

Highlights from this issue

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DON'T MISS THIS AMERICAN PIE

This issue coinciding with the ATS meeting in San Diego has a North American theme. In addition to a number of strong research papers, there is a HOT off the breath by Detterbeck (*see page 406*) on minimising VOMITING in cancer management (*see Airwaves passim*, or previous issues for the classically challenged) and two great reviews on the genomic origins of asthma from the Channing laboratory (*see page 481*), and an –omics perspective on sleep disordered breathing in children (*see page 474*). This last, from a future ATS president, is a really hot topic – snoring is no joke but a clear and present danger to children's health and even life, as well as programming children towards chronic morbidity. Finally, light blue touch paper and retire – and Paul Enright certainly lights the fuse in his editorial (*see page 401*). Let battle commence!

THICK AS A PLANK?

The late great Richard Asher, commenting on the lines from an ancient hymn 'Teach me to live that I may dread, The grave as little as my bed' remarked that the bed was actually a very dangerous place indeed for the patient to be in. The same is even truer for the intensive care unit. So how do we avoid premature extubation with demoralising consequences and the converse fault of leaving the patient ventilated too long, prone to nosocomial pneumonia, pulmonary embolism and the other undesirable spin-offs of modern, high octane care? DiNino *et al* (*see page 431*; *Editors' choice*) suggest that using ultrasound to measure the change diaphragm thickness in the apposition zone between end inspiration and end expiration had a very good positive predictive value, although not so good negative predictive value, for successful extubation. In fairness, some of the failures were unrelated to diaphragm function, lowering the negative predictive value; this test can never replace a holistic review by a good intensivist. In an editorial, Gerard Criner (*see page 402*), while rightly highlighting some limitations of the study and emphasising that prospective validation studies are needed, welcomes this as a potentially exciting tool in the intensive care unit. Long live old-fashioned respiratory mechanics.

DO OUR PATIENTS WITH COPD DREAD THE GRAVE AS LITTLE AS THEIR BED?

Or maybe not so much the grave, but the actual passage to paradise. Readers who missed the terrific talk at the winter BTS by Robin Taylor on end of life care in patients with lung disease shouldn't miss his case based discussion on this subject (*see page 498*). The scenario described is, we suspect, familiar to many. Doing something – however unlikely to help we might privately think it is – is easier and less emotionally turbocharged than taking the time to think about end of life care and palliation, and actually (God forbid!) actually TALK to patients and families. Even worse, you may have to LISTEN to them! What can be done to ensure that care at the end of life is proportionate and compassionate rather than burdensome and futile? Robin suggests that symptom palliation should have a much higher priority, and that a ceiling of care document might help direct other potentially distressing treatments whose goal is prolongation of life. Whether such a document would be acceptable to Daily Mail readers and the great and the good in the Department of Health remains to be seen. What is beyond doubt, and is rightly emphasised, is that senior clinicians, who often have a long-term relationship with the patient and their family, are the individuals who have to step up to the plate and walk the walk with them. We would like to see more contributions on this increasingly important but neglected topic.

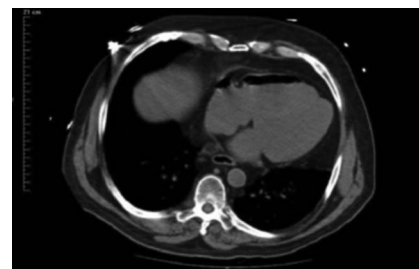
COPDGENE AND SUBTYPES OF COPD

Regular readers of Airwaves will know that your editors regard the terms asthma and COPD as roughly equivalent to arthritis and anaemia in their diagnostic precision and clinical utility, ranking only slightly above the effusions of our glorious political leaders (which we assume is a trans-Atlantic problem). In order to move on from these unsatisfactory terms we need to embrace methods to assess different aspects of airway disease and make sense of this new information in the hope that new, clinically important sub-groups will emerge. A hot area has been the use of cluster analysis to identify sub-groups of patients in a relatively unbiased way. Interestingly (and

encouragingly), when this method is applied to populations assessed using familiar measures, very familiar patterns of disease emerge. The great American physician Frances Rackemann would have been pleased. The findings of cluster analysis become potentially much more interesting when less familiar measures are assessed. Castaldi and colleagues show this well in a large study based on the COPDGene cohort, our Hot Topic paper this month (*see page 416*). They focus on airway and lung damage in patients with COPD assessed by CT and identify four groups of patients differing in the nature of the lesion (airway thickening vs emphysema) and the distribution of the changes. The subtypes showed strong association with relevant clinical measures and known COPD-associated genetic variants. The association with genetic markers differed by subtype suggesting that researchers interested in genome wide association studies (GWAS) will also have to master the field of cluster analysis in order to make progress. They should also take note of the importance of accurate clinical phenotyping, as in this manuscript – asking 'has someone, somewhere over the rainbow said you have asthma' does not cut it, no matter how sophisticated the GWAS. Garbage into the GWAS, Garbage out – politicians take note (not that they will).

THE RADIOGRAPH WITH THE HOLE IN THE MIDDLE

Why did this patient feel the power? Work it out before turning to *Images in Thorax*, (*see page 501*).



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