Current aspects of spontaneous pneumothorax


Pneumothorax is defined as the presence of air in the pleural cavity [1]. As early as 1819, LAENNEC [2] described the symptoms and signs of a patient with a pneumothorax. Although most pneumothoraces were then caused by tuberculosis, he also found pneumothoraces during autopsies of patients with apparently healthy lungs; he named these "pneumothorax simple".

Aetiology and pathogenesis

Today, pneumothoraces are divided into spontaneous pneumothorax, occurring without a preceding event, and traumatic pneumothorax, due to direct or indirect trauma. Iatrogenic pneumothoraces, resulting from diagnostic or therapeutic medical procedures, are also categorized as traumatic pneumothoraces.

Spontaneous pneumothoraces are divided into primary and secondary spontaneous pneumothoraces. Secondary spontaneous pneumothoraces are associated with underlying pulmonary pathology, usually chronic obstructive pulmonary disease (COPD). Acquired immune deficiency syndrome (AIDS) and Pneumocystis carinii infections appear to play an increasing role in the aetiology of secondary spontaneous pneumothoraces [3].

No underlying pulmonary disease is present in patients with primary spontaneous pneumothorax. However, blebs and bullae seem to play a role in the pathogenesis, since they are frequently found during thoracoscopy, thoracotomy or sternotomy (table 1). Thoracoscopic studies have shown the presence of blebs and bullae in 48–79% of patients with unilateral primary spontaneous pneumothorax [4–8]. With the development of video-assisted techniques, the recognition of blebs and bullae during thoracoscopy has improved. In more than 76% of patients, patients with a pneumothorax simple pathology seem to play a role in the pathogenesis, since they are frequently found during thoracoscopy, thoracotomy or sternotomy (table 1). Thoracoscopic studies have shown the presence of blebs and bullae in 48–79% of patients with unilateral primary spontaneous pneumothorax [4–8]. With the development of video-assisted techniques, the recognition of blebs and bullae during thoracoscopy has improved. In more than 76% of patients,

<table>
<thead>
<tr>
<th>Author</th>
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<td>T</td>
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<td>T</td>
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<td>10</td>
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<td>1993</td>
<td>17</td>
<td>83</td>
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</tbody>
</table>

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blebs and bullae were detected during video-assisted thoracoscopy (VAT) [9–12]. Surgical studies have shown blebs and bullae in almost all patients during thoracotomy or sternotomy [13–17]. Two studies have described bilateral surgical therapy in patients with unilateral spontaneous pneumothorax, demonstrating blebs and bullae on the contralateral side in 79–96% of the patients [13, 16]. Bilateral presence of blebs and bullae in patients with unilateral primary spontaneous pneumothorax has also been demonstrated with thoracic computed tomography (CT). On CT, blebs and bullae are designated as emphysema-like changes (ELCs). ELCs were found in 89% of patients on the ipsilateral side and in up to 80% on both sides [18, 19]. In the control group, consisting of healthy volunteers matched for age and smoking behaviour, ELCs were found in 20% of individuals [19]. ELCs were also found in 81% of patients with healed primary spontaneous pneumothorax, who had never smoked, in contrast to none of the controls who had never smoked [20]. Therefore, ELCs appear to be related to the occurrence of primary spontaneous pneumothorax.

Several theories exist regarding the development of ELCs in patients with primary spontaneous pneumothorax. As in centriacinar emphysema, the formation of ELCs in primary spontaneous pneumothorax is associated with the degradation of elastic fibres [21]. The elastolysis is caused by the imbalance between proteases and antiproteases and between oxidants and antioxidants, in which neutrophils and macrophages play an important part [22–24]. The respiratory bronchiolitis caused by the influx of these inflammatory cells, as in smokers, is closely associated with bronchiolar wall fibrosis and destruction of the pulmonary parenchyma, resulting in ELCs [25]. Bronchoalveolar lavage (BAL) in patients with primary spontaneous pneumothorax showed a close relationship between the total cell count, especially macrophages, in the BAL fluid and the extent of ELCs both in the affected and the nonaffected lung [26]. However, no study has demonstrated that ELCs are the actual cause of primary spontaneous pneumothorax.

