high-concentration oxygen therapy causes a clinically significant increase in PtCO₂ and they recommended the use of a titrated oxygen regime in the treatment of severe asthma. These results mirror those of a similar study performed recently in patients with COPD. In this randomised, controlled, prehospital study, participants allocated to titrated oxygen therapy were significantly less likely to have respiratory acidosis (mean difference in pH 0.12; SE 0.05; p=0.01; n=58) or hypercapnoea (mean difference in PaCO₂ 33.6 mm Hg; SE 16.3; p=0.02; n=59) than patients receiving high-concentration oxygen. Treatment with titrated oxygen was also associated with a 58% reduction in mortality, the primary outcome in this study.

As asthma and COPD are prevalent diseases in the Western world, and acute exacerbations of either are associated with an increased risk of death, it is beholden to health professionals to ensure that they do not contribute to this outcome. We now have strong evidence to support the BTS guidelines on emergency oxygen use, which recommend that it be approached in the same way as any other drug, recognising that adverse outcomes may eventuate from either inappropriately low or high concentrations. Should the guidelines be revised in the light of this new evidence to better align recommendations with the philosophy of keeping arterial oxygen saturations ‘within the target saturation range’ that aim to ‘achieve normal or near-normal oxygen saturation’ and move away from any suggestion that high-concentration oxygen should be administered in the absence of objective evidence of a physiological need? With the advent of low-cost portable oxygen saturation monitors, surely it is time we followed the guideline exhortations to measure the fifth vital sign, as in the words of Willy Wonka ‘it’s the only way if you want it just right’.

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Who will benefit from tracheostomy ventilation in motor neuron disease?

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The decision when to recommend tracheostomy ventilation in motor neuron disease (amyotrophic lateral sclerosis) has always been difficult. At one extreme is the view that when spontaneous ventilation or non-invasive ventilation is inadequate, a tracheostomy will save the patient’s life and lead to prolonged survival. This view has been more widely held in the USA than elsewhere. In the UK, a common position is the opposite, with a nihilistic attitude towards invasive respiratory treatment. The rationale behind this is that it is too intrusive, both for the patient and for the family and carers, and that once a tracheostomy is needed, palliative care is more appropriate.

Not surprisingly, there has been a wide geographical variation in the proportion of patients who proceed to a tracheostomy, and the review by Sancho et al is timely. The authors describe a 9-year experience in a specialist respiratory carer unit where the issues surrounding tracheostomy ventilation were openly discussed with each patient who might benefit from it. Out of 76 subjects 38 refused. Unfortunately, no further data are provided about these patients to compare their outcomes in terms of quality of life with the 38 who underwent a tracheostomy but their mean survival was only 0.83 months.

Interestingly, over half of those who underwent a tracheostomy did so during an acute severe chest infection in which non-invasive ventilation was either ineffective or not indicated. These patients were transferred from endotracheal intubation to tracheostomy ventilation. The indications were otherwise untreated ventilatory failure or the need for access to tracheobronchial secretions during and after the infection. The mean survival after tracheostomy was 10.76 months, which was similar to the mean survival when tracheostomy was carried out electively.

As has also been reported in a recent study, some of these patients eventually did not require continuous ventilatory support, but there is no mention of whether any could be weaned onto non-invasive ventilation once they recovered from their acute illness. Another report, however, suggests that almost half of those who undergo tracheostomy ventilation in this situation can eventually be weaned onto non-invasive support. Their survival is as good as those who still require tracheostomy ventilation but they are more likely to be able to return home.

These encouraging findings suggest that there is a need for a re-appraisal of the management of severe chest infections in motor neuron disease. A much more active approach needs to be taken by intensivists, neurologists and respiratory physicians involved in their care than has been the standard practice in the past.

The indications for elective tracheostomy ventilation were either an inability to provide adequate ventilatory support...
non-invasively or the need to protect the airway despite the use of manually assisted coughing. It has been demonstrated that non-invasive systems can almost invariably adequately ventilate the subject and it is a defective swallow or cough that usually leads to the need for a tracheostomy4 apart from the convenience of being ventilated through this route if respiratory support is needed virtually or completely continuously.

Complications of the tracheostomy itself were infrequent, but almost one-third of the patients required admission for treatment of a chest infection despite their tracheostomy. There was only one death from a respiratory infection in this series and overall 78.9% of patients survived for 1 year. Interestingly, nine patients died suddenly raising the possibility that there is a defect in sympathetic activation in response to stress as part of the neurological deficit in motor neuron disease.

These findings present an optimistic view of tracheostomy ventilation, but quite frequent psychiatric assessments were required, mainly for anxiety and depression. It could be argued that depression is a feature of motor neuron disease and the function changes.1 Furthermore, the findings of muscle atrophy and muscle biopsy6 corroborate by breathlessness and exercise limitations when forced expiratory volume in 1 s is corrected after double lung transplantation. Thus, muscle dysfunction has led to a particular focus upon the skeletal muscle compartment. Certainly, a surprisingly high proportion of patients with COPD terminate exercise complaining of muscle effort.4 Cross-sectional studies have typically demonstrated muscle weakness5 and reduced endurance,6 particularly of the lower limbs, in COPD compared with age-matched controls. This is corroborated by biopsy findings of muscle fibre atrophy and muscle fibre shift from type I to type II fibres.7 Skeletal muscle dysfunction seems to be clinically relevant in COPD, as

Muscle mass and strength in obstructive lung disease: a smoking gun?

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Over the past 2 decades, there has been increasing interest in the extrapulmonary manifestations of chronic obstructive pulmonary disease (COPD). This is supported by the clinical observation that patients show significant improvements in functional performance and health-related quality of life with pulmonary rehabilitation in the absence of lung function changes.1 Furthermore, the forced expiratory volume in 1 s is an imperfect predictor of mortality in severe patients with COPD,2 and when forced expiratory volume in 1 s is corrected after double lung transplantation, peak exercise remains only approximately 50% of predicted up to 1–2 years after surgery.3 This limitation in exercise and functional capacity has led to a particular focus upon the skeletal muscle compartment. Certainly, a surprisingly high proportion of patients with COPD terminate exercise complaining of muscle effort.4 Cross-sectional studies have typically demonstrated muscle weakness5 and reduced endurance,6 particularly of the lower limbs, in COPD compared with age-matched controls. This is corroborated by biopsy findings of muscle fibre atrophy and muscle fibre shift from type I to type II fibres.7 Skeletal muscle dysfunction seems to be clinically relevant in COPD, as

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