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EDITORIAL

Sleep apnoea screening in heart failure? Not until benefit is proven!

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In the current issue of *European Respiratory Journal*, in a German multicentre study, SCHULZ *et al.* [1] report a high frequency of sleep apnoea in patients with congestive heart failure (CHF). They also state that obstructive sleep apnoea (OSA) is more common in patients with heart failure than previously reported. Moving into the field of cardiology, the authors suggest that sleep studies should be routinely performed in patients with CHF and left ventricular dysfunction.

It is possible to investigate these patients using modern, portable, simplified recordings of breathing during sleep. However, before investigating every patient with CHF, we need to know if and how to treat these patients. What is the best therapy? What are the goals of treatment? Which criteria should be used to define central sleep apnoea in Cheyne–Stokes respiration?

OSA is characterised by snoring and daytime sleepiness due to obstructions of the upper airway during sleep. It particularly occurs in overweight patients with CHF [1–3]. However, these patients with heart failure have less subjective daytime sleepiness compared with individuals from a community sample, regardless of whether or not they have OSA [4]. Nasal continuous positive airway pressure (CPAP) is an effective therapy for obstructive apnoeas and only minor adverse effects have been reported. Nasal CPAP also reduces blood pressure and overnight norepinephrine excretion, as well as improving the left ventricular systolic function and quality of life in patients with CHF and OSA [5, 6]. However, only a few trials are available on this topic. Moreover, it is not known whether CPAP improves symptoms and survival, as this has not yet been studied in heart failure patients with OSA.

Central sleep apnoeas during Cheyne–Stokes respiration are typically seen in patients with heart failure, especially among males with atrial fibrillation [3]. The mechanisms, symptoms, consequences and treatment of central apnoeas are generally unknown. Some studies report increased mortality, while others do not [7]. Despite short sleep latency and sleep interrupted by arousals; these patients do not suffer from subjective sleepiness [8–10].

A variety of different treatment modalities reduce the frequency of central apnoeas during Cheyne–Stokes respiration. They include oxygen therapy, CPAP, adaptive servo-ventilation, noninvasive mechanical ventilation, cardiac resynchronisation therapy, theophylline, acetazolamide, carbon dioxide inhalation, sleeping in a lateral position and heart transplantation. Nasal CPAP is the most studied therapy. In the recent Canadian positive airway pressure trial, BRADLEY *et al.* [11] followed 258 patients with CHF and central sleep apnoea randomised to either CPAP or no CPAP for 2 yrs. The ejection fraction and distance walked in 6 min increased and norepinephrine levels were reduced by CPAP, but patient survival was unaffected in this study. The survival was, however, better in the control group during the first 18 months and was reversed to favour CPAP after 18 months. This is the largest randomised, controlled CPAP study ever performed. In spite of this, it was quite small compared with drug studies in this group of patients, and illustrates the problem of starting large controlled studies in the field of sleep apnoea.

Oxygen therapy has been shown to improve left ventricular ejection fraction and exercise capacity and to reduce sympathetic activity in patients with Cheyne–Stokes respiration [12–14]. Adaptive servo-ventilation effectively reduced central apnoea during Cheyne–Stokes respiration [15] and reduced daytime sleepiness in one study [16]. Acetazolamide before sleep not only reduced central apnoeas but also improved the subjective perception of sleep quality, daytime fatigue and involuntarily falling asleep during the daytime [17].

SCHULZ *et al.* [1] found a prevalence of OSA in 43% of patients with heart failure and central sleep apnoea in 28% of patients during Cheyne–Stokes respiration, using an apnoea-hypopnoea index of >10 as the cut-off for sleep apnoea. Patients were investigated with simplified sleep apnoea recordings, including pressure cannulae and plethysmographic belts around the thorax, but without electroencephalogram-recorded sleep time. JAVAHERI *et al.* [2] investigated 81 American males with CHF and left ventricular dysfunction using overnight polysomnography, the gold standard for sleep apnoea recordings. Using an apnoea/hypopnoea index of >15, they reported that 40% had central sleep apnoea and 11% OSA. MARED *et al.* [18] observed that 66% of Swedish males and females with CHF had Cheyne–Stokes respiration and only 4% had OSA. In a study from New Zealand, however, FERRIER *et al.* [19] observed that only 15% of patients had central sleep apnoea and as many as 52% of patients had OSA. Taken together, these studies

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report sleep-disordered breathing in 50–70% of patients with CHF. There were, however, large differences in the frequency of obstructive *versus* central apnoeas in these studies, which cannot be explained by differences in age, ethnicity, sex or medication, including angiotensin-converting enzyme inhibitors or β-blockers. The use of β-blockers, for example, was similar in the study by MARED *et al.* [18] and FERRIER *et al.* [19], but the frequency of central apnoeas and Cheyne–Stokes respiration differed considerably. All of these studies used different criteria to define central sleep apnoea in Cheyne–Stokes respiration. They all had different recording equipment and none used oesophageal pressure recordings, which is regarded as a superior method with which to distinguish central from obstructive apnoeas and hypopnoeas. Different equipment and inter-scorer variability could probably explain some of the discrepant findings in the aforementioned studies.

Sleep-disordered breathing of both the central and obstructive type is common among patients with heart failure and left ventricular dysfunction as highlighted by SCHULZ *et al.* [1]. Available studies report that sympathetic nervous activity and blood pressure are reduced, while ejection fraction and exercise capacity are improved, after the treatment of these apnoeas. Some patients with heart failure will doubtless improve with treatment of the sleep and breathing disorder. There are a variety of different treatment modalities available but there is a lack of solid evidence that these patients will benefit, in terms of outcome, from therapy directed towards sleep apnoea. Only one study investigated the treatment effect on survival and this study found no effect on mortality after continuous positive airway pressure [11]. We need to know how to treat sleep apnoea in patients with heart failure before screening all patients with this condition.

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