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Auto-CPAP therapy based on the forced oscillation technique

Auto-CPAP-Therapie auf der Basis der Oszilloresistometrie

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Key words: Sleep apnea syndromes – forced oscillation technique – compliance – automatic CPAP – CPAP therapy

Autoadjusting CPAP devices (APAP) are designed to continuously adjust the positive pressure to the required levels, and thus increase treatment quality and patient compliance. The results of APAP treatment strongly depend on the control mechanism of the respective APAP device. In agreement with other working groups, we have recently shown that the forced oscillation technique (FOT) is capable of detecting incipient upper airway obstruction prior to physiological reactions such as the onset of increasing esophageal pressure swings or microarousals. Therefore we studied efficacy and acceptance of a novel APAP device controlled exclusively by FOT.
100 consecutive patients with OSAS confirmed by polysomnography (mean AHI 47.9 ± 22.6) and daytime sleepiness (Epworth sleepiness scale, ESS 12.6 ± 3.9) were randomized to either APAP treatment (n = 50) or conventional CPAP treatment (n = 50). Polysomnographies were performed at the second treatment night and subjective sleepiness (modified ESS) was established in the morning. The respiratory disturbance was largely normalized in both treatment groups in the second treatment night (AHI 4.7 ± 5.3 vs. 3.7 ± 3.4; n.s.). Both groups showed largely improved sleep profiles and had markedly reduced ESS-scores (6.6 ± 3.6 vs. 7.0 ± 3.4; n.s.). The mean treatment pressure during APAP was significantly lower than during CPAP treatment (6.0 ± 2.0 vs. 9.0 ± 1.8 mbar; $p < 0.001$). There were no significant differences between APAP and CPAP treatment in any parameter of efficacy or acceptance.
APAP treatment with this device controlled exclusively by FOT is well accepted by the patients and permits an adequate treatment of OSAS without the need for individual CPAP titration.

Schlüsselwörter: Schlafapnoe-Syndrom, Oszilloresistometrie, CPAP-Therapie, Auto-CPAP

Auto-CPAP-Geräte werden entwickelt, um während der nasalen CPAP-Therapie beim obstruktiven Schlafapnoe-Syndrom (OSAS) den Behandlungsdruck zu jedem Zeitpunkt an die aktuellen Erfordernisse anzupassen. In Übereinstimmung mit anderen Untersuchern konnten wir zeigen, daß die Oszilloresistometrie eine hochsensitive Methode darstellt, um eine beginnende obere Atemwegsobstruktion beim OSAS frühzeitig zu erkennen, bevor physiologische Reaktionen, z. B. eine vermehrte Negativierung des inspiratorischen Oesophagusdruckes oder gar Arousals auftreten. Vor diesem Hintergrund untersuchten wir die Therapiequalität und Akzeptanz eines Auto-CPAP-Gerätes auf der Basis der Oszilloresistometrie (Somnosmart).
100 Patienten mit einem OSAS (mittlerer AHI 47,9 ± 22,6) und deutlicher Tagesmüdigkeit (Epworth sleepiness scale, ESS 12,6 ± 3,9) wurden fortlaufend randomisiert, entweder einer Auto-CPAP-Therapie (n = 50) oder einer konventionellen CPAP-Therapie (n = 50) zugeführt. In der jeweils zweiten Therapienacht wurden Polysomnographien durchgeführt, und die subjektive Schläfrigkeit wurde mit einer modifizierten ESS ermittelt. Die Atmungsstörung war in beiden Gruppen in der zweiten Untersuchungsnacht (AHI 4,7 ± 5,3 vs. 3,7 ± 3,4) weitestgehend normalisiert. Beide Gruppen zeigten weitestgehend normale Schlafprofile und deutlich reduzierte ESS-Scores (6,6 ± 3,6 vs. 7,0 ± 3,4). Der mittlere Behandlungsdruck unter Auto-CPAP war signifikant geringer als während der konventionellen CPAP-Therapie (6,0 ± 2,0 vs. 9,0 ± 1,8 mbar; $p < 0{,}001$). In keinem Parameter der Effektivität oder der Akzeptanz fanden sich signifikante Unterschiede zwischen den beiden Therapieformen.
Die durch Oszilloresistometrie gesteuerte Auto-CPAP-Therapie wird von den Patienten gut akzeptiert und stellt auch ohne individuelle Anpassungen eine adäquate Therapie des OSAS dar.

