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Evaluation of an auto-nCPAP device based on snoring detection

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Evaluation of an auto-nCPAP device based on snoring detection. F. Lofaso, A.M. Lorino, D. Duizabo, H. Najafi Zadeh, D. Theret, F. Goldenberg, A. Harf. ©ERS Journals Ltd 1996.

ABSTRACT: We evaluated an auto-nasal continuous positive airway pressure (nCPAP) prototype (MC+; SEFAM, Nancy, France) in which apnoea/hypopnoea detection was disabled and nasal mask pressure vibration detection was the only mode of pressure setting. The device was tested in 15 previously untreated obstructive sleep apnoea patients during a night with polysomnography.

We observed that a single night of auto-nCPAP improved the apnoea/hypnoea index (AHI) (12±21 *vs* 51±31 disordered breathing events·h^{-1} of sleep (mean±SD)), the awakening-arousal index (13±20 *vs* 40±26 arousals·h^{-1} of sleep), and duration of slow wave sleep (102±49 *vs* 71±56 min) but not of rapid eye movement (REM) sleep (55±31 *vs* 64±38 min). Auto-nCPAP was effective (apnoea/hypopnoea and arousal indices <10 events·h^{-1}) in all but three patients. Auto-nCPAP was ineffective in one patient, whose obstructive respiratory events were not preceded by nasal mask pressure vibration detection, and in two patients who were quasi-permanent mouth breathers.

Snoring detection may be effective in sleep apnoea syndrome with heavy snoring and without permanent mouth breathing, during the first night of nasal continuous positive airway pressure treatment.

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Nasal continuous positive airway pressure (nCPAP), which was introduced in 1981 by SULLIVAN *et al*. [1], has considerably improved the treatment and prognosis of patients suffering from obstructive sleep apnoea (OSA) [1[. In practice, the optimal nCPAP level is a trade-off between pressure-related side-effects and efficacy in preventing upper airway obstruction during sleep [2]. This optimal level is generally determined during a total or split night study, but follow-up is needed to verify that the selected level remains appropriate for the patient's needs, since the minimal effective pressure can vary over time depending on weight variations, sleep deprivation, nasal obstruction, and ingestion of alcohol or hypnotic agent [3]. For example, an increase rather than a decrease has been found in the efficient pressure level during the 3–8 months following the nCPAP titration night [4]. In addition, this minimal pressure can change during a given night, depending on body position and/or sleep stage [3].

To try to improve the efficacy in relieving upper airway obstruction and the acceptability of nCPAP, new nCPAP devices have been developed that use noninvasive indirect assessment of upper airway obstruction to continuously adjust the pressure around the minimal level that prevents abnormal breathing and arousal. The most sophisticated devices can detect respiratory events (apnoea/hypopnoea), flow limitation, and snoring at the mask (AutoSet; ResCare, Sydney) [5]. Other auto-nCPAP devices ensure apnoea-hypopnoea detection with (Virtuoso, Respironics Inc., Murrysville, USA; MC+, SEFAM, Villers-lès-Nancy, France) [6, 7], or without (Morphée plus, Pierre Médical Verrières le Buisson, France) [8] snoring detection.

Because most devices use these indices of upper airway obstruction in combination, it is difficult to evaluate the advantages and limitations of each index. A given system may be effective in some patients but ineffective or even harmful in others. For example, systems that use inspiratory-expiratory differences in pressure (or the nCPAP flow regimen when this parameter is servocontrolled to minimize pressure variations) (Virtuoso, Respironics Inc., Murrysville; MC+, SEFAM, Villers-lès-Nancy, France; Morphée plus, Pierre Médical Verrières le Buisson, France) to detect respiratory events cannot differentiate between obstructive apnoea, central apnoea, and intermittent mouth breathing. These nCPAP systems, therefore, automatically increase their pressure level during central apnoea (for example at sleep onset) and intermittent mouth breathing. These unprofitable increases in pressure may reduce the patient's tolerance to nCPAP [3]. Since all commercial auto-nCPAP devices use apnoea-hypopnoea detection for adjusting their pressure, no data were available on the efficiency of detection of snoring and/or detection of flow limitation used alone.

