

Chapter 4 – Part I

Mandible behaviour in severe obstructive sleep apnoea patients under CPAP treatment

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SUMMARY

The aim of this study was to investigate whether OSAS patients present different behaviours of mandible movements before and under CPAP therapy, and to compare the results against a healthy control group.

In this retrospective study, patients were selected according to criteria such as availability of polysomnography and related CPAP therapy control recordings, presence of mandible movement and mask pressure signals in the recordings, and tolerance to the applied positive pressure. Statistical analysis of four parameters, namely the apnoea-hypopnoea index (AHI), the arousal index (ArI), the average of the mandible lowering (aLOW) during sleep, and the average amplitude of the oscillations of the mandible movement signal (aAMPL), was performed on three groups: the OSAS group, the CPAP therapy group and a control group, the latter being composed of healthy subjects prospectively recorded.

The OSAS and CPAP groups each included thirty-four recordings, while twenty-four recordings from healthy subjects were contained in the control group. Significant difference ($p < 0.05$) was found in the OSAS group compared with both the CPAP group and the control group when considering the four parameters. Comparison of the parameters between the CPAP and the control groups showed that the aLOW and the aAMPL (respectively -5.8 vs -5.9 mm; $p : 0.97$ and 0.1 vs 0.2 mm; $p : 0.15$) did not differ significantly, while the AHI (4.0 vs 1.8; $p < 0.0001$) and the ArI (14.7 vs 6.0; $p < 0.00001$) differed.

In conclusion, when an efficient CPAP treatment is applied, the mandible behaves much more like in healthy subjects: the mouth is less open and presents fewer broad sharp closure movements, and repeated mandible excursions are absent or very small.

INTRODUCTION

As described in chapter 1, the mandible pattern during obstructive events in sleep includes a mandible lowering^{1,2,3} (mouth opening) and respiratory movements. The downward movements of the mandible (in the breathing frequency band [0.15-0.33] Hz) were observed in correlation with the oesophageal pressure swings during obstructive sleep events.³ This aspect is presented as partial results in the sixth chapter.

The purposes of this paper are to quantify the changes in the movements of the inferior jaw between the diagnostic PSG night and the CPAP treatment control night and to show that mandible behaviour under effective CPAP treatment is comparable to the one observed in healthy subjects. Therefore, in the present study, the analysis of the mandible movement signal was performed by decomposing the average (degree of mouth opening) and downward excursion (amplitude of respiratory effort) components of the signal during sleep periods only.

MATERIAL AND METHODS

1. Subjects and recordings

Diagnostic and CPAP treatment PSG recordings (DPSG and CPSG) were selected retrospectively among the recordings made from January 2009 to December 2010 according to these criteria: (1) DPSG and CPSG recordings were paired, i.e. each pair came from the same patient (3 to 6 months between DPSG and CPSG), (2) mandible movement and mask pressure signals had been recorded in addition to the standard PSG signals, (3) the DPSG recording highlighted a severe OSAS (AHI > 25/h), (4) the CPAP titration was performed on the basis of the Stradling et al.

equation pressure⁴, and (5) the CPAP was applied through a nasal mask for at least four hours of sleep in CPSG. Late 2010, healthy subjects were prospectively recorded in order to constitute the control group.

The PSG recordings were made using a S7000 polysomnograph (EMBLA Medcare, Denver, US) and included the following neurophysiology signals: three-channel electroencephalography (EEG, C3-A2, C4-A1, FZ-CZ), left and right electrooculography (EOG), submental electromyography (chin EMG) for sleep staging and arousal scoring, two (left and right) tibial EMG for periodic leg movement evaluation. Also recorded were cardiorespiratory signals comprising ECG, nasal cannula/pressure transducer (NAF) (Protech, Mukilteo, US), chest and abdominal inductance plethysmography belts, a plethypulse, a blood oxygen oximeter (SpO₂, Nonin, Plymouth, US), a snoring sound detection (piezoelectric sensor from EMBLA), and a body position marker (body position sensor Protech).

2. Mandible movement recording

The movements of the mandible were acquired by a distance-meter based on the principle of magnetometry (Jawsens[®], NOMICS, Liège, Belgium) previously described in the chapter 1. The sensor was connected to the distance-meter electronic module, which converted the distance between the two sensor probes into a voltage with a precision of 0.1 mm. The signal was digitalized with a sampling frequency of 10 Hz, transmitted by cable to the polysomnograph, and could be processed off-line.

