Highlights from this issue

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A LONG TIME AGO, IN A GALAXY FAR, FAR AWAY

may be the start of Star Wars, but there are no countries far, far away in the present day, as shown by the first concern of the NHS 111 help-line being whether you have got Ebola virus. We need to know about respiratory disease overseas, not just because it may turn up in an Accident and Emergency Department somewhere near you, but because it may shed new light on our home-grown issues. Our cover features the map of South America not because your editors are looking for a retirement haven with no UK extradition treaty, but because we have a review of the different asthmas and their prevalences across Latin America, highlighting the extraordinary diversity of syndromes and associations in that continent (***see page 898***). There is so much to learn from geographical pathology so long as we do not assume all asthma is asthma, rather than a spectrum of diverse airway diseases. From Africa, Pitcher *et al* highlight the very high prevalence of radiological abnormalities in very young HIV positive African children at initiation of treatment, especially LIP and TB (***see page 840***). Unsurprisingly, TB was ten-fold commoner than in other series – beware of extrapolation. Anti-retroviral therapy is highly beneficial – if it can be accessed. And in how many contexts can this sad statement me made in resource poor areas, while the West puts even more energy into expensive me-tooers.

SLEEPERS AWAKE – EVEN IF IDP'S BOLLINGER CLUB EXCESSES MAKE IT A LESS THAN HAPPY DAWN!

We have been impressed by the quality of submitted sleep manuscripts and super-impressed by Jean-Louis Pepin, our sleep Associate Editor. Since our sleep knowledge is exhausted by getting tucked up in bed, we have relied heavily on his sound judgement in deciding which to publish. A recurring theme is the extent to which sleep disordered breathing is an independent risk factor for morbidity and mortality due to vascular disease. This month we report a 3-3.5 fold increased risk of coronary artery disease in veterans with treated and untreated obstructive sleep apnoea (OSA) (***see page 888***); that patients with severe OSA are more likely to have incident coronary artery calcium evident on CT (***see page 880 and page 815***); and an association between atrial fibrillation and sleep disordered breathing which may be independent of other risk factors (***see page 873***). Our editorialist, Malcolm Kohler (***see page 817***) reminds us that there are no intervention data proving that treatment of OSA benefits these co-morbidities, and that there is not enough evidence for our colleagues from cardiology, neurology and nephrology to begin looking for OSA as a modifiable risk factor. A big prize to the investigator who carries out the killer clinical trial, but only if published in *Thorax*.

A REAL EVIL ALLIANCE?

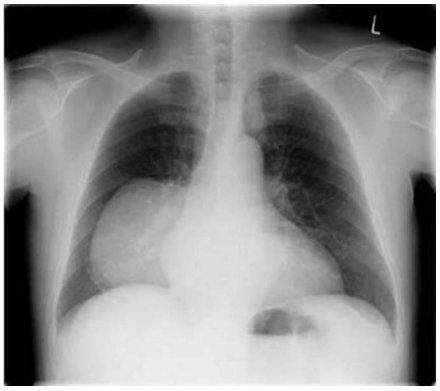
More damaging that the late unlamented coalition (sic) to the NHS may be the combination of bacteria and viruses in causing pneumonia. Viruses have long been known to be important in pneumonia in pre-school children, and in this issue Rhedin *et al* add human metapneumovirus to the cast of suspects (***see page 847***, Editors' choice). Interestingly, more than half the controls, as well as 80% of the patients, had one or more viruses isolated. Another stellar Associate Editor, Heather Zar, draws attention in an editorial to the lack of bacterial data, and cautions against extrapolating the findings uncritically to low and middle income countries (***see page 811***). Bacterial and viral co-infections are very common in childhood pneumonia – could it be that rather than being coincidental, viral-induced local immune paresis prepares the soil for bacterial pneumonia? For sure COPD patients treated with inhaled steroids, another cause of local immune paresis, get more pneumonia. Maybe work to be done by bright young clinical scientists in developed and developing world contexts?

INTERFERONCE?

Treatments which reduce eosinophilic airway inflammation such as anti-IL-5 substantially reduce the risk of attacks of airway disease. Why, when the main trigger for attacks is viral infection? Is this simply a reflection of removal of one of multiple inflammatory hits operating at the time of an attack or could it be due to a more fundamental link between eosinophilic airway inflammation and anti-viral immunity? Luke Hatchwell *et al* (***see page 854***, Hot Topic) provide strong support for the latter mechanism, and put Toll-like receptor 7 (TLR7) deficiency firmly in the frame, by showing that patients with moderate and severe eosinophilic asthma have decreased TLR7 and interferon γ 2/3 expression in bronchial biopsies. In mouse models IL-5-induced eosinophilia supressed TLR7 expression and TLR7 deficient mice were IFN-γ deficient and tended to exaggerated eosinophilic airway inflammation. We are particularly impressed by studies that link basic animal work with clinical disease in this way. This is great science from a terrific team. Most importantly, it is highly clinically relevant.

THINGS THAT GO LUMP IN THE CHEST

A bloody obvious diagnosis? If this picture is as baffling as a Shane Warne slider, turn to *Images in Thorax* on page 913.



AND FINALLY: IF YOU ARE GOING TO BE HANGED FOR A TREASONABLE WORD

you may as well throw in an offensive gesture as well (Earnest Bramah, *Kai Lung*). So can we thank our political masters for telling us how to apologise (not that we do for any single word we have published in *Thorax*); can we also hope these supreme experts in apology will tell us what response we can expect for pre-election unfulfilled promises (no more top-down NHS reorganisations, no increase in tuition fees, the list is endless)? Or perhaps not; as a French politician remarked, promises are only binding on those who believe in them.