hate scrolling'. In practice this leads to a staccato style and makes it read like an exam notes book. Clicking on an underlined word (here italicised) in the online version leads to a web page. Thus, one discovers that Jakob Nielsen is 'the web usability czar' and the page to which one is directed includes a photo of him which can be downloaded. Clicking on Paul Starr's *The social transformation of American medicine* leads to the Amazon site where 'our price is £17.50' but 'we are unable to offer this title'. Clicking on *Emma* leads to Amazon again but clicking on the author leads to the Jane Austen information page. Clicking on *David Halberstam* leads to a site operated by a firm called Royce Carlton who can organise that he (among others including Kay Redfield Jamieson, Oliver Sacks, Susan Sontag, Terry Waite and Jonathan Miller) come and give a talk to your organisation. Some of the links are simply distracting. To explain the attraction of complementary medicine, Muir Gray suggests that patients in some orthodox hospitals feel 'just like a number in a huge machine as awesome and impersonal as the *Metropolis* of *Fritz Lang*'. It is not clear to me why the resourceful patient should wish to know about a classical German film maker. I often find excessive referencing in medical history articles irritating, but these web links are references gone completely mad! It was a surprise to find that St Peter was not underlined in the following sentence: 'The doctor is expected to act like St Peter, holding the keys to illness, to unlock the door through which many wish to pass.' Perhaps he doesn't have a website?

The resourceful patient would find quite a lot to interest and educate him in this book if he could avoid being distracted by the web links. However, he would probably be equally well informed by reading a broadsheet newspaper every day and watching a few episodes of the American television drama *ER* (which is quoted twice with a web link).

RB TATTERSALL
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letters TO THE EDITOR

Please submit letters for the Editor's consideration within three weeks of receipt of the Journal. Letters should ideally be limited to 350 words, and can be submitted on disk or sent by e-mail to: Clinicalmedicine@rcplondon.ac.uk.

The use of oxygen in acute exacerbations of chronic obstructive pulmonary disease: a prospective audit of pre-hospital and hospital emergency management

Editor – The paper by Denniston *et al* (*Clin Med* September/October 2002, pp 449–51) strengthens previous evidence that patients with AECOPD are frequently given uncontrolled oxygen therapy which may cause respiratory acidosis. A majority of their patients (and ambulance crews) did not identify COPD as the cause of the patient's breathlessness and BTS Guidelines were not followed when COPD was correctly identified.

Because of similar problems in the North West Region, a North West Oxygen Group was formed to produce regional guidelines for emergency oxygen use in AECOPD and other causes of sudden breathlessness. The North West Oxygen Guidelines were agreed by the Regional Societies of Respiratory Physicians, Emergency Physicians and ICU specialists and published in the *Emergency Medicine Journal*.[1] These Guidelines are based on a review of the literature concerning the relative dangers of hypoxia and hypercarbia.[2] The guidelines aim at a target oxygen saturation of 90–92% for COPD patients treated in ambulances and on arrival in emergency departments prior to the availability of blood gas results. This will prevent most cases of hyperoxia and acidosis. One further issue is that patients with AECOPD are usually given oxygen-driven nebulised treatment in ambulances. The empty nebuliser chamber (with high flow oxygen-mask) is often left in place for long periods of time and may contribute to hyperoxia and acidosis. We have suggested that nebulised treatment should be limited to six minutes in these circumstances.

Like Denniston and colleagues, we are piloting a COPD alert card for patients with previous episodes of respiratory acidosis for whom a lower target oxygen saturation may be appropriate. These Guidelines have been approved by the Clinical Effectiveness Committee of the British Association of Accident and Emergency Medicine.

I can supply coloured flow-charts summarising the North West Guidelines together with a copy of the COPD Alert Card to any readers of the journal who wish to send me an e-mail at the following address: ronan.o'driscoll@srht.nhs.uk

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Editor – We agree with Denniston *et al* (*Clin Med* September/October 2002, pp 449–51) that the use of uncontrolled oxygen therapy in patients with acute exacerbations of chronic obstructive pulmonary disease (AECOPD) is common The authors indicate that they are investigating the use of a COPD card to be held by the patient. We gave a wallet-sized

laminated COPD alert card to 17 patients who had had a prior episode of acute hypercapnic respiratory failure aggravated by injudicious use of excessive oxygen. After receipt of the card there were 19 re-admissions with AECOPD but in only five (26%) of these attendances did the patients show the card. When the card was shown, however, 15 (80%) of the patients did receive an initial fractional inspired oxygen concentration (FiO_2) of no more than 0.28 in accordance with national guidelines for the management of COPD.

