

which represents an established treatment modality affording identification and treatment of 90 and 100%, respectively, of all pulmonary blebs or bullae by pleural abrasion and wedge resection with similar long-term results [6], thereby avoiding the use of talc.

The British Thoracic Society guidelines also recommend that talc pleurodesis should not be considered as initial treatment for PSP requiring surgical intervention. Open thoracotomy or video-assisted thoracoscopy with wedge resection and pleuroectomy is the first-choice treatment for PSP and, indeed, secondary pneumothorax [7]. The American College of Chest Physicians has the same recommendation: the instillation of sclerosing agents is only acceptable in patients who wish to avoid surgery and for patients who present increased surgical risk [8].

As thoracic surgeons we have the responsibility to preclude potential hazards for our patients that are caused by our own actions. The light-hearted employment of talc represents, in our view, such an avoidable hazard, for which we all have to carry the can with our patients.

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STATEMENT OF INTEREST

None declared.

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From the authors:

We would like to thank V. Steger and co-workers for their interest in our recent article [1]. The technique of talc pleurodesis for primary spontaneous pneumothorax has been applied for decades in many centres in Europe without any evidence of long-term problems [2–4]. We have already discussed the question of treating primary spontaneous pneumothorax with persisting air leak for >48 h in our study. This practice at our institution has stemmed from a previous report from our hospital which showed that when an air leak persists for >48 h, the probability of spontaneous resolution of the pneumothorax is low [5]. The recurrence rate of primary spontaneous pneumothorax is ≤40% [6]. Would the cardiologist not put a stent in the coronary arteries if the risk of myocardial infarction was “only 40%”? Therefore, we think that medical thoracoscopy with talc pleurodesis or video-assisted thoracic surgery (VATS) might already be performed in the first episode of pneumothorax. We agree with V. Steger and co-workers that the use of talc pleurodesis in the young population may make potential surgery for lung cancer more difficult. However, we think that smoking cessation would be the important approach to avoid future development of cancer in the young population.

VATS, under combined general anaesthesia and epidural anaesthesia with double lumen intubation to perform abrasion pleurodesis and stapling of visualised blebs, has been recommended as the first-line approach for the management of recurrent spontaneous pneumothorax with recurrence rates of 3% [7]. The patients in our study also had a similar outcome but with a simple, fast and cheaper procedure without the need for general anaesthesia. Furthermore, the results of our study suggest that stapling or electrocoagulation of blebs does not influence the outcome of talc pleurodesis and supports the approach that talc pleurodesis is sufficient for the management of recurrent or persistent primary spontaneous pneumothorax. A recent review article [8] has also concluded that systematic treatment of emphysema like changes with blebectomy or bullectomy is not indicated and that it is the treatment of the pleura (*i.e.* pleurodesis), and not that of the lung, which should be considered the real cornerstone of recurrence prevention.

Whether medical thoracoscopic talc pleurodesis or video-assisted thoracic surgery is applied depends mainly on the availability of pulmonologists with experience in invasive procedures.

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STATEMENT OF INTEREST

None declared.

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Mitochondrial dysfunction in COPD patients with low body mass index

To the Editors:

The potential importance of mitochondrial (dys)function in common chronic diseases is increasingly recognised [1]. Accordingly, we read with great interest the article by RABINOVICH *et al.* [2], which was recently published in the *European Respiratory Journal*. RABINOVICH *et al.* [2] showed that chronic obstructive pulmonary disease (COPD) patients with a low body mass index (BMI; $19.2 \pm 0.6 \text{ kg}\cdot\text{m}^{-2}$) have a dysfunctional mitochondrial electron transport chain in comparison with COPD patients with a “normal” BMI ($29.0 \pm 1.7 \text{ kg}\cdot\text{m}^{-2}$) and healthy controls ($27.9 \pm 1.9 \text{ kg}\cdot\text{m}^{-2}$). The function of the mitochondrial electron transport chain was assessed as acceptor control ratio (*i.e.* the efficiency of oxidative phosphorylation) in freshly obtained muscle biopsies.

We recently showed that cigarette smoke, which is the major cause of COPD in the Western world, can induce significant mitochondrial dysfunction [3]. This dysfunction was the consequence of a cigarette smoke-induced blockage of the mitochondrial electron transport chain, resulting in a decreased mitochondrial respiration rate and a corresponding decrease in adenosine triphosphate production.

In the study performed by RABINOVICH *et al.* [2], the prevalence of current and past smoking was not reported. As a consequence, the potential influence of smoking on the outcome of the study was neither analysed nor discussed. Due to our recent observations, and reports from other groups about the induction of mitochondrial dysfunction by cigarette smoke even in circulating cells that do not reside in pulmonary tissues [4], we thought it would be very interesting to know how the results of the clinical study performed by RABINOVICH *et al.* [2] would appear if current (and past) smoking were taken into account. Furthermore, it would be interesting to know how the significantly lower acceptor control ratio results reported in their study affect the principal function of mitochondria: adenosine triphosphate production.

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STATEMENT OF INTEREST

None declared.

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From the authors:

We would like to thank D-J. Slebos and co-workers for their interesting and assertive contribution to the present issue of the *European Respiratory Journal*. Following their arguments, one can reasonably state that because tobacco smoking is the main cause of chronic obstructive pulmonary disease (COPD), it might also play a role in a mechanism leading to the