Highlights from this issue

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LET US FIRST PRAISE FAMOUS MEN (AND WOMEN!)

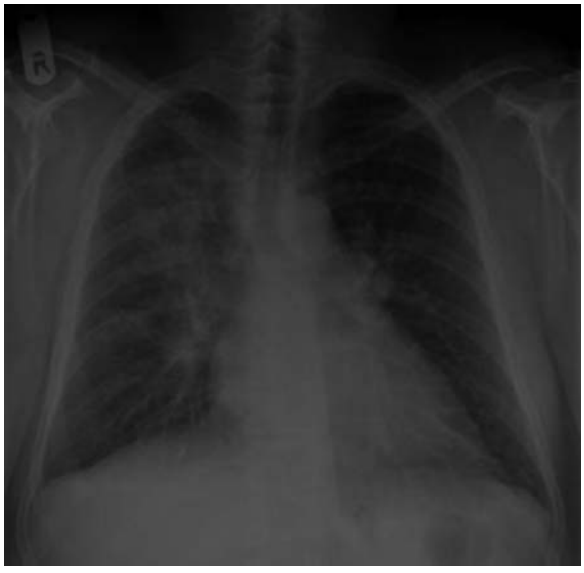
Specifically, Ben Nemery and his group, long renowned for their epidemiological and other insights. They have recently shown that the successive population interventions to restrict smoking have led to consistent reductions in preterm delivery (*BMJ* 2013;**346**: f441). This will likely lead to substantial lifelong improvements in lung function (see *Thorax* passim). Pity Ben did not publish in *Thorax* – he could then have had the whole manuscript in the Journal, not the 'one blink and you've missed it' form favoured by our sister periodical, presumably in the belief that their entire readership has an attention span measured in nanoseconds.

STEMMING THE TIDE

Another cause of long-term respiratory morbidity is extreme preterm birth, which is difficult to prevent and for which treatments are supportive and non-specific. The placenta and umbilical cord are generally discarded after preterm birth (or eaten by some animal species), but Pierro *et al* derived two different types of stem cells derived from the umbilical cord as a step towards treating the lung consequences of prematurity (Hot topic, ***see page 475***). Although of course the hyperoxic rats they studied are not the same as 450gm babies undergoing positive pressure ventilation, alveolar growth arrest is a feature of disease in both. They showed that both prophylactic and treatment strategies partially prevented and rescued alveolar growth respectively; and notably, both strategies would be feasible in clinical practice. Challengingly, the evidence of stem cell engraftment was meagre despite benefit, and the effects could be replicated by cell-free conditioned media derived from these cells. An accompanying editorial highlights the challenges of treating a disparate condition like bronchopulmonary dysplasia, but also the rich potential benefits extending well into adulthood that stem cell therapy could offer (***see page 402***). However, if the molecular basis of the benefits of stem cells could be established, maybe in the future the paediatrician awaiting the delivery of a very pre-term infant will have vials of surfactant in one hand and a 'lung growth tonic' in the other, ready to put down the endotracheal tube in the delivery room?

OSCILLATING IN THE BREEZE

High frequency oscillatory ventilation (HFOV) was one of those obviously excellent ideas which did not turn out as expected, and is apparently heading towards the medical graveyard where many such apparently good ideas are sadly already resting in peace, slain by the evidence. Recently, OSCAR and OSCILLATE (not two Muppets, but both excellent randomised controlled trials that enrolled over 1300 patients between them) suggested that HFOV was not merely not beneficial, but could actually be harmful (so OSCILLATE was even terminated prematurely because of safety concerns). So should we be hammering the last nails into the coffin of HFOV? No, say Matt Wise and colleagues in this month's *Hot off the Breath* (***see page 406***), adducing a number of arguments that more data are needed. Convinced? Take to the Correspondence columns if not!



BIG NEWS IN IPF RESEARCH

The demonstration of a link between a single nucleotide polymorphism (rs35705950) near the MUC5B gene and IPF is indeed big news (see *NEJM* 2011;364:1503-12). The polymorphism likely involves a promoter region and may have a pathogenic role mediated by altered mucus rheology and mucosal defence. Could this pathway also be involved in pulmonary fibrosis associated with sarcoidosis or scleroderma? Apparently not, according to Stock *et al* (***see page 436***; **Editors' choice**). The group also suggest that patients with IPF and rs35705950 have more benign disease. Mike Silverman, a mentor for both of us, regularly reminded us that every complex system has a key node, which when modified has a large effect on that system (for example, the gene responsible for swans being black not white and the gene that makes some lucky folk into paediatricians not adult doctors). The evidence to date implies that dysregulation of airway mucus is not in this category and is more likely one of multiple abnormalities of lung immunity and repair that lead to pulmonary fibrosis. However, as Mahida and Turner discuss (***see page 401***) it remains possible that a significant association has been missed due to lack of power, or because the true functional variant lies away from rs35705950. More work please!

THE HEART OF THE MATTER

This 46 year old man became acutely breathless and was treated for pneumonia initially, with no improvement. A loud systolic murmur was heard. So why did a cardiac lesion cause such almost entirely unilateral pulmonary abnormalities? Try to work it out before turning to *Images in Thorax* (***see page 498***)