Determinants of respiratory disturbance and oxygen saturation drop indices in obstructive sleep apnoea syndrome

M.A. Quera-Salva, C. Guilleminault, M. Partinen, A. Jamieson


ABSTRACT: Although chronic sleep fragmentation and oxygen saturation (SaO₂) drops alone do not induce obstructive sleep apnoea (OSA), both are part of the feedback loop leading to obstructive sleep apnoea syndrome (OSAS). To determine factors in respiratory disturbance and SaO₂ drops, we used polysomnographic and cephalometric data from 120 OSAS patients to construct a model which we then applied prospectively to 25 new OSAS patients, calculating the correlation between observed and predicted values. We found body mass index and the amount of stage 1 non-rapid eye movement sleep to be significant variables when considering both the respiratory disturbance index (RDI) and SaO₂ drops. Posterior airway space was also a significant variable for RDI. Forced expiratory volume in one second, expressed as the percentage of forced vital capacity (FEV₁/FVC), was significant when considering SaO₂ drops. Upper airway abnormalities were also significant variables in the models and must be considered when treating OSAS patients.

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Unfortunately little is known about the progressive development of obstructive sleep apnoea syndrome (OSAS). In a study of 120 consecutive OSAS patients, interviews with family members and patients revealed that 68% of the patients were regular snorers by twenty years of age, but less than 5% were reported to be overweight at that age [1]. In many patients there appears to be a more or less rapid progression from snoring to OSAS. When partial or complete upper airway obstruction occurs during sleep, the resulting disturbances will undoubtedly induce sleep states and stage switches, and when severe, OSAS will be associated with a significant sleep disruption. The sleep disruptions induced by the abnormal breathing pattern can be expected to feed back and to play a role in the progressive worsening of the syndrome.

Experimental repetitive sleep disturbance (sleep fragmentation) decreases hypoxic and hypercapnic responses during sleep in dogs [2]. Sleep deprivation prolongs the duration of obstructive sleep apnoea (OSA) in mild to moderately severe OSAS and leads to more pronounced oxygen saturation (SaO₂) drops [3]. It also causes small but significant drops in forced expiratory volume in one second (FEV₁), forced vital capacity (FVC), minute ventilation and maximal inspiratory pressure in severe chronic obstructive lung disease (COLD) patients [4, 5]. However, 'sleep disturbance' is rarely, if ever, considered in OSAS when the components responsible for partial or complete upper airway obstruction during sleep, or the importance of SaO₂ drops associated with abnormal breathing, are analysed. Should 'sleep disturbance' continue to be systematically eliminated from all models dealing with determinants of OSAS, or should we now integrate the repetitive disturbance of states of alertness? As a first step, we have tried to assess the relative importance to OSAS patients of different factors, including sleep disturbance, that may affect a) the number of apnoeas and hypopnoeas experienced (apnea = absence of air exchange at nose and mouth for a minimum of ten seconds; hypopnoea = 50% reduction in maximal thermistor output compared with baseline and associated with a decrease in SaO₂ to <92% from a baseline of at least 94%, or a drop in SaO₂ of at least 3% if baseline was <90%), and b) the number of SaO₂ drops. The variables, other than sleep disturbance, selected for the study came from a review of the literature: age, body mass index (BMI) [6–8], presence or absence of lung disease [6, 9] and size of the upper airway, particularly in the hypo- and nasopharynx [10–12]. Being also based upon clinical experience, the rationale for the selection of these factors appears sound: gas exchange may be impaired by lung diseases [9, 13] and, in a supine subject, by morbid obesity [7, 9, 14]; retrognathia is known to be
associated with OSA and Sao2 drops even in slender subjects with intact lungs [10]. A statistical model was derived from our study using a) stepwise multiple regression analysis and b) multiple regression analysis of data obtained on 120 OSA patients. This statistical model was then prospectively applied to a smaller group of 25 OSAS patients.

