CORRESPONDENCE

Using the BTS CAP audit to evaluate local data

We read Dr Lim and Dr Woodhead’s update on the British Thoracic Society (BTS) 2009/2010 community acquired pneumonia (CAP) audit with interest and noted the high inpatient mortality rate of 18.3%.1 As a contributing site, we received a useful summary of our data in comparison with the national data and local mortality rates for severe CAP (CURB65 3-5) were 21.4% versus 42.6% nationally. This provoked an examination of local severe CAP admissions between December 2009 and May 2011 (n=169) that found 25% mortality with age, gender and comorbidity distributions similar to national audit data. We suspect variations in case definitions may be important in understanding differences between local and national data.

The Thorax report focuses on adherence to local antibiotic guidelines. Nationally, 55.5% of patients received antibiotics to avoid treating patients with local prescribing policies (64% in severe CAP), but that no association between adherence and mortality.1 From our 18-month severe CAP data, over 89% of our patients received antibiotics in line with local policy. Local policies are assumed to reflect BTS guidelines, which advise intravenous co-amoxiclav plus clarithromycin as the first choice in severe CAP.1 Locally, we recommend intravenous benzylpenicillin plus clarithromycin in severe CAP as advocated by the British Society for Antimicrobial Chemotherapy.2 They suggest that it is likely to apply the least bacterial ecological pressure while still being clinically effective and they refer to the ‘collateral damage’ of resistance and Clostridium difficile infection, which results from injudicious broad-spectrum antibiotic use.3 Locally, the spectre of C difficile was a key factor when arriving at our antibiotic policy and although this represents only one of a range of measures which have been implemented, our C difficile rates have remained low since adopting this policy.

Studies have shown narrow-spectrum agents to be as effective as broader-spectrum antibiotics in severe CAP.4 A large Australian study comparing benzylpenicillin with ceftriaxone, in combination with a macrolide or doxycycline, found similar outcomes in severe CAP.4 Furthermore, the benzylpenicillin group had lower 30-day mortality (though high-risk patients were more likely to receive ceftriaxone).5 British data in this area are lacking and further high quality studies and in-depth interrogation of the BTS audit are desirable.

The BTS audit was excellent but national data should be interpreted carefully, as forming case definitions is difficult. Local data should inform locally appropriate policies, as suggested by Mandell, and this may include narrow-spectrum agents, which combine efficacy with favourable complication profiles.

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Authors’ response

We read with interest the comments of Dr Singh and colleagues.1 We note that in the data submitted from their local population they found a lower mortality for patients with high severity community acquired pneumonia (CAP) (CURB65 score 3–5) than was found in the National cohort.2 It is not possible to precisely explain this difference from the available information, but there are a number of likely explanations. The first is sampling error, which will be inevitable when small numbers of cases are submitted from a large number of general practitioners collected over a longer time period than the British Thoracic Society (BTS) audit suggest that their lower mortality score is real. A differential response to treatment is a consideration, but since only selected aspects of patient care were documented in the audit, this aspect cannot be properly examined. The most likely explanation of all is probably case selection, which may be slightly different in each contributing centre as we alluded to in the paper. Contributors may have used a number of different methodologies to identify and correlate community CAP cases, but data on how this was done were not collected. The 2010/2011 audit may go some way in answering this difference since additional data, such as nursing home residence, were obtained.

High severity (CURB65 score 3–5) CAP has a mortality of between 15% and 40%, so treatment should primarily be used that is most likely to limit this mortality. Ideally, antibiotic recommendations would be based on robust randomised controlled comparative treatment trials, but unfortunately such trials are lacking in this area. What data are available are mainly from weak, non-robust trials or retrospective observational data such as the study referred to by the authors.3 It is therefore impossible to be certain that one antibiotic regime is categorically preferable to another in this setting. The ecological importance of good antibiotic stewardship is acknowledged in the BTS guidelines and where possible narrow spectrum agents have been recommended in preference to broad spectrum agents.4 Since only one-third of admissions for CAP are of high severity, we consider the choice of penicillin plus clarithromycin by Singh et al compared with the BTS recommendation of co-amoxiclav plus clarithromycin to be one that is unlikely to make a significant difference to antibiotic resistance or Clostridium difficile rates. Of much more importance as a driver of resistance and C difficile rates are the failures to accurately diagnose CAP and to accurately assess CAP severity. These result in overuse of antibiotics, including combination regimens primarily indicated for high severity CAP, in patients who should not require such broad therapy as identified in the audit. Penicillin plus clarithromycin may be as effective as co-amoxiclav plus clarithromycin in high severity CAP but there are no robust trial data to demonstrate this. Co-amoxiclav has the advantage of a broader antibacterial coverage, which is important for this severely ill group and easy conversion to an oral formulation for the continuation phase of treatment. We acknowledge that local data on resistance and C difficile rates may mean that an antibiotic choice other than that recommended in the BTS guidelines may be appropriate for that local setting.

We agree with the authors that National audit data should be interpreted carefully as the data collection processes at each contributing centre may not be as robust as in carefully conducted cohort studies by interested experts, but one of the strengths of the National cohort is its size. For the first
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 Smith1,2 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 and Dr Zahir Udwaadia’s article ‘MDR, XDR, TDR tuberculosis’. Both were excellent articles but their proximity raised issues of clinical research in this important, but research-neglected area.

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

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 al1 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 (Pco2) in this population. Therefore, it cannot be assumed that the Pco2 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 ischemia.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,2 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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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 al1 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 (Pco2) in this population. Therefore, it cannot be assumed that the Pco2 levels would have continued to rise after 60 min as the authors suggest.

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