High inspired oxygen in hypercapnic respiratory failure

Sir,

Mbamalu et al's study (vol 68(3), 2007, p. 156) showed poor knowledge of oxygen (O_2) therapy among senior house officers. Two clinical scenarios involving hypoxic patients were presented; one with uncompensated acute respiratory acidosis and another with hypercapnic (type 2) respiratory failure. Respondents were deemed correct in treating the first patient with a high concentration of O_2 (FiO_2), but incorrect if they did so for the second. The explanation that in patients with type 2 respiratory failure 'there is a definite risk of suppression of the hypoxic drive … with the resultant development of carbon dioxide (CO_2) narcosis in the presence of higher inspired oxygen concentrations' is worthy of comment.

A high FiO_2 in a patient with type 2 respiratory failure will increase arterial CO_2 tension ($paCO_2$). Donald (1949) hypothesized that this is the result of blunting of the hypoxic ventilatory drive, leading to recommendations that oxygen therapy be limited in such patients. This has become entrenched in medical folklore.

Aubier et al (1980) argued to the contrary. In 22 patients with type 2 respiratory failure, supplemental O_2 produced only a small decrease in minute ventilation. $paCO_2$ rose more than could be explained by this decrease, and the additional rise was thought to be caused by O_2-induced ventilation/perfusion (V/Q) mismatch. Multi-compartment modeling of Aubier's data found that the increase in $paCO_2$ was caused by reduced minute ventilation, the Haldane effect and changes in V/Q matching (Hanson et al, 1996). However, Stradling (1986) argued that the small change in tidal volume in these patients would have produced a larger fall in physiological dead space which explains the raised $paCO_2$.

There is not space here to explore the reasons for these different results, but trainees should note this controversy, if only in the context of their postgraduate exams.

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Aubier M, Murciano D, Milic-Emili J, Touaty E, Daghfous J, Pariente R, Derenne JP (1980) Effects of the administration of O2 on ventilation and blood gases in patients with chronic obstructive pulmonary disease during acute respiratory failure. *Am Rev Respir Dis* **122**(5): 747–54
Donald KW (1949) Neurological effects of oxygen. *Lancet* **ii**: 1056–7
Hanson CW III, Marshall BE, Frasch HF, Marshall C (1996) Causes of hypercarbia with oxygen therapy in patients with chronic obstructive pulmonary disease. *Crit Care Med* **24**(1): 23–8
Stradling JR (1986) Hypercapnia during oxygen therapy in airways obstruction: a reappraisal. *Thorax* **41**(12): 897–902

Sir,

Mbamalu et al's article suggests that there is a deficiency in junior doctors' understanding of oxygen therapy. We believe that the questionnaire upon which the study is based is misleading and factually incorrect.

Question 1 requires a choice based on the colour of the Venturi device. It is far more accurate to stipulate the required FiO_2.

In question 2 the patient has acute type 2 respiratory failure and is markedly acidotic. A pH <7.25 as a result of respiratory acidaemia is associated with increased risk of admission to intensive care, intubation and death; pH in turn is inversely correlated with arterial O_2 tension (Plank et al, 2000). Current British Thoracic Society (1997) guidelines suggest that PaO_2 be maintained at >6.6 kPa if pH <7.26, or at >7.5 kPa if the patient is not acidotic. Oxygen should initially be given at 28% and titrated to balance hypoxia and respiratory acidaemia. National Institute for Clinical Excellence (2004) guidelines suggest that non-invasive ventilation (NIV) be offered if the pH remains <7.35 despite oxygen titration and maximal medical management.

With this in mind, question 2 is nonsensical as it asks 'what the maximum safe FiO_2 should be', whereas 'what FiO_2 should be prescribed initially' would have been more appropriate. The patient in the example should have been considered for NIV.

The discussion states that only those with chronic partially compensated respiratory failure are at risk of hypoventilation and hypercarbia as a result of suppression of the hypoxic drive. It is thought that mechanisms involving oxygen-induced atelectasis and resultant V/Q mismatch offer a more accurate explanation for oxygen-induced hypercapnia (Dunn et al, 1991). These mechanisms apply in both in acute and acute on chronic settings, and should not be managed differently with regard to oxygen therapy as suggested in this study.

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British Thoracic Society (1997) BTS guidelines for the management of chronic obstructive airways disease. *Thorax* **52** (suppl 5): S1–S27
Dunn WF, Nelson SB, Hubmayr RD (1991) Oxygen induced hypercarbia in obstructive pulmonary disease. *Am Rev Resp Dis* **144**: 526–30
National Institute for Clinical Excellence (2004) National clinical guideline management of chronic obstructive pulmonary disease in adults primary and secondary care. *Thorax* **59** (supp1 1): S1–S232
Plank PK, Owen J, Elliot MV (2000) One year period prevalence study of respiratory acidosis in acute exacerbation of COPD: implications for the provision of non-invasive ventilation and oxygen administration. *Thorax* **55**: 550–4

Sir,

We are grateful for these comments. Reade gives a balanced account of the pros and cons of oxygen therapy in relation to hypoxic drive in chronic hypercapnic therapy, and we agree that the jury is out on the mechanism by which oxygen acts in this situtation. We kept questions consistent with widespread clinical practice in the UK, and they were framed in a single best answer format, which does not preclude other options.

With regard to the comments by Fletcher and Warley, question 1 tested understanding of a range of oxygen delivery devices matched to appropriate flow rates, as available in an emergency department. Reference to colour coding in two stems tested the fact that flow rates are related to colour codes. The FiO_2 of 60% was stipulated in the question.

Question 2 required a single best answer to the options provided. It stipulated that blood gases were obtained on arrival and on room air, hence immediate consideration of NIV is invalid. Giving 28% oxygen initially to a patient with type 2 respiratory failure is dangerous in acute respiratory acidosis. The highest possible FiO_2 should be provided, and this can be titrated to clinical response and repeat arterial blood gases.

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