Although 86% of our patients still carry the card, most do not present them on admission. We now insert an alert poster in the front of case notes along with the card as some staff remain unaware of the dangers of giving excessive oxygen to COPD patients or the importance of the COPD alert card.

We suggest that the COPD card, although acceptable to the patient, will not influence management unless supported by vigorous and continuing staff and patient education.

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Editor – It was with considerable dismay that we read the audit by Denniston *et al* (*Clin Med* September/October 2002, pp449–451) purporting to show that high inspired oxygen concentrations administered to patients with acute exacerbations of their chromic obstructive pulmonary disease by emergency services resulted in a higher mortality. Our discontent is mainly directed at the peer review process of the journal that could have let this unsubstantiated, factually lacking and totally misleading travesty of a paper through to publication. Strong words, we acknowledge, but we do feel this paper does a great disservice both to the journal and to sick patients (including those with COPD) for whom oxygen is a life-saving therapy. All this audit does is simply recognise – and misinterpret – an association between a high FiO_2 requirement in sick patients and a higher subsequent mortality, in much the same way that patients requiring mechanical ventilation or defibrillation are much more likely to die than those that don't.

From what little data are provided, the authors reported **no** significant difference in $PaCO_2$ between the high and low oxygen treated groups yet the hydrogen ion concentration did differ significantly, indicating a metabolic component to the acidosis that was not even commented upon. A high arterial base deficit in emergency room patients is a well recognised adverse prognostic feature.[1] They also reported no significant difference in PaO_2 despite one group receiving a much higher FiO_2. We would thus assume that the PaO_2: FiO_2 ratio was significantly lower, indicating a much sicker group of patients receiving (needing?) high FiO_2. This supposition is supported by the far higher incidence of severe acidosis (H^+>55 nmol/l) in those receiving a high FiO_2 (25% vs 3%). Again, corresponding $PaCO_2$ values are not shown to determine the respiratory and metabolic components of the acidosis.

The authors provide no data as to the numbers of known chronically hypercapnic patients in either group, what the patients actually died of, how many in total required non-invasive or invasive ventilation, and whether those who died were offered these support options. There is not even a single mention of respiratory rate. Paradoxically, they do state that of those subsequently dying, four had a severe 'respiratory' acidosis (we think they confuse 'respiratory' for 'total' acidosis?!), two had mild acidosis, two were non-acidotic and one had an unknown acid-base status. It is thus hard to reconcile these data with loss of hypoxic drive as a cause of subsequent death. Is the extra oxygen producing another unstated toxic effect we are not made aware of?

We frequently attend the A&E department to treat such patients; despite the commonplace finding of a high $PaCO_2$, we routinely use a high inspired FiO_2 in conjunction with mechanical ventilation (non-invasive or invasive) as their major problem is fatigue often compounded by actelectasis due to shallow respiratory efforts, weak cough and sputum retention, rather than the semi-mythical loss of hypoxic drive. To allow them to remain hypoxaemic (ie below their normal baseline level) and thus struggle and tire further is contrary to all the precepts underpinning 'ABC' resuscitation and good clinical practice. Remarkably, our patients – the sicker end of the acute exacerbation of COPD spectrum – often do very well because – or in spite – of the high inspired oxygen they receive. As a simple rule of thumb, hypoxic drive is a non-issue in tachypnoeic patients; in the slow breathers the clinician has to distinguish between exhaustion, and/or other pathology or, very rarely, the possible issue of lost hypoxic drive.

Finally, our disappointment is confounded by the authors' inadequate literature review; they cite a previous lack of outcome data to confirm detriment yet make no mention of the prospective randomised study of 34 similar patients by Gomersall *et al*[2] published in January showing no harm in the group treated with adequate oxygen therapy. Your publication of this letter in a prominent position to counter the potential harm of the original paper would be greatly appreciated.

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MERVYN SINGER
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GEOFF BELLINGAN
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Editor – The retrospective study of the use of oxygen in acute exacerbations of chronic obstructive pulmonary disease by Denniston *et al* (*Clin Med* September/October 2002, pp449–51) is problematic. The authors claim that the increased incidence of severe acidosis and mortality in the group given 'uncontrolled' oxygen therapy was not due to a more severe exacerbation or more comorbidity, but

they present no data to support this. Only the triage category, age and smoking history of the two groups appear to have been compared. In addition, the authors do not state how the FiO_2 of the patients was determined; presumably it was simply estimated from the type of oxygen device to which the patient was attached.

