Highlights from this issue

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GAIN WITHOUT PAIN?

Exercise is not always enthusiastically pursued by your Editors, whose idea of a strenuous work out is serial elbow flexion with a large glass of wine (or Bollinger club Champagne for IDP) in the ipsilateral hand. The relationship between exercise and asthma has hitherto been not dissimilar to that between Greece and Angela Merkel – we all know exercise triggers asthma, and has been proposed to be causative, if carried out in polluted air or you are an elite Nordic skier. However, in this issue of *Thorax*, we publish a randomised controlled trial showing that asthmatic patients assigned to aerobic training got fitter, and reduced bronchial responsiveness and systemic inflammation, albeit with no effect on markers of airway eosinophilia (***see page 732***, Editors' choice). So in addition to being Editors' Choice, they get the Jeremy Hunt prize for the cheapest asthma treatment on the market. For the dedicatedly inert, LUTE and VERSE may be an alternative. Not Oscar Wilde being aesthetic, but two trials of the anti-IL13 monoclonal Lebrikizumab, which showed that periostin positive adult asthmatics randomised to active treatment had fewer asthma attacks and improved lung function (***see page 748***). The manuscript confirms the irrationality of assuming asthma is one disease and treating it accordingly, but the bad news? These authors get the Jeremy Hunt booby prize for what will undoubtedly be an expensive therapy. Cheers and no knighthoods all round.

SNAP, CRACKLE, PROST?

Question: which inhaled endogenous mediator has been shown to inhibit the early and late response to allergen and prevent allergen-induced airway eosinophilia? Answer: Prostaglandin E_2 (PGE_2). Inhaled PGE_2 also prevents bronchoconstriction induced by most indirectly acting bronchoconstrictor challenges, and endogenously produced PGE_2 may be responsible for the refractory period sometimes seen after these challenges. You might presume that the pharmaceutical industry would have invested heavily in this compound but, perplexingly, there has been almost no interest – too good to be true, or so true it might wipe away all competition? One explanation is that inhaled PGE_2 causes airway irritation and cough. Might work by Birrell and colleagues (***see page 740***, Hot Topic) cause Pharma to reach for the cheque book again? The team used knockout mice to show that the anti-inflammatory effects of PGE_2 are mediated by the EP4 receptor. Selective EP4 agonists may therefore have anti-inflammatory and bronchoprotective effects without causing airway irritation, which is mediated via the EP3 receptor. Alan Knox's editorial concluded that these findings are of major interest in directing prostanoid based therapeutic approaches (***see page 711***).

TART OF THE YEAR?

So how come Alan Knox had time to write an editorial? He took a break from the golf course and his defence of the Fowler Cup because Ian Pavord has run away overseas, and tweeted (or more like twitted) 'I'm sorry to be missing this to go to a meeting (the money was good!). I hope everyone has a great day. Please do look after "my cup" (who he? Ed) for another year'. Scotland voted to stay with the UK so Alan could defend, but Pavord voted with his wallet to miss the golf! Dream on Pavord, and brown envelopes and brown noses all round!

AN ANTIEMETIC FOR VOMIT SYNDROME?

We've highlighted the epidemic of VOMIT (=Victims Of Modern Imaging Technology, or poor punters who are scarred witless by innocent nodules seen on their chest CT scan) in previous issues, and also the consequent endless cycles of job creation schemes this spawns. This month we offer W S Gilbert's firehose of commonsense on their management in the form of the first BTS guidelines on investigation of solitary pulmonary nodules, a summary of which is available on ***page 794***. Key recommendations include the use of semi-automated volumetric measurement of nodules to allow malignant growth to be detected more quickly; the use of the Brock University tool for predicting malignancy risk; and the setting up of dedicated lung nodule follow up clinics or virtual clinics. Angela Morgan and Mark Slade (***see page 716***) view the guidelines as a huge step from chaos towards order. They make an excellent recommendation of their own by suggesting that a standardised UK dataset for pulmonary nodule follow-up is set up so that high quality, real world data is available to the research community. Now that would be a worthwhile job creation scheme.

THE RIGHT CALIBRE FOR THE JOB?

Imaging studies have suggested that asthma may be a more focal disease than once we thought (*Thorax* 2014;69:63–71). In this issue, Oguma *et al* report spectacular HRCT reconstructions of the airways of normals, and patients with asthma and COPD (***see page 719*** and cover), which were carefully validated on phantoms. Staggeringly, they could image 5th generation airways. The COPD patients had more irregular airways, but in asthma the airways were both thicker and narrower. It would be fascinating to see if there were regional differences in the distribution of these HRCT abnormalities, and even more fascinating if they could be correlated with the ventilation defects seen with hyperpolarised helium. This could be particularly interesting if true, so that thermoplasty might be targeted. The technique could also be a powerful tool for serial studies of remodelling if it could be shown to distinguish fixed changes from transient such as airway wall oedema. Added value would be greatest in children if the radiation dose permitted, given serial bronchoscopies cannot be performed. Will micro-CT, which has been so productive in vitro, become a reality in vivo as well, as image resolution extends to more and more distal airways?

FAT AND FLABBY?

No, not the editors-in-chief again, but a clue to the cause of what the bronchoscopist saw when investigating a 68 year old male smoker with haemoptysis (***see page 809***). No cheating please!