Residual sleepiness in patients with OSA on CPAP

To the Editors:

PEPIN *et al.* [1] recently published a very interesting study of residual excessive sleepiness (RES) in 502 patients with obstructive sleep apnoea treated successfully with continuous positive airway pressure (CPAP). They concluded that a significant percentage (about 6–12%, depending on inclusion criteria) had RES. They concluded that, since about 230,000 French patients are on CPAP, a minimum of 13,800 might suffer from RES, as defined by an Epworth Sleepiness Score (ESS) $\geqslant$11. In 2007, we looked at the same issue in 572 patients with OSA from the Oxford Sleep Clinic [2], but compared them with a community-based sample of 525 control subjects [3]. What was striking, was that there was no difference between the patients and the control subjects, implying that patients on CPAP had no greater a proportion with RES than the general population. This was not to say that the subjects in the control population were normal, there would have been subjects with a variety of sleep disorders on statistical grounds alone. The point we felt important was that the patients on CPAP appeared no different, thus arguing against there being any specific residual problem in patients with OSA receiving successful CPAP treatment. I have redrawn the graph (% of subjects *versus* ESS quintile) from our 2007 paper and included as a third column the raw data from PEPIN *et al.* [1] (fig. 1). Again, what is striking is that the ESS distribution from the French patients on CPAP is virtually identical to the Oxford groups of both CPAP patients and control subjects. The purpose of this correspondence is again to question whether sleepiness in successfully treated patients with OSA is any different to ''normal''.

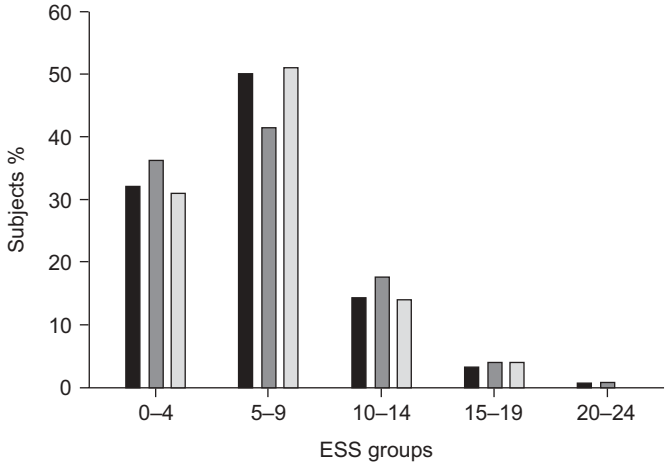

FIGURE 1. Percentage of subjects categorised in each Epworth Sleepiness Score (ESS) quintile. Data are for control subjects (■, [3]) and patients receiving continuous positive airway pressure (CPAP) treatment (■ from [2], and ■ from [1]).

J.R. Stradling
Oxford Centre for Respiratory Medicine, Churchill Hospital, Oxford, OX3 7LJ, UK.

Correspondence: J.R. Stradling, Oxford Centre for Respiratory Medicine, Churchill Hospital, Oxford, OX3 7LJ, UK. E-mail: john.stradling@orh.nhs.uk

Statement of Interest: None declared

REFERENCES

1 Pepin JL, Viot-Blanc V, Escourrou P, *et al.* Prevalence of residual excessive sleepiness in CPAP-treated sleep apnoea patients: the French multicentre study. *Eur Respir J* 2009; 33: 1062–1067.
2 Stradling JR, Smith D, Crosby J. Post-CPAP sleepiness—a specific syndrome? *J Sleep Res* 2007; 16: 436–438.
3 Stradling JR, Barbour C, Glennon J, *et al.* Prevalence of sleepiness and its relation to autonomic evidence of arousals and increased inspiratory effort in a community based population of men and women. *J Sleep Res* 2000; 9: 381–388.

DOI: 10.1183/09031936.00099609

Is there a metabolic effect of continuous positive airway pressure in sleep apnoea? Adherence should not be underestimated

To the Editors:

We read with great interest the excellent article by LÉVY *et al.* [1], which was published recently in the *European Respiratory Journal*. The authors conclude that continuous positive airway pressure (CPAP) treatment has little or no effect on the metabolic status of obese obstructive sleep apnoea (OSA) patients, presumably due to the major impact of visceral obesity [1].

While we agree on most issues covered in this article, we would also like to emphasise the role of adherence to CPAP

treatment. This may be an important, yet frequently overlooked factor, partly explaining, along with the short follow-up period, why most studies failed to show a significant effect of CPAP treatment on glycaemic control and metabolic profile of OSA patients. Indeed, in the studies included in this article that have examined this parameter, there was a considerable variation of mean CPAP use (range 3.6–6.6 $h \cdot night^{-1}$).

Moreover, only two randomised studies exist in the literature comparing the effects of therapeutic *versus* subtherapeutic CPAP treatment [2, 3]. In both studies, the average nocturnal therapeutic CPAP use was <4 h. Specifically, it was 3.6 h in the study by WEST *et al.* [2] and 3.9 h in the study by COUGHLIN *et al.* [3]. Hence, one may question whether insufficient CPAP use is a potential confounding factor in their negative findings.

