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SLEEP BREATHING PHYSIOLOGY AND DISORDERS • ORIGINAL ARTICLE

The changes of AHI after long-term CPAP in patients with comorbid OSA and cardiovascular disease

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Abstract

Purpose To evaluate the effect of long-term continuous positive airway pressure (CPAP) treatment on disease severity of obstructive sleep apnea (OSA).

Methods We analyzed results from the Sleep Apnea and Cardiovascular Events (SAVE) study involving participants recruited at the Guangdong Provincial People's Hospital, China. Participants were aged 45–75 years with a history of cardiac or cerebrovascular disease. OSA was confirmed by home sleep apnea testing (HSAT). Participants were randomized to receive CPAP plus standard cardiovascular care (CPAP group) or standard care alone (UC group) and followed for several years. At the study conclusion, surviving participants were invited to repeat HSAT. Changes in OSA indicators were compared by independent samples *t*-tests and subgroup analysis was implied among groups stratified by OSA severity.

Results One hundred two adults were recruited (51 per group) and followed for 48.0 ± 14.5 months. Daily CPAP usage in the CPAP group was 4.1 ± 1.9 h. AHI decreased from baseline to end-of-study in both CPAP and UC groups (− 5.0 (− 12.5,2.0), $P = 0.000$; − 4.0 (− 12.5,1.5), $P = 0.007$, respectively), with no between-group difference ($P = 0.453$). An improvement in nadir SpO_2 showed from baseline to end-of-study in the CPAP but not UC group (2.3% ± 6.1%, $P = 0.011$ and − 0.7% ± 7.6%, $P = 0.511$, respectively; between-group difference $P = 0.032$). Subgroup analysis shows that CPAP could improve AHI in patients with moderate OSA (− 8.0 (− 11.8, − 2.8) in CPAP group, − 2.0 (− 0.8,6.0) in UC group, $P = 0.022$) and improve nadir SpO_2 in patients with severe OSA (5.0 (− 0.8, − 0.8,7.0) in CPAP group, 0.0 (− 8.5,2.5) in UC group, $P = 0.032$).

Conclusion Long-term CPAP use did not result in clinically significant changes in AHI or ODI overall but showed variable effects stratified by OSA severity.

Clinical Trial Registration Registry: Clinical Trials.gov, title: Continuous Positive Airway Pressure Treatment of Obstructive Sleep Apnea to Prevent Cardiovascular Disease (SAVE), URL: www.clinicaltrials.gov, identifier: NCT00738179.

Keywords OSA · Long-term CPAP · AHI · Oxygen saturation

Introduction

Obstructive sleep apnea (OSA) is a disorder characterized by repetitive partial (hypopnea) or complete (apnea) collapse of the upper airway during sleep [1]. However, the mechanisms involved are not very clear. Continuous positive airway pressure (CPAP), the first-line treatment of OSA, has an immediate positive effect on breathing during sleep [2]. It improves or eliminates obstructive breathing events and nocturnal hypoxemia and reduces arousals [3, 4]. Whether or not these effects are strictly transient, or have, over time, a sustained impact on underlying AHI is unclear.

Loud snoring and severe OSA have been associated with upper airway inflammation and edema, sensorineural

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injury, and partial muscle denervation [5], as well as an increase in the arousal threshold to respiratory stimuli [6], which may progressively worsen OSA. Several of these abnormalities may be improved with CPAP treatment [5, 6] but it is uncertain if this is sufficient to improve OSA to a clinically significant extent. Several previous studies of patients treated with CPAP for a few months to approximately a year showed a small decrease in the frequency of sleep apnea and hypopnea events (apnea–hypopnea index, AHI) after withdrawal of CPAP compared with the AHI at time of diagnosis [7–12]. In one study, approximately 15% of individuals with OSA (AHI > 10 events per hour) who had used CPAP consistently for > 12 months did not experience significant recurrence of AHI after stopping CPAP for 4 days and 2 weeks [13]. However, there have been no randomized controlled studies that allow the effects of several years of CPAP treatment to be ascertained while controlling for other changes that might occur in patients with OSA who are untreated.