Methods

Definition of variables and indices

Recent pulmonary function tests and blood gases obtained on patients awake and seated were collected. Three variables are usually selected to define chronic obstructive lung disease (COLD): ratio of residual volume to total lung capacity, maximal mid-expiratory flow, and forced expiratory volume in one second expressed as the percentage of forced vital capacity (FEV1/FVC) [15, 16]. Considering the obesity of the patients, the literature [15, 16], and the definition of COLD, we decided to select FEV1/FVC for the purpose of the study and as the variable to enter the statistical model, since it is the only variable of the three not affected by marked obesity [15, 16]. Similarly, awake/seated arterial O2 and CO2 tension expressed in torr were also selected. Since a prior study involving 155 OSAS patients had shown that posterior airway space (PAS) (the space behind the base of the tongue) and the distance from mandibular plane to hyoid bone (MP-H) were the only two variables obtained from cephalometric roentgenograms [1, 17] that explained meaningful variations in the respiratory disturbance index (RDI), they were selected as indicative of marked airway anatomical abnormality. To measure the number of abnormal breathing events per hour of sleep, RDI was defined as \[ \text{apnoea + hypopnoea} \times 60 / \text{total sleep time (TST)} \] in minutes. Finally, BMI was calculated, using the method of KHOLSA and LOWE [8] (weight/height\(^2\), expressed in kg/m\(^2\)).

Oxygen saturation indices during sleep

Several Sao2 indices were calculated: the percentage of TST spent with an Sao2 <90%, the mean nocturnal sleep Sao2 (mean Sao2), and an arbitrarily defined Sao2 index called oxygen-80-index (O2-80-I). The mean nocturnal sleep Sao2 was calculated using the formula of BRADLEY and co-workers [6]. The highest and lowest Sao2 of each polygraphically recorded 'epoch' were measured. Because the pattern of desaturation and resaturation in OSA approximates a sine wave, the mean Sao2 of each polygraphic epoch, i.e., 30 s, was estimated by averaging the high and low values. Mean nocturnal Sao2 for TST was then calculated, using the mean values of all epochs. To further focus on events leading to significant Sao2 drops, even if short-lived, we calculated the number of Sao2 drops related to apnoea and hypopnoea ≤80% and, as with the RDI, calculated the number of Sao2 drops per hour of sleep (O2-80-I).

Sleep disturbance index

Several measures of sleep disturbance have been advocated. Using the criteria of RECHTSCAFFEN and KALES [18], sleep is scored in 30 s epochs. Wake is scored when at least half of an epoch can be scored as such. Cumulative time spent awake, as defined, is called 'wake after sleep onset' (WASO) and has been used as an index of sleep disturbance. However, this index may ignore sleep disturbances of shorter duration. Short EEG alpha arousals lasting 1–14 s have also been considered. To decide which criteria should be selected to define 'sleep disturbance' we first analysed forty randomly selected sleep apnoeas or hypopnoeas in forty different nocturnal polygraphic recordings. In 8% of the cases, two independent observers disagreed on the scoring of an EEG alpha arousal at the end of an apnoea or a hypopnoea, as they considered some of the termination of the partial or complete obstructions to be unassociated with an EEG alpha arousal. Conversely, there was <1% discrepancy in the independent scoring when stage 1 non-rapid eye movement (NREM) sleep (S1) was scored on a 10 s epoch started at the end of the apnoea or hypopnoea. As there was also a high correlation (Pearson correlation coefficient = 0.80) between WASO and percentage of S1, it was decided to consider the percentage of S1 sleep, as classically defined [18], as the index of sleep disturbance in our study.

Tests

Nocturnal polygraphy was systematically performed. We monitored respiration by inductive respiratory plethysmography and airflow using thermistors. Sao2 was monitored with an ear oximeter (Biox\(^{TM}\)). The following variables were monitored: EEG (C3/A2), electro-oculogram, chin electromyogram, and electrocardiogram (ECG) (modified V1 lead). Sleep and sleep states and stages were scored following the criteria outlined by RILEY et al. [17]. Spirometry and arterial blood gases were also obtained (awake, standing or seated) within three weeks of the time of polygraphic recording.

