

Highlights from this issue

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CF lung attacks: more than just transient trouble

The feeble word 'exacerbation' has been applied to deterioration in health status in CF patients usually requiring intravenous antibiotics, but with the expectation that all will be well, and the whole thing is minor nuisance value, like getting a plumber to fix a dripping tap. Not so. It is known that around a quarter of patients do not go back to baseline spirometry, and now an even more disturbing study in *Thorax* reports that CF patients more than two 'exacerbations' a year have a greater likelihood of a >5% rate of decline in FEV₁ over the study period, and are more likely to die or undergo a lung transplant. Perhaps unsurprisingly, those who had multiple exacerbations were more likely to be female, diabetic and have poor baseline lung function. As with all good studies, new questions arise. Are the multiple CF lung attacks a marker of intrinsically more serious disease, or is the problem adherence to treatment? Are the multiple CF lung attacks causal of an accelerated decline in lung function, or are the attacks and the decline a manifestation of underlying severe disease or poor adherence? More studies are needed to answer this question, but these compelling data add force to the contention that 'exacerbations' are in fact 'attacks' and should prompt action. Antibiotics are of course necessary first aid, but the need for their use on multiple occasions is a marker of a CF patient who is doing badly. As well as fingers in the dyke with antibiotics, a reality check as to why the flood waters are rising is urgent. *See page 680.*

Taking the plunge: lung transplantation

There is no doubt that we have got better at keeping patients with 'end-stage' CF alive. Median survival from the point of reaching an FEV₁ of 30% has risen from two to more than five years over the last two decades. Furthermore, overall survival has increased dramatically over the decades. Both these factors mean that the conventional life table predicted median survival approach is unsatisfactory. The

better survival of severely ill CF patients has made predicting the optimal timing of transplantation even more difficult. In this issue, Jackson *et al* have used sophisticated Weibull modelling to construct survival curves based on more than 13 000 patients from the US CF Registry. The model was then prospectively tested on a smaller Irish cohort (243 patients). The accuracy of the model increased with length of follow-up, perhaps not unexpectedly, but reassuringly given that most CF patients contemplating transplantation will have been followed up for a prolonged period. The details of the mathematics may be a tad beyond this Bear of Little Brain, but the approach is clearly powerful, and could well be used in other diseases in which the treatment goalposts are not so much moving as disappearing over the horizon. *See page 674.*

Lung cancer screening

Last month saw the on-line publication of the large, 53 000 patient National Lung Cancer Screening trial in the *New England Journal of Medicine* (see 10.1056/NEJMoa1102873). The bottom line was that CT screening reduced lung cancer mortality by 20% and all cause mortality by 6.7% compared to CXR screening. The down side was that over a quarter of screening CT scans showed an abnormality and 96.4% of these were false positive, meaning that over 6500 of the 26 722 patients randomised to CT screening developed VOMITS (victims of modern imaging technology syndrome; see April airwaves). Are these findings definitive or are there remaining questions? Kilpatrick Field and colleagues argue strongly that there are and that the planned UK Lung Cancer Screening trial should continue as planned (*see page 736*). We find their case persuasive. We must reach a clear consensus on the risks and benefits of screening and information from multiple trials conducted in different healthcare settings will help us do this. We don't want to be in the same position as breast cancer screening where nationwide UK screening was implemented on a whim, as a political expedient, before clear agreement on efficacy had been reached.

A fluid situation: rising incidence of (parapneumonic) empyema

Grijalva and colleagues add to growing evidence that the incidence of empyema is rising in all age groups (*see page 663*). Using high quality US national hospital admission data they estimate a twofold increase in incidence from 3.04/100 000 to 5.98/100 000 in 1996 and 2008. Information on the microbiological aetiology is patchy as around 40% of pleural fluid cultures are negative using conventional methods. Menzies and colleagues suggest that positive microbiology rates might be increased using simple blood culture methods (*see page 658*). The information we do have on microbiology suggests a different pattern of pathogens to community acquired pneumonia. Perhaps it is time to abandon the term parapneumonic empyema and begin to think differently about the pathogenesis of this condition. Thankfully we have increasing high quality evidence on which to base management decisions. In no small part this is thanks to the efforts of Professor Rob Davies who tragically died recently. We join his disciples Rahman & Maskell (*see page 649*) in paying tribute to Rob's magnificent achievements in this and other fields. As well as being a world class clinical researcher he was a thoroughly good guy. He will be sadly missed.

All that bulges is not obesity

This woman was unwell, with neutrophilia, and an abnormal CXR which shows an empyema (see cover also) causing mediastinal displacement, linking with other manuscripts in this edition of *Thorax*? *See the Pulmonary Puzzle, page 733.*

