patient-specific protocols (PSP) to instruct ambulance staff about specific O_2 requirements for known chronic T2RF patients during acute ambulance transfer to hospital and studied their use and effect on length of stay (LOS).

Method: Patients with chronic T2RF, or acute T2RF when unwell, were given a PSP documenting FiO_2 recommended to maintain SaO_2 88–92%, with instructions if these parameters could not be achieved. PSP were agreed and co-signed by two respiratory consultants and the LAS medical director. Copies were held in notes, by patients and "flagged" by LAS to alert ambulance staff on call-outs. Ambulance FiO_2 and oximetry, initial arterial blood gases, admissions and LOS were reviewed retrospectively for 12 months before and after PSP issue.

Results: 20 patients (six men; 14 women; 19 COPD, one nocturnal hypoventilation; 13 (65%) on LTOT; mean ± SE age 71.9 ± 2.1 years, FEV_1 0.68 ± 0.06l, MRC dyspnoea score 4.6 ± 0.1) received PSP (April–September 2006). They had 60 admissions (3.2 ± 0.4/patient, mean ± SE) in the year before PSP and 67 admissions (3.4 ± 0.4/patient) in the year after. Before PSP, ambulance FiO_2 was >28% in 26% of transfers (10/39), but after PSP, occurred in 13% transfers (8/61, p = 0.06). Inappropriately high ambulance FiO_2 resulting in SaO_2 >92% occurred in 64% of transfers (16/25 transfers; data not recorded in 14) before PSP, but occurred significantly (p<0.001) less frequently after PSP (12/55 transfers (12.8%); data not recorded in four). There was no significant decrease in episodes of acute-on-chronic T2RF (5/41 (12%) before; 7/51 (14%) after PSP) or total LOS (mean ± SE 35.2 ± 7.1 days before; 35.3 ± 5.2 days after). Bicarbonate increased from mean ± SE 31.9 ± 0.7 mmol/l before PSP to 34.1 ± 1.0 mmol/l in the year after. 11/20 (55%) of the patients died during the year following the study, none related to acute hypoxia.

Conclusion: Patients with chronic T2RF are at high risk of acute T2RF and death. Whereas PSP were effective in reducing inappropriately high FiO_2 during ambulance transfer in susceptible patients with chronic T2RF, they had no effect on LOS, episodes of acute T2RF or serum bicarbonate, all of which reflect disease severity.

Clinical investigation of interstitial lung disease

P145 NOCTURNAL OXYGEN DESATURATION IS COMMON IN INTERSTITIAL LUNG DISEASE AND OCCURS IN PATIENTS WITHOUT RESTING OR EXERCISE-INDUCED HYPOXIA

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In patients with interstitial lung disease (ILD), nocturnal oxygen desaturation is associated with poorer quality of life and may contribute to long-term vascular stress. Nocturnal desaturation is thought to be common in ILD patients, with previous studies reporting its prevalence at 50–88%.

Aim: To determine the prevalence of nocturnal desaturation in ILD patients and in the subgroup of patients without resting or exercise-induced hypoxia.

Methods: We reviewed all ILD patients (n = 176, mean age 57.8 ± 13 years, 97 male) who had undergone overnight oximetry during 2005–8. All patients had pulmonary function and resting oxygen saturation (SpO_2), and 125 had 6-minute walk testing (6MWT). Significant nocturnal desaturation was considered as spending ⩾10% of sleep with SpO_2 ⩽90%. We determine the prevalence of nocturnal desaturation and its frequency in those without resting or exercise hypoxia.

