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# Nocturnal asthma: snoring, small pharynx and nasal CPAP

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*Nocturnal asthma: snoring, small pharynx and nasal CPAP. C. Guilleminault, M. A. Quera-Salva, N. Powell, R. Riley, A. Romaker, M. Partinen, R. Baldwin, G. Nino-Murcia.*

**ABSTRACT: We studied two populations of patients who snored and had frequent nocturnal asthma attacks: ten overweight men presenting with typical obstructive sleep apnoea syndrome, and a group of five adolescents with regular snoring and an increase in negative inspiratory oesophageal pressure during stage II non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. All subjects presented cranio-mandibular abnormalities at cephalometric evaluation, with a narrow space behind the base of the tongue. Both populations were treated with nasal continuous positive airway pressure (CPAP) during sleep. Snoring and partial or complete airway obstruction were eliminated, as were the nocturnal asthma attacks. Two adolescents treated with upper airway surgery after nasal CPAP showed no nocturnal asthma at short-term follow-up. Nasal CPAP had no effect on daytime asthma. One hypothesis is that a subgroup of asthmatic patients with small pharynxes may have enhanced vagal stimulation during sleep compared with other asthmatic patients. This enhancement would be related to the repetitive Müller manoeuvres noted with airway obstruction during sleep. Combined with the local effects of snoring, this extra vagal stimulation would be a precipitating factor in nocturnal asthma attacks.**

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Three out of four asthmatics are reported to have frequent nocturnal (sleep-related) attacks [1]. Despite several studies, it remains unclear whether the worsening of asthma at night is linked to a circadian rhythm of bronchomotor tone and its chemical controls, to a specific sleep state, or to a combination of both [2–6]. There are probably asthmatic subgroups in which a specific risk factor may increase the chance of a nocturnal asthma attack. Our report focuses on a subgroup of patients whose nocturnal asthma was greatly helped by nightly use of nasal continuous positive airway pressure (CPAP), although daytime attacks were still noted. The patient population consisted of two groups: population A, who were seen at the sleep disorders clinic for suspected obstructive sleep apnoea syndrome (OSAS) and were also found to have frequent nocturnal asthma; and population B, composed of younger subjects who were studied prospectively after being seen in a pulmonary clinic. The findings obtained on these two groups are reported separately: studies A and B consider the respective patient populations.

## Study A - Subjects

Population A included ten male patients referred for symptoms of OSAS. Their mean age was 43.8±14.3 yrs and their mean body mass index (BMI), calculated according to the formula of Khosla and Lowe [7], was 31.1±4.6 $kg \cdot m^{-2}$, indicating a moderate to moderately severe obesity. All complained of daytime somnolence, expressed as persistent daytime fatigue and the need to fight off sleep in a quiet situation (reading, watching television, performing a desk job at work, or driving), for a mean of 17±9 months. Seven patients reported having short breathing difficulties and a choking feeling whilst awake but supine during the day. At night, all reported sweating, restless sleep, intermittent arousal with choking or gagging, nocturia, and a history of snoring with apnoea observed by their bed partner. Family members reported that patients' snoring began at a mean age of 22±4 yrs, a time when patients were clearly more slender (reported mean weight was 75±11 kg in their early twenties) than at the time of consultation. In four cases, loud snoring had caused problems in military barracks, on camping trips, or in intimate relationships. In all cases the snoring had worsened significantly over time, with the spouse having abandoned the bedroom between 1 and 18 yrs prior to consultation. Eight patients reported oesophageal reflux during the night several times a week, waking up with regurgitation, heartburn, and an acid taste in the mouth and/or throat. All patients reported significant dryness of mouth and throat, and thirst during the night.

