No reduction of blood pressure with CPAP in non-sleepy hypertensive OSA patients

Several randomised controlled trials have shown that continuous positive airway pressure (CPAP) treatment for obstructive sleep apnoea (OSA) reduces blood pressure (BP). This study assesses whether CPAP produces a similar clinically significant fall in BP in hypertensive OSA patients without hypersomnolence.

Materials and methods
A total of 35 non-sleepy hypertensive OSA patients (oxygen desaturation index (ODI) 18–38) were randomised to either therapeutic (auto-CPAP for 1 month) or sham-placebo CPAP (subtherapeutic CPAP, 1 cmH₂O) in a crossover design with a 2-week washout period. "Non-sleepy" was defined as an Epworth Sleepiness Score (ESS) <10. Hypertension was defined as either taking antihypertensive drugs, or a BP >140/90 mmHg on 24-hour ambulatory BP monitoring.

Results
A total of 32 patients completed the study. The changes in mean 24-hour BP on therapeutic and subtherapeutic CPAP, respectively, were -2.1±8.1 mmHg and -1.1±8.1 mmHg (difference 0.7 mmHg; 95% confidence interval (CI) 2.9–-4.4), which was not significant. There was a small significant fall in ESS (therapeutic (-1.4) versus sham (-0.3); difference -1.2 (95% CI -2.0–-0.4)).

Conclusion
In nonhypersomnolent hypertensive OSA patients, there is no significant fall in mean 24-hour BP with CPAP, suggesting that sleep fragmentation may be important in the pathogenesis of hypertension in OSA.

Editorial comment
This is a well-organised study; however, it should be noted that almost only male patients were included and that 77% were already treating their hypertension with drugs. These patients were taking β-blockers, diuretics, and angiotensin-converting enzyme inhibitor or angiotensin II blocker or a calcium channel blocker, reducing the potential to further decrease BP.

Moreover, polysomnography (PSG) was not performed to determine the apnoea–hypopnoea index. Instead, a surrogate marker (ODI) was used to express the OSA severity, in association with pulse transit time (PTT), showing that only mild-to-moderately severe OSA patients were included. No data on oxygen desaturation time were reported.

Another limitation is that a commercial auto-CPAP device was used in the therapeutic group, which has the potential to induce (recurrent) arousals and can, therefore, counteract the beneficial effects on BP as expected with fixed CPAP. Analysis of the PTT signal could have shed light on this, but no data on these PTT measurements were reported. The effectiveness of the auto-CPAP was derived from the machine’s internal microprocessor, and was not based on PSG, again leading to a level of uncertainty.

The definition of “non-sleepy” was weak, taking into account the poor correlation between objective hypersomnolence and ESS, as well as the underreporting of sleepiness in untreated OSA patients.

Overall, the number of patients was small. In addition, the treatment period was too short. Therefore, a conclusion cannot be made as to whether there is a BP advantage after chronic CPAP administration in non-sleepy OSA patients. Extensive, long-term multicentre trials are needed to elucidate the real impact of CPAP in non-sleepy OSA patients.

Last, but not least, the conclusions of this study cannot be extrapolated to other cardiovascular problems occurring in OSA before new well-designed CPAP trials have covered this area. Although the costs of such studies are considerable, they are clearly required given the high prevalence and public health consequences of OSA.

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