episode. All patients with SSP should be hospitalized [40]. Awaiting recurrence prevention treatment, air evacuation can be achieved by simple manual aspiration in young (<50 years old) patients with small pneumothoraces [41], but most authors and guidelines recommend immediate insertion of a chest tube. Small bore chest tubes and even pigtail catheters [81] are usually sufficient; large-bore chest tubes are recommended when large air leaks are suspected or when mechanical positive pressure ventilation is required [42]. Recurrence prevention using a thoracoscopic approach (medical or thoracoscopic) is recommended; in case a visible air leak is present (e.g., a ruptured emphysematous bulla), air leak closure using electrocautery or stapling is indicated. In any case, a pleurodesis procedure such as talc poudrage, pleural abrasion or partial pleurectomy should be performed [3, 82]. In patients in whom lung transplantation is a possible future option (e.g., cystic fibrosis, some cases of COPD), the transplant team should be consulted on whether to perform pleurodesis or not. For most transplant teams, previous pleurodesis does not represent a contraindication for later transplantation.

Catamenial Pneumothorax

Catamenial pneumothorax occurs typically within 24–72 h after onset of menstruation. It is often recurrent and may be more common than previously thought [2, 3]. In most cases, catamenial pneumothorax is related to pelvic or thoracic endometriosis [83, 84]. Recurrence prevention treatment is indicated after a first episode of catamenial pneumothorax, because recurrences are frequent. Hormonal suppression treatment is often added.

Traumatic Noniatrogenic Pneumothorax

Pneumothorax ranks second to rib fracture as the most common sign of chest trauma, occurring in up to 50% of chest trauma victims [85]. In half of these cases, pneumothorax may be occult; in chest trauma patients requiring mechanical ventilation, CT of the chest should therefore always be performed [3, 85]. Most surgeons and emergency physicians will place a chest tube in occult and nonoccult traumatic pneumothoraces. However, studies suggest that carefully selected patients may be treated conservatively ultimately requiring chest tube placement only in about 10% of cases [86]. If positive pressure ventilation is anticipated, placement of a chest tube is mandatory. In these cases and in case of an associated hemothorax (20% of patients), placement of a large-bore chest tube (28–36 french) is advocated.

Traumatic Iatrogenic Pneumothorax

Iatrogenic pneumothorax occurs most often following transthoracic needle biopsy (24%), subclavian vein catheterization (22%), thoracentesis (20%), transbronchial lung biopsy (10%), pleural biopsy (8%) and positive pressure ventilation (7%) [3]. Diagnosis of iatrogenic pneumothorax is often delayed, which should make physicians vigilant. Small and asymptomatic iatrogenic pneumothoraces often do not need any treatment, and resolve spontaneously. In larger or symptomatic pneumothoraces, simple manual aspiration or placement of a small catheter or chest tube attached to a Heimlich valve usually is successful [87]. Larger tubes may be necessary in emphysematous patients or when mechanical ventilation is indicated.

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