3. Manual scoring

All the recordings were analysed between a START time (lights out) and a STOP time (lights on). Sleep stages and breathing events were scored according to AASM rules.⁵ Arousals were scored as recommended by the ASDA.⁶ Apnoea and hypopnoea index (AHI) and arousal index (ArI) were then computed over the total sleep time (TST).

4. Mandible movement processing

First, for each recording, the zero value of the mandible movement signal was attributed to the fully closed mouth level. The polysomnograph had been set so that, as the distance between the two sensor probes increases, the level of the recorded signal actually decreases (simple signal reversal). Therefore, the more open the mouth gets (the more the mandible lowers), the more negative the value of the signal gets (the more the mandible movement signal lowers). Second, the average value of the mandible movement signal over all the sleep periods (aLOW) was computed. Third, the average over all the sleep periods of the instantaneous peak-to-peak amplitude of the component of the signal downward excursion in the breathing frequency band [0.15-0.33] Hz (aAMPL, see Senny et al. for more details about the signal processing⁷) was computed. Thus, while aLOW reflects the mean level of mouth opening during sleep, aAMPL reflects the mean amplitude of the mandible movements. Figure 1 illustrates the computation of these two parameters.

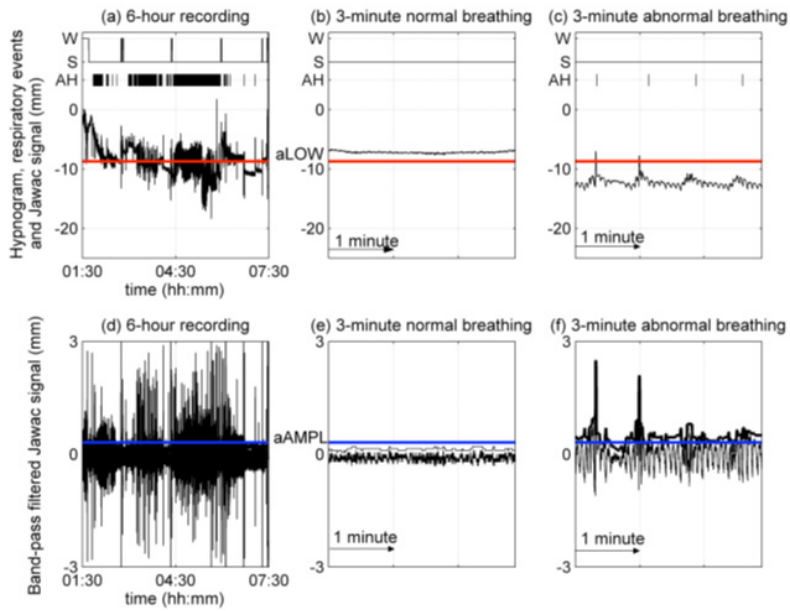


Figure 1 : Illustration of the computation of aLOW and aAMPL: (a) the mandible movement signal over 6-hour recording, the hypnogram (W-wake, S-sleep) and the respiratory events (AH), the red line is the aLOW, the average of the Jawac signal on the sleep periods; (b) a zoom on 180 seconds of the mandible movement signal in (a) showing the time behaviour of normal breathing; (c) 180 seconds of the mandible movement signal in (a) showing the time behaviour when obstructive apnoeas occur; (d) (e) (f) are the band pass filtering of the Jawac signal and the resulting aAMPL parameter, the average of the amplitude of movements on the sleep periods. The blue line is the average of peak to peak amplitude for this patient.

5. Statistical analysis

Four parameters (AHI, ArI, aLOW and aAMPL) were computed for each recording. The mean and standard deviation of these parameters were computed within each of the three groups (OSAS, CPAP, and healthy). Statistical difference between groups was assessed under the R software⁸ by reducing the input space dimensionality through principal component analysis followed by the non-parametric Wilcoxon rank-sum test.

RESULTS

The DPGS and CPDG recordings from thirty-four severe OSAS patients were collected and formed the groups 1 (before CPAP) and 2 (with CPAP). Twenty-four recordings from healthy subjects constituted the control group 3. Table 1 gives anthropometric information of the patients.