The Sleep Apnea and Cardiovascular Endpoints (SAVE) study was an international, multicenter, randomized controlled trial of patients with coexisting CV disease and moderate-to-severe OSA who were randomly allocated to receive CPAP plus usual cardiovascular care or usual cardiovascular care alone and followed for approximately 4 years. This secondary analysis in a subpopulation of SAVE participants provides an opportunity to assess the effect of long-term CPAP treatment on the severity of OSA.

Methods

Participants

Patients included in this sub-study were those who had provided written informed consent at the Guangdong Provincial People's Hospital to participate in the SAVE trial (NCT00738179) [14]. Participants were aged 45 to 75 years, and had established CV disease and co-occurring moderate-severe OSA. Patients were excluded if they had severe daytime sleepiness (Epworth Sleepiness Scale [ESS] score > 15), very severe nocturnal hypoxemia (oxygen saturation < 80% for > 10% of the monitoring time), or a predominantly Cheyne-Stokes respiration pattern on home sleep apnea testing (HSAT). Potential participants were required to adhere to a 1-week trial of sham CPAP (REMstar Auto M Series, Philips Respironics, Inc. with pressure set to subtherapeutic levels) to evaluate willingness and likely ability to adhere to the treatment. Those who recorded a daily average sham CPAP usage ≥ 3 h were included. Full details of the study have been outlined elsewhere [14, 15]. Details of the design have been published previously [16, 17].

HSAT monitoring and scoring

Specially trained persons were responsible for HSAT monitoring at baseline and at the end of the study. HSAT was conducted using a cardiorespiratory polygraphy device (ApneaLink, ResMed, Bella Vista, Sydney, Australia). ApneaLink is a type 3 portable sleep monitoring device. Respiration was monitored with a nasal cannula, chest and abdominal bands, and pulse oximeter. Participants learned how to operate the device at hospital and completed sleep monitoring at their home. Participants in the CPAP group were instructed not to use CPAP on the night of the end-of-study recording. Each study was automatically analyzed, after data were reviewed at a central core sleep laboratory to ensure it met pre-specified duration and quality standards. Obstructive sleep apnea was defined as a reduction ≥ 90% in airflow for ≥ 10 s during sleep with persistent ventilatory efforts while likely Cheyne-Stokes respiration indicative of central sleep apnea was identified by typical crescendo-decrescendo airflow patterns. Hypopnea was defined as a reduction of ≥ 30% airflow for ≥ 10 s, associated with a desaturation > 4% of oxygen. The AHI was calculated as the total number of apneas and hypopneas divided by the total recording time [18]. The oxygen desaturation index

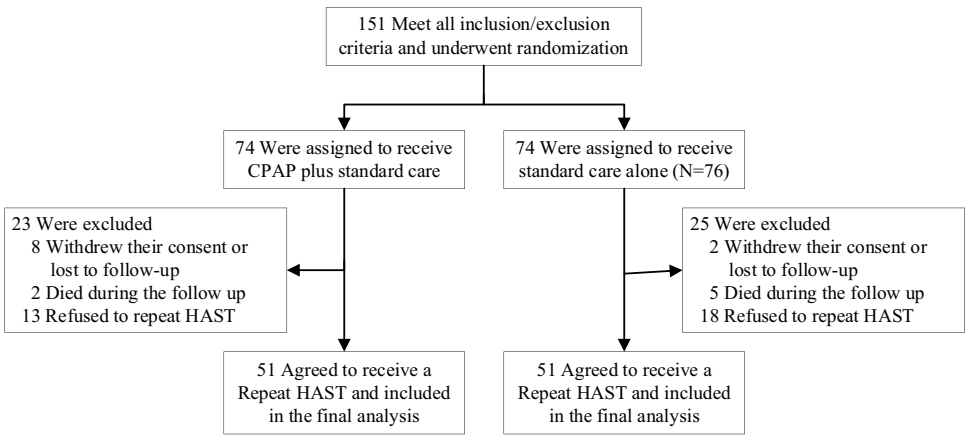

Fig. 1 Randomization and follow-up analyses. This is a second analysis for The Sleep Apnea and Cardiovascular Endpoints (SAVE) study. Participants were required to have a repeat home sleep apnea test (HSAT) at the end of the study

(ODI) was defined as the number of ≥ 4% oxygen desaturation events per hour. Moderate to severe OSA was defined as an ODI ≥ 12/h.