The purpose of this study was to determine whether an auto-titrating nCPAP system based on detection of snoring would yield meaningful improvements in sleep and

breathing during a single night of use. The working hypothesis was that such a device would have beneficial effects on the apnoea/hypnoea index (AHI), arousal index (ArI), and sleep architecture. To test this hypothesis, a prototype of a now commercially-available auto-nCPAP device (MC+, SEFAM, Nancy, France) was used, in which the nCPAP level was set based only on detection of snoring, *i.e.* in which detection of apnoea-hypopnoea was disabled.

Methods

Patients

This study was approved by the Research Ethics Committee of our institution. Patients were included in whom clinical suspicion of obstructive sleep apnoea syndrome (OSAS) was confirmed by a complete polysomnographic study using a 14-channel paper recorder (Electroencephalograph Nihon Kohden, Tokyo, Japan) and including electroencephalography (EEG) (C4-A1, C3-A2), electrooculography (EOG), chin electromyography (EMG), EMG of the tibialis anterior of both legs, oronasal thermistors, thoracic and abdominal movements, and arterial pulse oximetry (Nellcor BS; Nellcor Inc., Hayward, USA). Patients with restless legs, cardiac failure, cerebrovascular disease, or lung disease according to the reference spirometry values determined by the European Community were excluded. Criteria for inclusion were: 1) AHI ≥10 events·h^{-1} of sleep, with obstructive events >80% of total events; and 2) clinical indication of nCPAP as previously described by the American Thoracic Society (ATS) [2]. Fifteen male patients who fulfilled these criteria (mean age 58±9 yrs; mean body mass index (BMI) 33±5 kg·m^{-2}; mean Epworth sleepiness scale [9] 13.6±4.1; mean AHI 51±30 events·h^{-1} of sleep) gave their informed consent to participation in the study. All patients were permanent or quasi-permanent snorers, as described previously [10], and 11 of them had heavy snoring that resulted in social impairment even though they slept in a different room from other family members. None had received nCPAP. No sedatives, hypnotics or alcohol were taken by the patients prior to either study.

Pressure-setting algorithm of the tested device

Snoring was detected based on the occurrence of mask pressure vibration. Mask pressure was measured using a high frequency response pressure transducer placed inside the nCPAP device (REM+ control with MC+, SEFAM, Nancy, France). The pressure signal was band-pass filtered (30–280 Hz), and its amplitude was analysed using an automatic threshold system to detect amplitude variations as snoring. The pressure was increased by 1 cmH_2O if three consecutive snoring breaths were detected. The algorithm of snoring detection was then disabled for 5 min. On the contrary, when no vibrations were detected, airway pressure was reduced by 1 cmH_2O every 10 min. Thus, snoring was the only criterion used in this auto-nCPAP prototype to increase or decrease the pressure level.

Clinical trial

All patients received overnight auto-nCPAP treatment (REM+ with MC+, SEFAM, Nancy, France), with polysommnography to establish the efficacy of the device. The all-night polysomnographic study performed before inclusion to confirm the diagnosis of OSA served as the control. In all patients, polysomnography with auto-nCPAP included EEG (C4-A1, C3-A2), EOG, chin EMG, nasal flow measured by a Fleisch No. 2 pneumotachograph (Lausanne, Switzerland) connected to a differential pressure transducer (Validyne MP45 ±5 cmH_2O, Northridge, CA, USA), thermistance-evaluated oral airflow to differentiate mouth breathing from apnoea-hypopnoea, thoracic and abdominal movements, arterial pulse oximetry (Nellcor BS, Nellcor Inc., Hayward, USA), posture detected by a mercury tilt switch, and nasal mask pressure measured by a differential pressure transducer (Validyne MP45 ±35 cmH_2O). Polysomnography included oesophageal pressure (P_{oes}) for eight patients who agreed to swallow the oesophageal pressure catheter (Gaeltec, Dunvegan, Isle of Skye, UK).

Initial pressure of nCPAP was always set at the minimal level, *i.e.* 4 cmH_2O. The algorithm allowed nCPAP pressure to increase up to a maximal level of 18 cmH_2O.