Table 1 – Anthropometric data in the 3 groups of recordings					
	n	# men	# women	Age	BMI (kg/m ²)
Severe OSAS (Gr 1) and CPAP (Gr 2)	34	27	7	52 ± 9	33.0 ± 8.0
Healthy (Gr 3)	24	11	13	24 ± 4	21.8 ± 3.3

Table 2 gives the mean and standard deviation of the four computed parameters (AHI, ArI, aLOW, and aAMPL). It shows that the CPAP is effective in reducing the AHI and the ArI (mean AHI below 5 and mean ArI below 15 in group 2). Statistical difference between group 2 (CPAP) and group 3 (control) was found for mean AHI (4.0 vs 1.8, $p < 0.0001$) and mean ArI (14.7 vs 6.0, $p < 0.00001$), but the parameters computed from the mandible movement signal had similar values: the mean aLOW ($p = 0.97$) and the mean aAMPL ($p = 0.15$) were respectively -5.8mm and 0.1mm in group 2 (CPAP) while they were -5.9mm and 0.2mm in group 3 (control). The average lowering, aLOW, decreased from -9.1 mm in OSAS to -5.8mm under CPAP ($p < 0.05$). The average amplitude of the oscillations of the mandible, aAMPL, also drops from 0.45 mm in OSAS to 0.1 mm under CPAP ($p < 0.05$). Considering group 1 and 2, the principal component analysis reduced the dimensionality to one value that explained 96 % if the 4 parameters were considered and 98 % if only 2 parameters aLOW and aAMPL. The Wilcoxon was then applied and showed a strong difference between the OSAS and CPAP groups ($p < 0.0001$).

Table 2 : Mean and standard deviation of the parameters (AHI, ArI, aLOW, and aAMPL) in the 3 groups				
	AHI (n/h)	ArI (n/h)	aLOW (mm)	aAMPL (mm)
Severe OSAS (Gr 1)	49.2 ± 17.3 *°	51.9 ± 15.6 *°	-9.1 ± 3.5 *°	0.45 ± 0.3 *°
CPAP (Gr 2)	4.0 ± 3.1 **	14.7 ± 8.3 **	-5.8 ± 3.5	0.1 ± 0.05
Healthy (Gr 3- control)	1.8 ± 1.8	6.0 ± 2.0	-5.9 ± 2.0	0.2 ± 0.1

* statistically significant difference between Gr 1 and Gr 2 ($p < 0.05$);

° statistically significant difference between Gr 1 and Gr 3 ($p < 0.05$)

** statistically significant difference between Gr 2 and Gr 3 ($p < 0.05$)

Figure 2 gives the box plots of the 4 parameters for each group.

The AHI under CPAP was greater than 10/h in three cases while aLOW was in the range [-5;-2] and aAMPL in [0.10-0.12]. In the comparison of these 3 patients with the others, height (173.3 vs 173.5 cm; p : 0.97), weight (93.8 vs 99.2 kg; p : 0.69) and BMI (31.3 vs 33.5 kg/m²; p : 0.66) were not significantly different. However, despite the reduction in the mandible movements, respiratory events were still visible in the signal. The aAMPL parameter was 0.74 mm in one CPAP case but it was not related to any malfunctioning CPAP or leakage, the ideal pressure seemed to be underestimated. The aLOW parameter was increased in four CPAP cases in the range of [-14; -9] mm though the AHI, ArI and aAMPL fell below 7/h, 10/h and 0.15mm respectively. In this group, the height was significantly higher than others (182.3 vs 172.4 cm; $p < 0.05$) but weight and BMI were not significantly different: 105.5 vs 97.8 kg; p : 0.53 and 31.7 vs 33.4 kg/m²; p : 0.68 respectively. A sensor shifting (rotation) inducing an 8 mm offset was suspected in only one case.

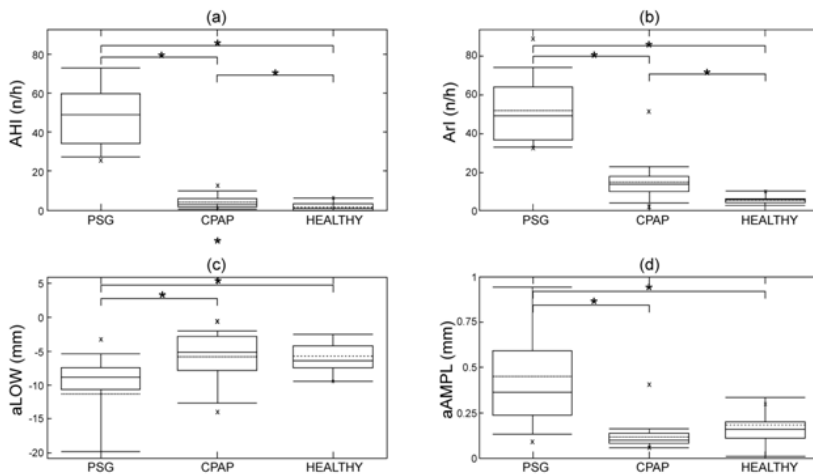


Figure 2 : Box plots of the four parameters AHI (a), ArI (b), aLOW (c), and aAMPL (d). The crosses indicate the extrema, the box height covers the interquartile range (between 25th and 75th percentile), the solid line in each box corresponds to the median and the dashed line is the mean. A * star appears between two groups when there is a significant difference with a p-value lower than 0.05.

