

## The breath of life

### Inspiratory muscles and dyspnoea in chronic heart failure

Breathlessness is common in chronic heart failure (CHF), but the role of inspiratory muscle dysfunction has not been clarified. Investigators at the Royal Brompton Hospital in the UK hypothesised that inspiratory muscle endurance, expressed as a function of endurance time ( $T_{lim}$ ), adjusted for inspiratory muscle load and inspiratory muscle capacity, would be reduced in CHF.<sup>1</sup>

Endurance was measured in 10 healthy controls and 10 patients with CHF. Although a marked reduction in endurance time was observed in CHF, much of this reduction was explained by the increased inspiratory muscle load to capacity ratio, suggesting that the major contributor to task failure was a maladaptive breathing pattern rather than impaired inspiratory muscle endurance.

### Quality of life measurement in sleep apnoea

It is accepted that symptoms of sleep apnoea have an effect on the patient's quality of life. When patients are treated for sleep apnoea, it is important to document the efficacy of the treatment. Most often, this evaluation is limited to determining whether the apnoea-hypopnoea index has been satisfactorily reduced, and not often enough is the effect on quality of life measured as well. If it is accepted that what matters most to patients is quality of life, an adequate method of evaluation should be used. Generic indices are not satisfactory for this evaluation as they measure quality of life at a particular moment in time, and not the change that occurs within treatment periods.

A disease-specific questionnaire, the Quebec Sleep Questionnaire (QSQ), has been designed as an evaluative instrument, which is sensitive to change with treatment. Sixty patients participated in a study undertaken to validate the QSQ for use in clinical trials.<sup>2</sup>

The results showed that there were significant differences in score changes between patients who were treated and those who were not. The QSQ was determined to be a valid measure of health-related quality of life in patients with obstructive sleep apnoea and is sensitive to treatment-induced changes.

### Doubling the dose of budesonide v. maintenance treatment in asthma exacerbations

Asthma exacerbations are common, and in recent years there has been an increase in the prevalence of asthma and asthma-related morbidity. Previous guidelines recommend doubling



the daily dose of maintenance inhaled corticosteroids to treat or prevent progression of exacerbations of asthma.

A cohort of patients were evaluated prospectively in a double-blind controlled trial. They were randomised to receive either a continued maintenance dose of inhaled corticosteroids, or doubling the dose at the time of an exacerbation.

A total of 98 patients experienced evaluable asthma exacerbations during the study period of 6 months. The primary outcome measure was defined as the proportion of patients who, after developing an exacerbation of their asthma, failed to regain control after introducing the additional inhaler, judged by the need for treatment with systemic steroids or an unscheduled visit to a physician or medical emergency department, or their asthma did not return to baseline.

The results showed that there was no difference between failures in the group that received a double dose and the group that received the maintenance dose (41% v. 40%). The authors concluded that in patients who regularly take an inhaled corticosteroid, doubling the maintenance dose may not affect the pattern of exacerbation.<sup>3</sup>

#### Systemic inflammation and COPD

Among the complications of chronic obstructive pulmonary disease (COPD) are atherosclerosis, cachexia, anorexia and osteoporosis. There is increasing recognition of systemic inflammation as a risk factor for these complications. A study was undertaken to determine whether systemic inflammation is present in stable COPD.

A systematic review was conducted of studies which reported on the relationship between COPD, and levels of systemic inflammatory markers: C-reactive protein (CRP), fibrinogen, leucocytes, tumour necrosis factor- $\alpha$  (TNF- $\alpha$ ) and interleukins 6 and 8.

Fourteen original studies were identified. In the pooled results, it was shown that reduced lung function is associated with increased levels of systemic inflammatory markers which may have important pathophysiological and therapeutic implications for patients with stable COPD.<sup>4</sup>

#### Urgent requirement for smoking cessation policies in developing countries

Developing countries face a rapidly escalating epidemic of tobacco use. The World Health Organisation predicts that, if current patterns of consumption continue, more than 500 million people alive today will be killed by tobacco by 2030. Tobacco control will thus need to be a priority for policy makers in developing countries. Unfortunately, many barriers must be overcome for cessation strategies to be implemented broadly and successfully.<sup>5</sup>

Obstacles to promoting smoking cessation in developing countries include:

- economic factors – many low-income countries grow exportable cash crops including tobacco
- lack of awareness by policy makers of the health consequences and costs of tobacco use
- low perception of risks among the public
- lack of policies that promote cessation
- smoking behaviour among health care service providers
- poor public health care systems
- lack of infrastructure and technology such as telephone quit lines
- industry action – aggressive marketing by cigarette manufacturers.

Developing countries can introduce broad policy approaches:

- interventions to reduce smoking among health care providers
- national government commitment in implementing a comprehensive approach to cessation
- developing a model for developing countries — these could be based on successful models used in developed countries
- changing the social acceptability of smoking.

The South African Government has, fortunately, taken large steps along the right road to tobacco control.

#### FNS

1. Hart N, *et al. Thorax* 2004; **59**: 477-482.
2. Lacasse Y, *et al. Thorax* 2004; **59**: 494-499.
3. FitzGerald JM, *et al. Thorax* 2004; **59**: 550-556.
4. Gan WQ, *et al. Thorax* 2004; **59**: 574-580.
5. Abdullah ASM, *et al. Thorax* 2004; **59**: 623-630.