Patient population and methodology

Criteria for entering the study were presence of clinical symptoms of OSAS, with heavy snoring at night and an RDI ≥ 10 during nocturnal polygraphic recording.

A. Initial patient population (Population A). Over a 6-month period, 120 patients (109 men and 11 women) seen consecutively met the above criteria. Their mean age was 49.0 ± 11.2 yrs; mean BMI was 31.1 ± 5.9 kg·m\(^{-2}\); awake mean Pao2 was 78 ± 6.9 torr and mean Paco2 was 38 ± 1.8 torr (see table 1).
6.6 \[(\log \text{FEY})

Some presented with abnormal FEY/FVC, but none was hypercapnic (table 1). Statistical analysis

Population A. We have, in fact, considered only eight independent variables in our modelling: BMI, age, RDI, \(\%\) FEY/FVC, \(\%\) O\(_2\)-80-I, \(\%\) TST<90\%, \(\text{PAS}\) \%, \(\text{Pao}_2\) mm, \(\text{Paco}_2\) mm. These independent variables accounted for 53% of the variance (\(R^2=0.529\)). The model accounted for 44% of the variance, while BMI accounted for 4% and \(\text{Pao}_2\) for 2%, for a total of 53%. The two other \(\text{Sao}_2\) indices considered were a) mean \(\text{Sao}_2\) and b) percentage of the time spent below 90% \(\text{Sao}_2\) \(\%\) TST<90\% \(\text{Sao}_2\). Using the same approach, with each of these indices as dependent variables and with stepwise multiple regression and multiple linear regression analyses, the results indicated a) BMI, FEY/FVC, \(\text{Pao}_2\), \(\text{Paco}_2\), and MP-H as independent variables with the following significance: BMI (p<0.0001); FEY/FVC (p<0.005); \(\text{Pao}_2\) (p<0.025). This model accounted for 41.2\% of the variance (\(R^2=0.412\)). The model was \(X = 84.5 + 8.2 \left(\text{FEY/FVC}\right) - 0.05(SI) + 0.1(BMI) = 0.02(\text{Pao}_2)\). BMI accounted for 30% of the variance, while BMI accounted for 3%, FEY/FVC for 3%, \(\text{Pao}_2\) for 2% (\(\text{PAS}\) and MP-H each accounted for 1%), and for the \% TST<90\% \(\text{Sao}_2\) model, \(R^2=0.53\) (i.e. accounted for 53\% of the variance). The statistically significant variables were: BMI (p<0.0001); FEY/FVC (p<0.003) and \(\text{Pao}_2\) (p<0.001). The model was 329 + 1.4 (BMI) + 0.90 (SI) - [130.58 \text{FEY/FVC}]. The partial \(R^2\)'s were: BMI = 0.41, \(\text{Pao}_2 = 0.17\), and \(\text{FEY/FVC} = 0.05\).

Population B. Of the prospective OSAS group, 6 of the 25 patients also presented with abnormal FEY/FVC. But once again, none was considered to have day-time hypercapnia based on awake, seated blood gas analysis (table 1).

Prospective validation of the statistical models

The same data (similar clinical evaluation, nocturnal polygraphic recordings, cephalometric roentgeno-

