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Haemodynamic effects of short-term nasal continuous positive airway pressure therapy in sleep apnoea syndrome: monitoring by a finger arterial pressure device

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ABSTRACT: We have evaluated the effects of short-term nasal continuous positive airway pressure (nCPAP) therapy on systemic blood pressure and heart rate in patients with obstructive sleep apnoea syndrome.

Twenty five consecutive patients were examined during baseline conditions (No-CPAP) and during one night of nCPAP treatment (CPAP). The mean value and the variation coefficient of cardiovascular variables, examined by a finger arterial pressure device (Finapres), were determined in wakefulness and sleep.

Without nCPAP an increase in blood pressure from wakefulness to sleep was observed in all patients from 138±3 mmHg to 146±3 and 155±4 mmHg, and from 80±1 mmHg to 82±2 and 84±2 mmHg, respectively, for systolic and diastolic values in non rapid eye movement (NREM) and rapid eye movement (REM) sleep. Conversely, heart rate decreased from 75±2 beats·min^{-1} to 70±2 and 69±2 beats·min^{-1}. In addition, variability of heart rate and blood pressure was greatly increased compared with the awake state. Short-term nCPAP therapy significantly reduced systolic pressure from 144±3 mmHg to 137±3 and 143±4 mmHg during NREM and REM sleep, respectively, associated with a decrease in heart rate (from 69±2 to 65±2 beat·min^{-1}). In total sleep and in all sleep stages a significantly reduced variability ($p<0.001$) was found. No changes were observed for diastolic pressure during CPAP night compared with baseline conditions.

The fall in systolic blood pressure without changes in diastolic pressure could be explained by a persistent vasoconstriction besides the removal of apnoeas. An additional mechanical effect of CPAP on the cardiovascular system is discussed.
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Obstructive sleep apnoea syndrome (OSAS) is characterized by recurrent upper airway obstruction during sleep, which results in episodic oxyhaemoglobin desaturations, transient arterial hypertension and tachycardia [1]. The nocturnal increase in systemic blood pressure has been implicated as a cause of daytime hypertension [2, 3], the most frequent cardiovascular complication in OSAS patients.

In recent years, nasal continuous positive airway pressure (nCPAP) has been recognized [4] as an effective therapy, preventing apnoeas and oxygen desaturations. Previous studies have demonstrated that the acute or chronic application of nCPAP preventing nocturnal hypoxaemia stabilizes heart rate and decreases pulmonary and systemic arterial pressure [4–6]. However, these studies were limited by the small number of patients subjected to invasive monitoring of arterial systemic pressure.

Recently, the non-invasive recording of blood pressure and heart rate by a finger arterial pressure device (Finapres) has been utilized as an accurate method to estimate means and variability of blood pressure [7, 8] and represents an acceptable alternative to invasive monitoring [9] even in OSA patients [10].

This study adopted finger arterial pressure monitoring to investigate the nocturnal trend of blood pressure and heart rate at baseline and after short-term nCPAP therapy in a group of OSAS patients.

Patients and material

Twenty five patients (24 men and 1 woman) with previously established diagnosis of OSAS were examined. Clinical and anthropometric parameters are shown in table 1. The mean age was 47±2 yrs, with a mean duration of the reported onset of snoring and diurnal sleepiness of 6±4 yrs. Mean body mass index (BMI) was 34±1; 17 patients were obese (BMI >30). According to a systolic pressure reading of >150 mmHg

or a diastolic value of >90 mmHg, as criteria for the definition of systemic hypertension, four patients were hypertensive (systolic pressure range 152–174 (mean±SD 163±9) mmHg; diastolic pressure range 92–96 (mean±SD 93±3) mmHg. All subjects were clinically stable on entering the study and all drugs acting on the cardiovascular system were suspended one week before the sleep studies. They all underwent a full evaluation including respiratory function tests and polysomnography. Spirometry indicated that 24% of patients had airway obstruction with a forced expiratory volume in one second/forced vital capacity (FEV_1/FVC) <70%. Six patients had resting hypoxaemia (defined as arterial oxygen tension (Pao_2) <8.7 kPa (65 mmHg)) with mean Pao_2 7.5±3 kPa (56±8 mmHg) and nine had hypercapnia (defined as arterial carbon dioxide tension ($Paco_2$) >6 kPa (45 mmHg)) with mean $Paco_2$ 6.5±1 kPa (49±5 mmHg).

