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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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 screening[4] 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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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 ($PtCO_2$) in this population. Therefore, it cannot be assumed that the $PtCO_2$ levels would have continued to rise after 60 min as the authors suggest.

We are unconvinced by the implication that the levels of normocarbia 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 normocarbia found in this study population of acute exacerbations of asthma.

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Authors' response: hyperoxia in acute asthma

We appreciate the comments by Snelson and Tunnicliffe[1] regarding our study of the effects of high concentration oxygen therapy in acute exacerbations of asthma.[2] We concur with the view that the effect of high concentration oxygen therapy on arterial carbon dioxide pressure ($PaCO_2$) is not clinically relevant in all patients presenting to the emergency department (ED) with acute severe asthma. However, we consider that the 3.9-fold greater risk of patients developing an increase in transcutaneous partial pressure of carbon dioxide ($PtCO_2$) ≥8 mm Hg (22% vs 6% in the high concentration vs titrated oxygen groups, respectively) is likely to be of clinical relevance in life-threatening asthma. Even in our study, which excluded patients who were unable to speak or perform spirometry due to breathlessness, all 10 patients who had a final $PtCO_2$ ≥45 mm Hg had received high concentration oxygen therapy. These findings suggest that the routine administration of high concentration oxygen therapy in the ED setting is a determinant of respiratory failure, a recognised marker of near fatal asthma. This probably also applies to the routine use of high concentration oxygen therapy during ambulance transfer in patients with severe asthma, as has been noted in chronic obstructive pulmonary disease,[3] but this was not assessed in our study.

While permissive hypercapnia is an approach to the management of mechanical ventilation for severe asthma, this relates to intubated patients, in whom the purpose is to reduce the risk of complications associated with hyperinflation.[4] It certainly does not apply to prehospital or ED care.

We agree that there are many potential risks associated with hyperoxia, including but not limited to reductions in coronary and cerebral blood flow, decreased cardiac output, increased oxidative stress, delay in recognising a clinical deterioration and rebound hypoxaemia if oxygen therapy is abruptly stopped. However, in respiratory disorders such as severe asthma where there is significant ventilation/perfusion (V/Q) mismatch, hypercapnia represents another potential risk of high concentration oxygen therapy that needs to be recognised in clinical practice.

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Increasing smokers' risk perception improves CT screening participation

We read with interest the article by Patel *et al*[1] and wish to comment on their findings with specific regard to smokers' risk perception, motivation and low participation rates in CT screening programmes.

Based on the studies to date, there is a consistent theme that smokers' participation in CT screening programmes for lung cancer is poor when their motivation is low and much greater when their perception of risk of lung cancer is high.[1 2] Despite overwhelming public health messaging, smokers continue to smoke, in large part, because they perceive their own risk from smoking to be low. This self-perception of low risk (termed optimistic bias) maintains a low level of motivational tension (the fear that smoking might indeed be harmful).[3] We propose that optimistic bias can be undermined, and motivational tension increased, when smokers are confronted with adverse 'personalised' risk data.[3] With advances in the understanding of the clinical and genetic factors underlying lung cancer susceptibility, we have developed a lung cancer susceptibility risk model.[4] This model assigns current and former smokers to moderate, high and very high risk. In a group of randomly selected current smokers, 84% took up the offer of risk testing and, surprisingly, quit rates 6 months after testing were 20%, 36% and 40%, respectively (28% overall).[5] Just as with triggering a decision to quit smoking, we suggest uptake of (and possibly adherence to) CT screening might be improved by risk testing that enhances risk perception, undermines optimistic bias and increases motivational tension.[3]

We tested this proposition in a scenario-based telephone questionnaire involving 350 current and former smokers (mean age 67, age range 44–86 years, 59% male and mean pack years 45). When told of a survival benefit with CT screening versus no screening, we found 68% agreed to undertake CT screening while 95% agreed to gene-based risk testing. Likelihood of participation in CT screening for lung cancer was 25% higher (absolute increase) in those testing high and very high risk compared with those at moderate (average) risk. Collectively, the results of these studies support our suggestion that optimistic bias can be undermined, and motivational tension increased, in current and former smokers through the use of personalised risk testing. We suggest that personalised risk testing, incorporating genetic markers of susceptibility, may help identify and motivate 'high risk' smokers to engage in CT screening.

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