Highlights from this issue

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HOLY COW!

Not a Passage to India, nor a ticket to the abattoir for yet another useless umbrella label for airway disease like ACOS (watch this space!), but the BATMAN study. A short-odds favourite for the best study name of the decade award, BATMAN (***see page 543***, ***Editors' choice***) tried to take the monitoring of paediatric asthma howling loudly into the 21st century by enrolling 280 children with asthma into one of 3 limbs: see every 4 months, ask how them how they were doing (Asthma control test); do the same thing monthly, but with a web-based tool; or add FeNO to the four monthly assessments. And the answer: asthma control days were the same in all 3 groups, although the web-based and FeNO groups were able to reduce their ICS dose over this impressive, year long study. Our editorialist, Louise Fleming (***see page 517***), draws the twin conclusions that it doesn't matter what you do so long as you do it lots of times, and you should use modern communication methods to do it, both profoundly depressing to your biomarker driven, old fogey editors in chief. Surely we can do better than a rather sophisticated 'e-hi-how are-you-doing' in managing the diversity of airway disease in children? Congratulations to the BATMAN team on a great study–Gotham city is proud of you. Prizes for those who can resuscitate biomarkers, especially if you can come up with an even better study name.

THE OLD MAN'S FRIEND

Pneumonia is often listed on the death certificate when the real cause is being time-expired, which is why 30 day mortality is problematic as an index of pneumonia severity. The CAPNETZ pneumonia cohort (***see page 551***) is notable for choosing mechanical ventilation and/or vasopressor support within 7 days of admission as indices of severity. Our editorialist, James Chalmers (***see page 515***), sees this as an important step forward. CAPNETZ showed that 5% of patients will require these interventions in the first few days following admission with community acquired pneumonia. These patients can be readily identified by the presence of a small number of abnormal variables: respiratory rate >30/min, PaO2/FiO2 ratio <250, multilobar infiltrates, confusion, uraemia, and hypotension requiring fluid resuscitation. Late deterioration, particularly after day 4, carried a very poor prognosis. The hope is that identification of these patients will lead to timely and effective intervention. Traditional pneumonia risk scores such as the PSI and CURB65 don't look like they will cut the mustard against this new index of pneumonia severity so a new risk score might be necessary. We suspect a catchy and memorable acronym might be a key to widespread clinical application of any new risk score. Our usual prize (dinner for two at McDonalds hosted by Professor Bush, going Dutch) for the winner.

REACHING THE PARTS THAT OTHER STEROIDS DO NOT REACH?

A few patients with asthma have persistent eosinophilic airway inflammation despite taking high doses of inhaled corticosteroids (ICS). The cause of this corticosteroid resistance is unclear but we do know that interventions that deplete circulating eosinophils such as systemic corticosteroids and anti-IL-5 are strikingly effective. Could the inflammation persist because it is predominantly located distally, in a site not well accessed by large particle ICS? David Hodgson and colleagues (***see page 559***) provide some support for this view by showing that additional small particle ICS Ciclesonide is better able to maintain the anti-inflammatory response to prednisolone than placebo. The study did not include a third arm of patients treated with a proportionately higher dose of their usual large particle ICS so we can't be sure that this effect is a function of better peripheral deposition of Ciclesonide. However, the findings are encouraging and provide a strong basis for a more definitive study powered to show clinical as well as inflammatory benefits.

PILLS FOR ILLS?

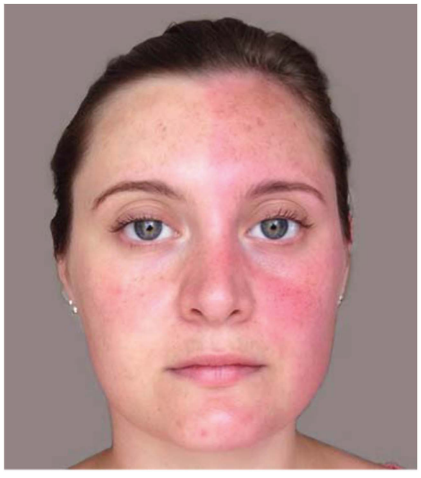
Paracetamol and asthma is our Hot Topic this month (***see page 528***). There is conflicting epidemiological evidence as to whether paracetamol ingestion by pregnant mothers is a marker of a propensity to infection, and the underlying reason for taking paracetamol, be it this or another mechanism, is what increases the risk of subsequent asthma in the baby, or that paracetamol ingestion is independently causative of asthma. Difficult to see how to do a human intervention study, but the Mighty Mouse may help us out. Debbie Lee and colleagues used an established neonatal allergic airway disease (AAD) murine model to determine whether paracetamol increased the risk or severity of inhaled house dust mite challenge-induced AAD. Mice were exposed to paracetamol during pregnancy, lactation or both. Despite detailed physiological and immunological testing of the pups, no signal could be found. So does paracetamol smell of roses? Probably, but two questions remain: wouldn't it be better to grin and bear it rather than pop a pill in pregnancy (and of course your all-male editors in chief are past masters at stoicism—just ask their spouses!); or if you must take a pill, take ibuprofen, with no safety question (a similar argument which led to the great Helen Clark banning Berotec in New Zealand—if we have something safe, what need we of an alternative that may not be)?

FLYING PIGS?

The UK will have a new government by the time you read this. Wouldn't it be nice if it and the opposition, whoever they might be, signed up with the Medical profession to a Duty of Candour? Don't hold your breath.

A MAKE-UP MALFUNCTION?

Was this woman in too much hurry to make up? Or half nervous (a subtle clue, aka dreadful pun—don't worry, only another few issues to survive)? Sort it out before turning to *Images in Thorax* (***see page 605***).



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