Highlights from this issue

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Andrew Bush and Ian Pavord, Editors

An APt response

Those who follow us on Twitter will know that there is now a Thorax mobile site, which we have been wanting for some time, and a bouquet to the publishers for delivering it. Those who think a Tweet is what our feathered friends do in the garden, do not read on, it will only cause you pain. We are told that the mobile site ‘has been specifically designed to accommodate the mobile behaviour of ‘keeping up’ and ‘looking up’ and works across all devices, including Blackberry, Android and iPhone. There are also links to the full site on the mobile site and to the mobile site on the full site, so that users can toggle easily between interfaces should they wish’. If this means more to you than it does to us, find out more at http://blogs.bmj.com/bmj-journals-development-blog/. Fountain pens out more at http://blogs.bmj.com/bmj-journals-development-blog/. If this means more to you than it does to us, find out more at http://blogs.bmj.com/bmj-journals-development-blog/. If this means more to you than it does to us, find out more at http://blogs.bmj.com/bmj-journals-development-blog/.

Stemming the tide of acute lung injury

This issue of Thorax includes two papers, an editorial and a Basic Science for the Chest Physician article on the hot topic of the therapeutic effects of stem cells. For those who feel challenged by the basic science, could we suggest you turn to the excellent review on (see page 533)? Stem cells could help by differentiating and replacing damaged cells or by paracrine effects, leading to tissue repair via locally released growth factors and cytokines. This latter mechanism seemed to be responsible for most of the beneficial effects of stem cells against a rat model of ventilator associated lung injury (see page 496) and gram negative pneumonia (see page 533). Pharmacological attempts to modulate aspects of the highly conserved and complex homeostatic response leading to acute lung injury have been a failure. Could stem cells be a new treatment paradigm? Sweeney and McAuley are cautious and remind us that there is a great deal more work to do (see page 475).

AREST-ED development?

Newborn screening for cystic fibrosis (CF) has led to a number of groups studying the evolution of lung disease with techniques such as infant lung function, HRCT and bronchoscopy. All of the studies agree that newborn screening does not equal problem solved. These ‘well’ babies with CF have structural and functional changes, and evidence of ongoing inflammation and infection. In this issue, the Australian AREST-CF group (see page 509) report on serial HRCT scanning in these babies. They demonstrate that many structural abnormalities progress, but interestingly ‘bronchiectasis’ did not persist in 26% of their paired scans, underscoring the importance of not assuming that airway dilatation, at least in young children, is permanent. This study adds to the evidence that lung disease evolves despite treatment even in apparently well, screened babies. So something must be done—in particular because the evidence base for treatment of these babies is exactly zero. So we need to use these and other results to design randomised controlled trials of treatment in CF babies as a high priority. In an accompanying editorial, Young and Owens (see page 471) highlight the critical importance of real attention to detail in CT protocols to minimise radiation. One lesson from these two manuscripts is the need to be careful not to move tests such as HRCT from a legitimate place as part of a research protocol to the general clinical arena without evidence of outcome benefit, particularly if the scans are not performed with meticulous attention to radiation reduction. CT scanning is not benign, babies are more vulnerable to the effects of radiation than adults, and we know there is an increased risk of at least some epithelial cancers in CF.

The second lieutenant of death

If tuberculosis is ‘the Captain of all these men of death’ then Streptococcus pneumoniae is the Second Lieutenant. Around 20 serotypes are responsible for human disease but knowledge is incomplete because, until recently, identification of the serotype required a positive culture. Berwick et al (see page 540) have used a sensitive urine multiplex immunosassay to show that serotypes responsible for pneumonia differ by age and co-morbidity, with invasive serotypes more commonly identified in the young and fit. In a linked editorial, Jeremy Brown suggests that vaccination with the new 13 serotype vaccine may be an effective prophylactic strategy as it would cover at least 57% of the serotypes causing pneumonia and 43% of those causing exacerbations of airways disease in adults (see page 473). The results of studies evaluating this possibility are awaited with interest.

An old-fashioned curvaceous beauty (of a diagnosis)

A 64-year-old woman with refractory asthma had this flow-volume loop. No phenotypes and no cytokines need apply; what is the diagnosis? Hone your old fashioned physiological skills before turning to the Pulmonary Puzzle on (see page 564).

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