Another fact that strengthens the hypothesis that inflammatory changes play an important role in the pathogenesis of primary spontaneous pneumothorax is the fact that smoking is related to a ninefold increase of relative risk of contracting a pneumothorax among females and a 22 fold increase of relative risk in males, with a dose-response relationship between the number of cigarettes smoked per day and the occurrence of primary spontaneous pneumothorax [27]. Numerous studies have demonstrated increased numbers of inflammatory cells, especially macrophages, in the small airways of smokers [28, 29]. These macrophages release potent chemotactic factors, resulting in accumulation of neutrophils in the small airways [28]. The influx of neutrophils is also enhanced by loss of functional activity of chemotactic factor inactivator due to the cigarette smoke itself [30].

The inflammatory changes in the distal airways of smokers suggest that endobronchial obstruction is part of the pathogenesis of primary spontaneous pneumothorax. Endobronchial obstruction due to accumulation of inflammatory cells between the pulmonary parenchyma and the bronchial tree can induce overpressure in alveolar tissue, resulting in rupture of pulmonary parenchyma. This is supported by the fact that changes in the transpulmonary pressure are related to increased incidence of pneumothorax in fighter pilots and divers [31, 32]. However, less excessive transpulmonary pressure changes also induce pneumothoraces. Atmospheric pressure changes of at least 1 kPa are associated with an increased incidence of spontaneous pneumothorax [33, 34]. It remains unclear whether ELCs are the site of rupture, because, according to Boyle’s law, during a rise or fall of intrapulmonary pressure, pressure changes inside ELCs will be smaller than in normal alveolar tissue due to the larger cubic capacities of ELCs.

Histopathological and electron microscopic analysis of tissue obtained during bullectomy in patients with spontaneous pneumothorax have revealed obstruction and stenosis of the distal airways due to bronchial wall inflammation and peribronchial fibrosis [35, 36]. These findings suggest an obstruction check-valve mechanism as the cause of spontaneous pneumothorax. In these studies, communications between pleural cavity and ELCs were not seen, although some types of blebs exhibited a marked absence of mesothelial cells [37]. Therefore, the site of rupture of the visceral pleura appears to be located outside ELCs.

Diagnosis

Spontaneous pneumothorax is usually suggested by clinical history and physical examination. Radiological investigation of the chest is needed to establish the diagnosis. Currently, it is common practice to perform chest radiographs during maximal inspiration and expiration in patients suspected of a pneumothorax. Two studies have been published evaluating the value of routine use of additional inspiratory chest radiographs in diagnosing pneumothoraces. All pneumothoraces were seen by two observers on inspiratory chest radiographs in 79 patients with spontaneous pneumothorax [38]. Two out of four observers missed one small apical pneumothorax on an inspiratory chest radiograph, when evaluating 128 chest radiographs, consisting of 100 paired inspiratory and expiratory chest radiographs with proven pneumothoraces and 28 normal chest radiographs [39]. If expiratory chest radiographs have additional value in diagnosing pneumothoraces, this should be especially so in difficult situations, such as in small apical pneumothoraces.

Several explanations are possible for the potential beneficial effects of expiration in detecting small apical pneumothoraces on chest radiographs in the upright position. A relative enlargement of the pneumothorax in relation to the size of the thoracic cage might occur during expiration. External compression of lung tissue during expiration could result in thickening of the visceral pleura and enhanced contrast between intrapleural air and pulmonary parenchyma. This last explanation appears to be invalid. During expiration, the density of collapsed lung tissue is not altered [40]. However, the density of the extrapulmonary tissue is increased during expiration, resulting in increased radiation exposure as monitored by the ionization chambers of standard radiological equipment. Increased radiation exposure resulted in increased blackening of the collapsed lung on expiratory chest radiographs.
Radiographs [41]. No difference in contrast was found between pulmonary tissue and intrapleural air on inspiratory and expiratory chest radiographs [41]. From these findings, one may conclude that the routine addition of expiratory chest radiographs does not improve the diagnostic yield from inspiratory chest radiographs in patients suspected of having a pneumothorax. It therefore seems justified to confine the radiological investigation to inspiratory chest radiographs. Moreover, inspiratory chest radiographs are needed to investigate other possible causes of the patient’s complaints, such as pulmonary effusions or parenchymal consolidations.