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Introduction

Since its first description by Sullivan in 1981 nasal continuous positive airway pressure therapy (CPAP) has become accepted as the standard treatment for the obstructive sleep apnea syndrome (OSAS). During nasal CPAP therapy air is applied via a nasal mask at a constant elevated pressure. This pressure is propagated through the nose into the pharynx where, acting as a „pneumatic splint", it prevents the upper airway from collapsing when it is set high enough to exceed the actual critical closing pressure of the upper airways (P_{crit}). Not only can CPAP treatment prevent nocturnal respiratory disturbances, but it also appreciably improves vigilance and the daytime performance of persons with OSAS [3]. Conventional CPAP therapy is usually initiated by CPAP titration, mostly under polysomnographic control, where the minimum pressure capable of preventing all obstructive events during sleep (minimum effective pressure, P_{eff}) is determined. Subsequently, CPAP treatment is applied at home over lengthy periods of time at this unchanging P_{eff}. P_{crit} and thus P_{eff} is dependent on many factors such as body position [8], sleep stage, nasal obstruction and upper airway edema, all of which may vary during the night. Furthermore, variations in patient weight, sleep deprivation or the use of alcohol, hypnotics or sedatives may contribute to changes in P_{crit} over time [19, 14]. As a consequence various auto-adjusting CPAP devices (APAP) have been developed that continuously adjust the positive pressure in response to varying needs. During APAP therapy the treatment pressure is varied continuously such that, any point in time, the pressure applied is high enough to prevent even partial collapse of the upper airways („upper airway resistance") and at the same time, this pressure is kept as low as possible to prevent pressure-dependent side effects as for instance leakage of air at the nasal mask and to spare the patient the sensation of uncomfortably high treatment pressure. The merit of APAP treatment in comparison with conventional CPAP treatment has, to date, not been definitively clarified [10, 18]. The results achieved with APAP treatment obviously strongly depend on the control mechanism of the respective APAP device. Currently available APAP devices employ, for example vibrations in the CPAP mask, reductions in airflow or flattening of the inspiratory flow contour as control parameters [1, 12, 7]. In agreement with other working groups, we have recently shown that the forced oscillation technique (FOT) is a highly suitable method to continuously record the impedance of the upper airways during CPAP treatment [6, 4, 13]. In particular we have demonstrated that even a simplified version of FOT is capable of detecting incipient upper airway obstruction prior to physiological reactions such as the onset of increasing esophageal pressure swings or microarousals [6]. Subsequently, it was shown that it is possible to control an APAP device exclusively via this simplified FOT [15, 5, 16].

In the present study, we now compare the outcome of FOT-controlled APAP treatment with that achieved with conventional CPAP therapy during the initiation of treatment in a larger population of patients with OSAS.

Methods

Subjects

Subjects from among the patients referred to our sleep laboratory for the initiation of nasal CPAP therapy, we consecutively recruited 100 adult patients who met the following inclusion criteria: diurnal somnolence defined by a score of no less than 8 on the Epworth Sleepiness Scale (ESS) [9], an apnea-hypopnea index (AHI) > 10/h on standard polysomnography (see below) and written informed consent to participate in the study. Patients who had already undergone CPAP treatment, or had central sleep apnea syndrome or Cheyne-Stokes respiration, those with severe nasal obstruction, or other conditions contraindicating CPAP treatment, were excluded. Also excluded were patients with obstructive pulmonary disease (FEV1 < 70 % predicted) or congestive heart failure (NYHA class III or IV).

Sleep studies

The polysomnographic studies were performed in our sleep laboratory by trained sleep lab technicians. All variables were recorded on a computer (SleepLab, Jaeger and Toennies, Wurzburg, Germany), and included EEG (C4/A1, C3/A2), bilateral EOG, submental EMG, snoring detected by microphone, ECG, thoracic and abdominal movements measured by inductive plethysmography and oxyhaemoglobin saturation using a finger oximeter (Microspan 3040G, Jaeger and Toennies, Wurzburg, Germany). During the diagnostic studies, oral and nasal airflow was measured by thermistors, during CPAP-Therapy a pneumotachograph (Weinmann, Hamburg, Germany) was used. The CPAP pressure was measured continuously at the nasal mask.

