Time course of CPAP effects on central sleep apnoea in chronic heart failure

In chronic heart failure (CHF), a number of factors combine to destabilise the ventilatory control system, such as hypocapnia and recurrent arousals, favouring the development of periodic breathing with central apnoeas and hypopnoeas (CSA). Continuous positive airway pressure (CPAP) ventilation causes a variable immediate fall in CSA frequency, but has beneficial mid-term effects on factors known to destabilise ventilatory control. The time course of this effect remains to be determined.

Materials and methods

This was a prospective study in 10 consecutive CHF patients with ischaemic, hypertensive or idiopathic dilated cardiomyopathy (left ventricular ejection fraction <45%). Patients had been stable for 3 months as documented by stable cardiac medication (including β-blockers) and no hospital admissions. In patients with apnoea/hypopnoea index (AHI) ≥15 and central apnoeas >75% of total apnoeas, CPAP was initiated during polysomnography and repeated 12 weeks later. An in-laboratory polysomnographic system was used.

Results

Throughout the second night on CPAP (baseline CPAP) the optimal CPAP from the first night was applied. AHI and oxygen desaturation index were significantly reduced by 47% ($p<0.05$) and 81% ($p<0.05$), respectively. Mean arterial oxygen saturation, arousal index and sleep efficiency were unchanged. After 12-weeks, CPAP compliance was 4.8±1.6 h per night and AHI fell by a further 42% ($p=0.03$) without further adjustments in CPAP.

Conclusion

In addition to its immediate effects, CPAP therapy leads to a time-dependent alleviation of CSA in some CHF patients. In such patients neither clinical nor scientific decisions should be based on a short-term trial of CPAP.

Editorial comment

CPAP is considered as a first-line treatment, but the reported effects on CSA in CHF are inconsistent. This prospective, observational, descriptive study clearly illustrates the mid-term effects of CPAP therapy on CSA, which alters factors linked to the pathogenesis of CSA, while in short-term trials no significant effect or a reduction of <50% was reported. Such altered factors are increased total body oxygen stores and nocturnal carbon dioxide partial pressure, reduced ventilatory drive and improved cardiac function.

This evaluation is the first to examine CPAP effects on CSA at two time-points on the same CPAP level through the night and during the treatment period in: during the 12-week study, AHI significantly decreased by 42% compared with the second night on CPAP. Based on these observations in CHF patients with CSA, CPAP causes progressive alleviation of CSA between the second night of its application and 12 weeks later on the same pressure level, without further adjustments of the CPAP level. This opens new perspectives to the approach of CPAP initiation for CSA in patients with CHF, where a time-dependent effect must be taken into account, which differs from that used to initiate CPAP in patients with obstructive sleep apnoea. First, it indicates that it is not possible to titrate CPAP within one night in order to immediately suppress CSA, since CPAP requires a longer period to exert its maximum effect on CSA. Secondly, inadequately high CPAP levels ("pressure toxicity") with poor effects on CSA and potentially harmful effects on haemodynamics in CHF patients can be prevented. Thirdly, since central apnoeas may play a key role in improving cardiovascular outcomes in CHF, it is crucial to know the time point when CPAP exerts its maximum effect on CSA in order to prevent a premature escalation to other forms of positive airway pressure support. A similar time-dependent effect was reported in patients with complex sleep apnoea syndrome (CompSAS) after CPAP application [1]. The authors concluded that CompSAS appears to represent a benign and transient phenomenon and is probably related to sleep fragmentation and sleep stage shifts that occur with initial CPAP titration. These mechanisms could perhaps also be applied in CHF with central apnoea.

This clinical evaluation was limited by its small sample size and the inability to provide a mechanism for the time-dependent effect of CPAP on CSA. Keeping this in mind, neither scientific nor clinical decisions (*e.g.* need for another form of positive airway pressure) should be based on the short-term effects of CPAP on CSA. A follow-up sleep study after a trial of 2–4 weeks on CPAP may be the most appropriate means to judge the efficacy of CPAP on CSA. More extensive studies based on polysomnography in well-described CHF patients would be welcome, given the high prevalence and public health consequences of this problem.

J. Verbraecken, Antwerp, Belgium

Reference

1. Dernaika T Tawk M, Nazir S, Younis W, Kinasewitz GT. The significance and outcome of continuous positive airway pressure-related central sleep apnea during split-night sleep studies. *Chest* 2007; 132: 81–87.

Message

It appears that improvement of CSA in response to CPAP occurs gradually. Therefore, a follow-up sleep study after a trial of 2–4 weeks of CPAP therapy may be the most appropriate means to judge the efficacy of CPAP on CSA.

Original article

Arzt M, Schulz M, Schroll S, et al. Time course of continuous positive airway pressure effects on central apnoea in patients with chronic heart failure. J Sleep Res 2009; 18: 20–25.

DOI: 10.1183/18106838.0504.355