Improving the investigation of suspected respiratory disease

**P178** THE EFFECT OF BAL INDUCED INFLAMMATION ON NASAL INNATE DEFENCE — REDUCTION IN EXPERIMENTAL HUMAN PNEUMOCOCCAL CARRIAGE

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Introduction and Objectives BAL causes pulmonary inflammation post procedure. It is not known if the inflammation affects the upper respiratory tract and nasal mucosa. We have developed an experimental human pneumococcal carriage (EHPC) platform. We wished to establish if prior bronchoscopy was associated with altered carriage rates in EHPC.

Methods Participants were screened for natural carriage of pneumococcus by nasal wash. Group A then proceeded to inoculation 7 days after initial screening whereas Group B underwent bronchoscopy with BAL prior to inoculation. Bronchoscopy with BAL was performed using fibre optic bronchoscope and instillation of 200 ml 0.9% saline in 50 ml aliquots followed by immediate manual aspiration via the working port of the bronchoscope. Participants were inoculated with 6B or 23F S pneumoniae (15 000–60 000 CFU/ml) within 14 days of bronchoscopy. Carriage was determined by the presence of pneumococci in nasal wash samples at 48 hr and/or 7 days post inoculation.

Results Thirty-seven participants were recruited, of which 19 proceeded to BAL prior to inoculation; 22 were inoculated with 6B and 15 with 23F. Baseline characteristics were not significantly different between Group A and B. Neither group had any symptoms at the time of inoculation. Both Group A and B were subdivided into 23F or those that received 6B. The inoculum dose was not significantly different between the BAL groups for either 23F or 6B. The mean length of time between bronchoscopy and inoculation was 10 days (±1). Carriage rates between Group A 6B and Group B 6F were significantly different (p=0.008); this difference was not seen between Group A 23F and Group B 23F. In adults challenged with SPN, carriage rates differ by type. In an experiment with high carriage rates, there was a significant decrease in carriage rates in subjects with preceding BAL.

Conclusions This study suggests that the inflammatory process caused by bronchoscopy with BAL, as highlighted in previous research, may influence innate mucosal defence. The inflammatory effect of bronchoscopy with BAL should be accounted for in future research allowing adequate time before performing interventions which may be affected.

**P180** ELECTROMAGNETIC NAVIGATION BRONCHOSCOPY AS A DIAGNOSTIC METHOD IN RESPIRATORY MEDICINE: EARLY CLINICAL EXPERIENCES

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Introduction and Objectives Electromagnetic navigation bronchoscopy (ENB) is approved for use as an adjunct to conventional bronchoscopy, aiding the diagnosis of peripheral lung lesions. It is a modern technique which improves bronchoscopic yield, thereby potentially preventing unnecessary operations or high-risk procedures. Our objective was to assess the use of this technique in regular clinical practice, and to identify factors which may influence its success.

Methods A retrospective data analysis of all ENB procedures carried out in a 120-bed speciality respiratory hospital in Solingen, Germany, between 2007 and 2011 revealed a total of 43 procedures. In each case, size and anatomical location of the tumour based on CT findings were noted. A positive result was documented if as a result of the procedure a clinical diagnosis could be reached.

Results ENB reached a clinical diagnosis in 15 of 45 patients (34.9%); eight malignant tumours, seven benign lesions, 28 left unclear. Of these 28, further investigations revealed a malignant process in nine...
P179 The changing numbers and indications of mediastinoscopy procedures performed following the introduction of endobronchial ultrasound at a UK tertiary centre
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However, a potential important confounding factor may explain a part of their results: undiagnosed pulmonary embolism (PE), mimicking (or induced by) COPD exacerbation. Troponin and BNP are factors associated with poor prognosis in PE. COPD is associated with an increased risk of deep venous thrombosis and PE (particularly during exacerbation) and with an increased risk of fatal PE. In particular, COPD is associated with increased risk of death from undiagnosed PE.

The real incidence of PE during exacerbation of COPD is not clearly known, ranging from 1.5% to 24.7% corresponding to the incidence of elevated troponin and BNP, as noted by Chang et al in their cohort. Therefore, it would be of great interest if Chang et al could provide us some precise answers:

- In how many of the 250 patients a PE has been evoked and/or eliminated?
- How many patients were under efficient anticoagulant drugs at inclusion?
- How many patients received thromboprophylaxis, as a significant number of patients included presented other PE risk factors such as malignancy or cerebrovascular diseases?

Because of the reasons of undiagnosed diagnosis of COPD patients with PE, and of the availability of preventive and curative specific drugs, COPD patients admitted with exacerbation and with abnormal cardiac biomarkers may require a PE screening and effective thromboprophylaxis if PE has been ruled out.

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Competing interests None.

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Authors’ response
We thank Bertoletti and colleagues for raising the important issue of pulmonary embolism (PE) in the exacerbation of chronic obstructive pulmonary disease (COPD). Although we did not routinely investigate for PE in our cohort, we excluded any patients with suspected or confirmed PE from the study. Unfortunately, it is difficult to detect thromboembolic events in this population and it is possible that we included some patients with subclinical pulmonary emboli. It is also plausible that this contributed to the association between elevated cardiac biomarkers and mortality. However, we think that this is unlikely to be the only mechanism.

Thromboprophylaxis was administered to some patients during their admission depending on their immobility and other risk factors, but this would not have influenced the NT-proBNP or troponin T results obtained on presentation. We did not collect information on pre-existing anticoagulation therapy on admission to the study.

Further research into the mechanism linking elevated cardiac biomarkers and mortality in COPD exacerbation is needed. We agree with Bertoletti and colleagues that investigating the contribution of concurrent PE is important, as this is something that can be treated.

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Thorax 2011;66:A128–A129 doi:10.1136/thoraxjnl-2011-200416c.151. P151 Cost of pulmonary rehabilitation is offset by reduction in healthcare utilisation. The author list and author affiliations for this poster should read: S Kibe, D Ford, S Hart. 1 Scarborough General Hospital, Scarborough, UK; 2 Castle Hill Hospital, Hull, UK.

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Thorax 2011;66;A133–A134 doi:10.1136/thoraxjnl-2011-200416c.165. P165 Factors influencing histological confirmation of diagnosis in lung cancer patients. The author list and author affiliations for this poster should read: S Chandramouli, M Cheema, J Corless. Wirral Lung Unit, Arrowe Park Hospital, Wirral CH49 5E, UK.

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