Highlights from this issue

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A Chinese cracker

Following on from Zarir Udwadia's *Hot off the Breath* article on totally drug resistant TB (Thorax 2012;**67**:286–8), China is now coughing and the world should be listening. Liang *et al* ***(see page 632)*** report on the factors leading to the staggeringly high prevalence of multi-drug resistant TB (12% of 1995 patients tested). Depressingly, the causes are largely preventable; among these were the need for re-treatment, delay in initiating treatment, financial burdens, poor knowledge of the disease, poor coordination of services and supervision of treatment and neglect of infection control. No reason at all to believe this is just a Chinese problem, which is why we published the manuscript. So what are the lessons that we show no sign of learning? First, treatment of newly presenting TB is an urgent matter to get right; cancer surgeons would not randomly hack out a bit of lung somewhere near a tumour and hope for the best. They go for the definitive surgery, and so should we with TB treatment. Second, yet again, **KISS**—**K**eep **I**t **S**imple, **S**tupid—good TB treatment was all worked out decades ago and is not rocket science. The Captain of these Men of Death is alive and kicking on the Bridge and shows no signs of going into retirement.

Who nose?

The fashionable concept of the united airway is anathema to us—for sure, the nose is in direct communication with the bronchial tree, but the gut is also a united tube and no-one would attempt to diagnose gastro-oesophageal reflux by peering into the patient's rectum. However, sadly we may have to re-visit our prejudices, because in this issue of the *Thorax*, de Groot *et al* have shown that children with symptoms of allergic rhinitis are more likely to have poorly controlled asthma, and that this association disappears if the children are treated with topical nasal corticosteroids ***(see page 582)*** . However, before we finally swallow a colossal slice of humble pie, we would echo the authors' call for an intervention study to nail or otherwise disprove this finding. In the meantime, given the work showing that allergic rhinitis per se importantly impacts on quality of life, we will continue to ask about nasal symptoms and offer treatment if they are present, but we will still think it is a bonus if the asthma improves as well.

Let's KISS again, like we did before

Guy Marks (see *Thorax* 2012;**67**:85–7) suggested that airflow limitation is seen as a risk factor and used to predict the likelihood of a future event, rather than defining a disease. However, the desire to leap, Lemming-like, from an arbitrary level of a physiological marker to the diagnosis of a disease is alas addictive. One issue with using a fixed FEV_1/FVC ratio to define COPD is that the measure declines more quickly with age in men. Jordan *et al* ***(see page 600)*** show very clearly that the epidemiology of COPD is different when using the fixed FEV_1/FVC ratio compared to the lower limit of normal, with older men over-represented when the former is used to diagnose disease. Vito Brusasco ***(see page 569)*** makes a plea for use of the lower limit of normal but do the advantages outweigh the increased complexity of deriving this measure? Q: So what is the statistical definition of abnormal? A: 1.96 SD Scores (Z-scores) below the mean. Q: What does 1.96 SD scores below the mean actually mean? A: that your result is 1.96 SD scores below the mean and NOTHING ELSE. KISS, KISS, KISS!! Using a fixed ratio is as sensible as defining short stature as a height less than five foot six inches, irrespective of age or gender.

More is worse

Multiple inflammatory hits to the airway is a bad thing, resulting in a more corticosteroid resistant and damaging airway inflammatory response and more severe disease. Potentially individual targeting of each hit is one of the most promising approach to modifying severe airways disease. Essilfie *et al* ***(see page 588)*** model this phenomenon in sensitised mice and show that the combination of *Haemophilus influenzae* infection and ovalbumin challenge resulted in more chronic infection, higher levels of neutrophilic airway inflammation, a diminished response to corticosteroids and greater airway dysfunction. IL-17 seems to be a key player in this process. Blocking antibodies are available and have recently been shown to be effective in psoriasis. Clinical trials in severe neutrophilic asthma are awaited with interest. But first (KISS yet again), what about actively seeking infection in patients with severe, particularly neutrophilic, asthma, including with molecular based technology, and treating with antibiotics? Remember the imbecile who actually thought duodenal ulcer was caused by spirochetes and could be cured with antibiotics? There is one born every minute.

To hear more on this topic, listen to the podcast with Peter Gibson available via the Thorax website.

Effusions, false effusions and pretty pictures

The cover photograph this month is not Professor Pavord's latest bid for the Turner prize, but something that came out of the pleural space of a 55 year-old woman. What is it, what caused it and what is the connection with the title of this piece? Try to answer the questions before turning to the *Pulmonary Puzzle* ***(see page 658)***.