airwaves

Wisia Wedzicha, *Editor in Chief*

MECHANISMS OF WEIGHT LOSS IN COPD

Chronic obstructive pulmonary disease (COPD) is characteristically associated with important systemic features such as weight loss, skeletal muscle atrophy, and systemic inflammation. Muscle atrophy leading to weight loss in COPD is associated with a poor prognosis, although our understanding of the mechanisms of weight loss in COPD is limited. In this issue of *Thorax* Agusti and colleagues report the results of a study of the upregulation of the nuclear transcription factor NF-κB in muscle biopsy specimens of patients with COPD. They found a 30% increase in NF-κB activity in patients of low body weight compared with those of normal body weight, and upregulation of the inducible form of nitric oxide synthase (iNOS) in the skeletal muscle of low body weight COPD patients. The authors also suggest that the higher levels of nitric oxide (NO) in the presence of upregulated iNOS can contribute to skeletal muscle atrophy either by enhancing skeletal muscle apoptosis or stimulating protein nitrotyrosination. These molecular mechanisms described will be an important basis for the development of new therapeutic modalities for weight loss in COPD.

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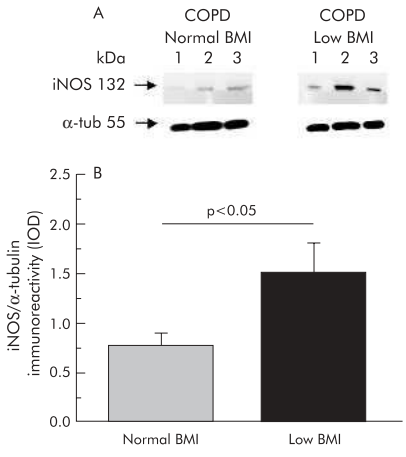

(A) Representative Western blot of iNOS in the quadriceps femoris muscle of three COPD patients with normal BMI and three patients with low BMI. (B) Mean (SE) iNOS expression (normalised by α-tubulin content) in each group.

QUALITY OF LIFE AND SLEEP APNOEA

Sleep apnoea has a considerable impact on patients' quality of life, although the effect of treatment is mainly measured by changes in the apnoea-hypopnoea index, symptoms, or Epworth scores, and few studies have used specific quality of life instruments. In this month's *Thorax* Lacasse and colleagues describe a 32-item sleep apnoea specific quality of life questionnaire that can be easily administered in clinical practice. The authors show that the Quebec Sleep Questionnaire (QSQ) is sensitive to treatment induced changes and thus can be used in clinical trials of sleep apnoea treatment. In the accompanying editorial Flemons discusses the development of quality of life questionnaires, describes some issues regarding the minimal important clinical differences, and concludes that the development of the QSQ establishes an important standard for specific sleep apnoea questionnaires.

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DYSPNOEA IN HEART FAILURE

Over the years there has been considerable controversy about the mechanisms of dyspnoea in heart failure. Although it was originally suggested that patients with chronic heart failure have inspiratory muscle weakness, later studies using more modern methodology have shown that muscle weakness is not the cause of dyspnoea in heart failure. Hart and colleagues show that, in patients with moderate heart failure associated with dyspnoea, endurance time is significantly shorter because of the breathing pattern adopted, resulting in an increased inspiratory muscle load relative to the inspiratory muscle capacity. Therapeutic strategies in chronic heart failure should therefore be aimed at correcting the maladaptive breathing pattern in order to reduce dyspnoea.

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WHY IS EMPYEMA INCREASING IN CHILDREN?

As reported in the paper by Eastham and colleagues, empyema and parapneumonic effusion complicating pneumonia have increased in children in the UK over the last 10 years. In the USA, *Streptococcus pneumoniae* is the most common bacterial pathogen found in childhood empyema, with serotype 1 accounting for up to half of culture positive cases. However, in the UK, serotype 14 has been the most common cause of pneumococcal pneumonia and current pneumococcal vaccines do not protect against serotype 1. Detailed bacteriological analysis was performed including the use of molecular methods, and *S pneumoniae* serotype 1 was also found to be the dominant serotype in empyema in this UK study. This paper has some important implications in that the results support the use of routine pneumococcal immunisation in childhood with appropriate vaccines. Further research is required into the reasons for the increase in the particular pneumococcal serotype.

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WHEEZING AFTER RSV INFECTION

Respiratory syncytial virus (RSV) causes lower respiratory tract infections in infants and leads to wheezing episodes after the initial infection, but little information is available on the time course of the wheezing episodes after RSV infection. In this issue of *Thorax* Bont and colleagues report a study from the Netherlands in which infants were closely followed after RSV infection requiring admission to hospital. Daily symptoms were monitored by the parents until the infants reached the age of 3 years. A significant decrease in wheezing was found during the first 12 months of follow up with most of the wheezing in infants with a previous RSV lower respiratory tract illness occurring in the winter months. These results suggest that respiratory viruses were the major cause of these wheezing episodes. Allergic risk factors did not predict the development of wheezing. This study emphasises the importance of regular daily follow up to understand the natural history of a disease.

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