Randomization

A special website (http://vmprdinf001.iih.usyd.edu.au/saveuat/pfts.dll?PVN=13) was designed for the randomization and data collection of this study. The randomization software was based in a system named FLEXETRIALS. All eligible subjects were randomly assigned to receive either

Table 1 Baseline characteristics of the study participants

Characteristic		CPAP group ($N=51$)	Usual-care group ($N=51$)	P values
Age (year)		61.3 ± 6.9	62.4 ± 6.8	0.412
Male sex (%)		44 (88%)	40 (78%)	0.199
Obstructive sleep apnea characteristics				
AHI (events/h)		22.0 (16.0,34.0) [a]	24.0 (17.0,42.0) [a]	0.379
AI(events/h)		15.0 (7.0,28.0) [a]	17.0 (9.0,32.0) [a]	0.588
HI(events/h)		7.0 (4.0,10.0) [a]	7.0 (4.0,12.0) [a]	0.473
AI/HI		2.7 (0.8,6.8) [a]	2.3 (1.0,5.7) [a]	0.932
ODI (events/h)		19.0 (15.0,32.0) [a]	26.0 (17.0,37.0) [a]	0.233
Nadir SpO_2		78.6 ± 6.0	78.1 ± 5.6	0.658
Mean SpO_2		93.9 ± 1.6	94.3 ± 1.6	0.285
Time < 90% SpO_2 (min)		22.0 (8.0,63.0)[a]	23.0 (12.0,41.0)[a]	0.804
Min pulse (beats/min)		50.5 ± 6.9	52.2 ± 6.4	0.195
Max pulse (beats/min)		95.3 ± 23.3	95.2 ± 17.7	0.977
Mean pulse (beats/min)		61.6 ± 7.6	63.9 ± 6.6	0.108
Anthropometric measurements				
Height (cm)		166.4 ± 6.8	165.3 ± 8.8	0.487
Weight (cm)		73.6 ± 10.7	72.2 ± 11.5	0.517
BMI (kg/m^2)		26.5 ± 2.9	26.4 ± 3.4	0.822
Neck circumference (cm)		38.8 ± 3.1	38.4 ± 3.3	0.534
Waist circumference (cm)		93.2 ± 8.6	91.7 ± 8.8	0.369
Hip circumference (cm)		99.6 ± 7.2	100.5 ± 6.1	0.514
Waist-hip ratio		0.9 ± 0.0	0.9 ± 0.1	0.014*
SBP (mmH_2O)		121.6 ± 13.3	119.8 ± 13.1	0.478
DBP (mmH_2O)		73.3 ± 11.3	72.7 ± 11.4	0.808
Lifestyle				
Current smoker		11 (22%)	10 (20%)	0.807
Current alcohol drinking		7 (14%)	10 (20%)	0.425
Exercise	Sedentary	10 (20%)	8 (16%)	
	Moderately active	22 (43%)	29 (57%)	0.379
	Active	19 (37%)	14 (28%)	
Medical history				
Coronary artery disease		25 (49%)	28 (55%)	0.552
Cerebrovascular disease		25 (49%)	16 (31%)	0.069
Coronary and cerebrovascular disease		1 (2%)	7 (14%)	0.027*
Diabetes mellitus		17 (33%)	12 (24%)	0.272

CPAP, continuous positive airway pressure group; *UC*, usual care group; *AHI*, apnea–hypopnea index; *AI*, apnea index; *HI*, hypopnea index; *AI/HI*, the radio of AI to HI; *ODI*, oxygen desaturation index; *SpO_2*, oxygen saturation; *BMI*, body mass index; *SBP*, systolic blood pressure; *DBP*, diastolic blood pressure; current smokers were distinguished from former smokers and never smokers. Current alcohol drinking was defined by answering yes to the question "Do you currently drink alcohol once a week or more?" Exercise level was measured by Godin Leisure-Time Exercise Questionnaire (LTEQ) and was divided into three categories as previously described [19]

*Significant at the $p < 0.05$ level

[a]Median (quartile 1; quartile 3)

CPAP therapy plus usual care (CPAP group) or usual care alone (usual-care group). Randomization was performed with the use of a minimization procedure to balance the group assignments according to site, type of cardiovascular disease (cardiac, cerebrovascular, or both), and severity of daytime sleepiness (Epworth Sleepiness Scale score < 11 vs. ≥ 11). This step was unblinded and subjects who met all inclusion/exclusion criteria were entered onto the study in the website by the principal investigator.