Table 1. – Clinical and anthropometric findings

		Mean	SEM	Range
Age	yrs	47	2.2	26–65
BMI	$kg \cdot m^{-2}$	34	1.1	25–50
SPB	mmHg	138	2.7	110–174
DPB	mmHg	80	1.4	70–96
Pao_2	kPa	10	2.7	6–12
$Paco_2$	kPa	6	1.2	5–8

BMI: body mass index (weight/height^2); SBP: diurnal systolic pressure; DPB: diurnal diastolic pressure; Pao_2: arterial oxygen tension; $Paco_2$: arterial carbon dioxide tension.

Methods

Two consecutive polysomnographic studies were performed in all subjects: the first as baseline evaluation No-CPAP and the second with nCPAP therapy (CPAP). During the CPAP night, positive airway pressure was rapidly increased above the starting level of 2 cmH_2O until apnoeas, snoring and intrathoracic negative effort were abolished. The mean level of nCPAP applied was 12.5±2.3 cmH_2O (range 8.5–16.5 cmH_2O).

Polysomnographic investigation included electroencephalography (EEG), electro-oculography (EOG), electromyography (EMG) of chin and intercostal muscles, microphone and electrocardiograph (ECG). Nasal and oral flow were recorded with thermistors, thoracic and abdominal respiratory movements with strain gauge and endothoracic pressure by oesophageal balloon. Continuous oxygen saturation was monitored by ear oximetry (Biox III).

Sleep was scored using the method of RECHTSCHAFFEN and KALES [11]. Central, mixed and obstructive apnoeas were defined according to standard criteria. Hypopnoeas were defined by an estimated 50% or more reduction of the oronasal flow. The apnoea index (AI: number of apnoeas·h^{-1}), apnoea+hypopnoea index (A+HI: number of apnoeas and hypopnoeas·h^{-1}), mean duration of respiratory events and percentage of different apnoeas were computed. The following arterial oxygen saturation (Sao_2) indices were defined: mean Sao_2 during 10 min of wakefulness ($WSao_2$) prior to sleep onset during No-CPAP night and during the last awakening during CPAP night; mean minimal Sao_2 after each event (mean low Sao_2) and mean Sao_2 during sleep (mean Sao_2).

Systolic (SBP) and diastolic (DBP) pressure and heart rate (HR) were continuously recorded by a finger arterial pressure monitor (Finapres). Haemodynamic parameters were examined at 30 s intervals and the mean value and coefficient of variation (standard deviation/mean) were determined during awake state and NREM and REM sleep. During CPAP night these parameters were examined only during the period of efficacious applied pressure. To avoid artifacts on blood pressure values caused by hand movements, we fixed hand position at the level of the sternum. Periods with body movements were excluded by computing.

Group means and coefficient of variation taken from the data of each patient were compared during No-CPAP and CPAP night using Student's t-test for paired data. Correlations between changes in blood pressure and heart rate and diurnal and nocturnal parameters were calculated using a multiple regression analysis. The level of statistical significance was taken as $p<0.05$. Results are given as mean±SEM.

Results

Respiratory variables

The patients had relatively moderate or severe OSAS in terms of the frequency of disordered breathing events. During No-CPAP night (table 2) the A+HI was 74±4 with an AI of 67±5. The vast majority of events were obstructive (83%) and mixed with a low percentage of central apnoeas (3%). The mean duration of apnoeas was 27±2 s and low Sao_2 of 78.1±2%. During CPAP night, mean oxygen saturation improved from 87.7±1% to 94.2±0.3%.

Haemodynamic variables

The haemodynamic variables during No-CPAP and CPAP night are reported in table 3 (next page).