In association with their OSAS-related symptoms, all patients presented with asthma during the day and the night, when attacks were much more frequent. The patients had a mean of one severe asthma attack during sleep every 17 days, and four patients had been admitted to the Intensive Care Unit at night more than four times during the previous year. At the time of sleep clinic evaluation, all patients had wheezing respiration at auscultation but were seen with normal cardiac auscultation and with no peripheral oedema or cyanosis. Their mean blood pressure was 140±16/90±11. All were treated with inhalers (corticosteroid and/or albuterol sulphate) and seven of the ten had received oral corticotherapy in the preceding year. Pulmonary function testing within two months of nocturnal polygraphic monitoring indicated that all patients had a forced expiratory volume in one second ($FEV_1$) between 48 and 62% (mean $\bar{x}$=54±6%) of predicted values and $FEV_1/VC$ was 51±10%. Both values could be moderately improved by bronchodilators ($FEV_1$=58±10% and $FEV_1/VC$=56±13%). Arterial blood gas readings on awake, seated patients showed an oxygen pressure ($Pao_2$) between 58 and 71 mmHg and a carbon dioxide tension ($Paco_2$) between 36 and 39 mmHg.

## Study A - Methods

### *Nocturnal polygraphic recordings*

*Baseline recordings.* The following polygraphic variables were measured in two nights of baseline monitoring: electroencephalogram (EEG), C3/A2, C4/A1; electromyogram (EMG), chin and right and left anterior tibialis muscles; electro-oculogram (EOG) [8]; and electrocardiogram (ECG) (modified $V_2$ lead). Respiration was monitored by uncalibrated respiratory inductive plethysmography, using the Respitrace™ (Ambulatory Monitoring, New York) system; airflow was monitored by nasal and oral thermistors; and oxygen saturation ($SaO_2$) was measured by ear oximetry. Variables were continuously monitored on a Grass polygraph.

*Nasal CPAP recordings.* The same variables, with the exception of thermistors, were monitored during a set-up for nasal CPAP. The "Sleep-easy" Respironics TM system was used to establish nasal CPAP. Positive end-expiratory pressure (PEEP), which allows control of OSAS and associated $SaO_2$ drops, was determined on the first night of nasal CPAP. The appropriateness of the PEEP value setting on the Respironics™ equipment was checked on night two, *i.e.* each patient was monitored again with the Respironics™ PEEP valves that had eliminated apnoeas and snoring the previous night. The Respironics™ PEEP value used was adapted for each individual's needs and the range of calibrated PEEP values used was between 10 and 15 $cmH_2O$. After 6–9 months of treatment, patients returned for a third night of monitoring, whilst using nasal CPAP.

### *Cephalometric roentgenograms*

These special lateral X-rays of skull and face were systematically obtained, following the technique described by RILEY *et al.* [9], using a Wehmer cephalostat on the day of the first baseline recordings and 12–14 months post-treatment with nasal CPAP. A simultaneous fibreoptic evaluation of the upper airway was performed on a recumbent, awake patient, together with a follow-up clinical interview.

## Study A - Results

Results of baseline polygraphic recording and cephalometric evaluations are presented in table 1. The respiratory disturbance index (RDI) is defined as the total number of apnoeas and hypopnoeas divided by total sleep time (TST) multiplied by 60 and is an index of abnormal respiratory events per hour of sleep [10].

Table 1. – Baseline polygraphic recording and cephalometric roentgenogram findings in population A

| Variable | Baseline | Nasal CPAP | |
|---|---|---|---|
| | | 2nd night | Follow-up 6–9 months |
| Polygraphic: | | | |
| TST min | 397±24 | 402±34 | 413±31 |
| RDI | 51±13 | 11±7 | 5±8 |
| %T<90% $Sao_2$ % | 18±5 | 1.5±1 | 0.5±0.3 |
| Lowest $Sao_2$ % | 71±7 | 88±2 | 91±2 |
| Cephalometric: | | | |
| SNA angle° | 79±2 | | 79±2 |
| SNB angle° | 74±3.5 | | 74±3.5 |
| MP-H distance mm | 28±7 | | 28±7 |
| PAS distance mm | 4±2 | | 5.5±2.5 |
| PNS-P distance mm | 43±5 | | 43±5 |

TST: total sleep time; RDI: respiratory disturbance index; %T <90% $Sao_2$: percentage of time spent with $Sao_2$ below 90% during sleep; SNA: sella-nasion-(point A) supramentale; SNB: sella-nasion-(point B) supraspinale; MP-H: mandibular plane-hyoid bone distance; PAS: posterior airway space; PNS-P: posterior nasal spine to end of soft palate. All values mean±SEM.