CPAP treatment and follow-up

All participants were followed-up at 1, 3, 6, and 12 months and annually thereafter. Participants were also contacted by telephone at 6 months between the annual clinic visits. Additional end-of-study visits were performed between September 2015 and January 2016 on all participants who remained actively enrolled in the trial. CPAP adherence was recorded at each follow up visit and reported as a time-weighted mean value for each participant across their whole study follow-up period.

Statistical analysis

Continuous variables were expressed in the form of the mean ± standard deviation (SD) or median (lower quartile: upper quartile) as appropriate. Categorical variables were presented as percentages. We compared baseline characteristics between CPAP and UC groups using a two-sample independent *t*-test or nonparametric test as appropriate. The changes in the HSAT indicators from baseline to end of study were compared between treatment groups to clarify the effect of CPAP on AHI, ODI, and measurements of SpO_2. Subgroup analysis was further implied to explore the effect of CPAP on different degrees of OSA severity. Data analysis was conducted using SPSS software (SPSS standard version 25.0, SPSS Inc.).

Results

There were 151 participants in the SAVE study at the Guangdong Provincial People's Hospital site. Forty-nine participants were unable to be included in the present analysis: 7 had died in the observation phase, 10 were lost to follow-up, and 32 refused the HSAT reevaluation, leaving 102 participants (83% male), aged (mean ± SD) 61.9 ± 6.8 years who were followed for 48.0 ± 14.5 months (Fig. 1). Table 1 lists the baseline characteristics of these participants. Figure 2 shows the nightly usage of CPAP during follow-up in the CPAP group. The mean nightly CPAP adherence over the whole duration of follow-up in those allocated to receive the treatment was 4.1 ± 1.9 h. Table 2 describes the changes in AHI, ODI, and other OSA indicators from baseline to end of study. Although there was a decrease in AHI in both CPAP and UC groups (mean (95% CI): − 5.0 (− 12.5,2.0) and − 4.0 (− 12.5,1.5) events per hour, respectively), no statistical difference was evident between the two groups. Similarly, there were no significant differences between treatment groups in ODI though there were significant intra-group differences from baseline to the end of the study. The decrease of AHI was mainly due to a decrease in the apnea index. Long-term CPAP showed a positive effect on nadir SpO_2. Supplementary Table S1 lists some anthropometric variables that are known to be related to AHI and ODI. There were small increases in neck and waist circumference in CPAP and UC groups but no between group differences.

Patients were categorized into three subgroups based on disease severity and the effect of long-term CPAP on OSA severity was further exploded. Table 3 shows that long-term CPAP showed a positive effect on AHI in moderate OSA group (− 8.0 (− 11.8, − 2.8) in CPAP group, − 2.0 (− 0.8,6.0) in UC group, $P = 0.022$) and improve nadir SpO_2 in severe OSA group (5.0 (− 0.8, − 0.8,7.0) in CPAP group, 0.0 (− 8.5,2.5) in UC group, $P = 0.032$). Supplementary Table S2 lists the baseline characteristics of these participants according to subgroups.

Discussion

The main finding of this study was that long-term CPAP treatment had no effect on the underlying severity of sleep disordered breathing as measured by either AHI or ODI. A small reduction in AHI and ODI occurred in both study groups with no between-group differences. However, CPAP