Table 2. – Respiratory variables during untreated night

	Mean	SEM	Range
Apnoea index $n \cdot h^{-1}$	67.3	4.8	25–124
Apnoea+hypopnoea index $n \cdot h^{-1}$	74.4	4.2	33–125
Mean duration apnoea s	27.1	1.5	16–41
Low Sao_2 %	78.1	1.6	64–91
Mean Sao_2 %	87.7	1.0	75–95
Awake Sao_2 %	93.3	0.4	89–96

Sao_2: arterial oxygen saturation.

Table 3. – Mean values and variability of blood pressure and heart rate during baseline (No-CPAP) and nCPAP (CPAP) night

	No-CPAP	CPAP	p
SBP W mmHg	138±3	144±3	0.04
	(0.05)	(0.03)	0.001
DBP W mmHg	80±1	85±2	0.02
	(0.07)	(0.04)	0.001
HR W b·min^{-1}	75±2	69±2	0.02
	(0.07)	(0.06)	0.004
SBP NR mmHg	146±3	137±3	0.05
	(0.11)	(0.05)	0.001
DBP NR mmHg	82±2	81±2	NS
	(0.13)	(0.06)	0.001
HR NR b·min^{-1}	70±2	65±2	0.03
	(0.12)	(0.5)	0.001
SBP R mmHg	155±4	143±4	0.01
	(0.16)	(0.05)	0.001
DBP R mmHg	84±2	82±2	NS
	(0.13)	(0.07)	0.001
HR R b·min^{-1}	69±2	65±2	NS
	(0.16)	(0.06)	0.001
SBP S mmHg	146±3	139±4	0.05
	(0.12)	(0.06)	0.001
DBP S mmHg	82±2	82±2	NS
	(0.13)	(0.07)	0.001
HR S b·min^{-1}	70±2	65±2	0.03
	(0.13)	(0.06)	0.001

Data are given as mean±SEM, with coefficient of variation in parenthesis. SBP: systolic arterial pressure; DBP: diastolic arterial pressure; HR: heart rate; W: awake state; NR: non rapid eye movement; R: rapid eye movement sleep; S: total sleep.

During the No-CPAP night, systemic arterial pressure increased from wakefulness to sleep with an average difference of 9±6 mmHg for SBP and 2±2 mmHg for DBP. The systolic blood pressure was 138±3 mmHg in awake state, 146±3 mmHg in NREM sleep and 155±4 mmHg in REM sleep. Diastolic pressure increased from 80±1 mmHg in wakefulness to 82±2 mmHg in NREM sleep and 84±2 mmHg in REM sleep. During apnoeas a progressive increase in blood pressure was associated with cyclic variations (fig. 1). HR fell by awake value of 75±2 beats·min^{-1} to 70±2 and 69±2 beats·min^{-1}, respectively, in NREM and REM sleep. In addition, the coefficient of variability was increased for all parameters without significant differences in NREM and REM sleep.

During the treated night, nCPAP treatment prevented the peaks of systemic hypertension and stabilized cardiovascular parameters at values lower than the untreated night (see fig. 2). In the group of patients as a whole, systolic pressure fell from awake value of 144±3 mmHg to nocturnal values of 139±4 mmHg ($p<0.05$), associated with a decrease in HR from 69±2 to 65±2 beats·min^{-1} ($p<0.03$). The diastolic pressure value on the CPAP night were 85±2 mmHg in wakefulness, 81±2 in NREM sleep and 82±2 in REM sleep. Comparison of the changes between No-CPAP and CPAP nights in different sleep stages (fig. 3) showed a significant decrease in SBP during NREM sleep (146±3 mmHg *vs* 137±3, $p<0.05$) and REM sleep (155±4 mmHg *vs* 143±4, $p<0.01$), whereas there were no significant changes in DBP in any sleep stage. HR decreased significantly in all patients during NREM sleep from 70±2 to 65±2 ($p<0.03$) without a significant fall during REM sleep.