Obstructive apneas were defined as the absence of oronasal airflow for at least 10 seconds. During diagnostic polysomnographies hypopneas were defined as a reduction in thoracoabdominal movement amplitude to 50 % or less of the preceding stable baseline for 10 seconds or longer [7]. During nasal CPAP therapy hypopneas were defined as a reduction of nasal airflow amplitude (measured by a pneumotachograph) to 60 % or less of the preceding stable baseline for 10 seconds or longer. The mean number of apneas and hypopneas per hour of sleep was calculated as the apnea-hypopnea index (AHI). Sleep parameters were determined using the criteria of Rechtschaffen and Kales [17], and arousals were defined in accordance with the ASDA definitions [2].

APAP and CPAP devices

The APAP machine investigated was the Somnosmart® (Weinmann, Hamburg, Germany). It employs a servo-controlled turbine, and provides a CPAP of 4 – 18 mbar. The treatment pressure is controlled by an algorithm based exclusively on a simplified FOT [6]. Details of this device and its algorithm have recently been described in details elsewhere [4, 5, 7, 15, 16]. The treatment pressure is controlled by an algorithm based exclusively on a simplified FOT: A membrane pump generates a sinusoidal signal at a constant frequency of 20 Hz. This membrane pump is connected to the CPAP system at the nasal mask via a rubber tube. The pressure inside the nasal mask is measured via another rubber tube connected to a piezoresistive transducer inside the CPAP device, and the pressure signal is filtered by a 20 Hz band-pass to isolate the forced oscillation components. Changes in the amplitude of this FOT-signal are considered to represent changes in upper airway impedance $|Z|$. Increasing values of this FOT-signal are considered to represent dynamic upper airway obstruction. Thus when the actual FOT value for is higher than 160 % of the „baseline value" the CPAP pressure starts increasing by 0.2 mbar/sec. until the FOT-signal has „normalised" again. When the FOT signal remains lower than 160 % of the baseline normal value for some time, the CPAP pressure is gradually reduced. The time delay to initiation of pressure reduction, and the rate of pressure reduction depend on the „pattern" of the FOT signal which had initiated the pressure increase. If for example the pressure increase has been initiated by a single

Table 1: Patient characteristics

	All patients (*n* = 100)	**APAP** (*n* = 50)	**CPAP** (*n* = 50)	**Mean difference**	**95 % CI for differences**	***p* value**
Age (years)	54.3 ± 11.0 (18–78)	54.0 ± 11.7 (30–78)	54.7 ± 10.3 (18–74)	0.7	–3.8 to 5.2	NS
BMI (kg/m^2)	31.8 ± 5.0 (23.8–47.8)	31.9 ± 4.7 (23.8–41.7)	31.7 ± 5.3 (24.3–47.8)	–0.2	–2.2 to 1.9	NS
AHI	47.9 ± 22.6 (11.0–103.0)	48.0 ± 23.7 (11.0–103.0)	47.8 ± 21.7 (14.0–100.0)	–0.2	–9.4 to 9.1	NS
AI	33.3 ± 22.6 (1.8–96.0)	37.5 ± 24.1 (2.0–96.0)	29.0 ± 20.3 (1.8–78.0)	–8.5	–17.6 to 0.6	NS
ODI	51.4 ± 21.7 (9.0–106.0)	49.6 ± 23.4 (9.0–103.0)	53.2 ± 19.9 (12.0–106.0)	3.7	–5.2 to 12.5	NS
Arousal I	48.8 ± 18.0 (8.7–87.1)	49.6 ± 16.5 (9.6–77.3)	48.0 ± 19.5 (8.7–87.1)	–1.6	–8.9 to 5.8	NS
TST (min.)	367.7 ± 51.7 (132.0–456.0)	369.9 ± 38.3 (264.0–450.0)	365.5 ± 62.8 (132.0–456.0)	–4.5	–25.6 to 16.7	NS
Stage 1 (%TST)	15.7 ± 12.9 (0.9–73.2)	15.0 ± 11.7 (1.3–52.9)	16.4 ± 14.2 (0.9–73.2)	1.4	–3.9 to 6.7	NS
Stage 2 (%TST)	61.9 ± 14.4 (13.7–95.3)	62.6 ± 12.7 (37.1–88.1)	61.1 ± 16.0 (13.7–95.3)	–1.4	–7.3 to 4.3	NS
Stage 3 (%TST)	8.7 ± 7.6 (0.0–35.0)	8.1 ± 6.6 (0.0–21.1)	9.3 ± 8.6 (0.0–35.0)	1.2	–1.9 to 4.3	NS
Stage 4 (%TST)	0.7 ± 2.3 (0.0–17.7)	0.3 ± 1.2 (0.0–7.4)	1.1 ± 3.0 (0.0–17.7)	0.7	–0.2 to 1.7	NS
REM (%TST)	13.1 ± 7.0 (0.0–34.7)	14.0 ± 6.8 (1.4–29.3)	12.1 ± 7.2 (0.0–34.7)	–1.9	–4.7 to 1.0	NS
ESS score	12.6 ± 3.9 (8.0–23.0)	12.7 ± 4.0 (8.0–23.0)	12.4 ± 3.8 (8.0–21.0)	–0.3	–1.8 to 1.3	NS