Figure 3 presents three examples with mandible movement signal, scored apnoeas/hypopnoeas (AH) and related hypnogram (W for wake, S for sleep), one example per group. In these examples the mandible movement behaviour is characterized by a permanent activity and a wide open mouth in severe OSAS, but this high activity vanishes and the mouth becomes more closed when CPAP pressure is applied. In the healthy subject, the mouth is stable and slightly open, without any regular mandible movement of high amplitude component during the night.

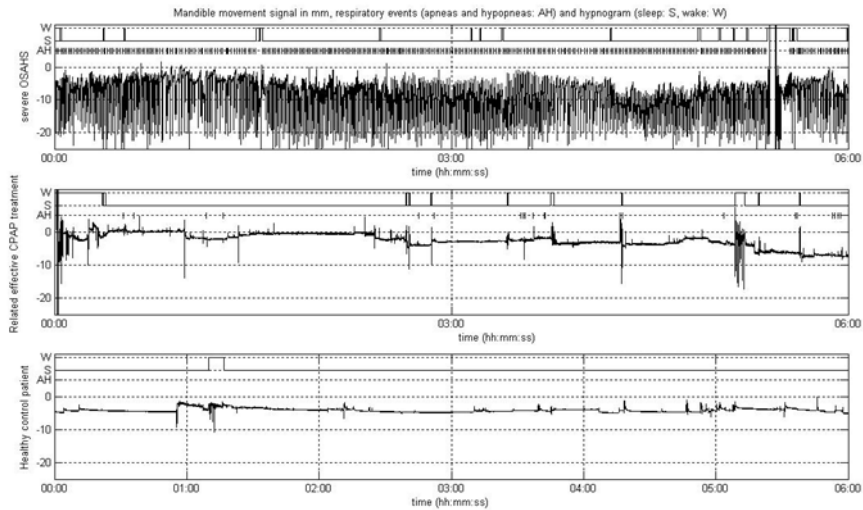


Figure 3 : Three examples, one for each group, of the mandible movement signal (in mm), the scored apnoeas and hypopnoeas (AH), and the sleep/wake state: (upper graph) recording extract characterized by permanent regular mandible movements of high amplitude and a wide open mouth - corresponding to a patient with severe OSAS - and, (middle graph) once an efficient CPAP pressure is applied (same patient), the mandible movement signal behaves much more like the signal from a healthy case (bottom graph).

DISCUSSION

Several studies showed that the mouth is more open in OSAS patients than in healthy subjects, especially during a respiratory event and at the end of inspiration.¹⁻

³ Our study showed that in severe OSAS (AHI > 25/h, n = 34), the opening of the mouth is of more than 9 mm in average (aLOW) and the mandible experiences regular mandible movements of high amplitude (0.45 mm average amplitude, aAMPL). This mandible behaviour was very different from the behaviour observed in the related CPAP recordings (same 34 patients, aLOW = -5.8mm and aAMPL = 0.1mm; $p < 0.05$ for both) and also from the behaviour observed in healthy subjects (n = 24 healthy subjects, aLOW = -5.9 mm and aAMPL = 0.2mm; $p < 0.05$ for both). When an efficient CPAP pressure is applied, the mandible behaves much more like in healthy subjects: no statistical difference is found in the parameters computed from the mandible movement signal between CPAP and control groups. Statistical difference between group 2 (CPAP) and group 3 (control) was found for mean ArI (14.7 vs 6.0, $p < 0.00001$). Arousals have few impacts on mandible movement under nasal CPAP treatment, possibly due to the necessity to keep the mouth closed under this condition. The parameters computed from the mandible movement signal had similar values : the mean aLOW and the mean aAMPL were respectively -5.8 mm and 0.1 mm in group 2 (under CPAP treatment) while they were -5.9 mm ($p = 0.97$) and 0.2 mm ($p = 0.15$) in group 3 (control). Despite a higher ArI than group 3, the aAMPL is low in group 2. The increased number of arousals (statistically significant) in the group 2 could be explained by the CPAP treatment, its secondary effects, periodic limb movements, the attended character of the polysomnography or residual RERA (in patients located in the overlap values of aLOW and aAMPL before and under CPAP treatment). Despite a long period between the 2 PSG (3 to 6 months between DPSG and CPSG), the average adherence to CPAP during this time is not known and we could not define how new the CPAP treatment in this group is. To assess the impact of CPAP treatment

