

Revisiting oxygen therapy in patients with exacerbation of chronic obstructive pulmonary disease

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Controlling oxygen delivery to limit oxygen saturation should reduce the incidence of hyperoxic hypercapnia

The report by Joosten et al in this issue of the Journal (page 235)¹ is a timely reminder of the importance of avoiding the induction of hyperoxic hypercapnia in patients with acute-on-chronic respiratory failure. The complication of acute hypercapnic respiratory failure precipitated by giving oxygen has long been recognised; most resident medical and nursing staff are aware of this problem. The natural intervention in patients presenting with acute-on-chronic respiratory failure is to relieve any hypoxia with supplemental oxygen, but this can be associated with carbon dioxide retention, narcosis, respiratory acidosis, and death. That the use of controlled oxygen flow rates could avoid this complication (and the need, in those days, for tracheostomy and invasive ventilation) was first recognised in the 1940s and 1950s.² Although hyperoxic hypercapnia can now be managed with non-invasive ventilation, the article by Joosten et al reminds us that it still has adverse consequences for morbidity, length of stay and the use of hospital resources.¹

Studies over the past 20–30 years have identified the characteristics of the patients most likely to have this problem, and have gone some way to identifying the mechanisms responsible. Chronic respiratory failure is the usual predisposing condition, and the most common cause of chronic respiratory failure is chronic obstructive pulmonary disease (COPD). Interestingly, hyperoxic hypercapnia is a phenomenon of acute exacerbations of COPD — giving oxygen to patients with stable hypercapnia rarely, if ever, causes clinically significant further hypercapnia.³ This may, of course, be related to the lower flow rates used for stable hypercapnia. Importantly, the degree of hypoxaemia at presentation is a better predictor of hyperoxic hypercapnia progressing to narcosis than is the initial degree of hypercapnia.⁴

Usual clinical teaching is that high concentrations of inspired oxygen remove the hypoxic drive to ventilation in susceptible hypoxaemic patients; the narcotic effect of the rising hypercapnia amplifies this effect, promoting further hypoventilation. However, a number of studies have cast doubt on this as the most important mechanism, at least up to the point of narcosis.^{5–7} The most comprehensive study of mechanisms, using the multiple inert gas elimination technique, suggests that relative hypoventilation is the defining event in those who retain carbon dioxide, but that worsening ventilation–perfusion mismatching and an accompanying increase in dead space ventilation contribute about 50% of the increase in carbon dioxide levels.⁸ This finding is clinically important because it identifies non-invasive ventilatory support as the appropriate intervention before narcosis progresses.

The dictum “hypoxia kills quickly, hypercapnia slowly” engages the clinician when confronted with this situation. How can hyperoxic hypercapnia be avoided without exposing these patients to the more acute risk of inadequate oxygenation? Oxygen delivery controlled to an appropriate flow rate appears to be the answer, but there are no large-scale studies to indicate how the oxygen “dose”

should be determined and monitored.^{9,10} In the absence of clinical trial evidence, it is reasonable to control oxygen flow rate to achieve an arterial oxygen saturation of 90%, but not above 93%–95%. This corresponds with an arterial oxygen tension of 60–70 mmHg at the start of the “flat part” of the oxyhaemoglobin dissociation curve, and ensures adequate arterial oxygen content and delivery in most circumstances. This is also consistent with the data of Joosten and colleagues, who found an arterial oxygen tension of less than 74.5 mmHg to be protective.¹ The ready availability of continuously reading pulse oximeters makes the above recommendation a practical procedure, and its wide application in wards, emergency departments and, particularly, ambulances should substantially reduce the incidence of the hazardous and largely unnecessary complication of hyperoxic hypercapnia.

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