**Recurrences**

The average rate of absorption of air from the pleural cavity is slow. In a study of 11 patients with pneumothoraces ranging 16–100% of collapsed lung in size, the mean rate of re-expansion was 1.8% per day, resulting in full re-expansion of the collapsed lung after 7 weeks (mean 3.2 weeks) [42]. Therefore, only patients with a pneumothorax <15% of the hemithorax in size can be successfully subjected to this kind of conservative treatment [1]. The recurrence rate of primary spontaneous pneumothorax treated with observation was 32% [43]. Since a minority of patients with primary spontaneous pneumothorax have small pneumothoraces, most patients are treated with invasive procedures [5, 43].

Needle aspiration or tube drainage will have the smallest influence upon the natural course of primary spontaneous pneumothorax because conditions in the pleural cavity will only be slightly altered. A survey of several studies (table 2) shows that the mean recurrence rate of primary spontaneous pneumothorax treated with bedrest, needle aspiration, or tube drainage was 30%, ranging 16–52% [43–52]. The majority of recurrences appeared within 6 months to 2 yrs [49, 50], although other studies could not confirm this finding [45, 47].

Several studies have been performed concerning possible relationships between patient characteristics, pulmonary disease, and the development of recurrences. Independent risk factors for recurrence in 122 patients with primary spontaneous pneumothorax were reported to be: pulmonary fibrosis detected on chest radiographs, physical characteristics; smoking behaviour; and age [50]. However, no association could be demonstrated between recurrence rate and COPD in 303 patients with primary and secondary spontaneous pneumothorax [53]. In 35 patients with primary spontaneous pneumothorax, who underwent thoracic CT scanning, the presence of ELCs had no predictive value for recurrences during follow-up [18]. No differences in presence of ELCs detected during VAT could be found in patients with first time primary spontaneous pneumothorax and recurrent spontaneous pneumothorax [54].

From these findings, one can conclude that the presence of ELCs in patients with primary spontaneous pneumothorax has no predictive value for the future development of recurrences. Therefore, investigations in order to diagnose ELCs should not influence the choice of treatment to prevent recurrences.

**Pulmonary function tests**

In the past, several studies have reported various results concerning the analysis of pulmonary function parameters in patients treated for primary spontaneous pneumothorax [55–58]. Almost all patients were treated with tube drainage. Since the patients had no previous pulmonary complaints, none of the studies described pulmonary function test parameters before the onset of the pneumothoraces.

The main issue in these studies concerned the question of whether patients with primary spontaneous pneumothorax had physiological evidence of emphysema. Although emphysema is a histopathological diagnosis, pulmonary function criteria, such as decreased diffusing capacity, reduced retractive force and increased total lung capacity, are known to correlate with emphysema [59].

Decreased diffusing capacity parameters have been reported in healthy smokers, caused by decrease of the gas-exchange surface due to destruction of alveolar walls [60]. Reduced diffusing capacity was found in 25–86% of patients with primary spontaneous pneumothorax [55–58]. More than 70% of the patients were smokers. The reduced diffusing capacity reflected the loss of alveolar surface, which is expressed by ELCs [57]. In previous reports, 10% of patients with primary spontaneous pneumothorax met all of the pulmonary function criteria of emphysema [55–58]. The mean age of these patients ranged 28–32 yrs, which is the lower limit of the age distribution at which emphysema becomes apparent [61]. The discrepancy between the minimal pulmonary

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Table 2. – Recurrence rates of patients with primary spontaneous pneumothorax, treated with bed-rest, needle aspiration or tube drainage