Results: On overnight oximetry, 128 (73%) patients had oxygen desaturation to ⩽90% at any stage of the night. However, 73 (42%) had significant nocturnal desaturation (⩾10% of sleep with SpO_2 ⩽90%). Mean minimum SpO_2 was 84.0 ± 8.6% and fall in SpO_2 was 11.4 ± 8.4%. Patients spent an average of 18.1 ± 26.9% of the night below 90%. On 6MWT, mean end SpO_2 was 87.9 ± 7.5% and 6MWT distance was 325.4 ± 124.8 m. Sixty-two (50%) had oxygen desaturation to ⩽88% during 6MWT. Patients had a mean body mass index of 28.4 ± 6.7 kg/m^2; TLco% 37.3 ± 16.6%; FVC% 66.3 ± 23.4% and SpO_2 95.4 ± 2.5%. As demonstrated in the table, nocturnal desaturation was present in 49 of 149 (33%) patients without resting hypoxia and in 37 of 63 (59%) patients without desaturation on 6MWT.

Conclusion: Nocturnal desaturation is frequent in ILD patients and is not uncommon in patients without resting or exercise-induced hypoxia. These results suggest that overnight oximetry is necessary to exclude significant nocturnal desaturation in ILD patients.

P146 NOCTURNAL DESATURATION IS ASSOCIATED WITH PULMONARY HYPERTENSION IN PATIENTS WITH MILD-TO-MODERATE INTERSTITIAL LUNG DISEASE

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Nocturnal desaturation is common in interstitial lung disease (ILD). In severe ILD, nocturnal desaturation can be attributed to the underlying disease. However, in less severe disease, nocturnal desaturation may be a marker for the presence of pulmonary arterial hypertension (PAH). It is possible that repetitive nocturnal hypoxia contributes to vascular stress and subsequent PAH.

Aim: We study the association between nocturnal oxygen desaturation and disproportionate PAH in ILD patients.

Methods: We reviewed 102 ILD patients (mean age 57.5 ± 12.7 years; 54 male) with overnight oximetry, pulmonary function and echocardiography during 2005–8. In order to study disproportionate PAH, we excluded patients with severe disease (TLco ⩽30%), in which PAH is not unexpected. We considered significant nocturnal desaturation as having SpO_2 ⩽90% for ⩾10% of sleep. Evidence of PAH on echocardiography was considered as systolic pulmonary arterial pressure (sPAP) ⩾40 mm Hg and/or right ventricular dilatation or functional impairment. The association between nocturnal desaturation and markers of pulmonary vascular impairment was assessed.

Results: Baseline parameters: On overnight oximetry, 41 (40%) had significant nocturnal desaturation. Mean minimum SpO_2 was 85.2 ± 7.3%, and fall in SpO_2 was 10.1 ± 6.9%. Patients spent 17.6 ± 26.3% of the night ⩽90%. Patients had a mean body mass index of 29.0 ± 6.1 kg/m^2; 6-minute walk test (6MWT) end SpO_2 90.7 ± 5.6%; 6MWT distance 345.9 ± 111.4 m TLco% 46.9 ± 13.8%; FVC% 73.8 ± 23.3%; composite physiological index (CPI) 44.9 ± 13.9 and SpO_2 94.9 ± 2.6%. Analysis: Patients with

Abstract P145 Table Prevalence of significant nocturnal desaturation (SpO_2 ⩽90% for ⩾10% sleep) in patients with and without resting and exercise-induced hypoxia

		Daytime SpO_2 <93% (n = 27)	6MWT end SpO_2 >88% (n = 63)	6MWT end SpO_2 ⩽88% (n = 62)
No significant nocturnal desaturation (n = 103)	100	3	37	34
Significant nocturnal desaturation (n = 73)	49	24	26	28

6MWT, 6-minute walk test.

significant nocturnal desaturation were more likely to have PAH on echocardiography (odds ratio (OR) 2.82, 95% CI 1.02 to 7.74, $p<0.05$) independent of disease severity as determined by CPI (OR 1.06, CI 1.01 to 1.11, $p = 0.01$). Patients with significant nocturnal desaturation had higher sPAP ($p = 0.03$), lower pulmonary acceleration time and lower PaO_2 ($p = 0.02$). Pulmonary function (including TLco%, FVC% and CPI) did not differ in patients with or without nocturnal desaturation.

