Airwaves

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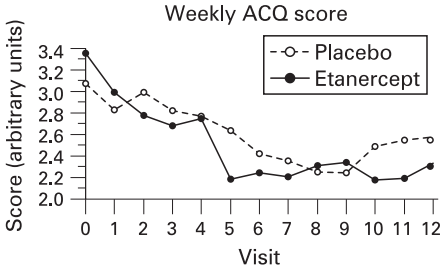
TNFα in refractory asthma

As Berry and Pavord point out in their editorial in this month's *Thorax*, asthma refractory to treatment with inhaled corticosteroids is an important health and economic problem and unmet clinical need. Tumour necrosis factor (TNF) α has been implicated in the mechanisms of refractory asthma, where TNFα production is increased, and this has led to interest in TNFα antagonists for asthma management. Morjaria and colleagues report a randomised controlled trial of a TNFα antagonist (etanercept) in corticosteroid refractory asthma. After 12 weeks there was a small improvement in asthma control, but no effect on quality of life, lung function, bronchial hyperresponsiveness and eosinophilic or neutrophilic airway inflammation. A fall in C-reactive protein (CRP) reflecting systemic inflammation was also noted. The authors conclude that larger trials are required that are adequately powered for the relevant outcomes. The editorial also concludes that future studies need careful patient characterisation with recruitment of the at-need population. ***See pages 571 and 584***



Changes in Asthma Control Questionnaire (ACQ) over the 12-week study period in the etanercept and placebo groups.

OSA, CPAP and vascular function

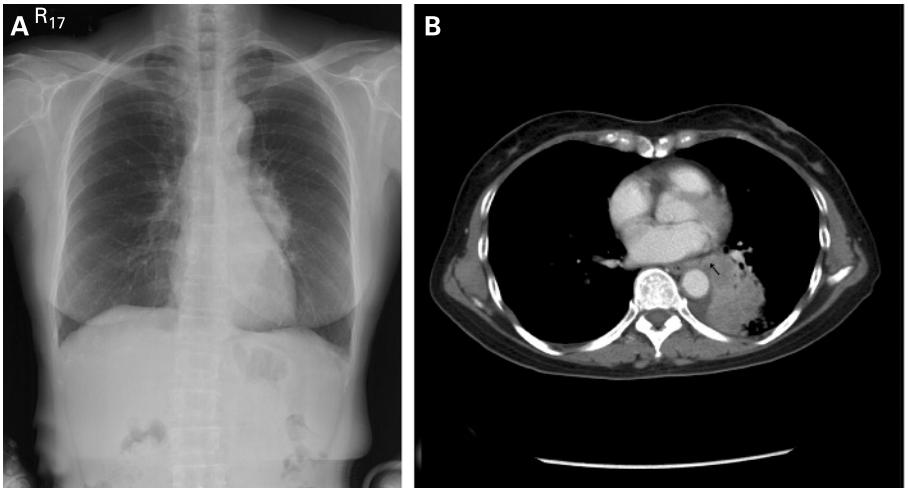
We now know that obstructive sleep apnoea (OSA) is associated with hypertension and increased cardiovascular risk. Rises in blood pressure occur at the ends of the apnoeas and hypopneas and are greater with more sleep-related hypoxaemia. In this issue, Cross and colleagues describe a randomised controlled trial of continuous positive airway pressure (CPAP) treatment in two OSA patients groups, one with significant arterial oxygen desaturation and the other with less desaturation. The results show that patients with OSA and frequent nocturnal desaturations have impaired endothelial dependent and endothelial independent vasodilation, which is related to the degree of hypoxaemia and improved by CPAP treatment. This study provides a mechanism for the increased cardiovascular risk seen in these patients. ***See page 578***

Furosemide and dyspnoea in COPD

Dyspnoea is an important symptom in chronic obstructive pulmonary disease (COPD), leading to considerable disability and impairment of health status. There have been reports that inhaled furosemide reduces intensity of dyspnoea in COPD. In this month's *Thorax*, Jensen and colleagues study the mechanisms of this effect with a randomised placebo cross-over study in patients with advanced COPD. The authors show improvements in dyspnoea intensity and exercise endurance with furosemide. These changes were multifactorial and associated with changes in airway function and ventilatory mechanics. Thus, further assessment of inhaled furosemide as a treatment for dyspnoea associated with disabling COPD is now required. ***See page 627***

Alpha-1-antitrypsin augmentation and neutrophil elastase

It is well known that neutrophil elastase is increased in the lungs of patients with such conditions as alpha-1-antitrypsin (A1AT) deficiency and pneumonia. In this issue, Geraghty and colleagues report on a study of bronchoalveolar lavage (BAL) from these patient groups and show that the BAL contained free neutrophil elastase and increased cathepsin-B and matrix metalloprotease 2 (MMP-2) activities. A1AT augmentation treatment in the A1AT-deficient patients reduced cathepsin-B and MMP-2 activity in the BAL with higher levels of secretory leucoprotease inhibitor and lactoferrin found. This study shows a novel role for A1AT augmentation treatment. ***See page 621***



(A) Chest radiograph showing left lower lobe consolidation and (B) CT scan with contrast medium showing highly suspicious feeding artery arising from the descending aorta (arrow). See Pulmonary puzzle, ***page 620***