<table>
<thead>
<tr>
<th>First author</th>
<th>Year</th>
<th>[Ref.]</th>
<th>Pts n</th>
<th>Treatment</th>
<th>Recurrence rate %</th>
<th>Follow-up yrs</th>
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<td>SEREMEITIS</td>
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<td>TD</td>
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<td>LIGHT</td>
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<td>TD</td>
<td>41</td>
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<td>122</td>
<td>TD</td>
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<td>10</td>
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<td>NA, TD</td>
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<td>ANDRIVET</td>
<td>1995</td>
<td>[52]</td>
<td>61</td>
<td>NA, TD</td>
<td>21</td>
<td>0.25</td>
</tr>
</tbody>
</table>

**Pts:** patients; **BR:** bed-rest; **NA:** needle aspiration; **TD:** tube drainage; **BTSRC:** British Thoracic Society Research Committee.
function abnormalities and the macroscopic findings of ELCs might be explained by the limitations of pulmonary function testing. The patients are not old enough to show physiological signs of emphysema, since previous reports demonstrated that ageing resulted in a homogeneous enlargement of the alveolar airspaces [62]. These changes preceded emphysema and were responsible for loss of lung elasticity. A follow-up study over several decades, analysing pulmonary function parameters of patients with spontaneous pneumothorax and healthy volunteers, will be needed to determine whether more patients with spontaneous pneumothorax will develop clinical emphysema than the healthy volunteers.

Treatment

General considerations

Debate continues regarding the best management of primary spontaneous pneumothorax. Evacuation of air from the pleural cavity and the prevention of future recurrences are the primary goals of treatment. Numerous therapeutic options are available, ranging from observation to posterolateral thoracotomy with bullectomy and pleurectomy [1]. Invasive procedures are needed if the size of the pneumothorax exceeds 15% of the volume of the hemithorax, or if the pneumothorax is progressive over time.

Today, choice of treatment depends on the patient’s presentation with a first time or recurrent spontaneous pneumothorax, the size of the pneumothorax, and the presence and extent of ELCs [1, 7, 8].

However, as mentioned previously in this review, the presence of ELCs in patients with primary spontaneous pneumothorax has no predictive value for the future development of recurrences and, therefore, should not influence the choice of treatment.

A recurrent spontaneous pneumothorax is one of the indications to perform more invasive therapy, such as pleural drainage with chemical pleurodesis, or even a surgical intervention. This is based on the assumption that the recurrence rate increases after previous pneumothoraces [14]. However, differences in recurrence rates after first time (57%) and second time pneumothoraces (62%), as well as after third time pneumothoraces (83%), were not statistically significant, in contrast to differences between first and third time pneumothoraces (p=0.04) [14]. Since the average time interval between the first time and the third time pneumothorax was 5 yrs, it is possible that recurrent spontaneous pneumothorax was not the risk factor for contracting a subsequent pneumothorax, but rather the age of the patient. Age has proved to be one of the predictors for future recurrences [49].

Therefore, the choice of treatment must not depend on the presence of ELCs or presentation with first time or recurrent spontaneous pneumothorax, but on the effectiveness of the treatment.

Several other factors, such as morbidity and the availability and costs of therapeutic options available in the clinic, are also of importance in making the right choice. Currently, cost-effectiveness is becoming increasingly important in the choice of treatment.

Chemical pleurodesis

Needle aspiration, tube drainage and the use of a Heimlich flutter valve have proved to be safe procedures in the treatment of primary spontaneous pneumothorax, with minor side-effects [14, 44–52]. However the mean ipsilateral recurrence rate of 30% was rather high. Chemical pleurodesis was shown to decrease the ipsilateral recurrence rate. In a prospective, multicentre, randomised clinical trial of 229 patients with spontaneous pneumothorax, tetracycline reduced the recurrence rate to 25%, compared to tube drainage with a recurrence rate of 41% [48]. In several other studies, tetracycline also resulted in effective pleurodesis, with average recurrence rates of 9–16% [8, 63, 64]. Intrapleural instillation of tetracycline induces the release of cytokines (interleukin-6, interleukin-8, tumour necrosis factor), which are markers of inflammatory response and attract neutrophils into the pleural space, resulting in pleural symphysis [65].