Conclusion: In mild-to-moderate ILD, patients with significant nocturnal desaturation are more likely to have PAH, independent of the severity of their underlying disease. It remains unclear, however, whether nocturnal desaturation is implicated in the pathogenesis of PAH.

P147 ATYPICAL MYCOBACTERIA IN SARCOIDOSIS BRONCHOALVEOLAR LAVAGE CELL PELLETS

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Introduction: Sarcoidosis is a systemic granulomatous disease of unknown aetiology. Previous molecular studies on biological material using mainly polymerase chain reaction (PCR) technology have produced conflicting results. Of the positive studies, the presence of either mycobacterial or *Propionibacterium acnes* (PA) DNA, both recognised as part of the phylum *Actinobacteria* (ACT), were more commonly detected. However, there is little or no information on the background global bacterial profile in these patients

Methods: Bronchoalveolar lavage cell pellet DNA was extracted from 15 patients with sarcoidosis (radiographic stages 1–4). Ten patients with systemic sclerosis and 10 patients with idiopathic pulmonary fibrosis served as disease controls. We used PCR targeting the majority of species in the *Actinobacteria* phylum, followed by sequencing to assess the type of ACT DNA when present. We also used 16S terminal restriction fragment length polymorphism (T-RFLP) to profile the background microbial community.

Results: We amplified ACT DNA and obtained high quality score sequencing results in 87% (13/15) of sarcoidosis patients and 80% of the control patients. NCBI blast sequence analysis of the sequences revealed the presence of DNA from different non-tuberculosis mycobacteria (NTM) only in sarcoidosis specimens (7/13, 54%). Of these, 6/7 (85%) had radiological stages 0/I, and the other had radiological stage II. PA DNA was isolated in all three disease groups. A wide diversity in pattern profile, numbers and relative proportions was observed by T-RFLP analysis in the three disease groups.

Conclusion: We observed the presence of NTM DNA only in patients with sarcoidosis, whereas the presence of PA DNA was not specific to sarcoidosis patients. Our results are compatible with the hypothesis of a mycobacterial cause for at least one form of sarcoidosis and the diversity of detected NTM suggest that more than one pathogenic agent may be implicated.

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P148 SURGICAL BIOPSY IN THE INVESTIGATION OF INTERSTITIAL LUNG DISEASE: THE LARGEST SERIES TO DATE

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Introduction: Although surgical lung biopsy is the gold standard investigation for the diagnosis of diffuse interstitial lung disease (ILD), it is not without risk and previous small series have suggested a mortality of up to 20%. To study this further, we looked at the outcome in all such cases undergoing lung biopsy referred to a large thoracic surgical unit serving a population of 2.5 million.

Methods: We interrogated the comprehensive thoracic surgery database between April 2001 and April 2008 for all 224 patients (mean age 54.6 years (SD 13.7), mean FEV_1 2.01 (0.85), mean FVC 2.54 (1.12), mean FEV_1/FVC 75% (21.9), median NYHA dyspnoea score 2, 94 (42%) lifelong non-smokers, 55 (25%) current smokers, 23 (10%) significant asbestos exposure and 112 (50%) male) undergoing lung biopsy for ILD. Preoperative clinical and radiological information was available for 199 patients.

Results: 185 cases were carried out via video-assisted thoracoscopic surgery and 22 converted to open biopsy. Average length of stay was 3.6 days (SD 3.59). Only 25 patients had complications and there were two deaths. Satisfactory tissue was obtained in all patients (see table), providing a primary diagnosis in 121 (54%), confirming a suspected diagnosis in 26 (16%) and a different diagnosis in 60 (27%). In particular, suspected malignancy was refuted in 13, confirmed in five and unexpectedly diagnosed in five cases.

Discussion: Surgical lung biopsy is extremely effective and safe in the investigation of ILD, and should be considered routinely when simple tests have failed to yield a diagnosis. Furthermore, the results may alter management in a high proportion of patients. However, the low complication rate should be interpreted with caution as it may be the result of careful patient selection.