Hypopnoea is defined by the following criteria: reduction in maximal thermistor output by at least 50% compared with baseline associated with either a decrease in $Sao_2$ to <92% from a baseline of at least 94%, or a drop in $Sao_2$ of at least 3% if the baseline is equal to, or below 90% [10]. We also calculated the percentage of time spent with an $Sao_2$<90% during sleep (%T<90% $Sao_2$), and measured the lowest $Sao_2$ during sleep.

Cephalometric roentgenograms were interpreted according to normative data summarized by RILEY *et al.* [9]. The following measurements were considered: sella-nasion-(point A) supramentale (SNA), sella-nasion-(point B) supraspinale (SNB), mandibular plane-hyoid bone distance (MP-H), posterior nasal spine (PNS) to end of soft palate (P), and posterior airway space (PAS) (space behind base of tongue). Patients had a retroposition of the mandible, a long soft palate, a narrow PAS, and an elongated MP-H compared with published normative data (see table 1). As frequently reported in OSAS, the patients had an abnormally narrow upper airway above and behind the base of the tongue, and this was confirmed by fibreoptic evaluation [11–13].

Despite the upper airway abnormalities, nasal CPAP appropriately controlled the OSAS (see table 1). Of eight patients previously reporting oesophageal reflux, six had complete abatement of symptoms and two had greatly diminished reflux, despite continuing xanthine treatment. No patient presented a nocturnal asthma attack during the 12–14 month follow-up. The number of infrequent daytime asthma attacks was unchanged.

## Study B - Subjects

Following the above results, a prospective investigation was performed on a younger population. The charts of 119 teenagers (14+ yrs of age) and young adults seen in a pulmonary division were reviewed. Among the criteria for chart selection was the report of at least one nocturnal asthma attack that had led to an immediate hospital visit, usually to the Emergency Room. We identified 52 charts. Another selection criterion was the report of snoring at night and/or presence of daytime somnolence or symptoms evoking it. We identified nine individuals who snored, two of whom had enlarged tonsils/adenoids and reported mouth breathing and one of whom had symptoms of daytime somnolence. Five of the nine individuals located agreed to undergo investigation.

Population B involved five male patients, mean age 17±2.5 yrs (range 14–21 yrs) with BMI=25±3 $kg \cdot m^{-2}$. All patients presented daytime and nocturnal asthma attacks (mean of one attack in 15±6 days). Four of the five had experienced a predominance of nocturnal asthma during the previous year. Their latest pulmonary function tests, performed away from an acute episode, indicated a mean $FEV_1$ of 66±9%, which was improved in all cases by bronchodilators (mean $FEV_1$=71±8%). Arterial blood gases obtained awake and seated indicated a mean $PaO_2$ of 85±3.5 mmHg and a mean $Paco_2$ of 36±1.8 mmHg. Snoring had been regular since the onset of puberty in three of the five subjects and had been noted before puberty in two. In all cases family members reported snoring to be a nightly event for at least two years but this had not led to any specific inquiries and was not considered as "tremendous" by the household. Apnoea had not been noticed by anyone, but all subjects slept alone. Investigation of symptoms in favour of OSAS indicated that three of the five had restless sleep, as indicated by the bed status every morning or from the reports of siblings who had, rarely, shared bedrooms. One subject had taken a daily 30 min nap for the past 4 yrs in addition to an 8 h nocturnal sleep. Two of the five reported moderate intermittent nocturnal sweating. None had symptoms of gastro-oesophageal reflux despite treatment by xanthines. Four of the five had undergone prepubertal tonsillectomy or tonsi and adenoidectomy; one of the five had 3+ (on a scale of 0–4+) tonsils; and one of the five had an obviously deficient chin and had been a near failure-to-thrive in pre-pubertal years.

## Study B - Methods

### *Polygraphic monitoring*

*Baseline monitoring*. All patients underwent polygraphic monitoring during sleep with monitoring of respiration using uncalibrated inductive respiratory plethysmography. Monitored variables were the same as for population A. On night two, with the exception of the youngest subject (a 14 year old), the other four patients were monitored with measurement of oesophageal pressure during sleep with an oesophageal balloon. Recording was obtained on a Hewlett-Packard (HP) recorder with simultaneous time code on Grass and HP recorders. The 14 year old was monitored with calibrated inductive respiratory plethysmography.