Values are mean ± SD, range in parentheses; 95 % CI = 95 % confidence interval; BMI = body-mass-index (kg/m_2); AHI = apnea/hypopnea index (number of apneas and hypopneas per hour); AI = apnea index (number of apneas per hour); ODI = oxygen desaturation index (number of desaturations ≥ 4 %); Arousal I = arousal index (number of microarousals per hour) TST = total sleep time; %TST = percentage of total sleep time; ESS = Epworth Sleepiness Scale; CPAP = continuous positive airway pressure; APAP = automatic positive airway pressure

short rise in the FOT signal, pressure reduction starts at a rate of 0.1 mbar/sec. without any delay as soon as the FOT value has „normalised". If, for example, the pressure increase has been initiated by more than one increment of the FOT value of more than 160 % of the baseline value within 50 sec., the pressure reduction starts after a delay of 120 sec. at a rate of 0.05 mbar/sec. Such a „cluster" of obstructive events occurs, for example, when the sleeper changes position from lateral to supine. In this situation the treatment pressure should not be reduced too quickly. When the FOT value falls below 90 % of the individual normal value, a central apnoea or a major leakage are assumed, and the pressure remains unchanged until the FOT value has „normalised" again. During this study the lower pressure limit was set to 4 mbar, the upper limit to 18 mbar. The device was set to increase the CPAP-pressure by a maximum of 0.2 mbars/sec. No individual adjustments were made.

For constant-pressure CPAP therapy we used an identical device operating in constant pressure mode without FOT measurements (Somnotron 4®, Weinmann, Hamburg, Germany).

Study design

The initiation of either conventional CPAP or APAP was performed during three consecutive nights in the sleep laboratory. In the first night a diagnostic polysomnography was performed and subjective sleepiness was determined with the aid of the ESS. All patients who met the inclusion criteria, and gave their informed consent to participate, were randomized either to APAP treatment or conventional CPAP treatment. After the second treatment night in the sleep laboratory sleepiness was again determined using a modified ESS with identical items, but referring to the patients condition on the actual day.

Initiation of APAP therapy

Initiation of APAP therapy was performed on two consecutive nights in the sleep laboratory. In the night following diagnostic polysomnography, each patient in the APAP group underwent APAP therapy with the Somnosmart®. On that occasion, the device was operated at the settings preset by the manufacturer, with a maximum possible treatment pressure range of 4–18 mbar. For all subsequent treatment nights, an individual upper pressure limit was determined. For this purpose, the 95 % percentile was calculated from the range of pressures applied during the first night, and this was taken as the upper pressure limit. If the 95 % percentile was < 8 mbar, the upper pressure limit was set to 8 mbar.