Abstract P148 Table

Diagnosis	No of cases (%)
Usual interstitial pneumonitis/cryptogenic fibrosing alveolitis	78 (34)
Non-specific inflammation	25 (11)
Organising pneumonia	22 (10)
Sarcoidosis	19 (8.4)
Carcinoma	14 (6.3)
RB-ILD	12 (5.4)
DIP	10 (4.5)
Hypersensitivity pneumonitis	9 (4)
LAM/histiocytosis X	8 (3.6)
Vasculitis/connective tissue disease	5 (2.2)
Emphysema	4 (1.7)
Tuberculosis	2 (0.9)
Other	16 (7.1)

P149 RELATIONSHIP BETWEEN ECHO-ESTIMATED PULMONARY ARTERY SYSTOLIC PRESSURE, LUNG FIBROSIS AND PARAMETERS OF GAS DIFFUSION IN PATIENTS WITH PULMONARY SARCOIDOSIS

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Introduction: Sarcoidosis, with its pathological hallmark of granulomatous inflammation, has a predilection for the lungs and the pulmonary vasculature. Pulmonary hypertension (PH) affects 6–10% of general sarcoidosis cohorts, an incidence that rises with worsening lung disease. A number of pathogenetic mechanisms for PH have been proposed, from large vessel obstruction to granulomatous obliteration of the interstitial circulation, microvascular injury and cardiac sarcoidosis. We sought to describe the frequency of PH and its relationship to lung function impairment and disease phenotype in this disease.

Methods: Demographic, radiological and physiological (pulmonary function, echocardiography and right heart catheter) datasets were constructed for comparison. High-resolution computed tomography

(HRCT) allowed the pulmonary manifestations to be categorised into fibrotic and non-fibrotic subtypes. Resting lung diffusion capacity for carbon monoxide (D_{LCO}) and its derived transfer coefficient (K_{CO}) were obtained. Pulmonary artery systolic pressure (PASP) was estimated by transthoracic echocardiography (TTE) using the Bernoulli equation. When possible, TTE and lung function measurements were taken within 3 months of each other.

Results: 32 patients (21 women, median age 59 years; 11 men, median age 54 years) were identified. 11/32 (34%) had HRCT features of fibrotic lung architectural distortion. Among patients with detectable tricuspid regurgitation (18/32, 56%), PASP was measured in 16/18 (89%). Overall, 6/18 patients with TR (33%) had evidence of PH on echo as defined by PASP >40 mm Hg. Within this subgroup, parenchymal fibrosis was evident in a third of cases. In patients in whom both PASP and D_{LCO} were measured (n = 14), the coefficient of determination (r^2) between the two indices was −0.11. The inverse correlation between PASP and K_{CO} produced $r^2 = 0.28$. Median values for PASP and percentage predicted $D_{LCO}c$ and $K_{CO}c$ were 27 mm Hg, 58% and 91%, respectively.

Conclusion: In this preliminary analysis of patients with pulmonary sarcoidosis characterised in anatomical and physiological terms, PASP correlated poorly with indices of gas transfer. The weak association between gas diffusion measurements and echo-estimated pulmonary arterial pressure suggests that complex interactions govern the process of granulomatous lung damage and vascular remodelling. Specific mechanisms of sarcoid vascular pathogenesis await detailed characterisation.

P150 NON-INVASIVE ASSESSMENT OF CARDIAC OUTPUT USING AN INERT GAS REBREATHING DEVICE IN PATIENTS WITH FIBROTIC LUNG DISEASE

