and metabolic functions, and circulating inflammatory markers might help to better characterise these patients.

Diagnosis of COPD

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The diagnosis of COPD relies on the demonstration of fixed airflow limitation in the absence of less frequent diseases causing airways obstruction. Guidelines have for decades used the ratio of FEV_1/VC or FEV_1/FVC as the measurements of choice; this has rarely been the cause of debate whereas the cut-off values for the ratio have been intensely debated. The fixed ratio of 0.7 causes under-diagnosis of COPD in younger adults and over-diagnosis in the elderly whereas the use of lower limit of normal presumes that less than 5% of elderly asymptomatic smokers can have COPD as 'normality' is defined according to statistics only.

More importantly, however, the current diagnostic guidance does not take into account that we define COPD as 1) a disease characterised by an abnormal inflammatory response, 2) a disease with a frequently occurring extrapulmonary component – and 3) that we know that one of the main subtypes of COPD, emphysema, often does not lead to airflow limitation in its earliest stages. We will need to decide whether we want to keep our simple diagnostic criteria and subsequently try to describe the individual patient's features by "sub-grouping" COPD or whether our diagnostic criteria should mirror how we define the disease.

Disease modification in COPD — impacting the clinical course

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The most recent definition of COPD clearly states that the disease is both preventable and treatable. The knowledge on interventions that may improve the clinical course of COPD is constantly expanding and the COPD patients of today are facing a totally different approach from the health professionals compared to the situation 20 years ago. This means that the clinical course of the disease need no longer result in a continuing decline of lung function, repeated exacerbations and development of respiratory failure. Whereas smoking cessation in the early stage of COPD and long term oxygen treatment in the end-stage disease were in the past thought to be the only two modalities affecting the long term clinical course of the disease, it is now documented that also pharmacological treatment with long acting antimuscarinic agents, long acting beta-2-agonists and inhaled corticosteroids, rehabilitation comprising physical exercise and immunisation against influenza affects the long term prognosis and improves the health related quality of life of the patients. Also the treatment of acute exacerbations with non-invasive ventilation has revolutionised the clinical practice by reducing both mortality and the need for intubation.

In order to implement these options with success, the timing of intervention is essential. In this context the more widespread use of spirometry, assessment of the severity of dyspnea and identification of patients with repeated exacerbation is mandatory. The pulmonary community has a major obligation in assuring that the knowledge from the successful controlled trials will be implemented in the clinical practice.

ABSTRACTS - OTHER LECTURERS

Exhaled NO and systemic inflammation biomarkers in COPD - a longitudinal study

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The usefulness of exhaled nitric oxide (eNO) measurement is still controversial in chronic obstructive pulmonary disease (COPD). However, modelling measurements of eNO from multiple flows may provide measures of parenchymal and airway inflammation and could potentially be used to monitor inflammation in COPD. In my PhD study I will investigate any possible correlation between local inflammation and systemic inflammation in COPD patients and relate it to presence/development of cardiac disease.

Ninety one (91) COPD patients recruited in the ECLIPSE study with FEV_1 ranging from 17-77% have been seen every three months in the first year and every six months in the following eighteen month period. I aim to use two compartment nonlinear modelling of multiple flow rates eNO at 10, 30, 50, 100 and 200 $mL \cdot s^{-1}$, but currently only raw eNOs are available and eNO_{50} is used to indicate airway inflammation. The following biochemical markers of inflammation in serum are used: IL-6, IL-8, TNF_α, Clara cell secretory protein-16 (CC-16), and Surfactant Protein D (SPD).

Neither eNO_{50} nor any of the systemic markers varied according to age, gender or GOLD stage except IL6 which increased by age. There was no correlation between eNO_{50} and any of the systemic markers. There was an association between TNF_α and PARC ($r=.26$, $p=.01$) but no other inter-relations between systemic markers. IL-6 was significantly elevated in subjects with ischemic heart disease (7.27 vs 2.13 pg/mL, $p<.001$) as was CC-16 (6.17 vs. 4.86 ng/mL, $p=.03$), none of the others markers were related to heart disease at baseline.