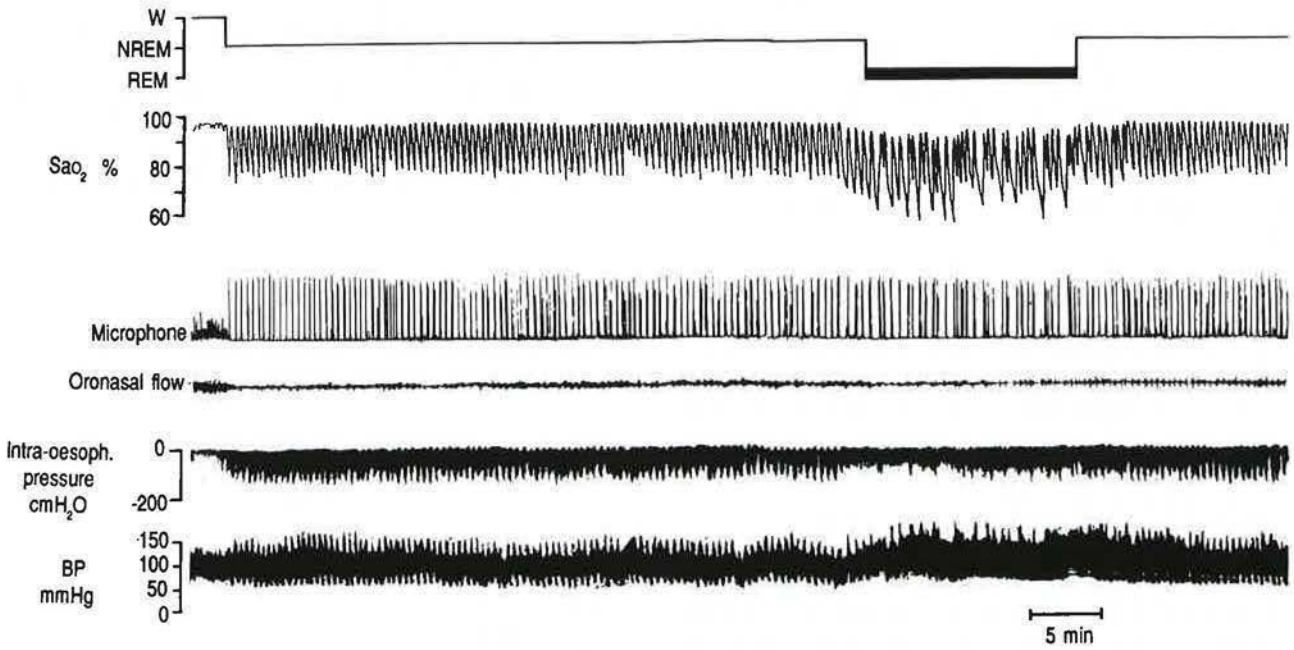

Fig. 1. – Slow recording of sleep stages, arterial oxygen saturation, microphone, oronasal flow, oesophageal pressure and systemic arterial pressure during the untreated night (No-CPAP) in male patient, Z.P. 30 yrs. During apnoeas, a progressive increase in blood pressure was associated with cyclic variations. The maximal peaks of hypertension were recorded during REM sleep. W: awake state; NREM: non rapid eye movement; REM: rapid eye movement; Sao_2: arterial oxygen saturation; BP: systemic arterial pressure.