*Further monitoring*. Following the baseline recording, nasal CPAP was initiated whilst measuring oesophageal pressure and snoring sounds for one night. A second night of nasal CPAP monitoring was performed, using inductive respiratory plethysmography and monitoring variables similar to baseline on a Grass polygraph.

### *Cephalometric roentgenograms*

Using the technique and landmarks described for population A, cephalometric X-rays were taken, using the Wehmer Cephalostat.

### *Follow-up*

Patients agreed to stay under nasal CPAP for six months and to undergo follow-up recordings. The patient with enlarged tonsils then underwent surgery, as did the patient with retrognathia [14]. Three patients elected to stay under nasal CPAP treatment after the six-month follow-up.

## Study B - Results

All subjects snored during sleep as indicated by sound and by chin EMG recordings. None of the subjects was monitored at the time of a nocturnal asthma

Table 2. – Night polygraphic recording and cephalometric variables in population B

| Variable | Baseline night | 2nd nasal CPAP night |
|---|---|---|
| Polygraphic: | | |
| TST min | 378±39 | 386±48 |
| RDI | 8±5 | 1±0.3 |
| %T<90 $Sa_{O_2}$ % | 0.5±0.5 | 0.00 |
| Lowest $Sa_{O_2}$ | 90±3 | |
| Mean highest negative inspiratory oesophageal pressure during REM sleep $cmH_2O$ | 49±11* | –** |
| Mean highest negative inspiratory oesophageal pressure during NREM sleep $cmH_2O$ | 47±8* | –** |
| Mean percentage of TST spent with each inspiratory breath presenting peak (inspiratory) negative oesophageal pressure below –10 $cmH_2O$ % | 79±11* | –** |
| Cephalometric: | | |
| SNA angle ° | 80±3.2 | |
| SNB angle ° | 74.5±3.8 | |
| MP-H distance mm | 30±9 | |
| PAS distance mm | 41±2 | |

TST: total sleep time; RDI: respiratory disturbance index; %T <90% $Sa_{O_2}$: percentage of time spent with $Sa_{O_2}$ below 90% during sleep; REM: rapid eye movement; NREM: non-rapid eye movement; SNA: sella-nasion-(point A) supramentale; SNB: sella-nasion-(point B) supraspinale; MP-H mandibular plane-hyoid bone distance; PAS: posterior airway space; *: four adolescents only are included; **: oesophageal pressure was monitored only during first CPAP night and the selected PEEP values always maintained it between 4 and 8 $cmH_2O$ peak negative inspiratory oesophageal pressure. All values mean±SEM.

attack. Table 2 shows the results of the polygraphic recordings made on night two using an oesophageal balloon. They indicate that even without apnoea during non-rapid eye movement (NREM) sleep, there was a great increase in negative inspiratory pressure during stage II NREM sleep. Awake, supine, and relaxed subjects had an oesophageal pressure recording oscillating between a mean of +1 and -5 $cmH_2O$. Negative inspiratory oesophageal pressure occurred in association with snoring, mostly in stage II NREM sleep. The mean highest increase in negative oesophageal pressure compared with baseline was 47±8 $cmH_2O$ (*i.e.*, negative inspiratory oesophageal pressure reached peaks between -45 and -60 $cmH_2O$ during stage II NREM sleep) in the four adolescents studied. During rapid eye movement (REM) sleep, negative inspiratory oesophageal pressure often increased and in all four patients, intermittent and rare obstructive apnoeas were noted. Table 2 shows the results of cephalometric evaluation: compared with normative data, all patients had a small airway behind the base of the tongue.

Nasal CPAP eliminated the regular snoring, pronounced negative inspiratory oesophageal pressure swings, and intermittent obstructive sleep apnoea seen during REM sleep. The Respironics positive end-expiratory pressure (PEEP) valves, which eliminated pronounced negative inspiratory oesophageal pressure, were calibrated between 5 and 10 $cmH_2O$, depending on the subject. During the six-month follow-up period, no subject using nasal CPAP with a PEEP valve as determined on a nightly basis had a nocturnal asthma attack. Although rare, daytime asthma whilst awake was noted in all subjects, the overall frequency of asthma attacks was greatly reduced after nasal CPAP treatment was begun (mean of one attack in 80±15 days), and no nocturnal asthma attack was reported.