Data analysis

Sleep staging was performed according to standard criteria [11]. EEG arousals were detected by an abrupt shift in EEG frequency lasting less than 15 s and including alpha activity and/or frequencies greater than 16 Hz, except spindles, and were scored according to standard criteria [12]. Alpha activity lasting more than 15 s was considered as indicating awakening. An abnormal breathing event during sleep was defined, according to commonly used clinical criteria, as either complete cessation of airflow lasting at least 10 s (apnoea), or an at least 50% decrease in airflow as compared to the previous breath lasting at least 10 s (hypopnoea). Abnormal breathing events were classified as obstructive or central according to whether respiratory effort continued or was correlated to the flow reduction. The average number of apnoeas and hypopnoeas per hour of sleep (AHI) was calculated from the sum of sleep-disordered breathing events. nCPAP was considered effective in preventing sleep apnoea syndrome (SAS) when: 1) AHI was reduced by more than 50%; and 2) both AHI and arousal-awakening index (sum of arousals and awakenings per unit time, Ar-AwI) were less than the normal values used in our laboratory [13], *i.e.* less than 10 events·h^{-1} of sleep. In addition, the percentage of total sleep time spent with oesophageal pressure swings lower than twice their mean value measured during quiet breathing in the awake state was evaluated [14].

Statistical analysis

Results of baseline polysomnography and polysomnography with auto-nCPAP were compared using Wilcoxon's test. This was carried out for the entire patient group and for subgroups with and without oesophageal pressure data. Results are given as mean±SD.

Results

The main sleep and respiratory parameters under basal conditions and under auto-nCPAP treatment are presented in table 1. An example of detection of nasal mask pressure vibration is depicted in figure 1, and an example of all-night slow recording of nCPAP pressure variations is presented in figure 2. The number of nCPAP pressure changes was 42±15 per night.

Table 1. – Sleep and respiratory data

	Basal condition	Auto-nCPAP	p-value
Sleep-onset latency min	31±29	23±38	NS
Stage 4 latency min	131±172	45±82	<0.05
REM sleep latency min	123±93	140±114	NS
TST min	369±86	346±100	NS
Wake after sleep onset min	81±77	118±105	NS
Stage I min	44±36	35±22	NS
Stage II min	190±47	154±66	<0.05
Slow wave sleep min	71±86	102±149	<0.05
REM min	64±38	55±31	NS
Ar-AwI events·h^{-1}	40±26	13±20	<0.05
Apnoea index events·h^{-1}	30±21	3±6	<0.05
AHI events·h^{-1}	51±31	12±21	<0.05
Central apnoea/hypopnoea % of A/H	5±3	6±4	NS
Lowest Sa,O_2 %	69±17	89±3	<0.05
Duration of Sa,O_2 under 90% min	98±126	39±101	<0.05

Values are presented as mean±SD (n=15). REM: rapid eye movement; TST: total sleep time; A/H: apnoeas/hypopnoea; Ar-AwI: arousal-awakening index; AHI: apnoea-hypopnoea index; Sa,O_2: arterial oxygen saturation; nCPAP: nasal continuous positive airway pressure; NS: nonsignificant.

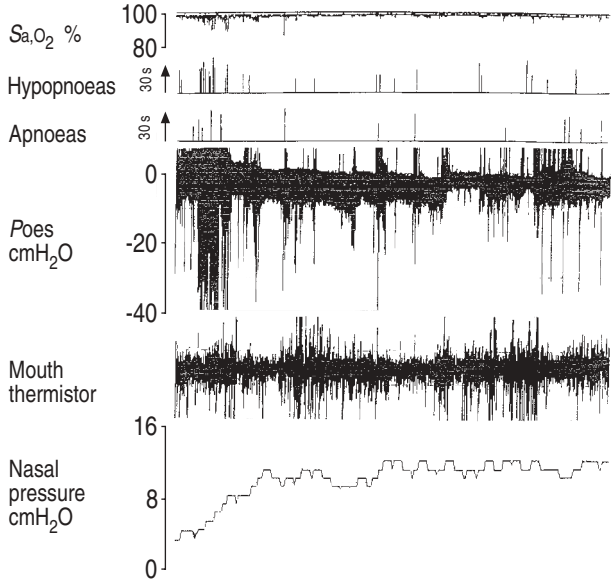

Fig. 2. – Typical overnight slow recording of arterial oxygen saturation (Sa,O_2), oesophageal pressure (*P*oes), oral thermistance (mouth thermistors) and nasal pressure variation with auto-nCPAP. In addition, the occurrence of each apnoea or hypopnoea is represented by a vertical line, whose length is proportional to the duration of the event. The automatic increases or decreases in mask pressure correspond to the abrupt steps observed on the pressure signal. During the first part of the night, when the auto-nCPAP pressure is at its lowest level, abnormal respiratory events and oesophageal pressure swings occurred. This inefficiency was due, in part, to disablement of the snoring detection algorithm for 5 min after each episode of snoring. To improve efficiency, the disablement period of the now commercialized device (REM+ auto, SEFAM, Nancy, France) has been reduced to 1 min. nCPAP: nasal continuous positive airway pressure.

