

Highlights from this issue

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CRIMES AGAINST HUMANITY

Thorax continues the theme of focussing on the world's major killers. Following issues on Tobacco and tuberculosis (TB), we present a themed issue on Pneumonia to coincide with World Pneumonia Day (12th November). There are commonalities about all three killers—they are brilliantly successful, global, cause millions of early deaths a year and most could be stopped in their tracks were it not for the cupidity, inertia and obstruction of their human accomplices. How many children's lives could be saved by simple basic measures like vaccination and antibiotics? Read the expert reviews focussing on the developing world (*see page 1052*) and Europe (*see page 1057*), as well as challenging papers from all round the globe. One could speculate what could be achieved in reducing pneumonia deaths if a tenth of the effort the human race puts into killing itself was channelled in that direction.

HOLD THE PRESSES: CANUTE TURNS BACK THE TIDE?

Deficiency of that Batman amongst vitamins, namely vitamin D, has been touted as causing everything from severe asthma to the recent financial crisis, and the nay-sayers appear powerless against this tsunami of manuscripts. Whatever the effects of vitamin D supplementation on asthma and Banker's bonuses may be, it does not protect against pneumonia. Remmelts *et al* (*see page 990*) report three case-controlled studies, totalling more than 33 000 adults with pneumonia and more than 100 000 controls, showing that there was no association between vitamin D supplementation and risk of pneumonia diagnosis. Indeed, in the third study, vitamin D supplementation was associated with an *increased* risk of pneumonia, even after adjusting for confounding factors. Of course, as with most huge studies, there are gaps in the evidence. We do not know how many patients were actually Vitamin D deficient—many cases did not have radiological confirmation—and hospital records, those well-known repositories of truth and accuracy, also had to be relied on. Nonetheless, this manuscript makes a compelling case that scattergun vitamin D supplementation will not help to prevent pneumonia.

PILING PELION ON OSSA

(Never let it be said that *Thorax* has abandoned classical erudition in favour of mere vulgar abuse!). There is enough misery from pneumonia without Doctors causing more cases. Evidence is mounting that among the risks of inhaled corticosteroid use is an increased risk of pneumonia and other infections, not least in patients with chronic obstructive pulmonary disease (COPD). The paper by Suissa *et al* (*see page 1029, Hot topic*) in this issue suggests that the risk of pneumonia might be greater with fluticasone than budesonide containing inhalers. Interpretation of their primary care database study is not straightforward as it is difficult to exclude with confidence confounding by diagnosis (patients with asthma are more likely to get budesonide) or severity (patients with more severe disease are more likely to get the newest and fanciest treatment). This latter possibility is an important one as it is likely to be an issue in the early stages of the introduction of all new treatments. Is there any way to sort this out? Answers in the form of letters or an opinion article would be most welcome. Old and young fogeys will maintain that the good old randomised control trial provides more solid data than primary care database studies, which themselves trump what Dante would surely have put in an even more outermost circle of Hell, namely comparative effectiveness research and other refuges of the terminally besotted with the latest trendiness. Whatever the ultimate conclusion of the fluticasone/budesonide/pneumonia risk debate, surely the main issue, as has been stressed in the *Journal* before, is that bad things will happen if potent immunosuppressive drugs are applied indiscriminately to an airway epithelium perpetually in the front line of the fight against invasive infection. We must start routinely to measure what we are treating in airway disease, including response to treatment. Current treatment of most patients with COPD with inhaled corticosteroids is as barmy as measuring anaemia by looking at a protruding tongue (or maybe an upthrust finger would better meet the case) and treating all such diagnosed 'anaemic' patients with vitamin B12 injections. Furthermore, in a developing world setting, the peddlers of expensive inhaled corticosteroids to those who can ill

afford them, who live in regions where community acquired pneumonia is endemic, for the most dubious of indications bear a heavy responsibility.

ANOTHER BLOW TO THE SPECIAL RELATIONSHIP?

Hot on the heels of the recent vote on Syria in the House of Commons comes a new source of Anglo-American discord. American guidelines recommend different antibiotics for healthcare associated as opposed to community acquired pneumonia as the condition is thought more likely to be associated with multi-drug resistant infections. Eva Polverino and colleagues (*see page 1007, Editors' choice*) question this dogma in a case-control study based in 12 Spanish hospitals. They found no difference in microbial aetiology in patients who meet accepted criteria for healthcare associated pneumonia. However, patients unsurprisingly did have more co-morbidity, more severe pneumonia and worse outcomes. Mark Woodhead (*see page 985*) cannot see a justification for retaining the community/hospital acquired pneumonia sub-division in healthcare systems where anticipated rates of multi-resistant pathogens are low and questions whether retaining this distinction might lead to worsening bacterial antibiotic resistance. Has another of Medicine's sacred cows been efficiently forwarded to the abattoir, or will the Empire strike back? We welcome views from both sides of the Atlantic (and anywhere else).

BRICKING IT?

Is it a lung, is it a liver or is it Professor Pavord's latest attempt at a culinary masterpiece? The source is a 76-year-old bricklayer and the answers are in the *Pulmonary Puzzle* (*see page 1078*). No cheating!