Notwithstanding the British Thoracic Society's guidelines to which the authors refer, there is sparse clinical evidence to support the 'magical' threshold FiO_2 of 0.28, nor for the ingrained dogma, first proposed by Campbell[1] on a purely anecdotal basis in 1967, that a high FiO_2 is a clinically important cause of worsening respiratory acidosis and mortality.

Bone *et al*[2] found in 1978 that 26% of acutely ill COPD patients became stuporous and required mechanical ventilation despite 'controlled' oxygen therapy, suggesting indirectly that any contribution of uncontrolled oxygen therapy was likely to be minor.

In a recent small, prospective, randomised study[3] in which oxygen therapy was titrated to either a low (6.6–9 kPa) or high (>9 kPa) arterial PO_2, the only deaths or deteriorations requiring mechanical ventilation were in the low PaO_2 group.

If the clinical relevance of uncontrolled oxygen therapy remains uncertain, the proposed mechanism by which it might cause CO_2 retention is even more so. It now seems unlikely that a dependence on hypoxia to drive ventilation is important. More recent studies suggest that a high FiO_2 can worsen ventilation-perfusion matching or otherwise increase dead space ventilation.[4] The Haldane effect, in which oxygen displaces CO_2 bound to haemoglobin, may also be involved.

Little wonder 'the need for controlled oxygen therapy … may be dismissed by intensivists'. Perhaps they are right!

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In response

We write in response to the letters of Singer and Bellingan and that of Skowronski. Our audit in 97 consecutive patients admitted over the months of March and April 2000 was carried out because of concern that previous patients had deteriorated over the period of emergency assessment, with elevated $PaCO_2$ and respiratory acidosis, improving after empirical reduction of high flow oxygen treatment (HFO). Their letters indicate that we have stepped into a controversial debate in which it seems there is a lack of agreement between intensivists, emergency specialists and respiratory physicians over the role of oxygen treatment in AECOPD. In general, it appears that the former two groups believe that the commencement of HFO in pre-admission scenarios is justified because of hazards of hypoxia when the diagnosis is not known. This perfectly correct treatment and counsel of safety for the majority, however, takes little account of the fact that amongst these, will be patients with AECOPD who go on to develop worsened type 2 respiratory failure during the transit and waiting period prior to admission under a respiratory or acute receiving physician. A recent study by Plant *et al* reported that oxygen-induced hypercapnoea and respiratory acidosis were a common problem in patients presenting with this condition.[1] Our audit confirmed this and showed that of 24 who received HFO and had a respiratory acidosis, 13 improved on being given FiO_2 <0.28. Two worsened but in eight there were no subsequent arterial blood gas measurements.

The letter of Singer and Bellingan alleges that our study simply recognises and misinterprets an association between a high FiO_2 requirement and higher subsequent mortality. We cannot deny that some patients in the high FiO_2 group may have been more ill and with a higher oxygen requirement than those in the low FiO_2 group. In this prospective audit we also cannot confirm that our nine deaths occurred in direct relation to their respiratory acidosis though this factor cannot have helped them. The causes of death in nine patients were recorded as follows: respiratory failure/COPD in six, pneumonia in two and myocardial infarction in one. Two in the controlled oxygen group were ventilated with full intubation or NIPPV. None of those who died were offered or received assisted ventilation.

Data collection in any audit is necessarily limited to allow completion within the constraints of busy hospital practice. Detailed retrospective comorbidities, pre-admission spirometry or arterial blood gases or similar stable state post-recovery measurements were available for few and are one of the limitations of our audit. We

Table 1. Blood gas tensions for 57 patients with acidosis had a higher mean oxygen tension than those without

	Severe acidosis (H^+ >55 nmol/l)	Mild acidosis (H^+ 45-55 nmol/l)	No acidosis
H^+ (nmol/l)	63.9 (56.7, 80.4)	49.8 (47.4, 51.6)	39 (36.3, 40.3)
$PaCO_2$ (kPa)	12.03 (11.1, 13.9)	7.8 (6.9, 8.8)**	5.94 (5.3, 7.4)**
$PaCO_2$ (kPa)	11.3 (7.9,13.6)	12.8 (9.9, 16.3)	8.9 (7.95, 12.5)*

Data presented as median (interquartile range); *p <0.05 compared to patients with mild acidosis: ** p <0.01 patients with mild vs. severe acidosis and no acidosis vs. mild acidosis.

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