One patient with enlarged lymphoid tissue and redundant palatal mucosa underwent tonsil and adenoidectomy, uvulectomy, and lateral resection of the soft palate in a modified version of palato-pharyngoplasty. Although he stopped using nasal CPAP following surgery, he has not experienced any nocturnal asthma during the 4.5 months since surgery. Oesophageal pressure recording indicated unobstructed breathing throughout the night, with a peak negative oesophageal pressure recording oscillating between -4 and -8 $cmH_2O$ during sleep. Another subject, who also discontinued nasal CPAP after undergoing maxillo-mandibular surgery for retrognathia, has had no nocturnal asthma attacks during the 2.5-month postsurgical follow-up. In both cases, cephalometric roentgenograms demonstrated an increase in upper airway size; posterior airway space increased by 8 mm after maxillo-mandibular surgery.

## Discussion

In 1979, HUDGEL and SHUCARD [15] published a case report demonstrating the co-existence of sleep apnoea and asthma resulting in severe sleep hypoxia. This finding is confirmed in our investigation of patient population A. However, if the beneficial effect of nasal CPAP in obstructive sleep apnoea was expected, the elimination of nocturnal asthma attacks was not. The systematic use of nasal CPAP on younger patients with nocturnal asthma but no classical OSAS (population B), confirmed the positive response to nasal CPAP on nocturnal asthma in this subgroup. Although not all asthma attacks were eliminated with nasal CPAP, as daytime asthma still occurred, we believe that its benefit is related to an indirect effect.

Why are nocturnal asthma attacks seen in some patients and not in others? We cannot appropriately answer this question for all asthmatics; however, we may obtain better information if we focus on our very specific subgroup. We hypothesize an abnormal enhancement of autonomic nervous system activity as a factor in the triggering of nocturnal asthma attacks.

The indirect evidence supporting the notion of vagal tone enhancement during sleep in our subgroup is easy to find. Cephalometric roentgenograms demonstrated that our subjects presented with a narrow upper airway [9]. This narrowing was associated with heavy snoring

and obstructive hypopnoea or with complete obstructive apnoea during sleep [5, 9, 16]. A consequence of partial or complete airway obstruction during sleep was the performance of partial or complete Müller manoeuvres, as indicated by oesophageal pressure monitoring. The classical Müller manoeuvre consists of an inspiratory effort against a closed glottis and is associated with significant bradycardia. Significant bradycardia is noted in adult obstructive sleep apnoea and during the obstructive hypopnoea seen with heavy snoring.

Obstructive hypopneas during continuous heavy snoring are seen particularly in children [16]. Müller manoeuvres are also involved in the development of the known sleep-related haemodynamic changes noted with obstructive apnoea or hypopnoea [17–19]. Atropine and other anticholinergic medications, or autonomic nervous system lesions eliminate the cardiovascular changes associated with partial or complete upper airway obstruction during sleep, which are mediated through marked vagal stimulation [18, 19].

Thus, patients with obstructive apnoea, or hypopnoea with continuous heavy snoring, have increased vagal tone during sleep. This sleep-related pathological enhancement of vagal tone can be eliminated by appropriate treatment of partial or complete upper airway obstruction during sleep, including tonsillectomy and adenoidectomy in pre-pubertal children.

On the other hand, several studies have shown that increased vagal tone is probably a factor in asthma, and an inhaled anticholinergic drug has been shown to reduce nocturnal asthma [20, 21].