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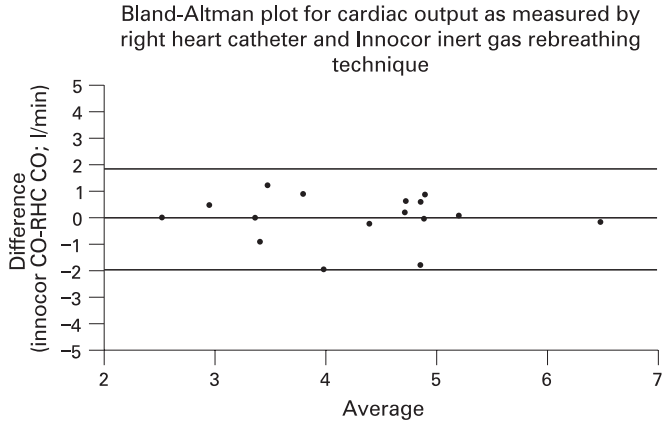
Background: Pulmonary arterial hypertension (PAH) is increasingly recognised in patients with diffuse fibrotic lung disease and is associated with increased mortality. Cardiac output (CO) is an important prognostic marker in PAH. Non-invasive CO assessment with inert gas rebreathing methods have been validated in patients with PAH,[1 2] but not in PAH associated with parenchymal lung disease. Inert gas rebreathing measures of CO may be less accurate in patients with lung disease because of intrapulmonary shunting and/or incomplete gas mixing.

Aim: This study aims to determine the accuracy of an inert gas rebreathing measure of CO in patients with diffuse fibrotic lung disease.

Methods: Twenty consecutive patients (10 men, mean age 59 ± 12 years) with fibrotic lung disease referred for right heart catheterisation (RHC) underwent non-invasive CO assessment. Fick's method was used to calculate CO_{FICK} at RHC. $CO_{INNOCOR}$ was measured within 24 h of RHC with an inert gas rebreathing technique (nitrous oxide and sulphur hexafluoride) with rapid photoacoustic spectroscopic analysis (Innocor; Innovision, Denmark). The relationship between invasive and non-invasive measures of CO was assessed by correlation and Bland–Altman analysis.



Abstract P150 Figure

Results: 13 of 20 (65%) had PAH on RHC: mean pulmonary artery pressure 27.2 ± 10.0 mm Hg; pulmonary vascular resistance 5.2 ± 3.5 Wood's units. Patients had fibrotic lung disease, including idiopathic pulmonary fibrosis (n = 8), non-specific interstitial pneumonia (n = 8), chronic hypersensitivity pneumonia (n = 2) and other (n = 2). Baseline parameters included: mean TLco 24.8 ± 8.4%, FVC 67.6 ± 22.1%, right ventricular systolic pressure 56.7 ± 19.4 mm Hg and BNP 8 (5–234) pmol/l. There was no evidence of systematic bias, as judged by Student's paired t test: mean CO_{FICK} 4.1 ± 1.1, mean $CO_{INNOCOR}$ 4.0 ± 1.3 l/min (p = 0.61). There was a strong positive correlation between CO_{FICK} and $CO_{INNOCOR}$ (R = 0.74, p = 0.001). Bland–Altman analysis revealed a mean difference of 0.11 ± 0.89 l/min (limits of agreement −1.69 to +1.91) (see fig).

Conclusion: In patients with diffuse fibrotic lung disease, cardiac output can be accurately and non-invasively measured by the Innocor inert gas rebreathing device. This may be important in the non-invasive assessment of PAH in these patients.

1. **Hoeper MM,** *et al*. *AJRCCM* 1999;**160**:535–41.
2. **Mclure LER,** *et al*. *Thorax* 2007;**62**:A17[abstract]

P151 DOES ASBESTOS EXPOSURE EXPLAIN THE EXCESS OF MEN WITH DIFFUSE PULMONARY FIBROSIS

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Introduction: Diffuse pulmonary fibrosis (DPF) is associated with a relatively poor prognosis and median survival of only 2–3 years. Over 50% of cases remain idiopathic. DPF has a male predominance with a 3M : 2F prevalence ratio, suggesting genetic or environmental factors may play an important role. Occupational history often distinguishes men from women; particularly occupational exposure to asbestos. We currently recognise asbestos as a cause of DPF in the context of a heavy exposure history.

Aim: To assess whether asbestos exposure of any degree may account for the excess number of male cases of DPF.

