Highlights from this issue

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A SPRING IN YOUR LUNG

Bronchoscopic lung volume reduction is very much a hot topic. Hitherto coils have been positioned for contraceptive purposes somewhat distal to the lungs; so are the Gynaecologists making a Putin-like bid for our territory? This month we publish 12-month outcomes after bronchoscopic insertion of a nitinol coil device in patients with emphysema (***Hot topic; see page 980***). Coils have potential advantages over other techniques as their spring like effect might address loss of elasticity (is there a whole body coil for your editors?) and dynamic airway collapse; they are applicable in the 60% of patients with interlobular collateral ventilation; and they can be inserted via a fibreoptic flexible bronchoscope in sedated patients (although patients participating in this trial had general anaesthetic). The trial reports reasonable safety, with problems probably relating as much to the bronchoscopy as to the technique. A large and sustained effect on quality of life score was seen, somewhat disproportionate to the effect on lung function. Interpretation of uncontrolled trials such as this is problematic as the placebo effect is much greater with procedures than drugs and it might be that this affects subjective more than objective outcome measures. The important priority is to get high quality data from randomised sham controlled studies but this is no straight forward matter. Finding strong and robust predictors of response would help. Our editorialists (***see page 973*** and front cover image) point out that resolving the issue of efficacy definitively is a priority as the cost effectiveness of bronchoscopic lung volume reduction looks borderline.

STAND AND DESATURATE

We suspect that most MRCP candidates will know that an abnormal fall in O_2 saturation on standing (orthodeoxia) is a feature of pulmonary arteriovenous malformations (AVMs) and hepatopulmonary syndrome but how many know what the normal range is for postural change in O_2 sat or how useful it is as a screening test? Two research letters this month address these questions in children (***see page 1045***) and adults (***see page 1046***) with AVMs and hereditary haemorrhagic telangiectasia. In children orthodeoxia was unusual, even in those with larger, more clinically significant AVMs. The test performed no better than oxygen saturation as a screening test (and of course screening attracts its fair share of controversy). In adults only 20–30% have an abnormal >2% fall in saturation. It is more likely to be seen in those with lower lobe AVMs and less likely in patients with obesity. Postural tachycardia might be a more valid test but even so it is doubtful if either test of any real value in identifying those at risk of important complications such as systemic embolization.

GOOD GOUT!

High serum uric acid and the possibility of gout may terrorise Professor Pavord after a typically port-saturated evening with the Bullingdon club, but now it seems that low serum uric acid may also be a BAD THING. Horsfall *et al* (**Editors' choice**; ***see page 1021***) in a small study merely encompassing more than a million patient years, showed that if you smoke and have a LOW serum uric acid you are more likely to develop COPD, and particularly lung cancer. In an accompanying editorial, Seif Shaheen (***see page 978***) reviews the anti-oxidant effects of uric acid, and the conflicting evidence in the literature, and suggests moving the field forward using Mendelian randomisation (a new technique to these editors) and even a controlled trial of supplementation of uric acid containing foods especially in those who fail smoking cessation, obviously with due regard to not pushing the levels up too high. Seems that the apple a day may keep the Doctor away by supplying uric acid, and not flavonoids?

SOMETHING TO PLAY FOR?

Maybe all is not lost as the baby exits the vagina and takes probably the most important breath of its life. Early determinants of lung function and their relationship to patterns of wheeze are another hot topic, with the generally gloomy assumption that all is quickly lost, and the mother's place is in the wrong (especially if she smoked in pregnancy). The Perth Birth cohort (try saying that quickly after a Bullingdon dinner!) reports that decrements in lung function as early as a month of age persisted until age 18 (***see page 1015***). Maternal smoking and early onset atopy were unsurprisingly the chief culprits, but interestingly, there was evidence of catch-up growth, particularly in boys, and encouragingly, in the offspring of mothers who smoked in pregnancy. Even flow-limitation, whereby tidal exhalation is close to maximal possible expiratory flow, seems to resolve. In an editorial, John Henderson (***see page 976***) sets these findings in the context of a not altogether consistent literature, and discusses the paradox that asthma did not seem to affect growth trajectory, even though it is associated with early airway inflammation and remodelling. Maybe after all, remodelling is protective? Or most asthma diagnoses in childhood are wrong? There is still much more to be learned, and the recent MEDALL initiative to combine big birth cohorts into mega-studies, in which endeavour John has been a major figure, will undoubtedly lead the way.

ANOTHER INTERACTION BETWEEN THE LUNG AND OBESITY?

A 70-year-old woman, who was known to have bronchiectasis, underwent resection of longstanding pulmonary nodules. What does the histology show? And what was her BMI? Turn to *Images in Thorax* (***see page 1061***) to find out.