How can we link together the different pieces of information outlined above? Many factors may play a role in the appearance of nocturnal asthma, but certain combinations may be more powerful than others. A clear circadian variation in peak flow rates has been demonstrated in asthmatic subjects [1–3]. Bronchoconstriction seems to be exacerbated in the early morning hours in stable asthmatics, and this circadian peak has been linked to observation of asthma during the nocturnal period [1–6]. However, other nocturnal factors could enhance the risks of nocturnal asthma attacks. In our subgroup of patients, the effects of snoring (with secondary induced local muscular and mucosal changes) on the increased vagal tone related to the Müller manoeuvre [18, 19, 22] are added to the circadian variation of bronchoconstriction and its nocturnal peak [1–6]. Finally, asthma attacks are associated with significant reduction of upper airway dimension during inspiration and expiration as shown by COLLETT *et al.* [23]. This reduction is most prominent in the oropharynx above the hyoid bone, an anatomical region that is already abnormally small in our population. There is a continuous risk of sudden development of significant flow impairment, due to the anatomical abnormalities in association with any atopic change. The slowing of expiratory flow through the upper airway, another element supposedly playing a role in asthma, will occur much faster during sleep in our subgroup of subjects than in other asthmatic subjects. By maintaining an unobstructed upper airway, eliminating snoring, eliminating the Müller-manoeuvre-related vagal enhancement during sleep, and acting on the speed of flow through the upper airway, the risk of nocturnal asthma attack should be reduced. This would explain the beneficial response observed with nasal CPAP in our subgroup of asthmatic patients. This concept would also receive support from our observation of the effect of upper airway surgery on nocturnal asthma attacks in two of our cases. In both cases there was elimination of upper airway narrowing and its secondary cohort of snoring, partial or complete upper airway obstruction during sleep, and Müller manoeuvre.

Independent of our speculation on possible mechanisms involved in the triggering of nocturnal asthma attacks on our subgroup of asthmatic patients, the observation of the beneficial effect of nasal CPAP and a similar observation by Sullivan (both simultaneously reported at the Fifth International Congress on Sleep Research in Copenhagen, June 1987 [24, 25]) emphasize the need for identifying, from the total asthmatic group, those patients whose nocturnal asthma attacks may be appropriately controlled.

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## References

1. Bagg LR, Hughes DTD. – Diurnal variation in peak expiratory flow in asthmatics. *Eur J Respir Dis*, 1980, 61, 298–302.
2. Ballard RD, Martin RJ, Kelly PL, Patel DK, Saathoff MC. – Nocturnal asthma: sleep-triggered bronchoconstriction or true circadian rhythm. *Sleep Res*, 1987, 16, 464 (abstract).
3. Clark TJH, Hetzel MR. – Diurnal variation of asthma. *Br J Dis Chest*, 1977, 71, 87–92.
4. Hetzel MR, Clark TJH. – Does sleep cause nocturnal asthma? *Thorax*, 1979, 39, 749–754.
5. Martin RJ, Cicutto LC, Ballard RD. – Diurnal variation of airway reactivity in normals and asthmatics. *Sleep Res*, 1987, 16, 489 (abstract).
6. Douglas N. – Asthma at night. *Clin Chest Med*, 1985, 6, 663–674.
7. Khosla T, Lowe FR. – Indices of obesity derived from body weight and height. *Br J Prev Soc Med*, 1967, 21, 122–128.
8. 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.
9. Riley R, Guilleminault C, Herran J, Powell N. – Cephalometric analyses and flow volume loops in obstructive sleep apnea patients. *Sleep*, 1983, 6, 304–317.
10. Guilleminault C, Cummiskey J, Motta J. – Chronic obstructive airflow disease and sleep studies. *Am Rev Respir Dis*, 1980, 122, 397–408.
11. Jamieson A, Guilleminault C, Partinen M, Quera-Salva MA. – Obstructive sleep apnea patients have cranio-mandibular abnormalities. *Sleep*, 1986, 9, 469–472.
12. Rivlin J, Hoffstein V, Kalbfleisch J, McNicholas W, Zamel N, Bryan C. – Upper airway morphology in patients with idiopathic obstructive sleep apnea. *Am Rev Respir Dis*, 1984, 129, 355–360.