Methods: We surveyed a population of 421 patients with interstitial lung disease (ILD) presenting consecutively over 12 years to one consultant within our department. 92% of relevant records were available. 253 cases of ILD with specific characteristics such as sarcoidosis, extrinsic allergic alveolitis, drug reactions, collagen vascular disease and other rare entities were excluded. We examined the records of all remaining 168 patients with DPF (comprising idiopathic pulmonary fibrosis, idiopathic interstitial pneumonias and fibrosis provisionally attributed to chronic aspiration or a pneumoconiosis). Asbestos exposure was graded as trivial (no recognised exposure or minor background exposure) or appreciable (definite exposure history, regardless of extent).

Results: There were 120 men and 48 women (5M : 2F) with DPF. 75 men and three women (25M : 1F) had DPF associated with an appreciable history of asbestos exposure. 45 men and 45 women (1M : 1F) had DPF and only trivial asbestos exposure.

Discussion: The excess number of men with DPF in this survey is matched by the excess number of men with a history of asbestos exposure judged to be more than trivial. If this link is causal, the excess of DPF affecting men may be attributed to asbestosis. This is against general consensus that a diagnosis of asbestosis can only be made with a history of substantial exposure.

Conclusion: We hypothesise that asbestos exposure is causally linked to many more cases of DPF than is currently accepted.

Chronic obstructive pulmonary disease: airways and systemic features

P152 SPUTUM IL-5 CONCENTRATION IS ASSOCIATED WITH A SPUTUM EOSINOPHILIA AND ATTENUATED BY CORTICOSTEROID THERAPY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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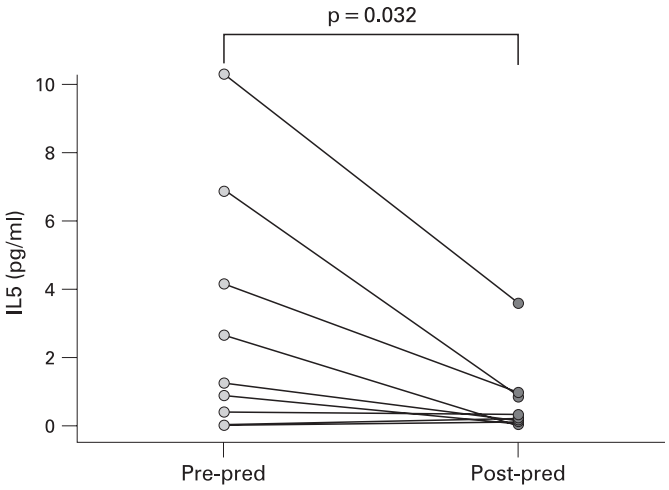
Background: Airway inflammation in chronic obstructive pulmonary disease (COPD) is predominantly neutrophilic, but some subjects demonstrate eosinophilic airway inflammation. Whether these inflammatory phenotypes have differential cytokine and chemokine expression is unknown.
Aims: To assess the sputum concentrations of cytokines and chemokines and their response to oral corticosteroid therapy in COPD subjects with or without a sputum eosinophilia.
Methods: Cytokine and chemokine concentrations were measured using the mesoscale device platform. To assess validity recovery of exogenous spikes was examined. The concentrations of the validated mediators were measured in COPD sputum from subjects with or without a sputum eosinophilia. In a subgroup with a sputum eosinophilia the response to prednisolone 10 mg for 1 month was examined.
Results: The recovery in sputum of exogenous spiked mediators was >80% in 17/26 cytokines and chemokines. In supernatants from eosinophilic (n = 39) versus non-eosinophilic (n = 59) sputa the geometric mean (95% CI) concentration was increased for IL-5 (9.0, 4.5 to 18 pg/ml vs 3.6, 2.7 to 6.3 pg/ml; p = 0.03) and CCL26 (23.4, 10.8 to 19.8 pg/ml vs 9.0, 5.4 to 16.2 pg/ml; p = 0.04). IL-5 correlated with sputum eosinophil counts (R^2 = 0.11, p = 0.001) and was attenuated following treatment with prednisolone (n = 9; mean difference 2.3 pg/ml (95% CI 0.2 to 4.3; p = 0.03).
Conclusion: We have validated the use of the mesoscale device platform for cytokine and chemokine measurements in the sputum supernatants in COPD. Sputum IL-5 was associated with a sputum eosinophilia. Whether this cytokine is important in the pathogenesis of COPD in a subgroup of patients warrants further investigation.