<table>
<thead>
<tr>
<th>Variable</th>
<th>OSAS n=120</th>
<th>Prospective group n=25</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age yrs</td>
<td>49.2±11.3</td>
<td>51.2±12.1</td>
<td>++NS</td>
</tr>
<tr>
<td>BMI</td>
<td>31.3±5.9</td>
<td>35.8±7.0</td>
<td>+p&lt;0.0001</td>
</tr>
<tr>
<td>(%) SI</td>
<td>42.4±20.7</td>
<td>52.2±20.1</td>
<td>+p&lt;0.02</td>
</tr>
<tr>
<td>RDI</td>
<td>53.0±25.0</td>
<td>68.6±32.0</td>
<td>+p&lt;0.01</td>
</tr>
<tr>
<td>(%) O(_2)-80-I</td>
<td>6.6±17.0</td>
<td>20.0±22.0</td>
<td>+p&lt;0.003</td>
</tr>
<tr>
<td>(%) TST&lt;90% (\text{Sao}_2)</td>
<td>8.5±9.7</td>
<td>11.4±10.2</td>
<td>++NS</td>
</tr>
<tr>
<td>Mean (\text{O}_2) %</td>
<td>92.9±2.7</td>
<td>91.7±3.0</td>
<td>+p&lt;0.05</td>
</tr>
<tr>
<td>(\text{PAS}) mm</td>
<td>5.2±3.1</td>
<td>5.0±3.6</td>
<td>+NS</td>
</tr>
<tr>
<td>MP-H mm</td>
<td>27.0±6.7</td>
<td>26.4±6.3</td>
<td>+NS</td>
</tr>
<tr>
<td>(\text{FEY/FVC})</td>
<td>78.0±5.0</td>
<td>76.1±16.4</td>
<td>+NS</td>
</tr>
<tr>
<td>(\text{Pao}_2) mm</td>
<td>78.0±6.9</td>
<td>74.6±5.53</td>
<td>+p&lt;0.05</td>
</tr>
<tr>
<td>(\text{Paco}_2) mm</td>
<td>30.8±1.8</td>
<td>37.9±3.2</td>
<td>+NS</td>
</tr>
</tbody>
</table>

+ t-test; ++ Mann-Whitney U test. The prospective group was more obese, with more perturbation of the oxygen desaturation indices during sleep and lower arterial \(\text{Pao}_2\) during the day.

Results

Population A. As in any group of OSAS patients, some presented with abnormal FEY/FVC, but none was hypercapnic (table 1).

The stepwise multiple regression analysis showed a statistically significant link between the percentage of SI, BMI, age and \(\text{PAS}\) as independent variables when RDI was the dependent variable. Using the multiple linear regression analysis, the variables with a significant coefficient were BMI (p<0.0001), SI (p<0.0001), age (p<0.003), and logarithmic transformation of \(\text{PAS}\) (p<0.02). These independent variables accounted for 66\% of the variance (\(R^2=0.665\)). The model obtained was RDI = 11.14 + 1.22(BMI) + 0.87(SI) - [0.45 (age) + 7.5 (log \(\text{PAS}\)]. SI represented 51\% of the variance, while BMI accounted for 9\%, age for 4\% and \(\text{PAS}\) for 2\%, for a total of 66\%.

We next considered different \(\text{Sao}_2\) variables. When \(\text{O}_2\)-80-I was the dependent variable, the stepwise multiple regression indicated BMI, SI, and \(\text{FEY/FVC}\) as the independent variables. The multiple linear regression analysis indicated the significance of these variables: BMI (p<0.0001); SI (p<0.0001); and logarithmic transformation of \(\text{FEY/FVC}\) (p<0.002). This model, however, accounted for only 53\% of the variance (\(R^2=0.529\)). The model was [log \(\text{O}_2\)-80-I] = 21.9 + 0.14 (BMI) + 0.06 (SI) - 6.6 [log \(\text{FEY/FVC}\). SI accounted for 40\% of the variance, while BMI accounted for 9\% and \(\text{FEY/FVC}\) for 4\%, for a total of 53\%. The two other \(\text{Sao}_2\) indices considered were a) mean \(\text{Sao}_2\) and b) percentage of the TST spent below 90\% \(\text{Sao}_2\) \% TST<90\% \(\text{Sao}_2\). Using the same approach, with each of these indices as dependent variables and with stepwise multiple regression and multiple linear regression analyses, the results indicated a) BMI, FEY/FVC, \(\text{Pao}_2\), \(\text{Paco}_2\), and MP-H as independent variables with the following significance: BMI (p<0.0001); FEY/FVC (p<0.005); \(\text{Pao}_2\) (p<0.025). This model accounted for 41\% of the variance, (\(R^2=0.412\)). The model was X = 84.5 + 8.2 \(\left(\text{FEY/FVC}\right) - 0.05(SI) + 0.1(BMI) = 0.02(\text{Pao}_2)\). BMI accounted for 30\% of the variance, while BMI accounted for 3\%, \(\text{FEY/FVC}\) for 3\%, \(\text{Pao}_2\) for 2\% (\(\text{PAS}\) and MP-H each accounted for 1\%), and for the \% TST<90\% \(\text{Sao}_2\) model, \(R^2=0.53\) (i.e. accounted for 53\% of the variance). The statistically significant variables were: BMI (p<0.0001); FEY/FVC (p<0.003) and \(\text{Pao}_2\) (p<0.001). The model was 329 + 1.4 (BMI) + 0.90 (SI) - [130.58 \text{FEY/FVC}]. The partial \(R^2\)'s were: BMI = 0.41, \(\text{Pao}_2 = 0.17\), and \(\text{FEY/FVC} = 0.05\).

