Highlights from this issue

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Hot topic: 'always, all I need is the air that I breathe to be healthy'

Pollution is clearly not a good thing under any circumstances, and if you have lung disease it is even worse. We have come a long way from the 'London Particular' of *Bleak House*, but there is a long way to go yet. Bronchiolitis Obliterans Syndrome (BOS) is the scourge of lung transplant recipients, and the commonest cause of late mortality. In this issue of the *Journal*, the Leuven lung transplant group modelled exposure to air traffic pollution and found that living near to a major road was an independent risk factor for BOS and death after transplantation, and also associated with greater BAL neutrophilia and IL-6 and plasma CRP levels. As was also discussed in the accompanying editorial, this report highlights two major issues. First, we must redouble our efforts to reduce traffic pollution. But perhaps more importantly, these data lead to the intriguing speculation that the initial irritant stimulus is from pollutants which trigger neutrophilic airway inflammation, which becomes self-perpetuating and leads to BOS. At the very least, particulate pollution may act in tandem with other stimuli to perpetuate the neutrophilic inflammation of BOS. And could other factors triggering airway neutrophilia, such as cigarette smoke, or reflux and aspiration, have a similar effect on BOS? Finally, maybe other neutrophilic airway diseases are exacerbated by particulate pollution by similar mechanisms. This provocative manuscript is certainly a hot topic. ***See pages 748 and 742.***

VOCs Pop: another way of phenotyping asthma?

'Asthma' is probably about as useful a pathophysiological label as 'chronic renal failure', both likely representing the stereotypical response of the respective organ to a variety of insults. Phenotyping asthma is an academically satisfying activity, and increasingly being used clinically. The lesson of the anti-IL5 studies is clearly that treating all severe asthmatics irrespective of the nature of their disease with an expensive and potentially toxic monoclonal is neither useful nor safe. In this issue, the role of exhaled breath volatile organic compounds (VOCs) in the diagnosis and phenotyping of less severely affected asthmatics has been explored. The group was able to distinguish asthmatics from normals, and divide the asthmatics into eosinophilic and neutrophilic sputum phenotypes, and also pick out those with uncontrolled asthma. Exhaled breath is easier to obtain than induced sputum (only just over half the asthmatics could produce a sputum sample in this study), and may be particularly attractive in young children or the very elderly. Those VOCs that were discriminatory may open novel pathophysiological avenues which would repay exploration. Clinically the next step is to use the technique diagnostically in all comers referred to the chest clinic, and to demonstrate a real life therapeutic benefit from this sort of categorisation. Crucial will be proper standardisation—we do not want to reproduce the disappointment of failing to replicate 'asthma genes' with failure to replicate VOCs and everyone having their own private, non-overlapping dataset. ***See page 804.***

Cardiac morbidity and COPD lung attacks

I'm sure this editor is not alone in encountering commonly the unexpected finding of an elevated cardiac troponin T in patients presenting with COPD lung attacks. Rather than muttering under our breath "ask a stupid question, get a stupid answer" perhaps we should take careful note of this finding. Hoiseth *et al* (***see page 775***) and Chang *et al* (***see page 764***) confirm that this finding is common and show, convincingly, that it identifies patients with a poor prognosis. Maclay et al (***see page 769***) add to this evidence by showing that platelet activation is present in patients with COPD, particularly when studied during a lung attack. Collectively these studies provide strong support for the view that COPD lung attacks are a high risk period for cardiac morbidity and mortality. Leo Fabbri and colleagues (***see page 745***) rightly emphasise the importance of a comprehensive assessment of cardiac status in the management of these high risk patients.

Stop smoking, lose weight (editors' choice)

One of my non-respiratory medical friends maintains uncharitably that I could be replaced in clinic by a tape recorder chanting repeatedly "stop smoking, lose weight". He would be impressed by the findings of Sutherland *et al* reported on (***see page 797***). With the help of the anorexic agent Sibutramine, 54 obese subjects with moderate-to-severe obstructive sleep apnoea (OSA) lost an average of 7.8 kg over 6-months. Weight loss was associated with an improvement in the apnoea-hypopnoea index which correlated most closely with a reduction in upper airway length assessed using CT. The authors have previously shown that increased buccal fat (or chubby cheeks to use a technical term) predicts OSA more closely than other anthropometric and craniofacial risk factors but could not confirm that less chubby cheeks were independently linked to improvement in OSA. Indeed relatively little of the inter-individual variability in the effects of weight loss on OSA could be explained by the measures assessed, suggesting the need for more studies. Nevertheless, my friend suggests I turn the tape recorder volume up.

Hello, hello, hello! what's going on AIR then?

No smoke or mirrors: this 37-year-old man was breathless (unsurprisingly) but actually breathing spontaneously—*with what*? **See *Images in Thorax*, *see page 840.***