on sleep architecture in OSAS, McArdle et al. performed a randomised (versus placebo capsules) prospective study.⁹ A full PSG monitoring was realised at home after one month of treatment. CPAP resulted in better sleep quality with fewer arousals.⁹ Although arousal frequency is often considered to have an important role in the pathogenesis of daytime sleepiness, other sleep characteristics could play a role. Among patients with OSAS, the arousal index has an inverse correlation with the amount of stage 3 and stage REM sleep (expressed in percentage of TST).¹⁰ These parameters, not related in our study, could partly explain the ARI differences between group 2 and group 3. Nevertheless, these (non-respiratory) arousals have few impacts on aAMPL and aLOW due to the nasal CPAP treatment: in this condition, the patient must close the mouth.

Four CPAP cases did not close their mouth under treatment but opened it instead. We suspected a sensor shifting of several millimetres in only one case. In the other three cases, air leakage by the mouth should be speculated but the AHI was below 7/h indicating an efficient treatment. The CPAP pressure was therefore not changed. Another patient did have an important oscillatory component under CPAP (average amplitude around 0.4 mm). This latter case could be explained by persistent snoring or by an underestimation of the ideal pressure though respiratory events were drastically reduced (AHI under CPAP = 1/h). In three patients for whom the AHI was greater than 10/h under CPAP, the two average mandible parameters were small (aLOW in [-5;-2] and aAMPL in [0.10-0.15]). In this group, the height was significantly higher than the others. For these patients, the mean aLOW is probably not sensitive enough. In automated method, the advantage of using relative value will be discussed further. Nevertheless, a fine visual observation of mandible movement signal allowed pointing out most abnormal respiratory events.

We had no knowledge about nose or mouth breathers (NBs and MBs) included in this study. Bachour et al. showed that the time spent breathing by the mouth in MBs under nasal CPAP decreased from 84.9% down to 19.9% in average after 3 months of CPAP treatment.¹¹ As a nasal CPAP mask was used in this study and the inferior jaw was thus free to move, changes observed in the mandible movement signal between DPSG and CPSG recordings were due to passive or natural physiological effects.

This study suffers from its retrospective and automated nature. Very restrictive inclusion criteria have been considered to select patients. We did not have full control on how the data have been acquired to ensure that the mandible sensor did not shift for instance which is critical for the computation of the average lowering parameter.

Questions are still open. Firstly, the pathologic threshold of mouth opening and oscillating pattern during sleep in OSAS patients which implies the question of the pathological condition of snoring and associated pulmonary diseases (with increased breathing effort). Secondly, the use of the relative or absolute values is a difficult choice. Relative value has advantage in comparison with the absolute value used here. Relative values will automatically include the anthropomorphic characteristics of each patient (e.g. individual differences in mandibular opening distances due to the combined effect of mandibular length and mandibular rotation). A disadvantage of the relative value is the necessity of having an exact calibration. In case of sensor displacement, the relative value will have less robustness. The absolute value of aAMPL is less influenced by the baseline modifications occurring during the night. In the future, by the improvement of the software, taking into account abnormal breathing patterns in the detected period of sleep (in between the START and STOP points) will improve the accuracy of the automated mean value provided. Another possibility is the use of both results under a visual control of the trends during the night (a shift of the sensor is easily detected by trained professionals).

Finally, the study of sleep architecture and proportion of sleep stages could have been helpful.

Our results suggest that, in severe OSAS patients, when the CPAP treatment is not efficient or when an air leakage occurs, the inferior jaw will move (repeated downward excursions) and the mouth will open to greater than 6mm average opening (aLOW).

The most common treatment for severe OSAS is the CPAP device.¹²⁻¹⁴ Several methods to set up the ideal pressure exist: manual titration, predictive equations (Hoffstein or Stradling for instance), and auto-titration.¹⁵ Therefore, an easy-to-use titration method and/or method for controlling CPAP effectiveness would be welcome. For example, an at-home CPAP efficiency control method consisting in a CPAP device, of which the pressure was initially defined by a predictive equation, coupled with a portable monitoring device to check whether both the CPAP device and the applied pressure are adapted, could be cost-effective compared with the full night in a hospital setting for manual titration and CPAP control. This should be extended to multiple nights to check the stability of the computed parameters. Moreover, cost reduction could also be achieved if a quick visual inspection of a signal allowed a reliable assessment of the CPAP efficiency.

The second part of this chapter will focus on the titration of CPAP treatment. In the future, other studies should focus on efficacy of mandibular advancement devices by using the monitoring of mandible movements.

