Highlights from this issue

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THINGS ARE WHAT THEY THEME TO BE

This issue is dedicated to the pulmonary circulation—the so-called ‘lesser circulation’, which contains more endothelial cells than all the rest of the body, has to match blood flow to ventilation while cardiac output rises many-fold on exercise while not permitting perfusion pressure to rise excessively, and protect crucial organs such as the brain from vascular occlusion by filtering circulating debris thus leading to infarction; this last without the lung itself being infarcted. The pulmonary circulation is a silent workhorse, with disease often manifesting late—and papers on a range of pulmonary vascular issues are presented this month.

WHEN A CLOT IS A LOT

How's this for a classic Friday afternoon conundrum? A patient presents with a large proximal pulmonary embolus, systolic blood pressure hovering around 100 mm Hg and CT evidence of right ventricular dysfunction. Often recent surgery or bleeding will be thrown into the mix just to make decision making really tough. My colleagues and I debate this issue endlessly and split roughly 50:50 into those who would and wouldn't give thrombolytic therapy. What would you do? Jiménez *et al* (see page 109, Editors' choice) help us by providing guidance on the prognostic significance of a CT showing evidence of right ventricular dysfunction. In short, it identifies patients who are more likely to have echo evidence of right ventricular dysfunction and a raised brain natriuretic peptide level but not those with a higher chance of death or a complicated recovery. The question of when and in whom to use thrombolytic therapy in acute submassive pulmonary embolus remains a vexed one. Luke Howard (see page 103) and John Simpson (see page 105) argue for and against but agree on two key issues: we need better risk stratification tools and better data. Those who hoped that the PE Thrombolysis Study (PEITHO) would provide definitive evidence may be disappointed. This study assessed death or hemodynamic collapse within seven days of treatment in 1006 patients treated with heparin plus placebo or heparin plus a weight-adapted bolus of tenecteplase. Press releases suggest that the primary end point was reduced by 56% in patients treated

with tenecteplase and heparin but major bleeding was increased by a very similar amount. Methinks Friday afternoon debates will continue.

UNFILTERED!

Not another ‘very sensible’ manoeuvre proposed by the tobacco industry and supported by Ministers (and now UKIP, making a welcome late bid for the title role in our 2013 production ‘Where's Wally’), but what happens to the bloodstream when the filtration function of the pulmonary circulation is bypassed. Donaldson *et al* (see page 161) estimate the frequency of hereditary haemorrhagic telangiectasiae as around 1 in 10 000, much commoner than previously thought. The prevalence of this autosomal dominant condition was higher in women, implying either men complain less (yeah, right!) or are too lazy to seek medical attention. Prevalence was almost twice as high in the West Midlands compared with South West England—ascertainment bias? We need to think of this not so rare condition before rather than after it manifests with stroke or cerebral abscess.

ANOTHER GRIM REAPER

Sickle cell anaemia (sorry, could not resist yet another feeble pun!) is a global scourge of young children, but for which medical treatment and bone marrow transplantation offer new hope. The disease is punctuated by crises, particularly affecting the chest. The incentive to treat aggressively is to give the patient a chance to survive in good condition to benefit from novel treatments. Mekontso Dessap *et al* (see page 144, *Hot topic*) report the HRCT findings in a large series of patients with acute chest syndrome; consolidation followed by ground glass pattern was most typical, whereas upper lobe nodules cast doubt on the diagnosis. Perhaps unsurprisingly, agreement between reporters was less than perfect, and the authors call for more work to be done. We agree, but this should be combined with work showing that outcomes are improved by expensive imaging. VOMIT syndrome type 2 (see Airwaves passim for type 1) is characterised by the desire to accrue images for their own sake, and is currently particularly rampant in the world of cystic fibrosis. Workers building on this current nice please note.

AND NOW FOR SOMETHING COMPLETELY DIFFERENT

Like dogs returning to a different sort of VOMIT, we end with two items about airway disease. Another *Thorax* *bête noir* is the influence of big Pharma over guidelines. On the basis of no evidence whatsoever, prescriptions of inhaled corticosteroids as first line management of chronic persistent asthma have fallen, and the use of combination inhalers risen. Perish the thought that this could at all be related to the relative cost of the two approaches, and aggressive marketing. Perish also the thought that your editors are so obtunded by blind prejudice as to be unprepared to consider new evidence. Lazarinis *et al* (see page 130) compared regular inhaled budesonide and on demand terbutaline, on-demand budesonids/formoterol, and on demand terbutaline alone for asthmatic patients with exercise induced bronchoconstriction. The two budesonide containing regimens were superior to terbutaline alone, calling into question the guideline recommended treatment of short-acting bronchodilator to treat exercise bronchoconstriction. The as-needed regime resulted in less budesonide use, unsurprisingly since only 3–4 exercise sessions were performed per week, a far lower level than that achieved by your super-fit editors (usually fleeing from outraged potential authors many more times a week!). More evidence of this sort and less fiscally based guidelines please.

SOME THINGS AIN'T WHAT THEY SEEM TO BE

Is it a glomerulus? Is it Professor Pavord's liver after a Bullingdon club dinner in his new domicile, Oxford? Or what is it? Turn to the compelling pulmonary puzzle to find out (see page 193).