

compliance may not be generalised to all kinds of therapy, as it seems that adherence to therapy may vary across different pathologies and therapeutic approaches [10].

Thus, regarding intermittent CPAP use, not only is information to believe it could improve adherence scarce, but also, it could even diminish patients' perception of the importance of regular use, deteriorating previous strategies developed to enhance adherence [6], so caution should be advised until further studies have been conducted.



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Information to believe intermittent CPAP could improve adherence is scarce <http://ow.ly/DgZAi>

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From the authors:

We appreciate the interest shown by M. van Zeller and M. Drummond in our paper [1], who suggest a more cautious interpretation regarding the possibility of withdrawing continuous positive airway pressure (CPAP) therapy for short periods and raise some concerns about how to select candidates properly who might safely undergo this.

In our study, we observed that about one-third of patients affected by obstructive sleep apnoea (OSA) syndrome and treated with CPAP did not experience recurrence of oxygen desaturations after four nights of CPAP therapy withdrawal, and about 10% did not after 2 weeks [1]. Therefore, we put forward the hypothesis that some patients may be able to stop CPAP for short periods without recurrence of OSA and its related pathophysiological consequences. As already addressed in our article [1], we agree that the use of pulse oximetry and its derivatives (oxygen desaturation index (ODI)) to assess sleep-disordered breathing has its limitations, although most researchers have found near equivalence of apnoea-hypopnoea index (AHI) to ODI. Neither conventional AHI measurements nor oximetric derivatives capture sleep

fragmentation from increases in upper airway resistance and we agree that these subtle impairments in upper airway function during sleep need much more sophisticated measurements. However, we feel that our paper provides useful information to both researchers who may wish to use the CPAP withdrawal model [2, 3] in their studies (and thus need to perform sample size estimations), and to those who have made similar observations in daily clinical practice and thus are interested in performing a larger and more detailed study looking at the temporal recurrence of OSA and its pathophysiological consequences.

M. van Zeller and M. Drummond also raised the question of whether weight loss would explain the lack of OSA recurrence after CPAP withdrawal in some of the included patients. In the patients who underwent 2 weeks of CPAP withdrawal, the body mass index (BMI) at the time of the study was not statistically different to that at diagnosis (difference 0.09 kg·m⁻², SD 1.59; p=0.96) nor was there a statistically significant reduction in neck circumference (difference -0.83 cm, SD 1.33; p=0.57). Furthermore, in multivariate analysis, we were not able to find any statistically significant association between current BMI and ODI after short-term CPAP withdrawal [1].

In the majority of studies looking at causes of OSA, the factors usually identified (neck size, craniofacial shape, tongue size and abdominal obesity) rarely explain more than 50% of the variation in OSA severity. This therefore implies that other factors (e.g. mucosal oedema of the pharynx, dilator muscle fatigue, etc.) are also important in the pathogenesis of OSA [4]. Such factors may well vary over time and may be improved even by intermittent use of CPAP therapy.



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Unconventional factors involved in OSA pathogenesis may vary over time and may be improved even by intermittent CPAP <http://ow.ly/DemVu>

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