CONCLUSION

When an efficient CPAP pressure is applied in severe obese OSAS patients, the mandible behaves much more like in healthy subjects, i.e. a more closed mouth (< 6mm), fewer broad sharp mouth closures, and absence of, or at least very small, repeated mandible downward excursions. Mandible movement behaviour could be a simple and reliable indicator of CPAP effectiveness.

Chapter 4 – Part 2

Evaluation of a new automated pilot for CPAP treatment based on mandible behaviour in the obstructive sleep apnoea syndrome

Expanded from Oral presentation at the World Congress on Sleep Apnea 2009, Seoul, Korea and Poster session at the Biowin Day 2010, Louvain-la-Neuve, Belgium.

SUMMARY

Mandible behaviour under CPAP treatment is much more like in healthy subjects. Based on this important information, the aims of this study were first, to assess the feasibility to pilot a CPAP by the mandible movement provided by the distance-meter Jawsens[®]; second, to compare the automated titration pressure obtained by this method and classical auto-piloted CPAP. By identifying the aLOW and the aAMPL in severe OSAS patients, a nasal CPAP titration could be guided (called Jaw-Auto CPAP) until the correction of the respiratory events.

Due to technical aspects, only twelve patients for which both recordings were analysable (3 women, 9 men; age: 55.3 ± 8.3 ; AHI: $60.6 \pm 34.3/h$; Stradling's pressure: 8.8 ± 1.9 hPa) were studied. There was no significant difference in pressure 95% percentile achieved (10.8 ± 2.07 for the Flow-Auto CPAP and 10.9 ± 2.86 for the Jaw-Auto CPAP; p : 0.908), in abnormal residual breathing (AHI $8.9 \pm 6.9/h$ for the Flow-Auto CPAP and 7.3 ± 4.5 for the Jaw-Auto CPAP; p : 0.309), or in sleep qualities that could be observed between the two different auto-piloted nasal CPAP devices.

INTRODUCTION

Assuming that mouth opening and repeated mandible downward excursions during sleep are part of a clinical expression of impaired nervous system control of the upper airways, a servo-controller could be connected up to an auto-piloted CPAP device in order to increase administered pressure until some correction in mandible position and behaviour are reached. The mandible had to be pulled up and stable.

The aims of the study presented here were, first, to assess the feasibility to pilot a CPAP by the mandible movement provided by the Jawsens[®]; second, to compare the automated titration pressure (Jaw-Auto CPAP) obtained by this method with the classical auto-piloted CPAP device (Flow-Auto CPAP).

MATERIAL AND METHODS

Polysomnographic and mandible movement recordings have been depicted in sections 1 and 2 of this chapter.

Severe OSAS patients ($AHI \geq 30/h$) were selected and underwent 2 successive full-night polysomnographies. The same machine (BREAS[®] ISleep 20i) was used in two situations. One night the auto-piloted is working with its original feed-back circuit and algorithm (Flow-Auto CPAP), where the input is the shape of inspiratory airflow curve. The other night, this feedback system is disconnected and replaced by an alternative one based on the behaviour of the mandible. The succession of treatments was randomised.

For the Jaw-Auto CPAP, a servo-controller fed by a distance-meter drove an nCPAP device (BREAS[®] ISleep 20i). The algorithm included

two key thresholds: sharp mouth closure related to arousal and the detection of superimposed regular and repeated mandible movements within the breathing frequency bandwidth [0.16-0.26] Hz. The jaw auto-CPAP (Jaw-Auto CPAP; figure 1) was adjusted following respiratory mandible downward movements and related arousal (sharp mouth closure), leading to increase pressure by 0.5 hPa (system inertia of 5 minutes). Lower and higher levels of pressure were set at ± 5 hPa around a level defined by Stradling's equation.⁴

Stradling's equation :

$$\text{cmH}_2\text{O or hPa} = 0.048 \text{ SpO}_2 \text{ dips/h} + 0.128 \text{ neck circumference} + 2.1$$

This equation was proposed in a study using a one-night sleep study recording body movement, heart rate as markers of sleep disturbance, with arterial oxygen saturation measurements (SpO₂) and snoring as markers of respiratory impairment, and measurements of pulse transit time to differentiate obstructive from central apnoeas. After this diagnostic procedure, patients were instructed to use an auto-titrating CPAP machine (Autoset-T, Resmed®) every night for a minimum of 28 nights. The 95th centiles for pressure were considered for each night and the average was assumed to provide the best estimation for the fixed pressure. A multiple linear regression technique identified only neck circumference and OSAS severity ($> 4\%$ SpO₂ dips/h) as independent predictors of nasal CPAP titration pressures. By this study, the authors conclude that the proposed equation is as precise as a one-night titration. Therefore, in our study, this equation was considered to define the range of pressure to use to start this automated CPAP-titration for each patient. Other parameters studied were AHI, ODI (oxygen desaturation index : $> 4\%$ SpO₂/h of sleep), ARI : arousal index, snoring, total sleep time, sleep efficiency (TST / time in bed), sleep efficacy (Slow-wave-sleep + REM / TST).

