Highlights from this issue

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MAYBE BILL CLINTON WAS RIGHT AFTER ALL?

When he said he did not inhale, he may have avoided more trouble than he realised. A unique and aggressive form of interstitial lung disease (ILD) was first described in children; in this issue we publish the results of epidemiological and pathological investigations in adults (***see pages 703, 694*** with an editorial by Tony Pickering ***see page 692***). All three are our Hot Topic package this month. In children (in whom this ILD was first described) and in adults, shrewd detective work enabled humidifier disinfectant to be pinpointed as the cause, and crucially, the John Snow effect (not the ex-England fast bowler!) was seen; as with cholera when the pump handle was removed, when the disinfectant was banned, no new cases arose. The pathology was of a bronchiolocentric acute lung injury, with subpleural and peripheral sparing, and the outlook exceedingly bad for established cases at all ages. Tony Pickering makes the crucial point that we must be alert to chemical exposures in the home as well as at work. Perhaps the lesson of the week–there is no such thing as a free inhalation!

LOOK INTO MY ALVEOLI.

The striking images on the left half of the cover this month show elastic fibre distribution in distal airways imaged *in vivo* using fibred confocal florescence microscopy (FCFM). This technique was investigated by Cosío *et al* in patients with COPD and controls (***see page 724***). FCFM allows distal airways and alveoli to be examined using a 1.4 mm diameter probe inserted through the biopsy channel of a standard bronchoscope. The authors confirmed that alveoli are dilated and contain fewer and thinner alveolar capillaries as well as numerous overfed alveolar macrophages. They also show for the first time that the elastin fibres in distal airways show an abnormal loose distribution in a significant number of patients (see middle left panel on cover). This finding was confirmed by biopsy. Whereas alveolar size related to conventional lung function tests, the FCFM measurements did not, making them even more interesting. Next stop perhaps comparison with multibreath washout techniques, and hyperpolarised helium studies. Will sight of these disrupted airways make people fear smoking more? We offer the usual prize for the most absurd alternative for the acronym FCFM.

LET'S COUGH AGAIN, LIKE WE DID LAST YEAR

Infection control in cystic fibrosis (CF) has become a live and emotive issue, with plenty of heat but light at a premium, and likely will be an increasing issue in primary ciliary dyskinesia and non-CF bronchiectasis as well. Knibbs *et al* have stirred the (sputum?) pot by showing that viable *Pseudomonas aeruginosa* can be detected at least 4 metres away from a coughing CF patient, and viable organisms persist for at least 45 minutes (***see page 740***). So airborne as well as direct contact transmission of *Pseudomonas aeruginosa* is at least possible. Andy Jones, in an accompanying editorial (***see page 690***) highlights the missing link; the answer to the question 'So what?' Do these aerosols actually pose a real threat of infection? As Andy points out, it is not ethically possible to answer the question. So where do we go from here? Clearly, surveillance for cross-infection, and attention to hand washing and other precautions to prevent transmission of infection by direct contact is mandatory. Does this research mean we should leave rooms empty for nearly an hour between patients, ensuring many air changes during that time? We must surely avoid complete clinic paralysis from the introduction of more and more steps to prevent smaller and smaller risks; patients must not be terrified into recreating Marcel Proust's famous cork-lined bedroom and reclusive life style (maybe Airwaves should be re-named Culture Corner?). Perhaps a radical suggestion—treat adults as adults and ask them to cough into a handkerchief rather than mimic the experimental coughing of this manuscript?

NO SUCH THING AS A FREE LUNCH AND NO SUCH THING AS A NORMAL PERSON?

There is a tendency to view lung attacks as an exclusive feature of obstructive and restrictive diseases. However, Tan *et al* (***see page 709***, **Editors' Choice**) show that acute episodes of respiratory morbidity also occur in patients with normal spirometry. Risk factors in this population included use of respiratory medication, female gender, self-reported wheeze and poor general health. The frequency of these events was about half that seen in subjects with spirometric evidence of airflow obstruction suggesting that in the population as a whole non-COPD lung attacks (or whatever these are) are responsible for a good deal of morbidity and considerable health care costs. Almost nothing is known about the pathogenesis of these events, but that does not seem to stop the expenditure of NHS money on what may in fact be self-limiting episodes. Gavin Donaldson (***see page 689***) suggests that episodes of respiratory viral and bacterial infection are the most likely explanation. The practical take home point is that some thought is required before starting patients presenting with an acute lung attack on the latest and most expensive long acting bronchodilators as many will have had normal spirometry before the acute event and will continue to have when it has settled down; they do not have 'early COPD', a concept designed to bloat the incomes of big PHARMA. More evidence that the most difficult diagnosis of all is normal subject, at all ages.

SEEKING THE BUBBLE REPUTATION, EVEN IN THE PATIENT'S CHEST?

So what was this hugely expanding bubble that the Thoracic surgeons found when they opened the chest of this 67 year-old man? One cough and it was out! (***see page 785***) for the solution.