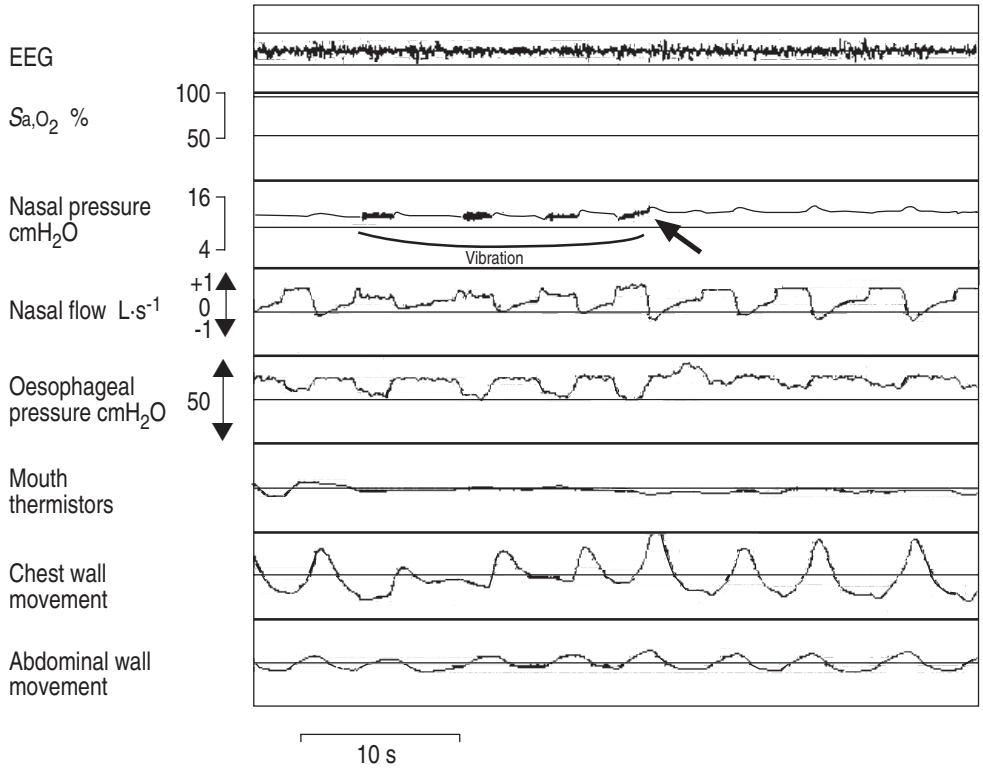

Fig. 1. – Typical snoring identified based on inspiratory vibration of pressure at the nasal mask followed by an increase in auto-nCPAP pressure (arrow) and by normalization of the oesophageal pressure swing. nCPAP: nasal continuous positive airway pressure; EEG: electroencephalogram; Sa,O_2: arterial oxygen saturation.

Auto-nCPAP was effective in 12 of the 15 patients, as assessed based on decreases both in AHI and Ar-AwI (fig. 3). In these 12 patients, mean and maximal nCPAP levels were 7.5±2.5 and 9.9±2.8 cmH_2O, respectively, and the lowest oxygen saturation improved from 69±17 to 89±3% ($p<0.05$). Three of the 12 patients were not heavy snorers.

In the eight subjects whose oesophageal pressure was recorded, 88±5% of the total sleep time (TST) was spent with oesophageal pressure swings lower than twice their mean value measured during quiet breathing in the awake state.

No arousals or awakenings that were not explained by respiratory events were observed within the 30 s after nCPAP pressure changes.

Interestingly, auto-nCPAP was ineffective in one non-heavy snorer, whose obstructive events were not preceded by detectable snoring, and in two heavy snorers, who were mouth breathers and had a mouth leak greater than 0.5 $L \cdot s^{-1}$ as detected by the nasal pneumotachograph. An example of inefficiency of auto-nCPAP during mouth breathing is shown in figure 4.