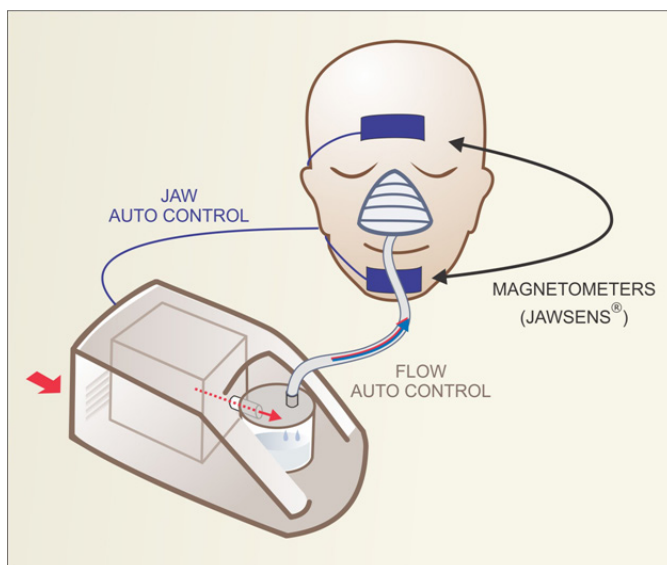


Figure 1 : The Jaw-Auto CPAP (in blue)
and flow-AUTO control (Flow AUTO-CPAP)

RESULTS

Thirty two patients entered the study. Twenty had to be discarded from analysis because either the Flow-Auto (8 cases), the Jaw-Auto (4 cases) or both (8 cases) reached the upper or lower limits during more than 30 % of the night. Table 1 gives the results in 12 patients for which both recordings were analysable (3 women, 9 men; age: 55.3 ± 8.3 ; AHI: $60.6 \pm 34.3/h$; Stradling pressure: 8.8 ± 1.9 hPa). Quality and duration of sleep were similar in the 2 methods of pressure titration.

	Pressure 95% percentile (hPa)	AHI (n/1h)	ODI n/1h	ARI n/1 h	Snoring min.	TST min.	Efficiency Index	SWS + REM TST
Flow-Auto CPAP	10.8 ± 2.07	8.9 ± 6.9	1.6 ± 2.64	10.7 ± 7.59	29.4 ± 39	427 ± 79	0.81 ± 0.06	0.52 ± 0.14
Jaw - auto CPAP	10.9 ± 2.86	7.3 ± 4.5	1.8 ± 2.76	8.3 ± 5.21	50 ± 81	433 ± 66	0.80 ± 0.09	0.54 ± 0.14
<i>p</i>	0.908	0.309	0.674	0.213	0.376	0.874	0.732	0.432

AHI : apnoea hypopnoea index, ODI: oxygen desaturation index, ARI: arousal index, TST: total sleep time, SWS: slow wave sleep, REM: REM-sleep.

DISCUSSION

In these twelve cases, we observed no significant difference in pressure (95 % percentile achieved) in abnormal residual breathing or in sleep qualities between the two different auto-piloted nasal CPAP (see table 1).

Interestingly, in this study, ArI were not different between the 2 methods and lower than the results described in the first part of this chapter for group 2.

This study suffers from many limitations. Firstly, the procedure used to compare the two auto-CPAP systems led to a failure in 62.5% of patients, which is not acceptable for publication. The reason was that the two security limits, fixed at 5 hPa, above and below Stradling theoretical pressure restricted the amplitude pressure variations, either in one system or both. The pressures reached the maxima and minima too rapidly and too often. The failures were evenly attributable to the two systems. This is also a limitation of our method: the choice of this prediction equation which has to define the correct range of pressure for a one-night CPAP titration. Moreover, the pre-installed auto-CPAP automated software was not adjustable. For the Jaw-Auto CPAP, the data to adapt the pressure