Conversely, we have recently demonstrated that CPAP use for >4 h per night is crucial in ameliorating HbA_{1c} and total cholesterol levels along with several inflammation markers after 6 months of CPAP treatment in nondiabetic OSA patients [4, 5]. Of note, it was exclusively shown in adherent patients that CPAP treatment had a beneficial metabolic effect [4, 5].

Clearly, the role of adherence to CPAP therapy in these inconsistent results should be highlighted rather than underestimated. Given that there is no consensus on the minimum duration of CPAP use for a beneficial metabolic effect, further research is eagerly awaited to establish the optimal use of this modality for ameliorating the metabolic consequences in OSA patients.

P. Steiropoulos*, N. Papanas#, E. Maltezos# and D. Bouros*
*Dept of Pneumonology, and #Outpatient Clinic of Obesity, Diabetes and Metabolism, 2nd Dept of Internal Medicine, Democritus University of Thrace, Alexandroupolis, Greece.

Correspondence: P. Steiropoulos, Dept of Pneumonology, Medical School, Democritus University of Thrace, 68100 Alexandroupolis, Greece. E-mail: pstirop@med.duth.gr

Statement of Interest: None declared.

REFERENCES

1 Lévy P, Bonsignore MR, Eckel J. Sleep, sleep-disordered breathing and metabolic consequences. *Eur Respir J* 2009; 34: 243–260.
2 West SD, Nicoll DJ, Wallace TM, *et al.* The effect of CPAP on insulin resistance and HbA1c in men with obstructive sleep apnoea and type 2 diabetes. *Thorax* 2007; 62: 969–974.
3 Coughlin SR, Mawdsley L, Mugarza JA, *et al.* Cardiovascular and metabolic effects of CPAP in obese men with OSA. *Eur Respir J* 2007; 29: 720–727.
4 Steiropoulos P, Tsara V, Nena E, *et al.* Effect of continuous positive airway pressure treatment on serum cardiovascular risk factors in patients with obstructive sleep apnea-hypopnea syndrome. *Chest* 2007; 132: 843–851.
5 Steiropoulos P, Papanas N, Nena E, *et al.*, Markers of glycemic control and insulin resistance in non-diabetic patients with obstructive sleep apnea hypopnea Syndrome: does adherence to CPAP treatment improve glycemic control? *Sleep Med* 2009; 10: 887–891.

DOI: 10.1183/09031936.00108109

From the author:

In response to our recent article on sleep, sleep-disordered breathing and metabolism [1], P. Steiropoulos and co-workers wish to emphasise the role of adherence to continuous positive airway pressure (CPAP) treatment in improving obstructive sleep apnoea (OSA) metabolic status. This is certainly a major factor of success regarding CPAP treatment effectiveness. Whilst CPAP compliance remains a major challenge [2], the optimal duration of treatment remains unclear. It has been suggested that improving vigilance and cognitive function would need a minimum of 5 h, apparently with additional benefits when the duration of treatment was further increased [3]. This is much less clear as regard cardiovascular and metabolic changes. There is apparently a relationship between CPAP duration and reduction in blood pressure [4]. There is very limited evidence regarding CPAP effects on glycaemic control. P. Steiropoulos and co-workers noticed that mean CPAP use in the two randomised control trials was <4 h, 3.6 [5] and 3.9 h [6], respectively. Thus, they question whether insufficient CPAP use may be a potential confounding factor in the published negative findings. They have recently demonstrated that CPAP use for >4 h per night is crucial in ameliorating HbA_{1c} and total cholesterol levels along with several inflammation markers after 6 months of CPAP treatment in non-diabetic OSA patients, with only adherent patients exhibiting beneficial metabolic effect [7]. They also recently reported that only patients using CPAP for >4 h had significant reduction in soluble and cellular immune response factors [8]. Their study on glycaemic control [7] is not a randomised controlled trial and as such has significant limitations. Moreover, good adherence to long-term CPAP treatment seems to significantly reduce HbA_{1C} levels but has no effect on markers of insulin resistance [7]. The study by HARSCH *et al.* [9] did not shown any effect on insulin sensitivity in OSA patients with a body mass index >30 $kg \cdot m^{-2}$. In this study, the mean compliance to CPAP was high, *i.e.* 5.2 ± 0.91 h [9]. In addition, there was no association between mean duration of CPAP use per night and change in insulin sensitivity from baseline to 3 months [9]. Insulin resistance and glycaemia are closely linked in obesity and diabetes pathophysiology. Thus, it may be concluded that CPAP compliance is certainly an important issue. However, obesity is a major confounding factor in OSA. Thus, it is not surprising that CPAP effects on glycaemic control could be modulated by the degree of obesity. It should certainly be further studied by large randomised controlled trials [10, 11].

P. Lévy
Physiology and Rehabilitation, Grenoble University Hospital and Inserm ERI17, Grenoble, France.

Correspondence: P. Lévy, Physiology and Rehabilitation, Grenoble University Hospital and Inserm ERI17, Grenoble, France. E-mail: PLevy@chu-grenoble.fr

Statement of Interest: None declared.

REFERENCES

1 Lévy P, Bonsignore MR, Eckel J. Sleep, sleep-disordered breathing and metabolic consequences. *Eur Respir J* 2009; 34: 243–260.