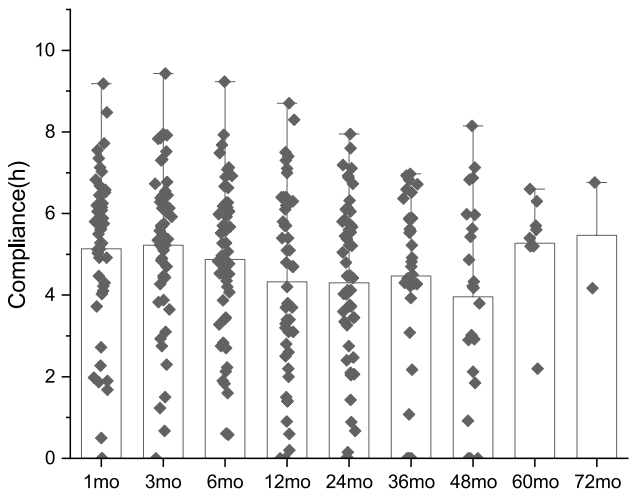

Fig. 2 Bar chart of CPAP adherence during follow-up. Each bar is presented as mean ± standard error with point markers indicating individual participants

Table 2 Baseline, end-of-study, and changes in OSA characteristics

Characteristic	CPAP ($N=51$)				UC (N = 51)				Between group
	Baseline	End-of-study	Delta End-of-study minus baseline	*P* values	Baseline	End-of-study	Delta End-of-study minus baseline	*P* values	*P* values
Age (year)	61.3 ± 6.9	65.2 ± 7.3	3.9 ± 1.2	0.000*	62.4 ± 6.8	66.5 ± 7	4.1 ± 1.2	0.000*	0.565
Obstructive sleep apnea characteristics									
AHI (events/h)	22.0 (16.0, 34.0)[a]	19.0 (9.0,27.0)[a]	− 5.0 (− 12.5, 2.0)[a]	< 0.001*	24.0 (17.0, 42.0)[a]	25.0 (11.0, 33.0)[a]	− 4.0 (− 12.5, 1.5) [a]	0.007*	0.453
AI (events/h)	15.0 (7.0, 28.0)[a]	10.0 (3.0, 19.0)[a]	− 4.0 (− 12.0, 0.5)[a]	0.001*	17.0 (9.0, 32.0)[a]	15.0 (6.0, 24.0)[a]	− 1.0 (− 8.0, 4.0)[a]	0.039*	0.197
HI (events/h)	7.0 (4.0, 10.0)[a]	6.0 (4.0, 11.0)[a]	− 1.0 (− 4.0, 2.5)[a]	0.539	7.0 (4.0, 12.0)[a]	6.0 (3.0, 9.0)[a]	− 1.0 (− 4.0, 1.0)[a]	0.051	0.354
AI/HI	2.7 (0.8, 6.8)[a]	1.4 (0.6, 3.0)[a]	− 0.6 (− 2.0, 0.3)[a]	0.207	2.3 (1.0, 5.7)[a]	2.0 (1.0, 3.7)[a]	0.1 (− 1.7, 0.9)[a]	0.296	0.110
ODI (events/h)	19.0 (15.0, 32.0)[a]	15.0 (11.0, 23.0)[a]	− 6.0 (− 12.0, 0.5)[a]	< 0.001*	26.0 (17.0, 37.0)[a]	23.0 (9.0, 31.0)[a]	− 6.0 (− 12.0, 0.0) [a]	0.001*	0.851
Nadir SpO_2	78.6 ± 6	80.8 ± 6.4	2.3 ± 6.1	0.011*	78.1 ± 5.6	77.4 ± 8.3	− 0.7 ± 7.6	0.511	0.032*
Mean SpO_2	93.9 ± 1.6	94.4 ± 1.6	0.4 ± 1.4	0.028*	94.3 ± 1.6	94.2 ± 1.6	0.0 ± 1.2	0.909	0.082
Time < 90% SpO_2 (min)	22.0 (8.0, 63.0)[a]	11.0 (3.0, 43.0)[a]	− 7.0 (− 24.0, 1.0)[a]	0.002*	23.0 (12.0, 41.0)[a]	19.0 (7.0, 55.0)[a]	− 2.0 (− 17.0, 10.0)[a]	0.650	0.063
Min pulse (beats/min)	50.5 ± 6.9	49.2 ± 5.7	− 1.3 ± 3.8	0.021*	52.2 ± 6.4	52.1 ± 6.7	− 0.1 ± 6.4	0.914	0.264
Max pulse (beats/min)	95.3 ± 23.3	88.9 ± 10.3	− 6.4 ± 23.1	0.054	95.2 ± 17.7	93.9 ± 17.4	− 1.3 ± 27.1	0.739	0.307
Mean pulse (beats/min)	61.6 ± 7.6	59.1 ± 8.0	− 2.5 ± 6.5	0.009*	63.9 ± 6.6	63.9 ± 9.2	0.0 ± 6.8	0.967	0.068