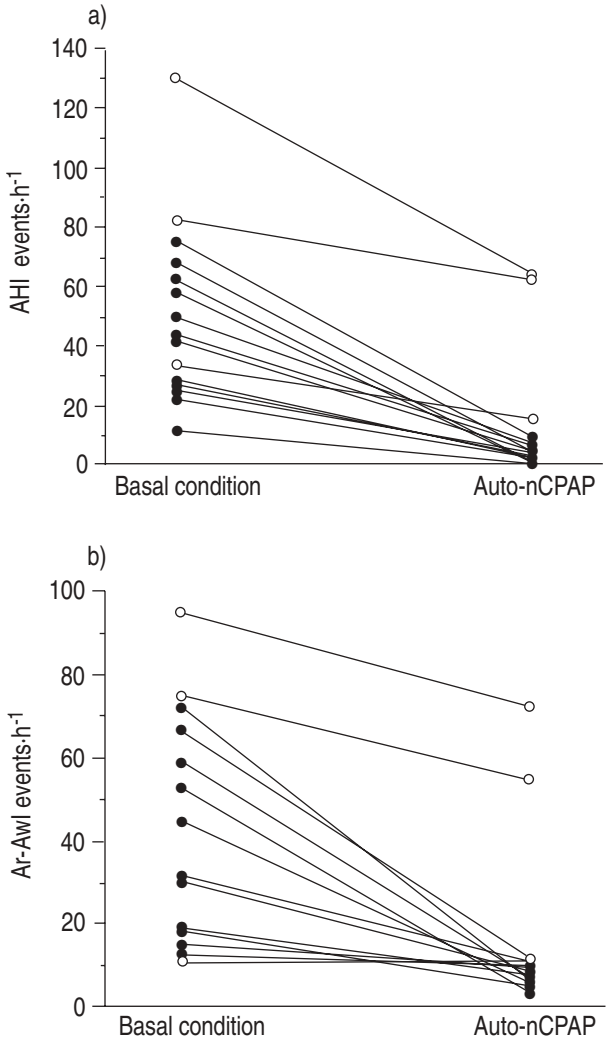

Fig. 3. – Effect of auto-nCPAP on: a) the apnoea-hypopnoea index (AHI); and b) the arousal-awakening index (Ar-AwI). Both were reduced to the normal range (—●—) (10 events,h^{-1}) in all but three subjects (—○—). nCPAP: nasal continuous positive airway pressure.

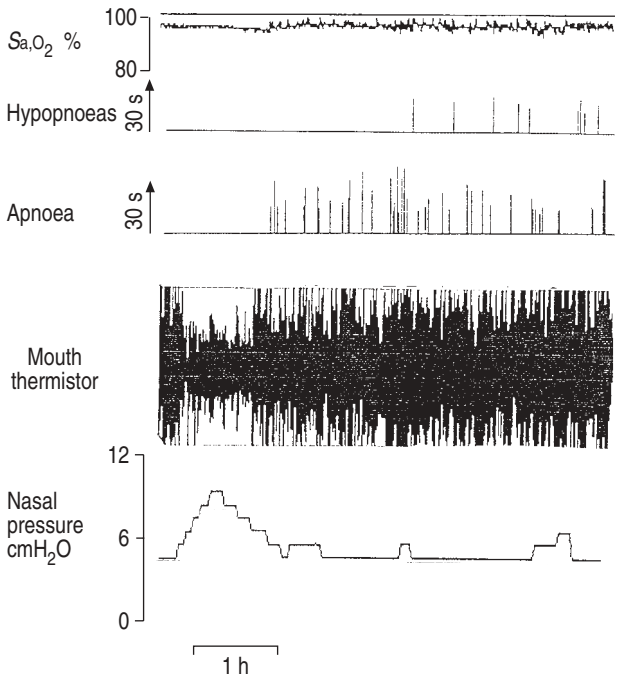

Fig. 4. – Overnight slow recording of nasal pressure variations, arterial oxygen saturation (S_{a,O_2}) oral thermistance (mouth thermistor), and duration of respiratory events, in a quasi-permanent mouth breather, with auto-nCPAP. Note that during the first part of the night, nasal pressure can vary, whereas no respiratory event occurs when mouth breathing is reduced. In contrast, during the second part of the night, nasal pressure remains at its lower level, whereas respiratory events occur when mouth breathing is increased. The scale of the oral thermistor tracing is the same as in figure 2.