A good alternative is talc-suspension, also known as talc slurry. In a randomized prospective study of 96 patients, pleurodesis by talc slurry resulted in the lowest recurrence rate of 8%, compared to 13% with tetracycline and 36% with simple tube drainage [47].

More commonly, talc is used by means of poudrage during thoracoscopy. Thoracoscopy can be performed by one port of entry with direct vision through a rigid telescope [4–7]. During the last few years, new imaging and video-techniques have become available, making thoracoscopy with a video-camera and multiple ports of entry possible [66]. Four to six grams of talc is insufflated by means of a talc atomizer or a disposable single-use spray canister. From recent reviews and several other following reports, a recurrence rate of 8% was found in 1,030 patients [8, 12, 67–69]. Pleural symphysis is achieved by increased activation of pleural coagulation, expressed by increased production of antithrombin III complex and plasminogen activator inhibitor, and by decreased pleural fibrinolytic activity expressed by decreased levels of the degradation product of cross-linked fibrin (d-dimer) [70].

There has been some doubt about the safety of pleurodesis with talc, especially with talc slurry. Two cases have been reported with acute pneumonitis and adult respiratory distress syndrome (ARDS) after the administration of talc slurry [71, 72]. This rare complication occurred when a large amount of talc (more than 10 g) was administered [71], or when vascular injury was present [72]. Others have suggested that it might be due to re-expansion oedema after the procedure [67].

Minimal long-term side-effects have been demonstrated with talc poudrage, with no major effect on the lung parenchyma, little impairment of pulmonary function, and pleural thickening on chest radiographs [57]. No mesothelioma was found in two studies following patients for 18–35 yrs after pleurodesis with talc [57, 73].

Surgical therapy

Conventional surgical therapy consists of (limited) posterolateral thoracotomy with bullectomy and pleurectomy (table 3). Pleurectomy was used to treat recurrent spontaneous pneumothorax for the first time in 1956, in nine patients [74]. In the following years, partial pleurectomy or pleural abrasion became standard therapy,
especially for recurrent spontaneous pneumothorax, and resulted in average recurrence rates of 1% [9, 75–77]. Combined pleurectomy and bullectomy showed average recurrence rates of 2% [7, 13, 14, 17, 78, 79]. A few authors have recommended bilateral surgical therapy, because 15% of the patients receiving unilateral therapy developed a contralateral pneumothorax during the years after the initial treatment [13, 16].

Recently, there has been a rapid development of minimally invasive surgery, particularly in video-assisted thoracic surgery (VATS). VATS is performed in the operation room during general anaesthesia with double-lumen intubation. Multiple ports of entry are used to introduce rigid telescopes with video attachment and various other instruments. Pleurectomy and bullectomy can be performed by VATS.

Reviewing the results of VATS in 805 patients with spontaneous pneumothorax, the mean recurrence rate was 4% (table 4) [10–12, 80–91], which was higher than the mean recurrence rate of 1.5% after conventional surgical therapy (table 3) [7, 9, 13, 14, 17, 74–78]. However, several studies that have reported comparative analysis of VATS versus conventional thoracotomy have shown reduced operation time, drainage time and complication rates after VATS, resulting in shorter hospital stay and a more rapid return to the normal daily activities of the patients [84, 92, 93].

VATS has also proved to be more cost-effective compared to conservative treatment by observation or pleural drainage in patients with spontaneous pneumothorax. Irrespective of the presence of ELCs, drainage and hospitalization time were shorter and complication and recurrence rates were lower in patients with first time or recurrent spontaneous pneumothorax treated with VATS compared to conservative treatment. When costs due to the waiting time before VATS were excluded, the total costs of VATS were lower than the cost of conservative therapy [94].