CPAP, continuous positive airway pressure group; *UC*, usual-care group; *AHI*, apnea–hypopnea index; *AI*, apnea index; *HI*, hypopnea index; *AI/HI*, the radio of AI to HI; *ODI*, oxygen desaturation index; *SpO_2*, oxygen saturation

*Significant at the $p < 0.05$ level

[a]Median (quartile 1; quartile 3)

Table 3 Changes in OSA characteristics among three groups

Characteristic (delta end-of-study minus baseline)	Mild OSA ($N=16$)			Moderate OSA ($N=49$)			Severe OSA ($N=37$)		
	CPAP ($N=9$)	UC ($N=7$)	*P* values	CPAP ($N=26$)	UC ($N=23$)	*P* values	CPAP ($N=16$)	UC ($N=21$)	*P* values
AHI (events/h)	2.0 (−4.5, 9.0)	−4.0 (−5.0, −1.0)	0.408	−8.0 (−11.8, −2.8)	−2.0 (−8.0, 6.0)	0.022*	−9.5 (−24.5, 2.0)	−12 (−23, 1.5)	0.988
AI(events/h)	1.0 (−0.5, 5.0)	−1.0 (−2.0, 1.0)	0.091	−5.0 (−12.0, −0.8)	0.0 (−3.0, 5.0)	0.003*	−10.5 (−17.8, −1.5)	−8.0 (−20.5, 4.5)	0.916
Nadir SpO_2	−1.0 (−6.5, 3.0)	1.0 (−1.0, 6.0)	0.351	2.5 (−1.0, 7.0)	0.0 (−5.0, 4.0)	0.169	5.0 (−0.8, 7.0)	0.0 (−8.5, 2.5)	0.032*
Mean SpO_2	1.0 (0.5, 2.0)	1.0 (0.0, 1.0)	0.408	0.5 (−0.3, 1.0)	0.0 (0.0, 1.0)	0.557	0.5 (0.0, 1.8)	0.0 (−1.5, 0.0)	0.037*

CPAP, continuous positive airway pressure group; *UC*, usual-care Group; *AHI*, apnea–hypopnea index; *AI*, apnea index; *HI*, hypopnea index; *AI/HI*, the radio of AI to HI; *ODI*, oxygen desaturation index; *SpO_2*, oxygen saturation

*Significant at the $p<0.05$ level

[a]Median (quartile 1; quartile 3)

did result in a small positive effect on the nadir night-time oxygen saturation. Further subgroup analysis showed that CPAP may have a variable effect according to the severity category of OSA.

These results appear to be at variance with several previously published studies [7–13] which reported small reductions in the frequency of obstructive breathing events after several months of CPAP. However, these studies were designed as before and after studies and lacked a control group.

We can only speculate as to possible reasons for the small reductions in AHI and ODI observed in both CPAP and UC groups. It could be due to regression to the mean, or, alternatively, a Hawthorne effect whereby participants, simply by virtue of being enrolled in a clinical trial, altered one or more other behaviors (e.g., medication adherence, improvements in alcohol, diet or exercise) that improved OSA severity. However, in the present sub-study population a small increase rather than decrease in weight was observed in the CPAP-treated participants [15]. The decrease of AHI and ODI could also be explained by the natural history of OSA. However, evidence from previous longitudinal studies is highly variable with one study reporting an increase in AHI but no change in SpO_2 parameters over 3 years [20]. Another study of 100 patients with OSA followed for 5 years showed no change in AHI [21], and a further study of 152 patients with moderate to severe OSA followed for 7 years showed a decrease in AHI but worsening of sleep oxygenation [22].

