are unaware of simple measurers of exacerbation severity which could have been applied in this study.

Their main criticism concerns the possible occurrence of a metabolic acidosis and the lack of significant difference in CO_2 tension between those receiving FiO_2 >0.28 and FiO_2 <0.28. In the group receiving an FiO_2 >0.28, only one patient had a predominantly metabolic acidosis (H^+ 56 nmol/1). A further five patients had a predominantly respiratory acidosis with a metabolic component (two severe and three mild acidosis). None of the patients receiving controlled oxygen had a metabolic component to their acidosis. Table 1 shows that in patients receiving an FiO_2 >0.28 at any stage during their presentation to hospital with AECOPD, the average carbon dioxide tension rose as the acidosis worsened; further, patients with acidosis had a higher mean oxygen tension than those without.

We note that Singer and Bellingan attend the A&E department to treat hypercapnoeic patients and frequently use high inspired FiO_2 with mechanical ventilation (invasive or non-invasive) to treat AECOPD as the major problems are fatigue, atelectasis, sputum retention, poor respiratory effort and cough. In the circumstances this course is perfectly correct, but as respiratory physicians we would seek to avoid this situation arising in the first place by careful titration of FiO_2 to achieve 85–90% if possible. The mechanism by which flow oxygen causes hypercapnoea (whether by altered ventilation perfusion, by the Haldane effect or by depressing hypoxic dive) is not at issue here. The fact is that we and others observe this phenomenon and believe it to be detrimental and potentially avoidable.

We are not alone in our concerns about the use of HFO. Murphy *et al* review the dangers of HFO in AECOPD showing evidence that the resultant hypercapnoea was associated with coma and death,[2] and their concerns are reflected in the guidelines produced by North West Oxygen Group (NWOG).[3] Howard and Harrison report similar findings in their prospective study in East Anglia (personal communication) identifying 27 episodes of hypercapnoea associated with HFO and hypoxia out of 175 admissions with AECOPD. The practice of liberal and unlimited oxygen administration to patients in the period leading to hospital admission in those with AECOPD is widespread and may cause additional morbidity and mortality. In some regions, ambulance services and A&E departments concur that there is a problem with COPD patients and have agreed to address it by a credit card type of self-identification as being at risk from high oxygen concentrations.

Slowranski's letter points out that we do not state how the FiO_2 of the patients was determined. This is a difficult area as oxygen prescription in hospitals is often in disarray.[4] We assumed that nasal oxygen at >2 litres/min by mask or nasal prongs was FiO_2 0.28 or greater. In many, however, 'asthma levels' of 6–10 l/min was administered and ambulance crew recorded percentages based on mask instructions whilst Lifecare masks suggests gradation from 2 l/min = 29% and 8 l/min = 60% oxygen.

We acknowledge that our prospective audit has shortcomings but it has served to further highlight a serious dichotomy between the approaches of different specialists to the problem of oxygen therapy in AECOPD. We suggest that this needs to be resolved by an adequately powered cooperative controlled trial of controlled oxygen so that guidelines can be agreed by all concerned.

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Cultural differences: practising medicine in an Islamic country

Editor – I read with pleasure Professor Al-Kassimi's article (*Clin Med* January/February 2003, pp52–3). However, an urgent correction is required in that the vaccine recommended for the Hajj is now the Meningitis ACWY not the AC. This is endorsed by both the Saudi government and the Department of Health. In the last two years many have died in the UK and abroad of Meningitis W135. Protection from this strain is given by the ACWY vaccine but not by the AC.

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Conversations with Charles

Editor – I have always enjoyed the wisdom and wit of Charles, but his latest offering (*Clin Med* November/December 2002, pp 595–6) makes me fear that the old boy is losing touch with reality. He advocates keeping information from patients and secrecy. He advances the argument that information feeds distrust. He fears that revealing the differential diagnosis may cause alarm. He worries that audit figures may be misinterpreted.

All these things are true but the cost of secrecy is far worse. Errors accumulate uncorrected. Patients understand that information is being withheld from them and find sinister explanations for this behaviour. The media smell something is being hidden and find grounds for wild conspiracy plots. Communication between patient and doctor is damaged. The patient is denied their right to develop greater understanding. The doctor denies himself or herself the opportunity to work with the patient as a co-producer of health.

Down with paternalism, long live openness and trust.

JOHN KEMM
Public Health Physician, Birmingham

In response

I asked Charles for his comments. He replied: 'I echo "Long live trust and openness", but openness should not be confused with unconsidered total disclosure. Trust allows discretion in disclosure and