Abstract P152 Figure IL-5 attenuation following corticosteroid administration.

P153 CAN SYSTEMIC MARKERS OF INFLAMMATION HELP PREDICT MORTALITY AT 1 YEAR IN PATIENTS HOSPITALISED WITH EXACERBATIONS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE?

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Mortality in the months after hospital admission for a chronic obstructive pulmonary disease (COPD) exacerbation is high. Many clinical and physiological markers such as FEV_1 and body mass index (BMI) are thought to predict the risk of dying after a COPD exacerbation. There are few data about potential COPD biomarkers in patients admitted to hospital. We hypothesised that markers of systemic inflammation measured at discharge would relate to the subsequent risk of death in the year post-hospitalisation.

We recruited patients who survived to hospital discharge after a COPD exacerbation to a case management study. As part of this study, blood samples were taken on the day the patients were discharged from hospital or the hospital at home service. Standard C-reactive protein (CRP), high sensitivity CRP (HSCRP), TNFα and IL-6 were measured as well as haemoglobin and creatinine.

107 patients were recruited, 55 female. Mean (SD) age 71 years (8), FEV_1 40% (16) predicted, BMI 24.0 (5.3) and inspiratory capacity median (interquartile range (IQR)) 53% (66–78) predicted. 18/107 (17%) of patients did not survive to 12 months.

Median (IQR) CRP was 4 mg/l (4–18), HSCRP 5.1 mg/l (1.8–17.6), 75 (70%) had CRP <10 mg/l, TNFα <1 pg/ml (<1, <1), IL-6 3.1 pg/ml (1.6–6.9), creatinine 85 mmol/l (73–97) and haemoglobin mean (SD) 13.3 g/dl (1.6).

FEV_1 (litres) died versus survived, respectively, 0.78 l (0.33) versus 0.97 l (0.33), p = 0.02; IC 57% (23) versus 67% (24), p = 0.01; body mass index 21.5 (5.1) versus 24.5 (5.3), p = 0.03, were all predictive of mortality at one year. Only creatinine was significantly associated with mortality, (died 77.5 (71–85) vs survived 87 (73.5–100), p = 0.04). There were no significant differences between the other biomarkers in relation to mortality.

There are still no clinically useful biomarkers that predict death following AECOPD. The observed difference in creatinine reflects low muscle mass, which is known to be associated with adverse outcome. In the clinical setting, this would probably only be useful in the context of very low levels.

P154 INFLAMMATION AND CENTRAL OBESITY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH AND WITHOUT METABOLIC SYNDROME

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Background: Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory disease of the airways that is characterised by partly reversible airways obstruction, which is predominantly caused by cigarette smoking.[1] It is known that excess body weight is associated with abnormal metabolic and inflammatory profiles that define the metabolic syndrome (MeS), which predisposes to cardiovascular disease.[2] The role of inflammation on features of MeS (central obesity) in smokers with and without COPD is less understood. The aim of this study is to understand the association of inflammation and central obesity in COPD.
Methods: 44 healthy controls (HC), 46 smokers without COPD (HS) and 41 with COPD were recruited for this study. They were matched for age and sex. Subjects were categorised according to the GOLD criteria for diagnosing COPD and International Diabetic Federation (IDF) criteria for defining MeS. All subjects underwent pulmonary function testing. Serum C-reactive protein (CRP) was measured. SPSS 14 was used as the statistical tool for the analysis.