Initiation of conventional CPAP therapy

For conventional CPAP-therapy we used the corresponding constant-pressure CPAP device from the same manufacturer (Somnotron4®). Following the diagnostic polysomnographies, manual titration of the CPAP pressure was carried out under polysomnographic control as previously described [7, 20]. For each patient, the minimal pressure was established at which most of apneas, hypopneas and snoring were abolished in all body postures and in all stages of sleep. Starting from an initial 4 mbar, the pressure was increased in steps of 1 mbar at intervals of at least 5 minutes when obstructive events (apneas, hypopneas or snoring) occurred. If no further obstructive events occurred during a period of 30 minutes, the pressure was then reduced again every ten minutes in steps of 1 mbar until such events re-occurred, whereupon the pressure was once more increased in the manner described above. After a second treatment night, the ESS score was determined again.

Data Collection and Statistical Analysis

The investigators responsible for the evaluation of the polysomnographic tracings (C.N. and C.C.) were unaware whether the patient had been treated with conventional constant-pressure CPAP or APAP. All figures are expressed as arithmetic means ± standard deviation. Group comparisons were performed using the t-test or the Mann-Whitney U-test as appropriate. The statistical calculations were performed with the aid of SPSS (versi-

Table 2: Results of treatment on the second treatment night

	APAP (*n* = 48)	CPAP (*n* = 47)	Mean difference	95% CI for differences	*p* value
AHI	4.7 ± 5.3 (0.0–27)	3.7 ± 3.4 (0.0–14.6)	–1.0	–2.8 to 0.9	NS
AI	2.2 ± 3.4 (0.0–21.0)	2.1 ± 2.3 (0.0–10.0)	–6.8	–1.3 to 1.1	NS
ODI	8.7 ± 9.4 (0.0–34.0)	7.3 ± 6.4 (0.0–29)	–1.4	–4.7 to 1.9	NS
Arousal I	20.7 ± 12.8 (4.6–87.1)	16.7 ± 8.8 (4.1–40.5)	–4.0	–8.4 to 0.5	NS
TST (min.)	369.5 ± 50.0 (286.0–438.0)	360.3 ± 50.0 (204.0–438.0)	–9.2	–27.2 to 8.8	NS
Stage 1 (%TST)	10.4 ± 7.1 (1.7–31.0)	11.1 ± 8.6 (0.7–36.0)	0.6	–2.6 to 3.9	NS
Stage 2 (%TST)	42.8 ± 10.4 (19.1–66.2)	44.8 ± 11.6 (23.3–66.3)	2.0	–2.5 to 6.5	NS
Stage 3 (%TST)	20.3 ± 6.5 (8.9–34.3)	18.5 ± 8.4 (1.3–42.6)	–1.8	–4.9 to 1.3	NS
Stage 4 (%TST)	3.7 ± 7.5 (0.0–34.4)	3.7 ± 4.7 (0.0–18.2)	–3.4	–2.6 to 2.5	NS
REM (%TST)	22.8 ± 7.7 (9.5–41.6)	22.0 ± 8.4 (4.1–41.6)	–0.8	–4.1 to 2.5	NS
ESS	7.0 ± 4.1 (0.0–19.0)	7.6 ± 3.5 (1.0–18.0)	–6.8	–1.3 to 1.1	NS
CPAP mean pressure (mbar)	6.0 ± 2.0 (3.8–12.8)	9.0 ± 1.8 (5.0–13.0)	3.1	2.2 to 4.0	$p < 0.0001$

Values are mean ±SD, range in parentheses; 95 % CI = 95 % confidence interval; AHI = apnea/hypopnea index (number of apneas and hypopneas per hour); AI = apnea index (number of apneas per hour); ODI = oxygen desaturation index (number of desaturations ≥ 4 %); Arousal I = arousal index (number of microarousals per hour) TST = total sleep time; %TST = percentage of total sleep time; ESS = Epworth Sleepiness Scale; CPAP = continuous positive airway pressure; APAP = automatic positive airway pressure

on 9.0.1). For differences between groups, a two-tailed *p* value of less than 0.05 was considered significant.

The study protocol was examined and approved by the ethics committee of the University of Erlangen.

Results

Table 1 shows the characteristics of the 100 patients randomized. At baseline no differences were to be seen between the two groups in terms of the biometric data, polysomnographic data, ESS and SF-36.