It is obvious that in each model, SI accounted for a large percentage of the partial variance.

Population B. Of the prospective OSAS group, 6 of the 25 patients also presented with abnormal FEY/FVC. But once again, none was considered to have day-time hypercapnia based on awake, seated blood gas analysis (table 1).
grams, and standard pulmonary function tests with arterial blood gas readings obtained with patients awake and seated) were collected and are presented in table 1.

RDI and Sao2 indices for this smaller prospective group were tested against the pre-established models. The degree of correlation (Pearson coefficient) between observed and predicted values were as follows: RDI = 0.70, O-80-I = 0.81, %TST <90% Sao2 = 0.88, mean Sao2 = 0.67. The statistical model on the average underestimated the mean RDI by 16±11 (apnoea + hypopnoea per hour); the mean O-80-I by 1.2±1.1 (drops per hour); and overestimated the mean % TST <90% Sao2 by 4.4±3.5%; and the mean Sao2 by 1.5±1.2% (fig. 1 and table 1).

**Discussion**

Several findings can be emphasized in our study:

A) Our OSAS population was a group of patients seen in a sleep disorders clinic and, a priori, the symptoms leading to consultation were related to sleep: i.e. presence of daytime tiredness, fatigue or sleepiness in the index case, or serious disturbance of the spouse's sleep due to the patient's heavy snoring; and despite a subgroup with a combination of OSAS and moderate COLD, day-time hypercapnia was not noted. Another important factor to note is that our initial patients were not extremely obese: the mean BMI was 31 kg·m² [8]. Standard data in the USA for men aged 40-65 yrs show that 26.7% have a BMI >27.8 kg·m² [20]. This patient population is representative of the overall OSAS patients seen in sleep clinics, and includes patients with a large variation in the severity of symptoms and abnormalities on the nocturnal recording.

B) We have obtained information on the determinants of Sao2 drops in our OSAS population. One may question why we decided to consider three different indices of Sao2 which were, not surprisingly, correlated (Pearson correlation coefficient = O-80-I and %TST <90% Sao2 = 0.70, X Sao2 and O-80-I = 0.80, X Sao2 and %TST <90% Sao2 = 0.83). The three indices approach Sao2 drops associated with apnoeas differently. For example, the percentage of time spent below a certain Sao2 ignores the fact that there are abrupt Sao2 drops and returns to baseline or near baseline levels with each apnoea or hypopnoea. This is better indicated by our O-80-I. Similarly, with mean Sao2, there is a blunting of the saturation changes with each apnoea or hypopnoea. We thus decided to calculate the three indices and study three models. We also resolved to select FEV1/FVC for our model, as it would not be affected by marked obesity.

Finally, blood gas values from patients who were awake and supine rather than seated would have been desirable; however, these data points were not available for all subjects.