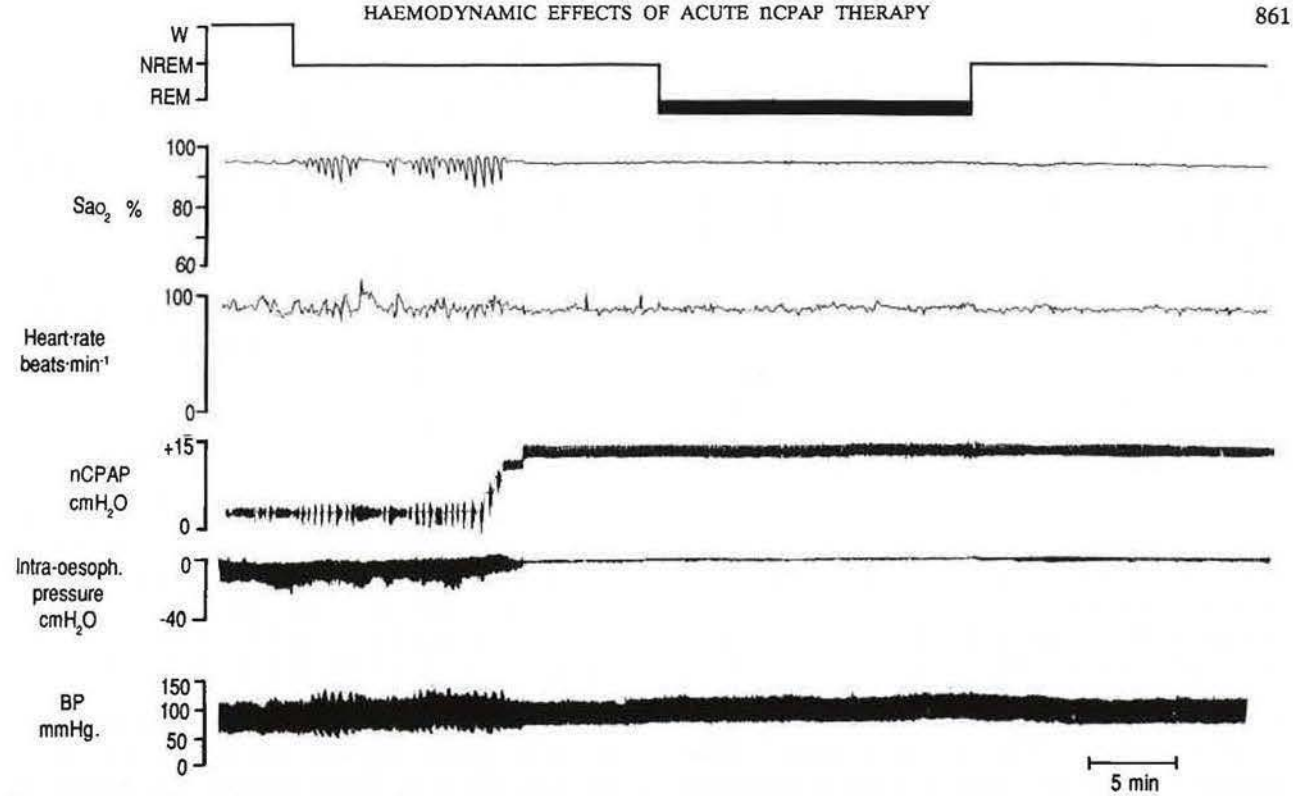

Fig. 2. – Recording of sleep stages, arterial oxygen saturation, nCPAP, oesophageal pressure, heart rate and systemic arterial pressure in the same patient as fig. 1 during the treated (CPAP) night. The progressive increase in nCPAP eliminates apnoeas and phasic desaturations and stabilizes blood pressure and heart rate at lower levels than on the untreated night. nCPAP: nasal continuous positive airway pressure. For further abbreviations see legend to figure 1.

