

Highlights from this issue

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The “Chest Clinic” has landed!

This issue of *Thorax* sees the birth of ‘Chest Clinic’, a new section of *Thorax* dealing with clinically relevant material of interest to practising Chest Physicians, both adult and paediatric. We will be publishing short updates of major BTS guidelines; audit projects, particularly if of national or international appeal; brief summaries of the protocols of ongoing or shortly to start key clinical trials; various formats of clinical case-based discussions; opinion pieces; Clinical Science for the Chest Physician; Images in *Thorax*; and Pulmonary Puzzles. We are delighted that David Halpin and Nick Maskell have agreed to join our team of Associate Editors and support the development of Chest Clinic. We hope this section will inform, entertain and stimulate constructive debate without detracting from the main function of *Thorax* as an international scientific journal. The success of Chest Clinic does depend on active support of our readers and a steady stream of excellent contributions. If you have an idea for this section, please email us. We’d particularly like to hear from experienced colleagues who are retired or nearing retirement. Is there anything you want to get off your chest? Perhaps a clinical observation which you think might be important, or a particularly memorable case (especially if you got it wrong!—nothing is more liberating than the knowledge that the great are just as accident prone as the struggling mortal). We’re delighted to consider unsolicited manuscripts. Please see our on-line instructions for more details of the format.

COPD phenotypes

Many feel that one of the major blocks to progress in our understanding of the science and treatment of obstructive lung disease is that the entities COPD and asthma are too heterogeneous to be properly understood. Long ago, the renal physicians learnt that ‘chronic renal failure’ was the description of the stereotypical end point of many conditions, and not a specific diagnosis—is the same true of ‘asthma’ and ‘COPD’? Progress requires them to be broken down into more homogeneous, pathogenically similar

subgroups. One way to begin to make sense of the heterogeneity is to apply relatively unbiased mathematical techniques such as factor and cluster analysis to understand how clinical measures interrelate and group between individuals. There has been increasing interest in this approach in airway disease. This is nicely reviewed by Fingleton and colleagues (*see page 363*). An example of this sort of study is presented by Garcia-Aymerich and colleagues (*see page 430*). The authors collected and analysed a large amount of clinical material from 342 patients hospitalised with COPD and followed the patients for 4 years. Two phenotypes differed mainly in the severity of their airflow obstruction and associated disability and mortality but a third had distinct features, with prominent obesity, diabetes and cardiovascular disease. ‘Systemic COPD’ will, we suspect, be recognised as a distinct and clinically important subgroup by many of our readers.

Drug-induced pneumonia

Did God do it, or was it the Doctor, is always a question worth asking. In recent years we have heard of increased risk of pneumonia with inhaled corticosteroids and a possible reduced risk in patients taking ACE inhibitors, particularly in those belonging to ethnic groups prone to developing ACE inhibitor induced cough. Singh *et al* (*see page 383*) add to the list of drugs associated with pneumonia by showing that the Thiazolidinediones (Rosiglitazone and Pioglitazone) are associated with an increased risk of pneumonia and lower respiratory tract infection. This may be because Thiazolidinediones have a corticosteroid like effect on the airways but another possibility is that they produce a pathologically amplified response to the infecting agent because of an interaction with pro-inflammatory PPAR δ receptors. A better understanding of the mechanism of these associations might provide important clues to the pathogenesis of pneumonia. In the meantime, *primum non nocere*, or for the non-connoisseurs of classical literature, try not to rock the boat more than is absolutely necessary.

A pill causes every ill (again!)?

Almost without thinking, presence of fever means reach for the anti-pyretic bottle, usually paracetamol or ibuprofen. A recent review (*JRSM 2010;108:403–11*) highlights that in animal models, anti-pyretics can increase the risk of mortality, and there is a paucity of human data. By contrast, Lauder *et al* report in a mouse influenza model that paracetamol treated mice have reduced airway inflammation, reduced methacholine hyper-responsiveness at least as measured by the rather controversial Penh technique, without impairment of viral clearance or protective T-cell mediated immunity. So we need human data—is paracetamol friend or foe? Difficult to see how we will get it, given that no-one will make money out of paracetamol and thus support a trial, and non-pharma research funding is ever harder to find. So if you are febrile with a viral infection, what do you do? Anti-pyretics, alcohol, or what? The only undisputed certainty is the need to whinge if you have a Y-chromosome. *see page 368*.

Not hard to swallow?

This 75-year-old man with a previous myocardial infarction became progressively more breathless over a 3-month period. What does this reconstruction of the great arteries show, does he need thrombolysis, inhaled corticosteroids or (radically!) neither of these, and what would spirometry show? Answers before turning to *see page 456*.