Data in the literature have already given important information on interaction between ventilatory variables and Sao2 values during sleep. Bradly and co-workers [6] found that mean Sao2 during sleep correlated with supine Pao2, expiratory reserve volume and percentage of time spent in apnoea. Catterall et al. [13] have emphasized the relationship between daytime Sao2 (patients awake and supine) and nocturnal Sao2 in patients with chronic obstructive pulmonary disease (COPD) with or without apnoea. In an experimental study of normal subjects, Findley et al. [21] showed that initial lung volume is an important determinant of hypoxia during apnoea. Undoubtedly, the previous studies have convincingly emphasized the roles of lung volume and wake up Sao2 in the sleep-related Sao2 drops associated with OSA. Bradely and co-workers [7] also emphasized the role of obesity (and its impact on lung volume) in the development of hypercapnia in morbidly obese OSA patients. Our data largely support these reports, but we have tried to add new dimensions and have analysed other than purely respiratory variables. This study confirmed our expectation that Sao2 indices are dependent on PEV1/FVC and, more notably, on BMI; and that RDI is dependent not only on obesity, but also on the size of the PAS. In each of the different models, however, the percentage of SI is a major component when one includes 'sleep disturbance' in a model.

The percentage of SI sleep, an index of sleep disruption and fragmentation, which also strongly correlates in our patients with WASO (i.e. awakening lasting longer than 30 s), is a controversial variable to consider in OSAS: the sleep fragmentation is considered as a consequence of disturbed breathing. However, as indicated in our introduction, sleep
Obstructive sleep apnea as indicated by the disappearance of the normal sleep as an organized state, the cost of disrupted airway muscles have different set-points during sleep than those during wake. Repetitive sleep disturbances and repetitive periodic breathing are too often forgotten in considering secondary impact on hypoxic and hypercapnic re­

interact as the syndrome worsens clinically.

Another issue is the increased daytime sleepiness and more pronounced blunting of hypoxic and hypercapnic responses. This will lead to longer OSAS and repetitive periodic breathing, may feed back to the sleeping brain and the controls of local reflexes, neuronal network, etc.

Conclusion

Our study indicates that the continuous disruption of sleep considered as an organized state is one of the RDI determinants in OSAS. Sleep disturbance alone obviously does not 'create' the initial obstructive apnoeas and hypopnoeas, even if severe sleep fragmentation may lead to periodic breathing.

Similarly, Sao₂ drops during sleep appear to be absent in the 'pure' young heavy snorers [22] and hypoxaemia does not induce obstructive apnoea in many COPD patients, even those also presenting OSA. Nevertheless, once the process begins and clinical symptoms of OSAS are noted, the determinants of Sao₂ drops include not only obesity and lung function [6, 7] but also the width of the upper airway and the severity of sleep disturbance.

These two components are also important in RDI determinants of the clinical syndrome. It must be acknowledged that width of the upper airway may be related to anatomical abnormalities and eventual dysfunction of local upper airway reflexes, and that 'sleep disturbance' is only the final, gross expression of the disintegration of normal sleep and basic brain function during sleep. The normal sleep EEG pattern is profoundly disturbed and, even if we cannot make any assumptions concerning specific neuronal dysfunctions, we can at least monitor the severity of the disturbance of stages and states.

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References

Role of diffuse airway obstruction

The Veterans

Institute, Los Angeles, 1968.

Brain Information Service, Brain Research

96-114.

apneic patients.

19. Wilkinson


Bradley TD, Martinez D, Rutherford R, Lee F, Grossman RF,

Moldofsky H, Zamel N, Phillipson EA. - Physiological determinants

of nocturnal arterial oxygenation in patients with obstructive


Bradley TD, Rutherford R, Lee F, Moldofsky H, Grossman

RF, Zamel N, Phillipson EA. - Role of diffuse airway obstruction

in the hypercapnia of obstructive sleep apnea. Am Rev Respir Dis,

1986, 134, 920-924.

Khosla T, Lowe FR. - Indices of obesity derived from body


Shepard JW Jr. - Gas exchange and hemodynamics during


Jamieson A, Guilleminault C, Partinen M, Quera-Salva MA.

Obstructive sleep apneic patients have cranio-mandibular

abnormalities. Sleep, 1986, 9, 469-477.

