time, we have an up-to-date picture of how CAP is currently managed in the UK, where this differs from guideline recommendations and where attention should be paid to lead to improvements in practice. We believe that the BTS guidelines are a reasonable translation of the available scientific evidence with regard to this topic, but we also acknowledge that they are not perfect and may not be appropriate for all settings. Inevitably, they are weakest where there is least evidence and choice of antibiotics is one such area. We would like to see the guidelines improve, but this can only occur with better evidence. This requires future funding for clinical research in this important, but research-neglected area.

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Competing interests None.

Contributors WSL and MW wrote the response.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 25 August 2011
Published Online First 20 September 2011

Thorax 2012;**67**:832–833.
doi:10.1136/thoraxjnl-2011-200980

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Acronyms, pneumothoraces and the impact of international health on the NHS

I read the latest Issue of *Thorax* with amusement and frustration.

I could not resist your challenge in your Editorial, 'Pre-drainage tension', triggered by letters from Drs Simpson and Leigh Smith^{1 2} to make a ridiculous acronym.³

My understanding of pneumothorax was that it is due to a loss of the negative intrapleural pressure that overcomes the elastic recoil of the pulmonary tissues. Once this

vacuum is lost then air is free to enter the lungs or intrapleural space with impunity. The actual amounts will vary according to many factors, including the strength of elastic recoil of pulmonary tissues, exact sites of leak and depth of inspiration. Perhaps we need an engineer to explain this?

However, on first reading of the letters I was concerned that all texts on the issue including life support and trauma courses would have to be REPRINTED (Rapidly Expanding Pneumothorax Requiring Immediate Needle Thoracic Elimination to avoid Death), or worse still would Stop Casualties Receiving Appropriate Pneumothorax Procedures to Eliminate Death (SCRAPPED).

Having tried to be ridiculous I was then struck by the juxtaposition of Kevin Southern's article on cystic fibrosis screening⁴ and Dr Zarir Udwadia's article 'MDR, XDR, TDR tuberculosis'.⁵ Both were excellent articles but their proximity raised issues of global health economics that must be addressed. Cystic fibrosis is a disease that has a very large budget, possibly larger with the advent of promising new treatments, but that affects relatively few. The tuberculosis figures from India are frightening. In the age of international travel it might be totally drug-resistant (TDR) tuberculosis that provides the West with a huge public health and mortality problem. When debating NHS reforms the impact of other healthcare systems on ours has not even been considered. Is it time to admit a national health service is not possible in the twenty-first century, but an international health service is not only possible but necessary? Is there a role for the British Thoracic Society to start public debates on these issues?

Thanks for a thought-provoking read.

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Competing interests None.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 5 April 2012
Published Online First 27 April 2012

Thorax 2012;**67**:833.
doi:10.1136/thoraxjnl-2012-201987

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Hyperoxia in acute asthma

We read with interest the recent article 'Randomised controlled trial of high concentration versus titrated oxygen therapy in severe exacerbations of asthma' by Perrin *et al*¹ and the accompanying editorial. We note that data presented in the online supplement suggest, unsurprisingly, response to treatment at 60 min in terms of respiratory rate and forced expiratory volume in one second, probably explaining the rise in transcutaneous partial pressure of carbon dioxide (P_tCO₂) in this population. Therefore, it cannot be assumed that the P_tCO₂ levels would have continued to rise after 60 min as the authors suggest.

We are unconvinced by the implication that the levels of normocarbida and hypercarbia (up to 50 mm Hg) demonstrated in this study are deleterious in acute asthma. Life-threatening respiratory failure in asthma is multifactorial, with ventilation–perfusion mismatch, lung hyperinflation and an increased work of breathing leading to respiratory muscle fatigue all being contributory factors.² A degree of 'permissive hypercapnea' is now regarded as best practice and a safe approach in the management of mechanical ventilation for respiratory failure in critical care, including the management of severe asthma. Conversely, hyperoxia is known to cause excess reactive oxygen species causing oxidative stress and free radical damage in exposed tissues,³ and has been implicated in worsening myocardial and cerebral ischaemia.⁴ Maintaining hyperoxia may also result in delays in recognising clinical deterioration.

We are in full agreement with current guidelines that therapy should target physiological levels of oxygen,⁵ but would argue that hyperoxia per se may be more harmful than the predominant normocarbida found in this study population of acute exacerbations of asthma.

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Competing interests None.

Provenance and peer review Not commissioned; internally peer reviewed.

Accepted 2 December 2011
Published Online First 20 December 2011

Thorax 2012;**67**:833–834.
doi:10.1136/thoraxjnl-2011-201383

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