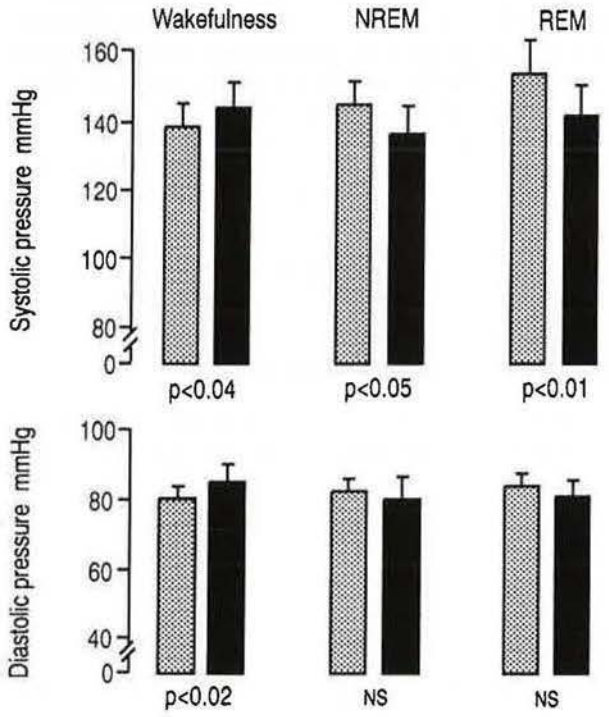

Fig. 3. – Mean±SEM of systolic and diastolic pressure during No-CPAP and CPAP night in wakefulness and sleep. During both NREM and REM sleep a significant decrease in systolic pressure was present without changes in diastolic pressure. Blood pressure increased in awake state during the CPAP night. ▨: No-CPAP; ■ : CPAP. For further abbreviations see legends to figures 1 and 2.

The coefficient of variability for all parameters was significantly reduced ($p<0.001$) in all sleep stages on the treated night (table 3). The variability was higher in sleep than in wakefulness and in REM sleep during No-CPAP night. During the treated night the variability decreased from wakefulness to sleep without differences in NREM and REM sleep.

No correlation was found between the changes in blood pressure, during either No-CPAP and CPAP night, and age, BMI, A+HI, mean low Sao_2, diurnal Pao_2 and $Paco_2$, diurnal systemic pressure and level of nCPAP pressure.

During awake state, HR decreased significantly ($p<0.02$) in the patients group as a whole. In 12 patients a slight and significant increase in blood pressure ($p<0.002$) was present in the morning after nCPAP study. To further analyse the changes in blood pressure during wakefulness, the subgroup of patients with a reduction of blood pressure (n=13) was compared with the subgroup (n=12) with an increase in blood pressure. The age, BMI, diurnal systemic pressure, AI, mean low Sao_2, diurnal Pao_2 and $Paco_2$ and level of nCPAP pressure did not differ between the two subgroups.

Discussion

This study confirms that patients with sleep apnoea syndrome have raised blood pressure during periods of obstructive apnoeas compared with awake state. Prevention of apnoeas by nCPAP partially reduced this sleep-associated hypertension, with a significant

decrease in variability. The major finding in our study was that the acute effect of nCPAP treatment did not significantly change diastolic pressure even though systolic blood pressure decreased.

In addition, a slight but significant increase in blood pressure in awake state after the treated night was found in 49% of patients. In contrast, heart rate was significantly reduced in awake and sleep state, suggesting an increased vagal tonus probably induced by lung stretch depressor reflex [12].

Our results suggest that the reduced blood pressure during short-term nCPAP therapy without a complete correction of the absolute values could reflect the impact of several factors, including an additional effect of mechanical ventilation on a persistent vasoconstriction.

Several factors [13, 14], *i.e.* enhanced sympathetic activity in response to hypoxaemia, mechanical stress induced by inspiratory effort and chemoreceptor stimulation by postapnoeic hyperventilation, have been reported to be responsible for the daytime and nighttime hypertension in OSAS. The relative contribution of each of these factors remains, however, unclear. Recently, the increased production of hormonal factors, *i.e.* atrial natriuretic factor and catecholamines, during obstructive apnoeas have been postulated as contributing factors, inducing a prolonged persistent vasoconstriction and a chronic fluid-retaining response. A reduction in atrial natriuretic factor [15] and a decreased haematocrit value and red blood cell count [16] have been described after short-term nCPAP therapy. These findings suggest that nCPAP probably has an acute effect entailing hormonal changes responsible for fluid shift from the extravascular to intravascular volume. In our patients, the correction of the hypoxaemia and negative intrathoracic pressure peaks excluded the effect of the chemical and mechanical factors on the changes in blood pressure during the treated night. On the other hand, the reported modifications of body fluid volume homeostasis could not explain the significant fall in systolic pressure found after a single night of nCPAP treatment.

In OSAS patients an increased sympathetic activity has been reported not only during apnoeas but also in wakefulness [17, 18]. The absent changes in diastolic pressure in our patient may indicate a permanent vasoconstriction in addition to the acute removal of hypoxaemia. We did not find any correlation between the change in blood pressure during treated and untreated night and resting awake blood pressure in our patients. Probably, this is a bias in our study due to the small number of hypertensive subjects investigated.