Discussion

Despite improvements in nCPAP devices, long-term compliance in patients under nCPAP therapy has varied from 45 to 90% [15–19]. There are many reasons why patients may not tolerate nCPAP. A number of minor side-effects related to a fixed high level of nasal pressure [3] have been reported, including: discomfort due to the mask; rib cage discomfort; difficulty in breathing against the machine; soreness and/or dryness of the nose, mouth or throat; nasal congestion; and poor sleep due to mask leaks and noise generated by the device [15–19]. To reduce the overnight mean nasal pressure value, new nCPAP devices that automatically and rapidly adjust the pressure to the minimal effective level have recently been developed. Results on the feasibility of treatment with some auto-nCPAP devices have been published as preliminary studies and/or abstracts [5–7, 20, 21]. Some devices have been designed to detect respiratory events, flow limitation and snoring [5, 20], whereas others have been designed to detect only respiratory events [21], or both respiratory events and snoring [6, 7].

To our knowledge, we are the first to use a device based only on detection of snoring. Because snoring is consistently present in OSAS patients [22], an auto-nCPAP device based only on detection of snoring may be effective in reducing obstructive sleep respiratory events and sleep fragmentation in patients with OSAS. It has been shown that snoring is associated with high frequency oscillation (ranging 40–90 Hz) of the soft palate, pharyngeal walls, epiglottis and tongue, resulting in airway

pressure and flow rate vibrations [23, 24]. Thus, the auto-nCPAP device used was controlled only by snoring detected by analysis of nasal mask pressure vibration. In the present study, this device was effective in reducing respiratory events and arousals during the first night under nCPAP in most of the patients.

Before discussing the implications of our findings, we will address several methodological issues. Definition of sleep fragmentation as an Ar-AwI of >10 events·h^{-1} of TST may seem arbitrary. Although the American Sleep Disorders Association (ASDA) has developed criteria for defining arousal, there is still room for subjectivity. Thus, normal subjects, snorers, and successfully treated SAS patients had less than 10 short EEG arousals per hour of sleep in some studies [21, 25, 26], whereas in another study non-OSA control subjects had an Ar-AwI of 21 events·h^{-1} [27]. Our selection of 10 arousals·h^{-1} of sleep as the threshold defining sleep fragmentation was based mainly on a recent study that we performed in 105 nonapnoeic heavy snorers [13]. In this study, slow wave sleep values were normal in middle-aged nonapnoeic snorers with an Ar-AwI of <10 events·h^{-1} (mean 7±2 events·h^{-1}), but were abnormal in those with an Ar-AwI of >10 events·h^{-1} (mean 15±5 events·h^{-1}). This finding proves that when the ASDA criteria were used in our laboratory arousals occurring more often than 10 times per hour significantly affected the architecture of nocturnal sleep.

A number of methodological characteristics of the present study may have led to underestimation of the efficacy of auto-nCPAP. Firstly, use of a thermistor during the diagnostic night instead of a pneumotachograph, which provides a quantitative signal, may have resulted in underestimation of the hypopnoea index. For example, hypoventilation with bradypnoea may be associated with an increase in the temperature of the expired gas, which may in turn increase the flow signal of a thermistor, despite the occurrence of hypopnoea that would have been detected by a pneumotachograph. Secondly, the presence of an oesophageal catheter in eight subjects during the nCPAP night may have contributed to the sleep disruption and may also explain why there was no rapid eye movement (REM) sleep rebound during the night with auto-nCPAP treatment; another explanation for this absence of REM rebound may be the relatively low level of REM deprivation before nCPAP titration. Thirdly, the snoring detection algorithm was disabled for 5 min after each episode of snoring, and this may have facilitated the occurrence of respiratory events, abnormal pressure swings, and sleep fragmentation. These events were particularly common during the first part of the night when the auto-nCPAP pressure was at its lowest level (fig. 2). However, similar inefficiency is observed during conventional nCPAP treatment, which involves use of a ramp of up to 45 min [28] to gradually increase the positive pressure to the prescribed level. The disablement period of the recently commercialized device (REM+ auto, SEFAM, Nancy, France) has been reduced to 1 min.