Lowe AA, Gionhaku N, Takeuchi K, Fleetham JA. - Three-
dimensional CT reconstruction of contour and tongue in adult

subjects with obstructive sleep apnea. Am J Ortho Dentofacial


Rivlin J, Hofstein V, Kalbfleisch J, McNicholas W, Zamel N,

Bryan AC. - Upper airway morphology in patients with idiopathic


Catterall JR, Douglas NJ, Calverly PMA et al. - Transient

hypoxemia during sleep in chronic obstructive pulmonary disease is


Wittels EH. - Obesity and hormonal factors in sleep and sleep


Boren HG, Kory RC, Syner JC. - The Veterans Administration-

Army cooperative study of pulmonary function. II. The lung


96-114.

Kory RC, Callahan R, Boren HG, Syner JC. - The Veterans

Administration-Army cooperative study of pulmonary function. I.


Riley RW, Guilleminault C, Herraw J, Powell NB. - Cephalometric analysis and flow volume loops in obstructive sleep

apnea patients. Sleep, 1986, 6, 303-311.

Rechtschaffen A, Kales A eds. - A manual of standardized
terminology, techniques and scoring system for sleep stages of

human subjects. Brain Information Service, Brain Research

Institute, Los Angeles, 1968.

Willkinson L. - SYSTAT: The System for Statistics. Systat,


Health United States 1985. - U.S. Department of Health and

Human Services, National Center for Health Statistics, Hyattsville,


Findley LJ, Rics AL, Tisi OM, Wagner PD. - Hypoxemia
during apnea in normal subjects: mechanisms and impact of lung

volume. J Appl Physiol: Respir Environ Exercise Physiol, 1983,

55, 1777-1783.

Guilleminault C, Winkle R, Korobkin R, Simmons B. -

Children and nocturnal snoring: evaluation of the effects of sleep-

related respiratory resistive load and daytime functioning. Ear J


RÉSUMÉ: Le syndrome d’apnée obstructive du sommeil est

caractérisé par des troubles respiratoires et par des chutes de la

saturatio n oxyhémoglobine au cours du sommeil. Pour détermi ner

les facteurs qui pourraient conduire à ces anomalies, nous avons

étudié, sur le plan statistique, des données provenant de 120 sujets

atteints de syndrome d’apnée obstructive du sommeil, qui avaient

subi un monitoring polysomnographique et des radiographies

céphalométriques. Nous avons ajouté des indices de trouble du

sommeil et des anomalies anatomo niques des voies aériennes

supérieures aux facteurs classiques (comme l’obésité et les maladies

pulmonaires) qui sont habituellement pris en compte dans une

analyse d’un modèle statistique. Après la construction de modèles

utilisant les indices des troubles respiratoires et des chutes de

saturation oxyhémoglobine, nous les avons appliqués de manière

prospective à 25 nouveaux cas de syndrome d’apnée obstructive

du sommeil, et nous avons calculé le degré de corrélation entre

les valeurs observées et les valeurs prédites. L’index de masse

corporelle et la largeur de la voix aérienne postérieure ont une

influence statistique sur l’index des troubles respiratoires. Toute-

fois, la quantité de sommeil de stade 1 non-REM, qui est un index

de désagrégation du sommeil nocturne, est également une variable

indépendante significative dans le modèle statistique lorsque l’index

des troubles respiratoires est la valeur dépendante. De même,

lorsqu’on considère les indices de chute de la saturation

oxyhémoglobinée pendant le sommeil, plusieurs autres variables

sont significatives dans le modèle statistique, comme l’index de

masse corporelle, le rapport VEMS/CVF, et le sommeil de stade 1

non-REM. Les modèles ne parviennent pas à expliquer l’apparition

de l’apnée obstructive partielle ou totale, puisque ni la fragmentation

du sommeil ni les chutes de saturation oxygénée seules

ne induisent le syndrome d’apnée obstructive. Toutefois, les deux font

partie de la courbe de feedback qui conduit au syndrome d’apnée

obstructive, dont les déterminants peuvent être appréciés par

l’utilisation des indices de perturbation respiratoire et de chute de

saturation oxyhémoglobinée. Nos résultats indiquent également

que les anomalies anatomiques de la voie aérienne supérieure sont

une variable importante dans les modèles statistiques et doivent

être considérées lorsque l’on traite les patients atteints de syndrome

d’obstruction des voies aériennes.