In addition, it is known that systemic and pulmonary vessels tend to undergo functional and/or anatomical remodelling after chronic exposure to hypoxia. After effective surgical treatment diurnal blood pressure falls to some extent but not to normal [19], in association with a return to normal levels of urinary catecholamines [20]. This indicates that sympathetic hyperactivity alone or associated with structural vessel changes in OSAS patients may contribute to the development of persistent hypertension.

Nevertheless, an additional mechanical effect of continuous positive airway pressure application cannot be excluded. The haemodynamic effects of CPAP therapy are still unclear. A potential fall in cardiac output similar to that seen with positive end-expiratory pressure (PEEP) [21] may occur as a result of decreased venous return or impaired left ventricular function. In a study examining the haemodynamic effects of nCPAP [22] in OSAS patients, a progressive decrease in cardiac output was found only in patients with pulmonary hypertension and left ventricular dysfunction at higher levels of nCPAP. Experimental studies have demonstrated that PEEP raises systemic pressure in addition to the invariable decrease in cardiac output due to the impedance of venous return [23, 24]. In association with the PEEP-induced increase in abdominal pressure transferring blood volume from the splanchnic to peripheral circulation, an activation of baroreflex sensitivity has recently been suggested [25]. The increase in vessel resistance due to baroreflex hyperactivity could explain the rise in systemic pressure despite the reduced cardiac output.

In our opinion, the persistent vasoconstriction related to OSAS disease together with the positive airway pressure effects on vessel capacity may explain the missing fall in diastolic pressure during sleep on short-term nCPAP therapy. Nevertheless, a probable time effect of the nCPAP therapy in normalizing vascular resistance can be suggested. In fact, a previous study [26] showed a reduction in systemic blood pressure during nCPAP night after six months of home treatment even in the absence of significantly reduced apnoea frequency. Further studies including nocturnal monitoring of blood pressure and hormonal samples are needed to clarify the role of different factors in the correction of diurnal and nocturnal blood pressure during short and long-term nCPAP treatment.

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References

1. Podszus T, Majer J, Penzel T, Peter JH, von Wichert P. – Nocturnal hemodynamics in patients with sleep apnea. *Bull Eur Physiopathol Respir*, 1986; 69 (Suppl. 146): 435–442.
2. Mondini S, Zucconi M, Cirignotta F, Aguglia U, Lenzi PL, Zauli C, Lugaresi E. – Snoring a risk factor for cardiac and circulatory problems: an epidemiological study. *In*: Guilleminault C, Lugaresi E, eds. Sleep/Wake Disorders: Natural History, Epidemiology and Long-Term Evolution. New York, Raven Press, 1983; pp. 99–105.
3. Stradling JR. – Sleep apnoea and systemic hypertension. *Thorax*, 1989; 44: 984–989.
4. Sullivan CE, Issa FG, Berthon Jones M, Eves L. – Reversal of obstructive apnea by continuous positive airway pressure applied through the nares. *Lancet*, 1981; i: 862–865.
5. Podszus T, Becker H, Figura M, Peter JH, von Wichert P. – The influence of nCPAP therapy on pulmonary arterial pressure in patients with sleep apnea. *Sleep Res*, 1988; 17: 234.