Despite the potential negative effects of the long disablement period on the efficiency of the device used in the present study, the AHI and the Ar-AwI were brought down to <10 events·h^{-1} in 12 of the 15 SAS patients. In addition, we observed similar results during the same period in 17 SAS patients treated with conventional nCPAP with one titration night and one follow-up night under a constant level of pressure. These patients did not differ from those of the present study in terms of age (62±9 yrs), BMI (32±9 kg·m^{-2}), AHI (59±24 events·h^{-1}), or AI (44±27 arousals·h^{-1}). During the follow-up night, the mean constant pressure was 9.5±2.4 cmH_2O, and 14 of the 17 patients had an AHI and an Ar-AwI of <10 events·h^{-1} of TST, with AHI (4.8±2.9 events·h^{-1}) and Ar-AwI (5.7±3.4 events·h^{-1}), similar to those found in the present study (unpublished data).

Clearly, preventing snoring does not always prevent respiratory events and consequent sleep fragmentation. Although the auto-nCPAP device used in the present study appeared to be effective in most of the patients, it failed in two mouth breathing patients and in one patient whose respiratory events were not preceded by detectable snoring. In the two patients with permanent mouth breathing, nasal mask pressure vibrations were seldom present despite the occurrence of respiratory events, resulting in nCPAP underprescription. We have no data on mouth breathing during nCPAP in our overall patient population; to overcome the problems raised by mouth breathing, SANDERS *et al*. [29] recently used an oronasal mask in 30 of 245 patients who underwent a trial of nCPAP or bilevel positive airway pressure. Use of an oronasal mask would probably improve the efficiency of our auto-nCPAP device, but to our knowledge auto-nCPAP devices have never been used with an oronasal mask. In addition, LIISTRO *et al.* [24] demonstrated that the frequency of pressure oscillations was lower during simulated snoring through the mouth (28 Hz) than through the nose (78 Hz), which suggests that our device that uses a band-pass filter (30–280 Hz) may fail to detect snoring in a mouth breather equipped with an oronasal mask. However, BERTHON-JONES [5] reported that use of a chin-strap with his auto-nCPAP device yielded good results in obese patients.

In one patient, whose obstructive apnoeas were seldom preceded by detectable snoring, the control algorithm tested in this study failed. However, this patient, who was considered by his bed partner as a quasi-permanent snorer, did not snore loud enough to be heard behind a dividing wall. In a very recent study [7], snoring was recorded by means of a stethoscope secured to the trachea in 13 patients who used the MC+ device controlled both by apnoea and snoring detection. Polysomnographic traces were then reviewed, with concomitant detection of sound with earphones in order to identify snoring. The authors observed that the device increased its pressure in response to only 84±6% of the snoring events. This suggests that the MC+ device may fail to respond consistently to low intensity snoring.

We observed that oesophageal pressure swings returned to normal except during 12% of the TST. Although a recent study found that during nCPAP titration arousals stopped before oesophageal high swings [30], we do not know what happens when the initial sleep deprivation is less acute. After having completed their slow wave rebound, patients may become more arousable and, due to its excessively low pressure level, nCPAP may convert obstructive sleep apnoea to airway resistance syndrome. Because flow limitation is a prerequisite to the occurrence of snoring [23], improving auto-nCPAP control by a system capable of detecting

inspiratory flow limitation may increase the efficacy of nCPAP in preventing partial airway obstruction [5]. On the other hand, because flow limitation has previously also been reported in healthy nonsnorers [31], use of an auto-nCPAP device controlled by flow limitation may induce an unprofitable increase in the nCPAP level. Although more simple than the algorithms based on flow limitation, the algorithm used in the present study to control the nCPAP setting was apparently effective during the first nCPAP night in heavy snorers without permanent mouth breathing or central apnoeas.

In conclusion, the results of this study suggest that nasal continuous positive airway pressure devices controlled by detection of snoring may prevent apnoea and/or arousal in heavy snorers with sleep apnoea syndrome. However, use of nasal continuous positive airway pressure devices controlled by snoring is not appropriate in non-heavy snorers, permanent mouth breathers, patients with central sleep apnoea syndrome, and patients with severe lung disease or respiratory failure. Further studies are needed to determine whether the beneficial effect observed during the first night persists during the following nights, when sleep deprivation is no longer present.

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