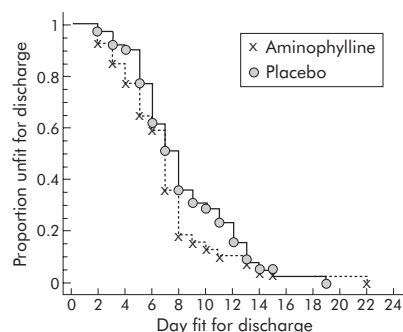


AMINOPHYLLINE AT COPD EXACERBATIONS

More effective treatment of COPD exacerbations is an important goal of COPD therapy. A number of guidelines have suggested that intravenous aminophylline can be used to treat COPD exacerbations when the response to usual treatment is inadequate. However, the evidence for this option is weak and the few previous trials of aminophylline at exacerbation have been small and generally underpowered for relevant outcomes. In this month's *Thorax*, Duffy and colleagues report the first adequately powered randomised trial of intravenous aminophylline in 80 patients admitted to hospital with COPD exacerbations, and the study is discussed in the accompanying editorial by Town. The study found that the addition of intravenous aminophylline to nebulised bronchodilators and oral corticosteroids in non-acidotic exacerbations produced small improvements in acid-base balance, but there was no effect on the subsequent clinical course. There were no benefits on the severity of breathlessness or length of hospital stay between the two groups. The authors conclude that intravenous aminophylline confers no benefit at COPD exacerbation and its use can potentially increase side effects.

See pages 709 and 713



Kaplan-Meier plot of the proportion of patients remaining in hospital in the two treatment groups

AIRWAY GLUCOSE AND MRSA

Nosocomial infection is increased in critically ill patients in the presence of hyperglycaemia. Philips and colleagues describe an interesting study of the relation of airway glucose to susceptibility to infection. Glucose was detected in the airways in over half of the critically ill patients studied, and these patients were more likely to have pathogenic bacteria in their airways. Patients with airway glucose were also more likely to have methicillin resistant *Staphylococcus aureus* (MRSA) infection. These novel observations are discussed in the accompanying editorial by Brown who concludes that further research is needed to study the potential mechanisms, confirm that airway glucose predisposes to infection, and evaluate whether lowering these glucose levels will reduce the risk of infection.

See pages 711 and 761

COMBINING INHALED STEROIDS AND LABA IN ASTHMA

Although asthma guidelines suggest that a long acting β agonist (LABA) should be added to inhaled corticosteroids if control is inadequate, there is uncertainty as to the dose of inhaled corticosteroids at which to start concomitant LABA therapy. In this issue of *Thorax*, Masoli and colleagues describe a meta-analysis of randomised trials that compared the efficacy of adding salmeterol to moderate doses of inhaled steroids (fluticasone 200 μ g or equivalent) with increasing the inhaled steroid dose. The results show that a greater benefit on asthma control was achieved with the combination therapy than with increasing the inhaled steroid dose. These results are discussed in an editorial by Tattersfield who concludes that the findings on the combined use of inhaled corticosteroids and LABA should now enable firmer advice to be incorporated into guidelines.

See pages 710 and 730

HOW DOES LONG TERM NIV WORK?

Long term home non-invasive ventilation (NIV) is a very effective treatment, particularly for restrictive wall disease, and the use of nocturnal ventilation can maintain daytime symptomatic and physiological improvements for many years. However, the exact reasons for these benefits are not known and, in this month's *Thorax*, Nickol and colleagues address the various hypotheses that have been used to explain the improvement. The authors conclude that the increased ventilatory response to carbon dioxide with NIV is the principal mechanism responsible for the long term improvement in gas exchange. No increases were observed in measures of lung function or inspiratory muscle strength.

See page 754

EARLY AND LATE HYPERINFLATORS?

We now recognise that dynamic hyperinflation contributes to the sensation of dyspnoea that leads to exercise limitation in patients with COPD. However, some COPD patients do not hyperinflate progressively with exercise but still become breathless. In this issue of *Thorax*, Vogiatzis *et al* describe the results of a study which investigated the pattern of change in chest wall volumes in patients who hyperinflate and in those who do not hyperinflate on exercise. They found that the patients fell into two groups: one group hyperinflated early in exercise and the other group hyperinflated late and, despite the group differences, the exercise capacity was similar. These results have important implications for the design of exercise programmes during pulmonary rehabilitation.

See page 723