Since the presence of ELCs and recurrent spontaneous pneumothorax at presentation are not predicting factors for future recurrences, it can be questioned whether VATS should be applied only in patients with recurrent or secondary spontaneous pneumothorax, as is the case in most studies. In 113 patients, mostly with recurrent spontaneous pneumothorax, univariate and multivariate analysis of clinical characteristics, such as age, smoking behaviour, presence of ELCs, pleurodesis and bleb ablation by stapling, showed that the resection of ELCs during VATS was the only predictor of future recurrences [88]. In a study of 97 patients, of which 73% were patients with first time spontaneous pneumothorax, similar multivariate analysis showed that the use of stapler devices during VATS was significantly associated with prolonged drainage time and hospital stay, but not with

### Table 3. – Conventional surgical treatment in patients with spontaneous pneumothorax

<table>
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<th>First author</th>
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<th>[Ref.]</th>
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<td>[79]</td>
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<td>B, PA</td>
<td>0</td>
<td>0.41</td>
</tr>
</tbody>
</table>

Total 977 1.5

Pts: patients; B: bullectomy; PP: partial pleurectomy; PA: pleural abrasion; PD: Pleurodesis.

### Table 4. – Video-assisted thoracic surgery (VATS) in patients with spontaneous pneumothorax

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<th>First author</th>
<th>Year</th>
<th>[Ref.]</th>
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<th>Treatment</th>
<th>Recurrence rate</th>
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Total 805 4

Pts: patients; EG: stapling with Endo-gastro-intestinal anastomosis (GIA) 3.0 Multifire (Autosuture; Zeist, the Netherlands); EL: Endo-loop; PS: pleural scarification; PP: partial pleurectomy; PD: Pleurodesis.
future recurrences [12]. From these findings, it may be concluded that it was not the presence of ELCs but the technique used to resect them that was responsible for the postoperative course and the occurrence of recurrent pneumothoraces.

First time or recurrent spontaneous pneumothorax showed no differences in postoperative outcome after VATS. In view of effectiveness, it is not justified to treat patients with first time spontaneous pneumothorax in a different and less effective way than patients with recurrent spontaneous pneumothorax.

Treatment of secondary spontaneous pneumothorax

Treatment options in primary and secondary spontaneous pneumothorax are similar. However, patients with secondary spontaneous pneumothorax are usually respiratorily compromised, necessitating rapid and effective treatment. In recent years, VATS has played an important role in the management of these patients [95]. Adequate pleurodesis with t alc or partial pleurectomy can be performed. If VATS is not available in the clinic, thoracotomy is mandatory to achieve pleurodesis and, if necessary, oversew the airleak. Surgical removal of blebs and bullae appears to have a negative effect on the postoperative course due to prolongation of the airleakage [95]. However, ablation of bullae by neodymium:yttrium-aluminum garnet (Nd:YAG) laser, carbon dioxide laser or argon beam laser showed a rapid sealing effect, which proved to be safe even in patients with severe bullous emphysema [96–98].

AIDS-related spontaneous pneumothorax is usually caused by Pneumocystis carinii infection and is difficult to treat. Treatment with tube thoracostomy has a high recurrence rate of 65% [1]. Effective treatment should be performed by chemical or surgical pleurodesis in combination with effective sealing of the air leakage by stapling, electrocautery or laser photoagulation [99].

Summary

Several new aspects have emerged during the last 177 yrs, in which spontaneous pneumothoraces have been recognized and treated.

Blebs and bullae are related to the occurrence of primary spontaneous pneumothorax, but are seldom the actual cause of the pneumothorax. The obstruction check-valve mechanism due to inflammatory changes of the distal airways is responsible for the development of the pneumothorax during transpulmonary pressure changes.

For diagnosing pneumothoraces with radiography, the additional use of an expiratory chest radiograph is of no value. The natural course of primary spontaneous pneumothorax shows a mean recurrence rate of 30%.

There is still no answer to the question of whether patients with spontaneous pneumothorax have (sub)clinical emphysema. The choice of treatment of spontaneous pneumothorax should not depend on the presence of ELCs or presentation with first time or recurrent spontaneous pneumothorax, but on the efficacy of the treatment. Pleurodesis by pleurectomy during video-assisted thoracoscopy shows the best results regarding cost-effectiveness in the management of primary and secondary spontaneous pneumothorax. Chemical pleurodesis with t alc is a good alternative.

References


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