was modified. Despite this choice, in the 12 patients, the CPAP effect in terms of control of abnormal breathing (AHI and ODI) is quite good. Secondly, the auto-CPAP used in our study was not the same as the one used in Stradling's equation definition procedure (Autoset-T, Resmed®). Thirdly, we did not assess body position. Body position could, in some cases with obstructive apnoea occurring predominantly in the supine position, explain a night-to-night variability in OSAS severity (due to reduced or increased proportion of time lying supine one of the nights). Hence the variability in the required pressure. Fourthly, the quality of the signal of the Jawsens® and the stability of the sensors, placed on the face above and below the nasal mask should have been studied. Fifthly, the reason for reaching maximal pressure defined by the equation used could be the CPAP removal inducing a significant leak. The use of the CPAP mask and the potential leaks during this PSG need to be monitored. Two other limitations are : the lack of special attention to distinguish nasal and mouth breathers and to check if differences exist between them and, lastly, the lack of distinction of central and obstructive residual apnoeas under CPAP treatment.

CONCLUSION

This preliminary study shows that an auto-CPAP (nasal mask) device can be fed with data from a complex behaviour of the mandible with good results. Beyond methodological problems, our results suggest that a greater role could be given to the nervous system control of breathing during sleep. The performance of the Jaw-Auto CPAP integrates a very complex behaviour coupling breathing with mouth closure. Moreover, it could provide information on the reasons for some CPAP treatment failures. Future studies are needed to improve this approach.

REFERENCES

1. Miyamoto K., Özbek M.M., Lowe A.A., Sjöholm T.T., Love L.L., Fleetham JA, Ryan CF. Mandibular posture during sleep in patients with obstructive sleep apnoea. *Arch. Oral Biol.* 1999; 44 : 657–664.
2. Hollowel D.E., Surrat P.M. Mandible position and activation of submental and masseter muscles during sleep. *J. Appl. Physiol.* 1991; 71(6) : 2267–2273.
3. Poirrier R., Chakar B., Lacroix A., Dive D., Hansen I., Frank G. Relationship between mouth opening and intrathoracic pressure during sleep apnea. In : *Somnologie, supplement of the 5th World Congress on Sleep Apnea 1997*; 2 : 37.
4. Stradling J.R., Hardinge M., Paxton J., Smith D.M. Relative accuracy of algorithm-based prescription of nasal CPAP in OSAS. *Respir. Med.* 2004; 98 : 152–154.
5. The Report of an American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. *Sleep* 1999; 22 : 667-89.
6. ASDA (American Sleep Disorders Association and Sleep Research Society) Atlas task Force. EEG arousals: Scoring rules and examples. *Sleep* 1992; 15 : 173–84.
7. Senny F., Destiné J., Verly J., Ansay P., Poirrier R. Automatic scoring of sleep apnea and hypopnoea by the analysis of mandibular movements. In *Proceedings of the 3rd IASTED international conference on Biomedical Engineering 2005*; 14-18 : 373–378.

8. R Development Core Team (2011) R : a language and environment for statistical computing, reference index version 2.13.0 R Foundation for Statistical Computing. Vienna. SBN 3-900051-07-0. Available at: <http://www.R-project.org>. [Accessed: 09 December 2011].
9. MacArdle N., Douglas N.J. Effect of continuous positive airway pressure on sleep architecture in the sleep apnea-hypopnoea syndrome. *Am. J. Respir. Crit. Care Med.* 2001; 164 : 1459-1463.
10. Collard P., Dury M., Delguste P., Aubert G., Rodenstein D.O. Movement arousals and sleep-related disordered breathing in adults. *Am. J. Respir. Crit. Care Med.* 1996; 154 : 454-459.
11. Bachour A, Maasilta P. Mouth breathing compromises adherence to nasal continuous positive airway pressure therapy. *Chest* 2004; 126 : 1248-1254.
12. Loube DI, Gay PC, Strohl KP, Pack AI, White DP, Collop NA. Indications for positive airway pressure treatment for adult obstructive sleep apnea patients: a consensus statement. *Chest* 1999; 115: 863–866.
13. Statement ATS. Indications and standards of use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes. *Am J Respir Crit Care Med* 1994; 150: 1738–1745.
14. Ballester E, Badia JR, Hernandez L, Carrasco E, de Pablo J, Fornas C, Rodríguez Roisin R, Montserrat JM. Evidence of the effectiveness of continuous positive airway pressure in the treatment of sleep apnea/hypopnea syndrome. *Am J Respir Crit Care Med* 1999; 159: 495–501.
15. Masa JF., Jiménez A, Duran J., Capote F, Monasterio C, Mayos M, Teran J, Hernandez L, Barbe F, Maimo A, Rubio M, Montserrat JM. Alternative methods of titrating continuous positive airway pressure. A large multicenter study. *Am J Respir Crit Care Med* 2004, 170: 1218-1224.