6. Mayer J, Becker H, Branderburger U, Penzel T, Peter JH, Podszus T, Weiner M, von Wichert P. – Blood pressure variability and nocturnal pressure profile in sleep apnea under nasal continuous positive airway pressure (nCPAP) therapy. Duron B, Levi Valensi P, eds. *In*: Sleep disorders and respiration. Colloque INSERM, Libbey Eurotext, 1988; 168: 241–243.
7. Kurki T, Smith NT, Dec-Silver H, Quinn A. – Noninvasive continuous blood pressure measurement from the finger: optimal measurement conditions and factors affecting reliability. *J Clin Monit*, 1987; 3: 6–13.
8. Imholz BPM, Wieling W, Langewouters GJ, van Montfrans GA. – Continuous finger arterial pressure: utility in the cardiovascular laboratory. *Clin Autonomic Res*, 1991; 1: 43–53.
9. Parati G, Casadei R, Groppelli A, Di Rienzo M, Mancia G. – Comparison of finger and intra-arterial blood pressure monitoring at rest and during laboratory testing. *Hypertension*, 1989; 13: 647–655.
10. Goldman MD, Goldberg R, Loh L, Chuachmsai N, Watanabe S, Fry JM. – Evidence of sleep-related disorder from finger arterial pressure. *Sleep Res*, 1989; 18: 32.
11. Rechtschaffen A, Kales A. – A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Publication 204. US Public Health Service, 1968.
12. Schreuder JJ, Jansen JRC, Versprille A. – Contribution of lung stretch depressor reflex to nonlinear fall in cardiac output during PEEP. *J Applied Physiol: Respirat Environ Exercise Physiol*, 1984; 56: 1578–1582.
13. Shepard JW. – Gas exchange and hemodynamics during sleep. *Med Clin North Am*, 1985; 69: 1243–1263.
14. Ringler J, Basner RC, Shannon R, Schwartzstein R, Manning H, Weinberger SE, Weiss JW. – Hypoxemia alone does not explain blood pressure elevation after obstructive apneas. *J Appl Physiol*, 1990; 69: 2143–2148.
15. Krieger J, Laks L, Wilcox I, Grunstein R, Costas L, McDougal J, Sullivan CE. – Atrial natriuretic factor release during sleep in obstructive sleep apnoea. *Clin Sci*, 1989; 77: 407–411.
16. Krieger J, Sforza E, Barthelmebs M, Imbs J-L, Kurtz D. – Overnight decrease in hematocrit after nasal CPAP treatment in patients with OSA. *Chest*, 1990; 97: 729–730.
17. Hedner J, Ejnell H, Sellgren J, Hedner T, Wallin G. – Is high and fluctuating muscle nerve sympathetic activity in the sleep apnea syndrome of pathogenic importance for the development of hypertension. *J Hypertension*, 1988; 6: S529–531.
18. Ejnell H, Hedner J, Caidahl K, Sellgren J, Wallin G. – Increased sympathetic activity as possible etiology of hypertension and left ventricular hypertrophy in patients with obstructive sleep apnea. *In*: Peter JH, Penzel T, Podszus T, von Wichert P eds. Sleep and health risk. Berlin, Springer-Verlag, 1991; pp. 341–347.
19. Guilleminault C, Simmons FB, Motta J, Cummisky J, Rosekind M, Schroeder JS, Dement WC. – Obstructive sleep apnea syndrome and tracheostomy. *Arch Intern Med*, 1981; 141: 985–988.
20. Fletcher E, Miller J, Schaaf J. – Urinary catecholamines before and after tracheostomy in obstructive sleep apnea. *Sleep Res*, 1985; 114: 154.
21. Kumar A, Falke KJ, Geffin B. – Continuous positive-pressure ventilation in acute respiratory failure: effects on hemodynamics and lung function. *N Engl J Med*, 1970; 283: 1430–1436.
22. Podszus T, Steindl S, Becker H, Peter JH, von Wichert P. – Influence of continuous positive airway pressure ventilation on left ventricular function. *Sleep Res*, 1989; 18: 285.
23. Cournand A, Motley HL, Werkö L, Richards DW Jr. – Physiological studies on the effects of intermittent positive pressure breathing on cardiac output in man. *Am J Physiol*, 1948; 152: 62–74.
24. Scharf SM, Ingram RH Jr. – Influence of abdominal pressure and sympathetic vasoconstriction on the cardiovascular response to positive end-expiratory pressure. *Am Rev Respir Dis*, 1977; 116: 661–670.
25. Fessler HE, Brower RG, Wise RA, Permutt S. – Effects of positive end-expiratory pressure on the gradient for venous return. *Am Rev Respir Dis*, 1991; 143: 19–24.
26. Mayer J, Becker H, Penzel T, Brandenburg V, Peter JH, Ploch T, *et al.* – Blood pressure in cases of sleep apnea after discontinuation of long-term nCPAP therapy. *Sleep Res*, 1989; 19: 350.