Five patients did not tolerate APAP (2 patients) or CPAP (3 patients) and broke off participation after the first treatment night. For the remaining 48 patients in the APAP group, and the remaining 47 patients in the CPAP group, treatment was initially carried out as planned. The mean upper pressure limit for APAP treatment was 10.1 ± 2.6 mbar (8–18 mbar). During the second treatment night in the sleep laboratory in each group nocturnal respiration was nearly normalized. The AHI in the APAP group did not differ significantly from that in the group receiving conventional CPAP therapy (4.7 ± 5.3 vs. 3.7 ± 3.4). Nor did the apnea index (AI) and the oxygen desaturation index (ODI) differ between the two groups (Table 2). In both groups equally, sleep profile was largely normal, with differences being seen neither in arousal index nor in the distribution of sleep stages (Table 2). In the APAP group, sleepiness, as measured with the ESS was appreciably reduced in comparison with baseline (7.0 ± 4.1 vs. 12.7 ± 4.0; $p < 0.001$), as also in the conventional CPAP group (7.6 ± 3.5 vs. 12.4 ± 3.8, $p < 0.001$). The ESS scores did not differ between the two groups.

The mean treatment pressure during APAP was significantly lower than during CPAP treatment (6.0 ± 2.0 vs. 9.0 ± 1.8 mbar; $p < 0.001$).

Discussion

Since initial studies in small groups of patients had indicated that, in principle effective treatment of OSAS is possible with the aid of an FOT-controlled APAP device [15, 5], the present study was designed to compare the efficacy of the FOT-controlled APAP device in a larger group of patients with that of conventional CPAP treatment.

Although the participants in the study were not informed as to which study arm they had been assigned, we are unable to exclude the possibility that, during wake periods the patients might have become aware of the automatic variations in pressure associated with the APAP device. Since, however, the participants were not informed of the underlying rationale of the study, and at no time had an opportunity to make a comparison with the respective other device, it may be assumed that the subjective assessment of the patients was largely unbiased.

In our study we used a „modified ESS" to evaluate our patients' subjective sleepiness. This method has potential limitations, since it is completely dependent on the patients subjective impression of his actual sleepiness. Thus using this „modified ESS", we cannot completely rule out minor differences in daytime performance and sleepiness between our two treatment groups.

In summary, we have been able to show that treatment with the FOT-regulated APAP device can be just as successful as with conventional CPAP treatment. As in earlier studies, it was again found that the mean treatment pressure applied during APAP treatment is appreciably lower than that used during CPAP therapy [5]. This „pressure saving" effect of auto-CPAP therapy has also been demonstrated for some other auto-CPAP devices. Theoretically, this lower mean treatment pressure might mean that patients find this form of treatment less inconvenient.

In earlier studies, we observed that, during APAP treatment with the device investigated herein, inadequate pressure increases can, very rarely, occur, e.g. when during nocturnal wake periods active movements in the pharynx with a temporary decrease in the cross-sectional diameter of the upper airways occur. This observation prompted us in the present study to define an upper pressure limit for long-term APAP treatment. To determine this upper pressure limit, we employed the 95 % percentile of the pressure values applied during the first APAP night. In a small study on 11 patients, Randerath et al. were able to show that the FOT-controlled APAP device was effective even without an individually defined upper pressure limit [15]. Presumably, the inadequate pressure increases we initially observed occur in particular during treatment initiation during which there are frequent periods of waking [11]. During the course of home treatment, as patients increasingly become accustomed to the treatment, such events will lose significance.

Although we initiated APAP treatment in our sleep laboratory, we made no use of polysomnographic data to define any individual settings of the APAP device. Accordingly, initiation of treatment with the device investigated herein would probably also be possible without concomitant polysomnography, e.g. in the patient's home. This would have the positive effect of appreciably reducing the staff requirements and costs associated with conventional sleep lab initiation. This is a further point that might be investigated in further studies comparing home with sleep laboratory therapy initiation.

To summarize the present study has confirmed that, over the short term, equally good therapeutic results and compliance can be achieved with the FOT-controlled APAP device as with conventional CPAP therapy.

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