13. Rojewski TE, Schuller DE, Clarke RW, Schmidt HS, Potts RE. – Video-endoscopy determination of the mechanisms of obstruction in obstructive sleep apnea. *Otolaryngol-Head and Neck Surg*, 1984, 92, 127–131.
14. Riley RW, Powell NB, Guilleminault C, Nino-Murcia G. – Maxillary-mandibular and hyoid advancement: an alternative to tracheostomy in obstructive sleep apnea. *Otolaryngol-Head and Neck Surg*, 1986, 94, 584–588.
15. Hudgel DW, Shucard DW. – Co-existence of sleep apnea and asthma resulting in severe hypoxemia. *J Am Med Assoc*, 1979, 242, 2789–2790.
16. Guilleminault C, Winkle R. – A review of 50 children with OSAS. *Lung*, 1981, 159, 275–287.
17. Coccagna G, Mantovani M, Brignani F, Parchi C, Lugaresi E. – Continuous recording of the pulmonary and systemic arterial pressure during sleep in syndromes of hypersomnia with periodic breathing. *Bull Eur Physiopathol Respir*, 1972, 8, 1159–1172.
18. Guilleminault C, Tilkian A, Lehrman K, Forno L, Dement WC. – Sleep apnea syndrome: states of sleep and autonomic dysfunction. *J Neurol Neurosurg Psych*, 1977, 40, 718–725.
19. Guilleminault C, Winkle R, Melvin K, Tilkian A. – Cyclical variation of the heart rate in sleep apnea syndromes: mechanisms and usefulness of 24-hour electrocardiography as a screening technique. *Lancet*, 1984, 1, 126–136.
20. Coe CI, Barnes PJ. – Reduction of nocturnal asthma by an inhaled anticholinergic drug. *Chest*, 1986, 90, 485–488.
21. Cropp GJA. – The role of the parasympathetic nervous systems in the maintenance of chronic airway obstruction in asthmatic children. *Am Rev Respir Dis*, 1975, 112, 599–605.
22. 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. *Eur J Pediatr*, 1982, 139, 165–171.
23. Collett PW, Brancatisano AP, Engel LA. – Upper airway dimensions and movements in bronchial asthma. *Am Rev Respir Dis*, 1986, 133, 1143–1149.
24. Chan CS, Yan K, Woolcock AJ, Sullivan CE. – The effect of nasal CPAP on patients with asthma and obstructive sleep apnea. *Am Rev Respir Dis*, 1986, 133, 343 (abstract).
25. Guilleminault C, Quera-Salva MA, Powell N, Riley R, Romaker A, Partinen M, Baldwin R, Nino-Murcia G. – Nocturnal asthma, upper airway anatomical abnormality and nasal CPAP. *Sleep Res*, 1987, 16, 477 (abstract).

*Asthme nocturne: ronflements, pharynx étroit et CPAP nasale. C. Guilleminault, M.A. Quera-Salva, N. Powell, R. Riley, A. Romaker, M. Partinen, R. Baldwin, G. Nino-Murcia.*

RÉSUMÉ: Deux populations de patients qui ronflaient et présentaient des attaques fréquentes d'asthme nocturne ont été étudiées: Dix hommes en surcharge pondérale, atteints d'un syndrome typique d'apnée obstructive du sommeil et un groupe de cinq adolescents ronflant régulièrement et ayant une augmentation de leur pression oesophagienne inspiratoire négative au cours des stades du sommeil 2, non REM et REM. Tous les sujets presentaient des anomalies craniomandibulaires lors de leur évaluation céphalométrique et un espace étroit derrière la base de la langue. Les deux populations ont été traitées par CPAP nasale durant leur sommeil. Le ronflement, ainsi que les épisodes d'obstruction partielle ou complete des voies aerieunes, ont été éliminés, tout comme d'ailleurs les crises d'asthme nocturne. Deux des adolescents, traités par chirurgie des voies aeriennes superieures après CPAP nasale, n'ont plus présenté de crises d'asthme nocturne lors d'un suivi à court terme. La CPAP nasale n'a eu aucun effet sur l'asthme diurne. Pour expliquer ce phénomène, nous suggerons qu'un sous groupe de patients asthmatiques à pharynx étroit pourrait avoir une stimulation vagale augmentee au cours du sommeil, en comparaison avec les autres asthmatiques. Cette augmentation serait à mettre en relation avec les manoeuvres de Müller effectuées a répétition au cours des obstructions des voies aeriennes supérieures se produisant durant le sommeil. Cette stimulation vagale additionnelle, combinée aux effets locaux du ronflement, pourrait être un facteur precipitant des crises d'asthme nocturne.
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