Compared with the UC group, the CPAP group showed an improvement in nadir SpO_2 (and marginal improvement in time spent below 90% SpO_2) despite there being no between-group difference in the frequency of obstructive events. One possibility is that CPAP treatment caused a reduction in the ratio of apnea to hypopnea events such that the decrease in ventilation per event might tend to be less. An increase in the size of the pharynx (awake) [23] and a reduction in the proportion of sleep apneas versus hypopneas [9, 10] have been previously reported following prolonged CPAP treatment. In this study, however, there was no change in the ratio of apneas to hypopneas within or between groups. Another possibility is that the average length of obstructive events was decreased because of a decrease in arousal threshold (i.e., subjects aroused more quickly to an obstruction [6]). Finally, a change in sleep architecture could also explain the results: there are two reports of less REM sleep and more non-REM sleep after withdrawal of long-term CPAP compared with pretreatment sleep studies [8, 10]. Unfortunately, the information recorded by the ApneaLink device did not allow us to explore these latter two possible mechanisms.

Whatever the explanations for the changes observed between and within groups in SpO_2 and AHI, it is important to acknowledge that they were very small and are unlikely

to be clinically significant. The observed decrease in AHI of 5 events per hour in both groups is well below the agreed minimal clinically significant change in AHI of 15 events per hour [24]. Furthermore, previous studies point to the transient nature of any improvement in the underlying severity of OSA as a result of CPAP treatment [7]. Our results, combined with those of previous studies do not therefore support the proposition that brief "vacations" from CPAP can be safely taken in patients with moderate to severe OSA who are prescribed long-term CPAP treatment. Our study suggests that CPAP treatment alone is unlikely to resolve the physiological abnormalities underpinning OSA, and therefore needs to be prescribed as an indefinite treatment.

Our subgroup analysis found that CPAP only decreased AHI and AI in patients with moderate OSA and increased nadir and mean SpO_2 in patients with severe OSA. Few studies have focused on this kind of phenomenon and the possible meanings hidden behind need to be further investigated.

We acknowledge that this post hoc sub-study of the main SAVE trial has several limitations. First, it was conducted at a single site on a relatively small sample. Greater precision would have been possible had we used more participants' data from across the recruitment network. However, CPAP participants at this site had considerably higher adherence to CPAP than for the study population as a whole, and the mean follow-up period was longer. The sample size provided ample statistical power to definitively answer whether or not a clinically significant change in OSA was present after sustained CPAP treatment. Second, the study population had established CV disease and was somewhat older compared with patients attending sleep clinics; and patients with very severe nocturnal hypoxemia or a predominantly Cheyne-Stokes respiration pattern were excluded. The study results may not therefore extrapolate to general sleep apnea clinic populations. Lastly, we used a type 3 portable sleep monitoring device in this study. The device was chosen because of the unavailability of polysomnography (PSG) in many recruitment sites and the cost and difficulty of setting up and standardizing PSG measurements across multiple centers and countries. The ApneaLink™ automated screening device provided a reliable and generalizable alternative. It was validated against PSG in a high cardiovascular-risk Chinese population before the SAVE trial and found to have high diagnostic accuracy for moderate to severe OSA [25]. It is unlikely therefore to have influenced the overall findings in this study. Nonetheless, the parameters collected to evaluate the severity of OSA were limited, preventing, for example, an evaluation of whether or not the length of obstructive events changed after CPAP treatment.

In summary, long-term CPAP use in patients with comorbid OSA and cardiovascular disease did not result in clinically significant change in AHI or ODI when compared with patients without CPAP, but showed a small positive effect on nadir SpO_2.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s11325-022-02633-y .

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Author contribution The study was designed by DM, QO, KL, and YC; data were collected by YC, QO, BC, YX, QW, and ML; data were analyzed by YC and results interpreted by all authors. The manuscript and figures were drafted by YC and critically revised by QO, DM, and KL. QO serves as the guarantor of the paper, taking responsibility for the integrity of the work as a whole, from inception to publication of the article.

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Data availability The detailed participant data are available from the corresponding author upon reasonable request.

Declarations

Ethics approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Consent to participate Participants signed ethics committee-approved consent forms prior to participation.

Consent for publication Not applicable.

Conflict of interest The authors declare no competing interests.

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